



# Left Atrial Appendage: Extending the Search for New Sources of Atrial Fibrillation Triggers

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## Introduction

The discovery of the role of the pulmonary veins (PVs) in atrial fibrillation (AF) has facilitated us to perform an anatomy-based ablation, “PV isolation”. Although several new mapping technologies have been developed, activation mapping during ongoing AF is still challenging. To improve the efficacy of AF ablation, therefore, we attempted to find the second “hot spot”. The superior vena cava, ligament of Marshall, coronary sinus and posterior LA have been reported as second critical areas for AF ablation. However, there remain patients who are refractory to catheter ablation targeting all of those above areas. Di Biase et al. recently demonstrated that the left atrial appendage (LAA) is one of the structures which is attributable to the recurrence of arrhythmias after PV isolation.<sup>1</sup> In their study, the LAA was targeted using focal ablation technique or electrical isolation, if triggers from the LAA were documented. Electrical isolation of the LAA was effective in as high as 85% of the patients, as compared to a success rate of only 32% using focal ablation targeting the earliest activation site. Isolation of the LAA was feasible in all patients with a relatively low rate of complications (pericardial effusions, 1.8%). Thus, LAA isolation can be an option in patients who have had recurrences despite PV isolation.

The emerging issue is which ablation technique

should be performed first after the PV isolation. Di Biase et al. performed LAA isolation in only patients in whom firing was documented in the LAA usually with administration of isoproterenol. In that study, LAA firing was documented in 27% of the patients with recurrences, which accounts for only 6.7% of all the patients that underwent AF ablation in their centers. More than a decade ago, we administered isoproterenol to induce triggers for PV focal ablation. Thus, we know isoproterenol is not always effective for the induction of arrhythmias. Even among the patients who did not exhibit any LAA firing, a certain number of those patients may benefit from LAA isolation.

Another issue with the LAA isolation is the risk of future thromboembolisms. Almost half of the patients exhibited excellent LAA contraction at 6-months of follow-up. This may be due to passive blood flow or electrical reconnection. In the patients with depressed LAA contraction, however, the continuation of anticoagulation therapy or LAA occlusion needs to be considered. In patients with excellent LAA contraction at the mid-term follow-up, it is unknown whether or not the LAA contraction will remain that way for the next 10 years. There remain issues to be solved concerning the LAA isolation; however, we recognized that the LAA is an important source of triggers in AF in addition to the thoracic veins.

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References

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