Very Low Risk of Thromboembolic Events in Patients Undergoing Successful Catheter Ablation of Atrial Fibrillation With a CHADS2 Score ≤3

A Long-Term Outcome Study

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Background—Long-term cessation of oral anticoagulation (OAC) after catheter ablation of atrial fibrillation (AF) has been deemed controversial. The safety of this management strategy in patients without recurrent AF and with historically elevated risks for thromboembolism remains largely unknown. In this study, we sought to evaluate the long-term results of OAC cessation after successful catheter ablation of AF.

Methods and Results—OAC and antiarrhythmic drugs (AADs) were discontinued irrespective of AF type or baseline CHADS2 (congestive heart failure, hypertension, age ≥75 years, diabetes mellitus, prior stroke or transient ischemic attack) risk score in 327 patients (mean age, 63±13 years; 79% men) with drug-refractory AF after catheter ablation (mean CHADS2 score, 1.89±0.95; median, 2.0). Patients with a CHADS2 score of 2 (45.4%) and 3 (23.2%) accounted for 68.8% of this cohort. In patients with a high risk of recurrence or prior thromboembolic complications, OAC was continued for up to 6 to 12 months postablation and antiplatelet therapy was administered to all patients who maintained sinus rhythm upon OAC interruption. After a follow-up of 46±17 months (range, 13–82 months), 82% remained AF free (off AADs). Significant predictors of late AF recurrence (P<0.05) were nonparoxysmal AF (hazard ratio [HR], 1.83), female sex (HR, 2.19), age ≥60 years (HR, 1.81), left atrial size >40 mm (HR, 3.52), CHADS2 score ≥2 (HR, 1.81), and early recurrences (HR, 5.52). No symptomatic ischemic cerebrovascular events were detected during follow-up despite interruption of OAC in 298 (91%) patients and AADs in 293 (89%) patients.

Conclusions—No significant thromboembolic-related morbidity is observed when AADs and OAC are discontinued after successful catheter ablation of AF in patients with a CHADS2 score ≤3 who are maintained on antiplatelet therapy during long-term follow-up. (Circ Arrhythm Electrophysiol. 2011;4:615-621.)

Key Words: ablation ■ atrial fibrillation ■ stroke ■ anticoagulants

Atrial fibrillation (AF) is a common cardiac arrhythmia associated with significant morbidities, including that of thromboembolic complications.1,2 Radiofrequency catheter ablation (RFA) has rapidly become a common mode of treatment for symptomatic AF3 partly because of the poor efficacy and significant side effects associated with antiarrhythmic drug (AAD) therapy.4–8 However, widespread adoption of RFA as a definitive therapy for treatment of AF remains limited by the requirement for multiple procedures in certain patients and the perception that oral anticoagulation (OAC) should not be withheld in patients with multiple CHADS2 (congestive heart failure, hypertension, age ≥75 years, diabetes mellitus, prior stroke or transient ischemic attack) risk factors.9,10 Despite recent investigations, most guidelines still recommend maintaining long-term OAC according to the baseline thromboembolic risk factors, independent of the results of ablation.4,11 This recommendation is based on the belief that the baseline risk of thromboembolic events remains unchanged despite a successful ablation procedure.12 Nonetheless, it is reasonable to speculate that elimination of AF may reduce the preprocedural risk of thromboembolic stroke and similar complications. The present study sought to evaluate long-term results of a standardized ablation technique and a protocol of OAC interruption based on periodic rhythm assessment regardless of patients’ baseline risk for thromboembolic events.
Methods

Study Population
Between November 2003 and July 2009, 352 consecutive patients underwent RFA for drug-refractory AF at our institution. Of these, 327 (92.9%) had complete follow-up information for a minimum of 1 year and were included in this single-center, retrospective analysis. The mean age of the cohort was 63±13 years (range, 17–87 years); 259 were men.

The definitions of paroxysmal AF (PAF), persistent AF, and long-standing persistent (LSP) AF were based on the prevailing consensus documents. Patients with PAF comprised 60% of the population (n=195), patients with persistent AF comprised 27% (n=89), and patients with LSP AF comprised 13% (n=43). Baseline patient characteristics are summarized in Table 1. All patients provided written informed consent before the procedure, and the protocol was approved by the institutional ethics committee.

