Editorial

Discontinuing Anticoagulation after Atrial Fibrillation Ablation: Has the Evidence Really Changed?

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Referring to: Nayak SS et al. Clinical outcomes of continuing vs discontinuing oral anticoagulation after atrial fibrillation ablation: an updated systematic review and meta-analysis of observational studies and randomized controlled trials.[1] Atrial fibrillation (AF) is one of the most commonly encountered heart conditions, and its prevalence is increasing as the population ages.[2] Beyond its symptomatic burden, AF is associated with serious complications, of which thromboembolism is probably the most significant one. Generally, antithrombotic therapy decisions require careful assessment of thrombotic versus bleeding risks. In AF, these risks often arise from overlapping factors, including age, hypertension, prior stroke, vascular disease, and renal impairment, which contribute to both CHA_2DS_2-VA and HAS-BLED scores. According to the latest guidelines, anticoagulation should be considered in patients with AF and a CHA2DS2-VA score of 1 (class IIa) and is recommended in patients with ≥2 scores (class I).[2]

Anticoagulation decisions are not straightforward in every clinical scenario. One challenging scenario involves patients who have undergone AF ablation. Since the first reports of pulmonary vein isolation in the late 1990s, AF catheter ablation has become the most effective strategy for maintaining sinus rhythm. Despite major advances in ablation technology, success rates remain suboptimal. In a recent systematic review and meta-analysis of 477 cohorts comprising more than 70 000 patients, single-procedure freedom from atrial arrhythmia at 12 months without antiarrhythmic therapy was only 66%.[3] Consequently, deciding whether to discontinue anticoagulation after AF ablation has long been a dilemma. The most current European Society of Cardiology (ESC) guidelines published in 2024 recommend continuing anticoagulation for at least 2 months after AF ablation in all patients, regardless of rhythm outcome or CHA_2DS_2-VASc score, and thereafter deciding based on the patient's CHA_2DS_2-VASc score rather than the perceived success of the ablation procedure. Nonetheless, the level of evidence for this recommendation is C (consensus of expert opinion and/or small studies and retrospective registries).[2]

The systematic review conducted by Nayak et al[1] aims to fill this evidence gap. This systematic review, published in EP Europace in July 2026, evaluated post-AF ablation anticoagulation. The authors pooled 31 studies comprising 50 327 patients, including 3 randomized clinical trials. Extension of oral anticoagulation beyond the blanking period was not associated with a reduction in stroke or transient ischemic attack (TIA) (odds ratio [OR], 1.48; 95% confidence interval [CI], 0.84 to 2.60; P=.16) but was associated with a substantially higher risk of major bleeding (OR, 4.62; 95% CI, 2.42 to 8.85; P<.01). In the subgroup with CHA_2DS_2-VASc score of 2 or higher, continuation was associated with a lower risk of stroke or TIA (OR, 0.42; 95% CI, 0.18 to 0.95; P=.04), which the authors highlighted as their main finding. Moreover, the authors conducted a thorough search, the reporting followed PRISMA, and the analytic approach-including prediction intervals, leave-one-out analyses, Galbraith plots, and meta-regression-was more rigorous than is typical. The authors also shared the checks behind their pooled results, which careful readers should start with.

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Initially, the findings appear to suggest that continued anticoagulation does not reduce thromboembolic risk. Nevertheless, further scrutiny reveals concerns. In the leave-one-out analysis, excluding a single registry shifts the estimate to a significant association between continued anticoagulation and increased thromboembolism (OR, 1.44; 95% CI, 1.03 to 1.99; P=.02), a result that lacks biological plausibility and likely reflects confounding by indication.[1] Heterogeneity metrics support this interpretation: an I^2 of 90.7% and a τ^2 of 1.77 indicate substantial heterogeneity, suggesting the studies do not estimate a common effect. The broad prediction interval (0.09–24.07) further implies that future study results may vary widely, which limits the clinical relevance of the pooled findings. Another source of heterogeneity is the timing of anticoagulant discontinuation; some studies used a 3-month cutoff, while others waited 1 year to stop anticoagulation. This diversity limits the direct translation of the findings into clinical practice.

Where heterogeneity is lower, the signal changes. In the analysis restricted to TIA (I^2=51.7%), continuation approaches a protective association and becomes significant following the removal of a single outlying study (OR, 0.37; 95% CI, 0.17 to 0.79). Furthermore, in studies with similar baseline CHA_2DS_2-VASc scores, continuing anticoagulation significantly lowered the risk of stroke or TIA (OR, 0.50; 95% CI, 0.34 to 0.72; I^2=0%).[1] That subgroup comprises only 5 studies; in the remaining 26, baseline risk either differed significantly or was not reported. This represents the central limitation of the dataset: in more than four-fifths of the studies, patients who continued anticoagulation were not comparable to those who stopped regarding baseline thromboembolic risk; therefore, their event rates cannot be interpreted as a treatment effect.

