S

stroke and thromboembolism are recognized as one of the most serious complications of atrial fibrillation (AF) because they are associated with a substantial risk of morbidity and mortality.1 Oral anticoagulation (OAC) with warfarin is highly effective in preventing thromboembolic events in patients with AF.2 However, treatment with OAC necessitates frequent monitoring and dietary and drug restrictions and may be associated with serious side effects, of which hemorrhagic stroke is the most devastating. Therefore, adequate risk stratification of AF patients and meticulous evaluation of benefits and risks are mandatory. Various risk factors have been identified as contributing to the risk for stroke in AF and risk stratification schemes such as the CHADS2 score have been established and validated.3 Although this score identifies patients who benefit most and least from anticoagulation, the threshold for use of anticoagulation is controversial, especially for those at intermediate risk (stroke rate, 3% to 5% per year).4 Updated guidelines tend to an earlier anticoagulation strategy withholding OAC only in patients who are truly low risk.5

The optimal anticoagulation treatment of patients undergoing successful AF catheter ablation is even less well defined. Limited data are available regarding the risk of thromboembolism with and without warfarin after AF ablation. Challenges arise from the definition of success, with noncontinuous monitoring techniques underestimating true arrhythmia recurrence rates,6 an increasing amount of asymptomatic AF after ablation,7 controversial data on long-term efficacy of AF catheter ablation,8,9 and on how much AF is necessary to provoke thromboembolic events. For these reasons, current recommendations rely on the patient’s individual baseline thromboembolic risk irrespective of a potentially curative ablation procedure to decide on long-term anticoagulation strategies.5,10

Patients having a CHADS2 score ≥2 are generally not recommended to discontinue OAC. But does the individual thromboembolic risk really remain unchanged after a successful ablation procedure? Data on this are limited and most welcome because one might speculate that elimination of AF may reduce the preprocedural risk of thromboembolic stroke.11 Eliminating restrictions and bleeding risks associated with long-term OAC would be desirable to patients as well as doctors.

