CONTEMPORARY REVIEW

Techniques for the provocation, localization, and ablation of non–pulmonary vein triggers for atrial fibrillation

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The end point of current catheter-based ablation approaches for the treatment of atrial fibrillation (AF) is the elimination of all the possible triggers with the least amount of ablation necessary. Once all the triggers have been eliminated, the incremental value of any additional lesion sets remains to be proven. Pulmonary vein (PV) isolation is the cornerstone of catheter ablation approaches for eliminating AF triggers. However, up to 11% of patients demonstrate reproducible sustained AF initiation from non-PV foci. In these patients, triggers can typically be elicited using standardized induction protocols, which include cardioversion of spontaneous and/or induced AF and infusion of high-dose isoproterenol. Non-PV triggers typically arise from discrete anatomical structures that include the mitral and tricuspid periannular regions, the crista terminalis and Eustachian ridge, the interatrial septum, the left atrial (LA) posterior wall, the left atrial appendage (LAA), and other thoracic veins such as the superior vena cava, the coronary sinus, and the ligament of Marshall. Localization of non-PV foci involves a detailed analysis of specific intra-atrial activation sequences using multipolar catheters in standard atrial locations coupled with information from the surface electrocardiogram P wave when possible. Multipolar catheters positioned along the coronary sinus and crista terminalis/superior vena cava region together with direct recordings from the right and left PVs allow a quick localization of non-PV foci. Elimination of non-PV triggers by means of focal ablation at the site of origin or isolation of arrhythmogenic structures (eg, LA posterior wall or superior vena cava) has been associated with improved arrhythmia-free survival.

KEYWORDS Atrial fibrillation; Catheter ablation; Mapping; Non–pulmonary vein triggers

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Introduction

Catheter ablation of atrial fibrillation (AF) represents an important therapeutic strategy in patients with symptomatic arrhythmia refractory to antiarrhythmic drug medications, with benefit consistently demonstrated in multiple randomized clinical trials. The main goal of the procedure is the elimination of all the possible AF triggers with the least amount of ablation necessary. Once all the arrhythmia triggers have been eliminated, the incremental value of any additional lesion set including substrate modification approaches,[1] ablation of putative localized sources of AF (eg, rotors or AF drivers) identified by means of computational mapping techniques,[2,3] or autonomic modulation targeting periatrial nerve ganglia[4] remains unclear, as none of these approaches has been convincingly demonstrated superior to trigger ablation alone in adequately designed randomized trials. After the initial description of focal triggers from the pulmonary veins (PVs) as an initiating mechanism for AF in humans, electrical isolation of the PVs has proven to be an effective catheter-based treatment to eliminate most recurrent AF. Reproducible initiation of sustained AF from non-PV foci can be elicited in up to 11% of unselected cohorts referred for AF ablation (Figure 1).[5] However, when induction of any nonsustained atrial arrhythmia (including frequent atrial premature depolarizations) is considered in the definition of non-PV triggers, the prevalence has been reported to be up to 60% (Supplemental Material and Supplemental Table 1).[6] Mapping studies of non-PV foci have demonstrated that triggers are typically clustered in discrete anatomical regions such as the inferior mitral annulus (MA), the posterior LA, the interatrial septum particularly at the fossa ovalis/limbus region, the crista terminalis (CT) and Eustachian ridge, the coronary sinus (CS), and the superior vena cava (SVC).[5] Other sites implicated in AF initiation include the LAA,[6] the left superior vena cava (LSVC),[7] and its remnant—the ligament of Marshall (LOM).[8] All these sites have been shown to contain cardiomyocytes that can exhibit arrhythmogenic activity. This may arise from the combined effects of enhanced automaticity, triggered activity,[9] and localized microreentrant circuits.[10] In an elegant preclinical study, Patterson et al[9] showed the occurrence of afterdepolarization in canine PV tissue due to increased Ca^{2+} transient