Adherence is another concern. Most studies classified patients by whether they were intended to receive anticoagulants rather than whether they were adherent.[1] Only 64% to 82% of patients take these drugs as prescribed, and poor adherence can triple stroke risk.[4] This misclassification may mask true benefits and exaggerate bleeding risk.

A key claim in the analysis is the proposed threshold of a CHA_2DS_2-VASc score of 2. However, this threshold was not included in the prespecified analyses, and the meta-regression (β=−0.45; P=.17) did not show a significant association between mean CHA_2DS_2-VASc score and treatment effect.[1] Thus, the suggested cutoff remains hypothetical and insufficient to warrant changes in clinical practice.

The findings regarding bleeding risk are more robust, indicating consistency across study designs and leave-one-out analyses. The magnitude of this risk, however, requires careful interpretation: 12 of the included studies were published in 2016 or earlier and predominantly reflect vitamin K antagonist therapy. Because novel oral anticoagulants (NOACs) reduce intracranial hemorrhage by approximately 50% compared with warfarin,[5] bleeding risk estimates derived mainly from the warfarin era are not directly applicable to patients receiving NOAC therapy in contemporary practice. Granted that the authors extracted data on anticoagulant type; still, they did not report agent-specific analyses. Among the randomized trials, all of which used NOACs, the estimated risk was notably lower (OR, 2.87; 95% CI, 1.01 to 8.13).[1]

Ultimately, clinical decisions should be guided by net clinical benefit, which compares the absolute reduction in thromboembolic events with the absolute increase in major bleeding, rather than by thromboembolic and bleeding odds ratios considered separately. This study does not provide such a conclusive result.

A major, increasingly recognized concern is that the CHA_2DS_2-VASc score does not capture all variables that contribute to thromboembolism. The 2023 American College of Cardiology/American Heart Association (ACC/AHA) guideline explicitly names several: obesity, hypertrophic cardiomyopathy, reduced estimated glomerular filtration rate (eGFR) with proteinuria, and left atrial enlargement.[6] Historically, AF burden and pattern have not influenced anticoagulation decisions. Recent continuous-monitoring studies have linked higher AF burden to increased thromboembolic risk. By way of example, the KP-RHYTHM study showed a more than 3-fold higher risk in the highest tertile.[7] Conversely, the ARTESiA and NOAH-AFNET 6 trials, which focused on patients with subclinical AF, found that AF burden did not predict stroke risk or modify the benefit of anticoagulation.[8,9]

Atrial cardiomyopathy has also been better characterized.[10] There is growing consideration that atrial cardiomyopathy, rather than AF itself, may drive thromboembolism, positioning AF as a potential marker. Nonetheless the ARCADIA trial demonstrated that in patients with atrial cardiomyopathy but without AF, apixaban did not outperform aspirin.[11] This finding illustrates the persistent uncertainty regarding the relationships among atrial cardiomyopathy, AF, and thromboembolic risk.

This context shapes post-AF ablation anticoagulation decision-making. In CIRCA-DOSE, with loop recorders in every patient, median AF burden fell by more than 99%, yet only approximately half remained free of any atrial arrhythmia at 1 year.[12] Burden reduction is not a cure and certainly not abolition of the substrate. Nayak et al[1] questioned whether anticoagulation should continue after what appears to be a successful ablation. Still, determining the true recurrence of AF is difficult because intermittent Holter monitoring often overestimates how many patients are free of AF.[12] As a result, the term “successful ablation” is partly misleading.

Where does this leave practice? Most studies used the CHA_2DS_2-VASc score, where female sex added 1 point. The 2024 ESC guidelines removed the sex category, now treating female sex as a risk marker rather than a risk factor, which we implemented in our practice.[2] At a CHA_2DS_2-VASc score of 0, discontinuation after the blanking period is uncontroversial; at 2 or more, continuation remains standard. Both approaches are consistent with guidelines that anchor long-term anticoagulation to baseline risk rather than procedural success.[2] The genuine uncertainty lies at a score of 1. For those patients, we would continue anticoagulation. Ablation does not abolish the atrial cardiomyopathy that underlies thromboembolism; it suppresses the arrhythmia that renders the myopathy visible. Randomized evidence is not yet sufficient to justify withdrawal. The OCEAN trial compared rivaroxaban with aspirin rather than no therapy,[13] and ALONE-AF was underpowered, and its event rates were too low to exclude a clinically relevant difference.[14] Concerning bleeding events, the SINGLE-AF trial reported that major bleeding on a direct oral anticoagulant occurred in only 0.3% of patients over 2 years, which was numerically lower than in the group that did not receive anticoagulation.[15] Hence, our recommendation is to continue a NOAC following successful ablation when the CHA_2DS_2-VASc score is 1 or higher, except in cases where the bleeding risk clearly outweighs the thrombotic risk or a major bleeding event has occurred.

What could alter current practice? Adequately powered randomized trials employing continuous rhythm monitoring, reporting net clinical benefit, and stratifying outcomes by baseline risk. Until such evidence is available, the most defensible approach remains consistent with current guidelines: anticoagulate based on baseline thromboembolic risk instead of solely on the apparent electrocardiographic success of ablation.

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