Ablation Strategy
The RFA technique used in this study has been previously described in detail. Briefly, patients were taken to the electrophysiology laboratory in a fasting state. After induction of general anesthesia, transesophageal echocardiography was performed to exclude presence of left atrial (LA) thrombus. Access was obtained in the right femoral vein (2), left femoral vein (1), and right jugular vein (1). A 10-F phased-array ultrasound imaging catheter (AcuNav; Mountain View, CA) was placed in the right atrium to guide catheter manipulation, and a 20-pole catheter ultrasound imaging catheter (Acunav; Mountain View, CA) was placed along with empirical isolation of the superior vena cava.

Transseptal access, guided by intracardiac echocardiography, was obtained using 2 separate punctures after administration of heparin (titrated to an activated clotting time of 350–400 s). LA and pulmonary vein (PV) mapping and ablation was guided by a 20-mm decapolar circular mapping catheter (Lasso; Biosense Webster; Baldwyn Park, CA). A Biosense Webster 8-mm-tip ablation catheter was used in the procedures of the initial 151 (46.2%) patients. Power titration was guided by microbubbles as seen on intracardiac echocardiography up to 70 W. The remaining 176 (53.8%) patients underwent ablation with a 3.5-mm-tip open irrigation catheter (Biosense Webster), with an irrigation rate of 30 mL/min during ablation. Power was set to 35 W with a maximum tip temperature no higher than 43°C. On the posterior LA wall, power was downtitrated according to the luminal esophageal temperature recorded by an esophageal temperature probe (EsoCath; CardioCommand Inc., Tampa, FL). RFA was discontinued if the esophageal temperature rose to ≥39°C. The power was then subsequently decreased to allow electrogram elimination.

PV antrum isolation was guided by intracardiac echocardiography and circular mapping along with empirical isolation of the superior vena cava whenever phrenic nerve capture could not be demonstrated with high-output pacing (20 mA at 2 ms). In patients with PAF, the proximal PV opening and a segment of the posterior LA wall contained within the PV area were targeted, aiming at bidirectional PV block and complete electric signal elimination. In patients with persistent and LSP AF, ablation was extended to the entire posterior LA wall, roof, coronary sinus, and LA septum, guided by presence of fractionated electrograms as recorded by the circular mapping catheter or the ablation catheter itself. At the end of the procedure, high-dose isoproterenol infusion (20 μg/min) was used to confirm PV isolation and to identify extra-PV trigger sites. After ablation, patients were monitored in the hospital overnight and typically discharged the next day.

Management of Anticoagulation Before Ablation
In most patients (79%), warfarin was discontinued 5 days before the procedure, whereas bridging therapy with enoxaparin (1 mg/kg twice daily) was initiated and continued until 24 hours before ablation. Enoxaparin was reinitiated immediately after ablation (0.5 mg/kg twice daily) and continued until a therapeutic international normalized ratio ≥2 was reached. In 70 (21%) patients, the procedure was performed while on warfarin at a therapeutic international normalized ratio level (2–3). In these patients, no bridging therapy was required.

CHADS2 Score Before Ablation
The mean CHADS2 score in this study population was 1.89±0.95 (median, 2.0). A CHADS2 score of 2 (n=149 [45.4%]) and a score of 3 (n=76 [23.2%]) accounted for 68.8% of the total cohort. Lower CHADS2 risk scores of 0 (n=33) and 1 (n=61) were noted in 28.7%, whereas a higher CHADS2 score (4) was encountered in 2.5% (n=8).

Postablation Management and Follow-Up
Patients were discharged on an AAD (preferably propafenone or sotalol) and on OAC with warfarin. Follow-up consisted of office visits with routine ECGs and 24-hour Holter monitoring at 1, 3, 6, 9, and 12 months. Whenever feasible, 7-day Holter recordings were obtained at 6 and 12 months. At 3 months, PV imaging (CT or MRI) and 2D echocardiography was performed to identify PV stenosis and to confirm LA contraction, respectively. Patients and their referring physicians were contacted by phone to assess for recurrence of symptoms and rhythm documentation, even when noncompliant with office follow-up. AADs generally were discontinued 4 weeks after catheter ablation, except when sustained arrhythmias were detected. In these situations, use of AADs was extended for up to an additional 3 months. Patients were instructed to monitor their pulse daily and to attempt to document their cardiac rhythm whenever symptoms or pulse irregularities were perceived.