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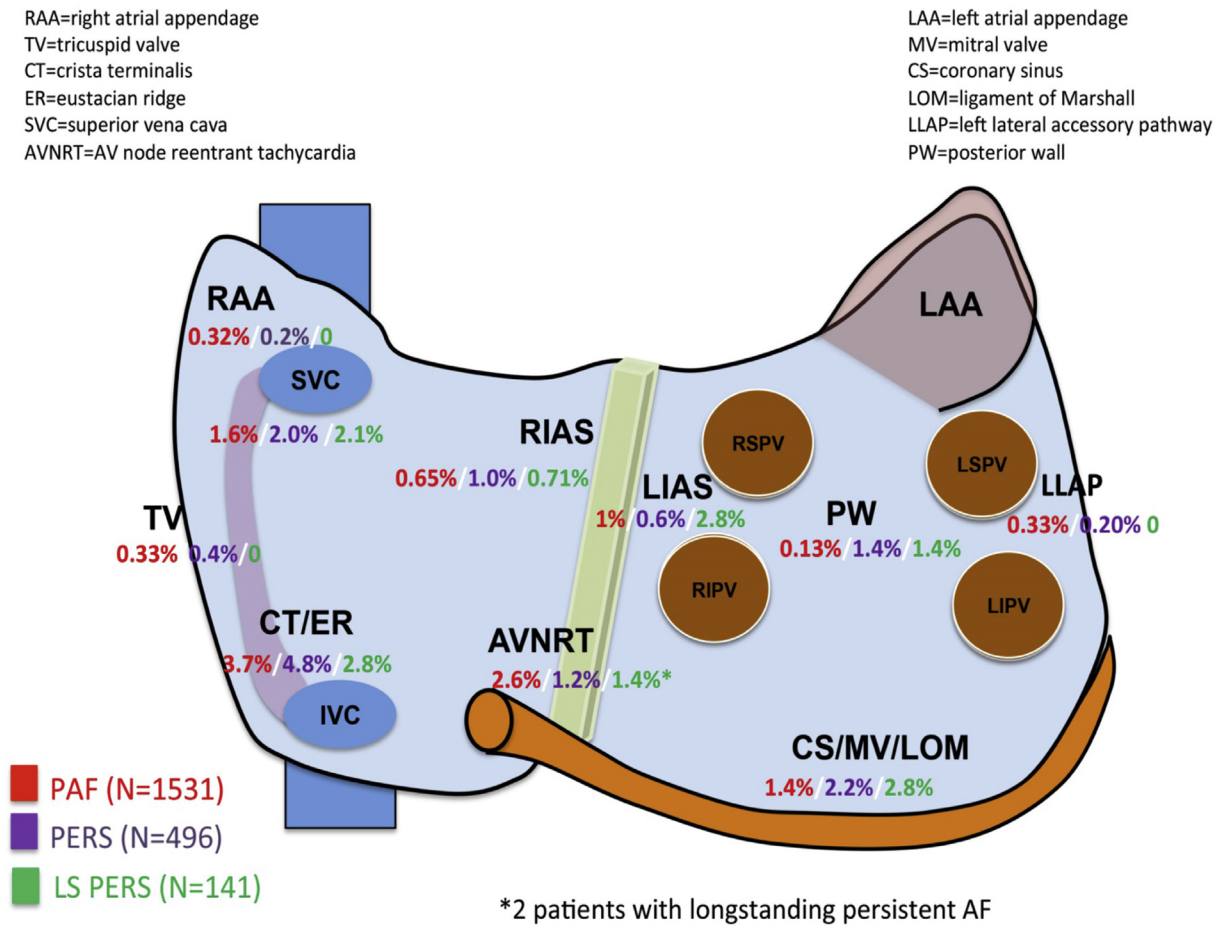

Figure 1 Diagram showing the prevalence and distribution of sustained non–pulmonary vein triggers in patients with paroxysmal (PAF), persistent (PERS), and long-standing persistent (LS PERS) atrial fibrillation (AF) undergoing first time ablation. Adapted from Santangeli et al.[5]

current and increased Na-Ca exchange current. This is in line with prior studies showing enhanced triggered and automatic activity in other thoracic veins, such as the CS.[11] More recently, Hocini et al[10] demonstrated the presence of multiple reentry circuits in the PVs with multipolar electrode recordings. These findings have been replicated with optimal mapping studies.[12]

Finally, a minority of subjects, particularly younger patients with lone paroxysmal AF, may have other mechanisms for AF induction (eg, atrioventricular reentrant tachycardia or atrioventricular nodal reentrant tachycardia).[13] The present article will describe our standardized approach to induce, localize, and ablate non-PV AF triggers using standardized stimulation protocols and integrating electrocardiographic (ECG) criteria with intracardiac recordings from multipolar catheters positioned at specific locations.

Catheter position for trigger mapping

A standardized position of multipolar catheters is also important to correctly identify triggers on the basis of defined intracardiac activation sequences (Figure 2). The decapolar CS catheter should be positioned with the most proximal electrode at the CS ostium; this can be validated either fluoroscopically, visualizing the proximal electrode just anteriorly to the epicardial fat in the posteroseptal space—as assessed in the right anterior oblique view—or with intracardiac echocardiography (ICE). We also couple the decapolar CS catheter with a similar decapolar catheter positioned along the CT and extending up to the SVC. The distal electrode pair of this catheter should be positioned in the SVC, just above the junction with the RA. Ideally, this should be validated with ICE; the distal electrode pair should be positioned at the lower border of the right pulmonary artery as directly visualized by ICE. In this position, the distal electrode pair of the RA catheter will typically record a far-field RA potential followed by a discrete near-field SVC potential. One can anticipate that the recordings from these 2 multipolar catheters allow for rapid and accurate regionalization/localization of triggers from the anatomic sites of origin in their direct proximity.

The information obtained from the CS and CT/SVC catheters are integrated with recordings from the circular mapping catheter and from the ablation catheter positioned in specific locations in order to increase the mapping resolution and validate the site of origin of triggers from atrial structures that are not directly recorded. For instance, a common mapping approach adopted during