Ablation for Atrial Fibrillation

Can We Decrease Thromboembolism Without Increasing the Risk for Bleeding?

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Stroke is one of the most feared untoward sequelae associated with atrial fibrillation (AF) ablation procedures, whereas bleeding is the most common.1,2 We typically explain to patients that intensive anticoagulation during ablation is necessary to decrease the likelihood of stroke, even though some increase in bleeding and vascular complications will occur. In the present issue of Circulation, Wazni et al3 report performing AF ablation in patients with a therapeutic International Normalized Ratio (INR) and successfully decreasing thromboembolism without increasing the risk of bleeding.

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The hitherto standard approach taken by most ablationists is to discontinue Coumadin for 3 to 5 days before ablation and proceed after normalization of the INR. Although simple, 3 issues require attention with this approach. First, in the period when the patient is off warfarin to prevent thrombus formation, low molecular weight heparin is administered. This approach is generally successful because, when transesophageal and/or intracardiac echocardiography is performed just before left atrial ablation, only rarely is thrombus found. Second, a more difficult issue is reanticoagulation after ablation where there may be periods of inadequate anticoagulation, which allows thrombus to occur. Third, heparin used as a bridge postablation has a higher propensity to promote postprocedural bleeding than warfarin itself.4 Wazni et al suggest the solution of simply continuing warfarin and obviating the need to address the above problems.3

Does their approach allow the brinkmanship necessary to resolve this dilemma by simultaneously decreasing thrombotic and hemorrhagic risk, and should AF ablation be performed with a therapeutic INR? Let us first examine what approaches have been previously tried to achieve this desired result and then analyze the potential limitations of their study.

The risk of thromboembolism is primarily related to catheter placement and ablation in the left atrium, whereas bleeding and vascular complications arise principally when accessing the vasculature. Thus, first minimizing (until sheaths are placed) and then intensifying anticoagulation when the left atrium has been entered is the precept underlying current ablation practice.5

Intensity of Anticoagulation

Studies have shown that once vascular access has been obtained, more aggressive anticoagulation (maintaining activated clotting time of 350 to 400 seconds) is more effective in the prevention of thromboembolism without significant bleeding or vascular complications.6,7

Timing of Anticoagulation

Initially with AF ablation, because of minimal experience and fear of complications with transseptal puncture, heparin was delayed until the second transseptal sheath had been placed. With intracardiac ultrasound,6 it became evident that thrombus may have already developed on sheaths, catheters, and in the right atrium (and potentially transferred to the left atrium) because of this delay. More recently, experienced operators have favored complete heparinization after vascular access and clearly before transseptal puncture (C. Bruce, MD, et al, unpublished observations, 2005).

Targeted Heparinization

Because sheaths and catheters placed in the left atrium are the primary sites where thrombus develops, heparin infusion at high concentration8 is used to minimize soft thrombus formation without augmentation of systemic anticoagulation. Even if one is to completely ignore the risk of bleeding, is it possible with anticoagulation, however intense, to completely eliminate the risk of ablation-related thromboembolism? To answer this question, one must appreciate the difference between coagulum and thrombus formation (Figure). Although these terms have been inexact and variably defined in previous studies, coagulum is directly heat-denatured fibrinogen, whereas thrombus formation requires the presence of thrombin to activate fibrinogen-producing clot. Because coagulum formation is independent of thrombin, heparin cannot prevent its formation. Because coagulum itself may embolize or form a nidus for propagative clot formation and its occurrence is related to overheating at electrode surfaces, ablation and imaging-related approaches have been tried to avert this complication.9,10

Power Titration

Coagulum formation is heralded by the simultaneous development of low power requirement with high temperatures and rising impedance. Several studies, however, have shown that
simply titrating ablation manually to avoid formation of coagulum on the electrode is ineffective.\(^2\,^{12}\)