Management of Anticoagulation After the Procedure
OAC with warfarin was continued for 3 months after catheter ablation in the absence of symptoms or documented sustained arrhythmias,
defined as lasting >2 minutes. After 3 months, OAC was discontinued irrespective of AF type, duration, or baseline CHADS2 risk score. In some patients in whom a high risk of recurrence was suspected or in those with previously documented thromboembolic complications, OAC was continued for up to 6 to 12 months after RFA (done at the investigator’s discretion). On cessation of OAC, patients were transitioned to antiplatelet therapy (aspirin 81–325 mg/d or clopidogrel 75 mg/d).

The initial 8 weeks after RFA was considered a blanking period; as such, any AF recurrence during this period was documented as an early recurrence and ignored for the purpose of the final analysis. AF recurrence was considered significant if encountered beyond 8 weeks postablation and was labeled as a late recurrence.

Whenever faced with symptomatic or asymptomatic documented sustained late AF recurrences, OAC and AADs were resumed and a second RFA procedure offered to the patient. During redo procedures, the same RFA techniques as the index procedure were used (PV disconnection and extensive LA ablation as described previously). In addition, any organized atrial arrhythmias (atrial flutter or tachycardia) were mapped accordingly and targeted for ablation. OAC was continued indefinitely in patients with recurrent AF who required AADs for maintenance of sinus rhythm.

**Statistical Analysis**
Continuous variables are reported as mean±SD, and categorical variables are given as proportions. The χ2 test was used to compare differences across AF types and subgroups. Multivariable Cox regression was used to identify significant predictors of recurrence while controlling for clinically relevant covariates.

Confounders were entered in the clinical model on the basis of known or expected clinical relevance. Significant interactions and multicollinearity of covariates were identified. The hazard ratios (HRs) and 95% CIs of AF recurrences were computed. Recurrence-free survival over time was calculated by Kaplan-Meier method (log-rank test). Tests were 2 sided, and P<0.05 was considered significant. Analysis was performed using SPSS version 18.0 (SPSS Inc; Chicago, IL) statistical software.

**Results**
Among the cohort, 97 (29.7%) patients had documented recurrence of sustained atrial arrhythmias after the primary RFA procedure, resulting in a 70.3% freedom from AF. AF was the presenting arrhythmia at recurrence in 78 (24%) patients, and organized atrial arrhythmia was found in 19 (6%) patients. Recurrence rates differed according to the AF type and are depicted in the Figure. Recurrent AF was encountered in 48 (24.6%) patients with PAF, 32 (36%) patients with persistent AF, and 17 (39.5%) patients with LSP AF (P=0.04). Patients were followed for a mean of 46±17 months (median, 36 months; range, 13–82 months). Two thirds (218/327) of the patients had complied with biannual visits beyond the first year after RFA.

Fifty patients (51.5% of the patients with AF recurrence) underwent a redo RFA procedure. Recurrence of sustained atrial arrhythmias after a second procedure was documented in 12 (24%) patients, which again differed according to the AF type at presentation: 20% (6/30 patients) for PAF, 15.4% (2/13) for persistent AF, and 57.4% (4/7) for LSP AF (P=0.08). After the second procedure, the probability of AF-free survival was 82% without AADs.

**Timing of Recurrence**
Early recurrences (<8 weeks) occurred in 69 (21%) patients after the index RFA procedure and in 7 (14%) after the redo procedure. The mean time to late recurrence documentation was 11.9±11.3 months (median, 8 months; range, 3–60 months). Late recurrences (>8 weeks) occurred in 97 patients. Of these, 34 (35%) patients were given the diagnosis after the first year after the index RFA (very late recurrence), as summarized in Table 2.

