EDITORIAL COMMENT

Should All Patients Be Started on Antiarrhythmic Drugs After Atrial Fibrillation Ablation?*

Edward P. Gerstenfeld, MD

It has been recognized since the early days of catheter ablation to treat atrial fibrillation (AF) that some patients can have early recurrences of AF in the weeks after ablation. This has been attributed to the effects of inflammation and pericarditis after ablation; several studies have shown that anti-inflammatory agents such as colchicine (1) may reduce these early recurrences. In paroxysmal AF patients, early recurrences clearly increase the risk of late recurrences, while in patients with persistent AF, such an association is less certain.

Early AF after ablation can be a source of frustration for patients, as they have just gone through an extensive procedure to treat their AF, only to suffer recurrent bothersome episodes. Such recurrences can also increase the burden and cost to providers, as patients require additional medication adjustments, cardioversions, and sometimes hospital admission. The cost to the healthcare system after an expensive ablation procedure should not be underestimated.

During a meeting in 2006, I was asked to give a lecture on treatment of patients after AF ablation. Upon reviewing the literature, I discovered that there were no studies addressing the question of whether antiarrhythmic drugs (AADs) were helpful or harmful after ablation. A few patients in our practice had developed proarrhythmia from use of Vaughan Williams Class IC agents after ablation leading to rapid atrial tachycardias, so it was not readily apparent that these agents were clearly beneficial. After a brief discussion with my partners, the 5A (Antiarrhythmics after Ablation of Atrial Fibrillation Study) study (2) was born. Our hypothesis was that the adverse effects of AADs would outweigh the benefits, and that routine use of AADs after ablation would not be warranted. Because we were only interested in the post-ablation period, we randomized patients to continue or stop AADs after ablation. The results demonstrated a clinically small but statistically significant benefit to AAD continuation after ablation, reducing the composite endpoint of arrhythmia recurrence lasting >24 h, hospitalization, or adverse event from AADs through 6 weeks after ablation. Of note, most patients in the study were on AADs before ablation, while the minority (15%) were started on new AADs after ablation.

In this issue of *JACC: Clinical Electrophysiology*, Noseworthy et al. (3) revisit this question using data culled from a large insurance claims database. They identified a large number of patients who underwent AF ablation and were not on AADs before ablation, and were newly initiated on AADs on the day following ablation. To perform this analysis they examined diagnosis codes for patients who had not filled an AAD prescription for 90 days before ablation, and began a new AAD the day of or following ablation. They found that there was a significant reduction in hospitalization within 90 days after ablation for those patients who were initiated on AADs before ablation, while the minority (15%) were started on new AADs after ablation.

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From the Section of Cardiac Electrophysiology, University of California, San Francisco, California. Dr. Gerstenfeld has received research grants from Biosense Webster and St. Jude Medical; and honoraria from Biosense-Webster, St. Jude Medical, Cardiofocus, and Boston Scientific.
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Atria. Most persistent AF patients will have also tried higher drugs and patients need time to reverse remodel the atria after ablation, because early AF recurrence rates are empirically high. Most electrophysiologists will empirically start patients with persistent AF on AADs versus persistent AF. Most electrophysiologists will know what proportion of patients had paroxysmal AF versus persistent AF. Most electrophysiologists will empirically start patients with persistent AF on AADs after ablation, because early AF recurrence rates are higher and patients need time to reverse remodel the atria. Most persistent AF patients will have also tried at least 1 AAD before ablation. However, even if we assume the majority of patients in the study have paroxysmal AF, we still cannot determine a physician’s reason for starting the AADs from a retrospective database. It is possible that physicians started some patients on AADs because they recognized markers of higher AF recurrence in some patients, such as left atrial enlargement, left atrial scarring, frequent atrial premature atrial complexes after ablation, or a difficult procedure. Therefore, it is difficult to know if the authors’ results apply to all patients undergoing AF ablation.