In this issue of Circulation: Arrhythmia and Electrophysiology, Saad et al12 evaluate the long-term results of OAC cessation after successful catheter ablation of AF in 327 consecutive patients with various CHADS2 scores (CHADS2 0: 10%; CHADS2 1: 19%; CHADS2 2: 45%; CHADS2 3: 23%; and CHADS2 4: 2.5%). Corresponding with current recommendations,5,10 all patients received OAC for 3 months irrespective of the individual thromboembolic risk. This procedure is based on a presumed prothrombotic state, induced by activation of clotting cascade and atrial stunning early after AF ablation, placing previous low-risk patients at a temporarily elevated thromboembolic risk.13 After 3 months, OAC was replaced with antiplatelet therapy (aspirin or clopidogrel), providing that patients were asymptomatic and no arrhythmia recurrence (AF >2 minutes) was documented (frequent pulse checks, ECG and 24-hour Holter monitoring at 1, 3, 6, 9, 12 months and in two-thirds of patients biannually thereafter). Of note, duration of anticoagulation was extended to 6 months in 46% of patients, to 12 months in 28% of patients, and indefinitely in 9.5% of patients with a history of left atrial thrombus, systemic embolization, or frequent atrial arrhythmias. No symptomatic ischemic cerebrovascular events were detected during follow-up despite interruption of OAC in 91% of patients, whereas 3 hemorrhagic strokes (0.9%) occurred in warfarin-treated patients. Interesting, but do these data provide a major breakthrough in favor of OAC withdrawal after ablation? No. This is due to a number of significant limitations of the study from Saad et al that must be recognized and considered. (1) The study had an unusually low inclusion rate: over a period of almost 6 years, a relatively small number of consecutive patients were included. (2) The study was designed as a retrospective, single-center data analysis. (3) Information was lacking regarding the adherence of the study population to the follow-up regime foreseen with respect to ECG monitoring and completeness of follow-up: only two-thirds of the patients complied with the biannual visits beyond the first year after ablation. Even more important, follow-up data appear to be completely lacking in a significant number of patients (25/352, 7.1%). (4) Standards were lacking regarding guiding anticoagulation therapy throughout the study: continuation of anticoagulation therapy on investigators discretion “in some patients” in whom “a high risk of recurrence was suspected” is certainly not an adequate standard to guide therapy in a study on anticoagulation and embolic risks.
Nevertheless, the presented findings are supported by prior data and challenge current treatment recommendations. Likewise, Bunch et al describe the safety of discharging selected low-risk patients (CHADS2 scores, 0 and 1; n = 123) undergoing AF ablation with aspirin in a retrospective single-center study. Over a follow-up of 1 year, no strokes occurred in the aspirin group, but 4 strokes (CHADS2 scores: 2, 2, 3, and 4) occurred in the warfarin group (n = 507). Oral et al reported a similar stroke risk in patients after catheter ablation compared with a matched population without history of AF. In this retrospective analysis of 755 patients (82% with CHADS2 score 0 and 1), OAC was discontinued in 75% of patients after 3 to 6 months and replaced with aspirin in the case of freedom from AF (>60 seconds; assessed with event monitors, serial ECG and/or 24-hour Holter ECG every 3–6 months for 1–2 years). None of these patients had thromboembolism during a follow-up of 25±8 months, whereas 2 late thromboembolic events occurred in patients adequately anti-coagulated (CHADS2 scores, 0 and 2). The largest study (n = 3355) on this, even though retrospective as well, was published by Themistoclakis et al. OAC was discontinued after 3 to 6 months in 80% of patients (87% with CHADS2 score 0 and 1) irrespective of CHADS2 score unless patients did not have any recurrence of atrial tachyarrhythmias (>1 minute assessed with Holter and/or transtelephonic monitoring every 3–6 months), severe pulmonary vein stenosis, or severe left atrial mechanical dysfunction (absence of A-wave). During a follow-up of 2 years, 2 patients on aspirin (CHADS2 scores, 0 and 1) and 3 on warfarin (CHADS2 scores, 1, 2, and 2) had an ischemic stroke. The authors emphasized that none of the patients with discontinued OAC and a CHADS2 risk score of ≥2 (n = 347) had an ischemic stroke. Major hemorrhage was observed in 1 patient off warfarin and 13 patients on warfarin. Although these retrospective studies suggest that the risk-benefit ratio favored the suspension of OAC after successful AF ablation over medium-term follow-up in selected patients, these results have never been confirmed by a large prospective randomized trial and are limited to rather low-risk patients. New data such as the results of the study from Saad et al apparently extend these findings to intermediate-risk patients (two-thirds of patients had at least 2 thromboembolic risk factors) with a longer follow-up of almost 4 years. However, these conclusions must be handled with caution because recent studies indicate a clear inferiority of aspirin to reduce thromboembolic events when compared with warfarin even in low-risk or elderly patients with AF, and a significant reduction in bleeding complications has never been proven. Actually, the rare occurrence of thromboembolic events after AF ablation would necessitate a large prospective, randomized study to accurately evaluate the safety and efficacy of anticoagulation versus antiplatelet therapy versus discontinuation of OAC in patients after AF ablation. The advent of new anticoagulants such as oral direct- thrombin inhibitors and Factor Xa inhibitors that are easier to use and potentially safer than warfarin will probably lower the threshold for anticoagulation and direct the risk-benefit discussion in favor of anticoagulation as long as any uncertainties remain. However, if we believe that AF catheter ablation is curative and has the potential to reduce stroke risk, current anticoagulation treatment, based on risk-stratification schemes only, probably results in an overtreatment of patients. At this point in time, it is certainly too early to say that catheter ablation eliminates the need for OAC. Current evidence indicates that in the absence of major stroke risk factors, cessation of OAC in patients without documented arrhythmia recurrence may be feasible and safe. However, the more burning question is whether ablation highly reduces or even eliminates the risk for thromboembolic events in patients with higher risk scores, that is, CHADS2 score of 0 or 3. Although this issue is addressed in the study from Saad et al, their findings add little conclusive evidence, due to the study limitations mentioned above. Thus, until hard and convincing data from prospective multicenter studies are available, anticoagulation should remain standard therapy for patients after ablation with CHADS2 score ≥2.

Disclosures
Dr Hindricks received modest lecture honoraria from St Jude Medical, Biotronik, Medtronic, and Biosense and is a member of the St Jude Medical and Biosense Advisory Board.

References


Key Words: Editorials ■ anticoagulation ■ atrial fibrillation ■ catheter ablation ■ stroke ■ thromboembolism
Does Catheter Ablation Eliminate the Need for Oral Anticoagulation?
Charlotte Eitel and Gerhard Hindricks

Circ Arrhythm Electrophysiol. 2011;4:595-597
doi: 10.1161/CIRCEP.111.966218
Circulation: Arrhythmia and Electrophysiology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2011 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-3149. Online ISSN: 1941-3084

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circep.ahajournals.org/content/4/5/595

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Arrhythmia and Electrophysiology 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: Arrhythmia and Electrophysiology is online at:
http://circep.ahajournals.org/subscriptions/