**Predictors of Long-Term AF Recurrence**
On univariable analysis, patients with recurrent AF were more likely to have a history of non-PAF (HR, 1.83; P=0.02), to be women (HR, 2.19; P<0.001), to be aged >60 years (HR, 1.81; P=0.005), to have a larger LA size (HR, 3.52 for LA size >40 mm; P=0.006), and to have multiple thromboembolic risk factors (HR, 1.81 for CHADS2 ≥2; P=0.005). Additionally, early recurrence was strongly predictive of late recurrence of AF (HR, 5.52; P<0.001) as 66.7% of the patients with early recurrence experienced late recurrence.

Structural heart disease, present in 72 (22%) patients, and impaired left ventricular ejection fraction (<55%), present in 45 (14%) patients, were not predictive of AF recurrence (Table 3). Use of open irrigation ablation catheters (HR, 0.52; P=0.003), on the other hand, was predictive of sinus rhythm maintenance.

**Table 2. Type of Recurrence in Patients With AF After First and Second Catheter Ablation Procedures**

<table>
<thead>
<tr>
<th>Recurrence</th>
<th>Risk of Recurrence</th>
<th>After First Ablation</th>
<th>After Second Ablation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n (95% CI)</td>
<td>n (95% CI)</td>
<td></td>
</tr>
<tr>
<td>Early</td>
<td>69 (16.9–25.8)</td>
<td>7 (6.3–25.7)</td>
<td></td>
</tr>
<tr>
<td>Late</td>
<td>97 (24.9–34.8)</td>
<td>12 (13.7–37.2)</td>
<td></td>
</tr>
<tr>
<td>&gt;1 y</td>
<td>34 (7.4–14.4)</td>
<td>1 (0.1–9.5)</td>
<td></td>
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</tbody>
</table>
In multivariable Cox regression analysis, only female sex (HR, 1.95; \( P = 0.002 \)), LA size > 40 mm (HR, 2.71; \( P = 0.03 \)), and early recurrence (HR, 4.62; \( P < 0.001 \)) predicted late recurrences (Table 3).

### Complications

Procedural complications were uncommon. Groin hematomas occurred in 16 (4.8%) patients. Deep venous thrombosis and phrenic nerve paralysis occurred each in 1 patient. Cardiac tamponade also occurred in 1 patient and was effectively managed by pericardiocentesis without the need for surgical intervention. LA thrombi were detected on intracardiac echocardiography in 2 patients and were uneventfully aspirated through the transseptal sheath. Asymptomatic PV stenosis was detected in 4 patients (0.01% of pulmonary veins) by CT/MRI (moderate in 3 and severe in 1 [left inferior PV]). All 4 patients were managed conservatively.

### Postprocedural Anticoagulation and Cerebrovascular Events

The duration of anticoagulation therapy after ablation was extended to 6 months in 151 (46%) patients and to 12 months in 92 (28%). These patients generally were older (age, 66±11 years); predominantly men (72%); and more likely to have persistent or LSP AF (n=96 [64%]), structural heart disease (n=57 [38%]), and larger LA dimensions (mean, 45±4 mm).

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### Table 3. Cox Models for Late Recurrence After Catheter Ablation

