Letter From Briceno et al Regarding Article, “An Unexpectedly High Incidence of Stroke and Left Atrial Appendage Thrombus Formation After Electrical Isolation of the Left Atrial Appendage for the Treatment of Atrial Tachyarrhythmias”

To the Editor:

We find somewhat puzzling the conclusions of the paper by Rillig et al.\(^1\) First, this is a small nonrandomized series. Second, the 3 patients with thromboembolic events had documented atrial fibrillation recurrence. Among those with left atrial appendage (LAA) thrombus, the recurrence rate is unclear. Thus, are the thromboembolic events related to the procedural technique itself or to atrial fibrillation recurrence? One could argue that these patients were on anticoagulation anyway. But this is not the case, the patient with a transient ischemic attack was off of anticoagulation, and among the 10 patients with LAA thrombus, one was off anticoagulation, 3 on vitamin K antagonists (2 had subtherapeutic international normalized ratios), and the other 6 were on novel oral anticoagulants.

Furthermore, are these complications related to loss of LAA mechanics? No. At least it seems unlikely in view of the fact that there was no differences in LAA flow velocities (0.18 versus 0.2 m/s) or isolated left atrial area (33.4 versus 29.4 cm\(^2\)) between groups. Flow velocities seem low, and we wonder if the baseline flow velocities were already abnormal. In addition, the technique used to achieve LAA isolation is different from that used by our group. They performed a wide encircling ablation of the pulmonary veins followed by a mitral isthmus line and an anterior line in the majority of patients. We feel this type of ablation may compromise atrial contractility and, possibly, be more prothrombotic. Our technique of LAA isolation is more conservative and guided by the circular mapping catheter placed in the ostium of the LAA. We have previously reported large data series, including the BELIEF randomized trial,\(^2\) illustrating that LAA isolation is not associated with higher risk of thromboembolic events even in the presence of impaired LAA function as long as optimal anticoagulation is maintained.\(^3\)

Overall, we find the conclusions from this paper misleading. Causality inferred from small uncontrolled series must be interpreted with caution.

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Disclosures

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References


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