Shortly after moving to my current institution, a fellow asked what AAD I wanted to prescribe a patient after AF ablation, presuming that I followed the outcome of my own SA study. However, I somewhat embarrassingly admitted that I do not routinely start patients without previous AAD use on a new AAD. Why is this the case? There are several, largely practical, reasons. First, I typically recommend a trial of an AAD before ablation, so patients who have elected to undergo primary ablation without recent AAD use have either already experienced side effects from these drugs, or are reluctant to take AADs because of anticipated side effects. These patients are referred for nonpharmacologic therapy because they wish to avoid AADs, so placing them all on AADs after ablation seems unnecessary. Class IC drugs can also lead to proarrhythmia, particularly after ablation when the atria may be predisposed to slow conduction around small ablation scars. Monitoring during AAD initiation can also be a problem. In the early days of AF ablation, we used to keep patients in the hospital for several days after ablation to wait for therapeutic anticoagulation with warfarin. During this time, an AAD could be initiated and the patient monitored for proarrhythmia. Today, due to higher use of novel anticoagulants and uninterrupted warfarin, patients are discharged the morning after ablation and such a window for monitoring does not exist. This likely accounts for the lack of use of dofetilide and sotalol in the authors study, as these agents typically require prolonging hospitalization to monitor for proarrhythmia. Finally, AADs do have side effects in many patients, including fatigue, loss of appetite, loss of taste, and unclear thinking. It seems unfair to expose all patients to these side effects without a clear benefit. Even in the authors study, one could argue that a 3.1% reduction in admission for recurrent AF or flutter is worth exposing all patients to AAD side effects. Finally, the authors find that Class IC agents and dronedarone did not reduce hospitalization, and only amiodarone was beneficial. When discussing AAD options for patients with AF, we discuss the multitude of long-term side effects of amiodarone. Although the authors’ data support only short-term use, young patients are often reluctant to take amiodarone, even for a short period of time, and the side effects of fatigue, loss of appetite, and hypo/hyperthyroidism are real and can affect many patients even early in the course of therapy. Additional considerations are that it is difficult to ascertain whether the procedure has been a “success” until AADs are stopped. A patient with frequent paroxysmal AF who shows up for the 6-week visit off of AADs with no AF is likely to do well, while if a patient is on an AAD, prediction of longer-term results are delayed.

So how would I integrate the findings of the SA study and the present paper into management of patients after ablation? My practice is the following. 1) All patients with persistent AF on AADs before ablation resume AADs after ablation for 3 to 6 months. Patients with persistent AF who undergo primary ablation without prior AAD use are typically started on an AAD after ablation. My personal preference is to admit such patients for initiation of dofetilide after ablation, and stop it after 6 months if there is no recurrent AF. 2) For paroxysmal AF patients, if the procedure is straightforward and there is a paucity of ectopy on telemetry overnight after ablation, I discharge them with an AV nodal blocker but no AAD therapy. Patients are all discharged with event monitors, and in the case of recurrent AF the arrhythmia can often be managed as an outpatient with adjusting medical therapy or initiation of a previously tolerated AAD, as appropriate. Certainly for bothersome recurrent AF symptoms, the manuscript would support initiation of amiodarone,
which could be started as an outpatient and stopped after 3 to 6 months. 3) For the patients that are the subject of this paper who are not on AAD before ablation, I typically do not initiate an AAD for all the reasons stated above. The lack of benefit in the paper (3) of IC agents seems to support this approach. However, for patients with significant left atrial scarring, multiple inducible flutters, or frequent ectopy of bursts of AF on telemetry after ablation, initiation of an AAD is reasonable. For young patients with normal renal function, I still would lean toward using a Class IC agent or dofetilide as initial therapy. But if episodes persist, then amiodarone for a short period is reasonable and supported by the current manuscript.

Fortunately, using current ablation techniques including wide-area antral pulmonary vein isolation with contact force catheters or the use of cryoballoon, my impression is that early AF recurrences are much lower than they were in the past. Most of us have evolved fairly straightforward practice strategies for managing post-operative AF—increasing atrioventricular nodal blockers and observation is often sufficient. The analysis in the paper (3) is useful, but the limitations of a retrospective review, in addition to the potential side effects of amiodarone, are not enough to alter my current approach. One would need a more detailed prospective study measuring quality of life and cost to conclusively determine whether an empiric strategy of amiodarone for 3 months after ablation was worthwhile. However, the authors data has taught us that in some patients at risk for early AF recurrence, empiric AAD therapy for several months after ablation is reasonable and may reduce hospitalizations. Meanwhile, the quest to reduce early AF recurrences after ablation will continue.

REPRINT REQUESTS AND CORRESPONDENCE: Dr. Edward P. Gerstenfeld, Section of Cardiac Electrophysiology, University of California-San Francisco, 500 Parnassus Avenue, MU East, 4th Floor, San Francisco, California. E-mail: egerstenfeld@medicine.ucsf.edu.

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