<table>
<thead>
<tr>
<th></th>
<th>Cox Model</th>
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<tr>
<td></td>
<td>Univariable</td>
<td>Multivariable 1</td>
<td>Multivariable 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>HRadj</td>
<td>( P )</td>
<td>HRadj</td>
<td>( P )</td>
</tr>
<tr>
<td>Type of AF</td>
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<td>0.744</td>
<td>1.83</td>
<td>0.033</td>
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<tr>
<td>PAF</td>
<td>195</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LSP AF</td>
<td>43</td>
<td>1.69</td>
<td>0.021</td>
<td>1.79</td>
<td>0.019</td>
</tr>
<tr>
<td>Persistent</td>
<td>89</td>
<td>2.19</td>
<td>&lt;0.001</td>
<td>1.79</td>
<td>0.019</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td>0.054</td>
<td>0.643</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>259</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>68</td>
<td>1.79</td>
<td>0.019</td>
<td>1.79</td>
<td>0.019</td>
</tr>
<tr>
<td>Age</td>
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<td>0.054</td>
<td>0.643</td>
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<tr>
<td>&lt;60 y</td>
<td>113</td>
<td>1</td>
<td>1</td>
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<tr>
<td>60–69 y</td>
<td>109</td>
<td>1.81</td>
<td>0.024</td>
<td>1.22</td>
<td>0.475</td>
</tr>
<tr>
<td>≥70 y</td>
<td>105</td>
<td>1.74</td>
<td>0.038</td>
<td>1.32</td>
<td>0.356</td>
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<tr>
<td>LVEF</td>
<td></td>
<td>0.054</td>
<td>0.643</td>
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<tr>
<td>≤55%</td>
<td>45</td>
<td>1.57</td>
<td>0.091</td>
<td>1.65</td>
<td>0.102</td>
</tr>
<tr>
<td>≥56%</td>
<td>282</td>
<td>1</td>
<td>1</td>
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<tr>
<td>LA size</td>
<td></td>
<td>0.054</td>
<td>0.643</td>
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</tr>
<tr>
<td>&lt;40 mm</td>
<td>51</td>
<td>1</td>
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<tr>
<td>≥40 mm</td>
<td>276</td>
<td>3.52</td>
<td>0.006</td>
<td>2.36</td>
<td>0.075</td>
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<td>Thromboembolic risk (CHADS(_2) ≥ 2)</td>
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<td>0.643</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>233</td>
<td>1.81</td>
<td>0.005</td>
<td>1.029</td>
<td>0.909</td>
</tr>
<tr>
<td>No</td>
<td>94</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
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<tr>
<td>Structural heart disease</td>
<td></td>
<td>0.054</td>
<td>0.643</td>
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<tr>
<td>Yes</td>
<td>72</td>
<td>1.22</td>
<td>0.398</td>
<td>1.57</td>
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<tr>
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<td>1</td>
<td>1</td>
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<td>SVC ablation</td>
<td></td>
<td>0.054</td>
<td>0.643</td>
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<tr>
<td>Yes</td>
<td>199</td>
<td>1.19</td>
<td>0.419</td>
<td>1.21</td>
<td>0.388</td>
</tr>
<tr>
<td>No</td>
<td>128</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
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<tr>
<td>Irrigated catheter</td>
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<td>0.054</td>
<td>0.643</td>
<td>1</td>
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</tr>
<tr>
<td>Yes</td>
<td>155</td>
<td>0.52</td>
<td>0.003</td>
<td>0.67</td>
<td>0.147</td>
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<tr>
<td>No</td>
<td>172</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
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<tr>
<td>Early recurrence</td>
<td></td>
<td>0.054</td>
<td>0.643</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>69</td>
<td>5.52</td>
<td>0.000</td>
<td>4.67</td>
<td>0.000</td>
</tr>
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<td>No</td>
<td>258</td>
<td>1</td>
<td>1</td>
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</table>

On the final model, variables with \( P > 0.05 \) were excluded. HR indicates hazard ratio; HRadj, adjusted hazard ratio; SVC, superior vena cava. Other abbreviations as in Table 1.
Symptomatic ischemic cerebrovascular events were not detected during follow-up, despite interruption of OAC in 298 (91%) patients and AADs in 293 (89%), respectively. Long-term maintenance of OAC therapy was recommended in 22 (9.5%) patients without recurrent AF because of a history of LA thrombus, systemic embolization, or frequent nonsustained atrial arrhythmias recorded on Holter monitoring. Antiplatelet therapy was used indefinitely in 308 (94%) patients and was rarely discontinued mostly in low-risk patients (CHADS2 score of 0 or 1).

Symptomatic hemorrhagic strokes were detected on CT imaging in 3 (0.9%) patients while receiving OAC, which resulted in significant motor and neurological deficits. One patient required decompressive surgery. These events occurred 2, 9, and 11 months after the RFA procedure.

### Discussion

#### Main Findings

In this study, we report a single-center experience and long-term results of a standardized catheter ablation strategy and postprocedural management of 327 patients undergoing RFA for symptomatic drug-refractory AF. The proposed ablation and postablation strategy resulted in an 82% AF-free survival rate without use of AADs and a remarkably low incidence of cerebrovascular events during a mean follow-up of 4 years (range, 1–6.8 years).