**Intracardiac Ultrasound**

Linear phased-array imaging from the right atrium with close observation of the electrode surface during ablation has been used to help titrate energy delivery and decrease coagulum formation.\(^{13}\,^{14}\) Further, earlier detection of thrombus formation when formed has allowed timely, appropriate suction and removal. Profuse microbubble formation detected at ultrasound sometimes allows downward power titration before impedance rise and coagulum formation.\(^{15}\)

**Charge Delivery During Ablation**

Another technique being investigated (B. Lim, MD, PhD, et al, unpublished observations, 2005) is placement of a negative charge on catheters and electrodes during ablation.\(^{17}\) Fibrinogen, a negatively-charged molecule, may be prevented from undergoing the conformational changes that precede coagulum formation with this approach. Given the quagmire with regard to preventing stroke that confronts ablationists, we welcome the demonstration by Wazni et al\(^3\) that, by simply continuing warfarin, a marked decrease in spontaneous echo contrast (SEC) and thrombosis occurs, but with no increase in bleeding risk. Should ablation with therapeutic INR now be praxis? While considering how best to incorporate this important and practical report into our own ablation practice, we should note the following.

**The Learning Curve**

In their present report, the group’s earlier experience involved patients bridged with low molecular weight heparin, and their suggested approach of continued warfarin use represents their most recent experience. Because of the complexity of AF ablation and the significant learning curve associated with safe multiple vascular accesses, catheter manipulation, and the technique of guiding ablation with intracardiac ultrasound, this may have affected their results. Ablationists early in their practice without considerable experience with these approaches should consider whether they would presently adopt the recommendation to not discontinue warfarin prior to ablation.

**Dealing With Complications**

Regardless of the operator’s experience, complications such as cardiac perforation do occur. When intravenous heparin has been used for anticoagulation, reversal with protamine and reinitiation is straightforward. With continued warfarinization, however, should perforation occur, reversal of anticoagulation involves the use of fresh frozen plasma and/or vitamin K with longer-lasting effects and considerable difficulties with reanticoagulation. As the authors point out, the period soon after ablation is a critical period for thromboembolic risk, and this increased difficulty with reanticoagulation when warfarin has been reversed may paradoxically increase thromboembolic risks.

**Type of Atrial Fibrillation**

Wazni et al report their findings with continued warfarin use only in patients with persistent AF.\(^3\) When paroxysmal AF is present, whether the potential increased risk of bleeding with their approach would still outweigh the less significant thromboembolic propensity in paroxysmal AF patients is not known from the present study.

**Spontaneous Echo Contrast**

A puzzling finding in the present report\(^3\) is the dramatic decrease in SEC (smoke) in the group where warfarin had not been discontinued. SEC detected with ultrasound is produced as a result of the interaction between erythrocytes and plasma protein (including fibrinogen), and its video density increased with stasis.\(^{18}\,^{19}\) Because SEC has been shown not to be “mini-thrombi” in prior studies,\(^{20}\) there should be no effect or change in the incidence of SEC when continuing warfarin rather than bridging with low molecular weight heparin. Why then was there this highly significant decrease in SEC in the group where warfarin was continued? An intriguing possible explanation is that SEC detected with intracardiac echocardiography is fundamentally different from that which has been studied and detected with transesophageal echocardiography and may in fact represent very small thrombi whose formation is mitigated with this brief, stable use of warfarin. Nevertheless, given the previous documented evidence that warfarin does not affect smoke even when it decreases the incidence of thrombus,\(^{20}\) we are left with the possibility that, in the present longitudinal study, the later patients had a less significant milieu for thrombosis than the prior groups.

Despite these potential limitations, Wazni et al\(^3\) have clearly answered the important question of whether complex left atrial ablation can be performed when the patient’s INR is still therapeutic. They have therefore established an optional approach for ablationists in our quest to decrease thromboembolism without increasing the risk of bleeding.

**Disclosures**

The author is coholder of a patent for a technique to decrease coagulum formation during radiofrequency ablation and may receive royalties from this invention.
References


8. Deleted in proof.


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