In this series, OAC and AAD therapy were withdrawn after RFA if neither symptomatic nor asymptomatic atrial arrhythmias could be detected by frequent pulse checks, ECGs, or serial Holter monitoring performed every 3 months during the first year postablation. All patients were maintained on antiplatelet therapy regardless of their preprocedure CHADS2 risk score.

After this protocol, neither ischemic strokes nor systemic emboli were detected. In addition, no AF-related mortality occurred. In fact, the only documented major adverse events were hemorrhagic strokes in 3 patients who had continued to receive OAC. These observations may have important clinical implications.

#### Previous Studies

The present study evaluated the long-term results of catheter ablation therapy for symptomatic AF in conjunction with OAC cessation. It is of particular relevance in light of recently published studies that have put into question the long-term efficacy of catheter ablation in the treatment of AF.

Tzou et al. reported long-term data on 123 patients free of AF while off AAD at 1 year after RFA. Interestingly, they showed a steady 7%/year late recurrence rate, with 71% of patients AF free at 5 years. Persistent AF, older age, larger LA size, and presence of a greater number of AF triggers predicted very late recurrences.

Weerasooriya et al. recently described a 5-year experience with 100 patients undergoing a median of 2 RFA procedures. Arrhythmia-free survival after a single procedure was 40%, 37%, and 29% at 1, 2, and 5 years, respectively, evident by clinical or Holter documentation of atrial arrhythmias lasting >30 seconds. After 2 procedures, the results improved to 87%, 81%, and 63% freedom from AF at 1, 2, and 5 years, respectively. Although most recurrences were in the first 12 months, a slow, but steady decline in AF-free survival was noted thereafter.

Meanwhile, there is a body of data in support of favorable long-term outcomes when OAC is discontinued in patients who undergo catheter ablation of AF. Bhargava et al. reported their multicenter experience with 1404 patients during 57±17 months of follow-up. Using a similar RFA ablation technique and postprocedural management, 78% of patients with PAF and 67% with non-PAF were AF free after a single ablation procedure. In patients undergoing a redo procedure, 92% and 84%, respectively, remained AF free. Similar to the present findings, Bhargava et al. reported few thromboembolic complications (stroke rates, ≈0.3%) when using a strategy of withholding OAC at 6 months with documentation of sinus rhythm.

Oral et al. reported on 755 patients with PAF and LSP AF who underwent catheter ablation and in whom OAC was discontinued. In their cohort, 55% had ≥1 stroke risk factor. Of the 70% who remained in sinus rhythm, OAC was discontinued in 68% after 3 to 6 months postablation (mostly in patients aged <65 years and in those with no history of prior stroke). Thromboembolic events occurred in 9 (1.2%) patients, of whom 6 (0.9%) had an early event (within the first 30 days after RFA); 6 had ≥1 risk factor for stroke and a subtherapeutic international normalized ratio <2.0. No further late embolic events were detected after 2 years of close follow-up.

The largest reported series was recently published by Themistoclakis et al. in a multicenter retrospective analysis of 3355 patients followed for a mean of 28±13 months. Similar to the present study, OAC therapy was withdrawn and replaced with aspirin in 80% of patients within 3 to 6 months after successful ablation. Among these, only 2 (0.07%) patients had an ischemic stroke compared with 3 (0.45%) who had a stroke while receiving OAC. No embolic events were recorded in the moderate-risk patients (CHADS2 ≥2). However, it should be emphasized that such patients only represented 13% (n=347) of the entire cohort. In fact, most (82%) patients in this study were at low risk for thromboembolic complications (CHADS2 ≤1) and did not necessarily have a compelling indication for OAC therapy. This population is different from the present study, in which more than two thirds were at moderate risk for stroke (CHADS2 ≥2).

Another important finding of the above mentioned study is that continuation of OAC was associated with a 13-fold increase in major hemorrhagic events (2% on OAC versus 0.04% off OAC therapy), supporting the notion that OAC has significant risks and that the risk-to-benefit ratio may favor discontinuation, even in patients with a moderate risk score. Similar results have been reported after surgical ablation for AF. Ad et al. evaluated 385 patients (17% with previous thromboembolism) after surgical ablation followed for an average of 32 months and recorded 4 embolic events (4.2 first events per 1000 patient-years); on the contrary, bleeding events occurred in 69 patients (72.8 first events per 1000 patient-years), predominantly in higher-risk patients (mean CHADS2 score, 2.3). No correlation was found between baseline CHADS2 score and embolic events, reinforcing the need for randomized data addressing anticoagulation strategies after ablation.
Implications
Several recent reports suggested that cessation of OAC after a successful catheter ablation procedure in treating drug-refractory AF may result in a very low incidence of thromboembolic complications in lower-risk patients. The present series confirms and expands these findings, suggesting that OAC may be safely discontinued in patients who remain free of symptomatic or asymptomatic atrial arrhythmias (detected by periodic ECG and serial Holter monitoring) despite a preprocedural CHADS2 score ≤3.

Undoubtedly, acceptance of RFA procedures for AF would likely increase if OAC therapy could be safely discontinued in patients who remain free of symptomatic or documented asymptomatic AF after catheter ablation. However, at this time, larger randomized trials are required to confirm and validate these findings. In case of a positive outcome, ablation procedures might even be extended to patients with asymptomatic AF whose sole objective may be the possibility of eliminating OAC therapy. The present study not only suggests that it is safe to stop OAC after a successful ablation procedure, but also emphasizes that severe hemorrhagic complications can be an issue more frequently in the general community than recognized in clinical trials.23 This perceived fear justifies the observed underutilization of warfarin in patients with AF with clear indications.24

It is also important to emphasize that the patients in the present cohort continued on antiplatelet therapy after discontinuation of OAC. Data from clinical trials advocate that warfarin is superior to antiplatelet therapy.25 However, such trials did not include patients previously treated by RFA. There is evidence, such as that reported by Bunch et al,26 showing equivalence of outcomes when low-risk patients are discharged on antiplatelet therapy rather than on warfarin after ablation. In Bunch et al, 690 patients were discharged on antithrombotic therapy with either warfarin or aspirin (in lower-risk patients [CHADS2 score, 0–1]) after AF ablation using an irrigated-tip catheter. After 1 year of follow-up, no events occurred in the aspirin group, suggesting that antiplatelet therapy might be enough in low-risk patients.

As suggested by Dagres et al,27 it is possible that we may be overtreating low-risk patients with warfarin after RFA ablation. These authors reported on 844 patients (mostly with a CHADS2 score of 0–1), demonstrating that anticoagulation after RFA, contrary to current recommendations, is hardly guided by stroke risk profile (90% of patients were still on OAC at 12 months). The most important factor influencing anticoagulation use was AF recurrence and not the CHADS2 risk score.

Limitations
We cannot exclude the likelihood that certain patients in our series did not experience undocumented asymptomatic AF during follow-up. In fact, this has been shown to be quite frequent among patients who undergo catheter ablation of AF.23 However, the data suggest that asymptomatic patients with undocumented AF who are transitioned from OAC and AADs to antiplatelet therapy are not at a higher risk for thromboembolic stroke and complications after successful catheter ablation therapy.

Our protocol involved strict follow-up, including review of ambulatory ECGs and Holter recordings and phone contact with referring physicians and patients not compliant with office visits.

As such, the findings do not mean that OAC can be stopped in all patients undergoing successful RFA; it should be considered only for those who are similarly screened and selected.

The study population included patients with relatively low to moderate risk for thromboembolism as reflected by a CHADS2 score ≤3. Thus, extrapolating the findings of this study to a higher-risk population (CHADS2 >3) does not seem prudent. Larger studies are necessary to evaluate the role of OAC after catheter ablation of AF in higher-risk patients to further address this question.

Conclusions
No significant thromboembolic-related morbidity is observed when AADs and OAC are discontinued after successful catheter ablation of AF in patients with a CHADS2 score ≤3 who are maintained on antiplatelet therapy during long-term follow-up.

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Disclosures
None.

References

CLINICAL PERSPECTIVE

Very Low Risk of Thromboembolic Events in Patients Undergoing Successful Catheter Ablation of Atrial Fibrillation With a CHADS² Score ≤3: A Long-Term Outcome Study

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