Do Not Forget the Mechanisms and Ablation Techniques of Atrial Fibrillation Beyond the Pulmonary Veins

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Editorial Comment

Ever since the pulmonary veins (PVs) were identified as the major trigger of atrial fibrillation (AF), catheter ablation of the PVs has become the mainstream nonpharmacologic therapy for the treatment of AF.[1,2] Currently, the techniques used for ablation of AF include one or a combination of the following techniques: isolation of the PVs (segmental or circumferential approach) with or without demonstration of PV-left atrial conduction block, left atrial linear ablation (mitral isthmus, roof, and posterior wall), ablation of the complex fractionated electrograms, and ablation of the autonomic ganglions.[3-6] In this decade of AF ablation, we have really been performing AF ablation with the "learning while burning" strategy. In this issue of the *Journal of Cardiovascular Electrophysiology*, Pak et al. used the noncontact balloon mapping technique to identify AF triggers and to demonstrate the atrial activation characteristics following the triggers.[7] They found that the persistent AF patients had a higher incidence of non-PV triggers compared with the paroxysmal AF patients, and the preferential conduction following the trigger activation was mostly located along the roof area. These novel results improve our current knowledge of AF mechanisms and provide further insight into the ablation techniques for AF.

AF Beyond the PVs

The non-PV triggers are important for the initiation of AF, and non-PV substrates are important for the initiation and maintenance of AF.[8-12] The reason why we continue to perform provocation testing to find the AF triggers before and after PV isolation is to search for possible non-PV foci.[8-11] The non-PV foci may contribute to 10–20% of the triggers in patients with paroxysmal AF and there may be an even higher incidence in persistent AF.[7-12] To improve the clinical outcome of AF ablation, detailed mapping and ablation of non-PV foci are necessary. Both the right and left atria, including the LA appendage, LA posterior wall, ligament of Marshall, superior vena cava, crista terminalis, coronary sinus, interatrial septum, etc., are possible trigger sites.[8-10] It is important to recognize that in a small proportion of patients, AF can be cured after only ablation of the non-PV triggers, and the right atrial triggers are important for very late (more than 1 year after the first ablation procedure) recurrence of AF.[11,13]

Kumagai et al. performed one elegant study with a basket catheter inside the PV and around the PV-left atrial junction, and demonstrated that the AF drivers were around the PV-left atrial junction.[14] Recently, the Taipei and Bordeaux groups have reported novel findings of AF drivers located away from the PV-left atrial junction, and AF can be cured after ablation of those non-PV drivers.[11,12] Lin et al. used the noncontact balloon mapping technique to identify single or double loop reentry with fibrillatory conduction in the right atrium, and ablation of the critical substrate for the maintenance of the AF could cure 85% of paroxysmal AF that was initiated by a driver (not initiated by an ectopic trigger).[11] Recently, Haissaguerre et al. also identified a trigger and driver of AF outside the PVs after isolation of the four PVs and linear ablation of the mitral isthmus and roof in patients with chronic AF.[12] They also used a flower catheter for extensive mapping, and proposed this type of AF was caused by a reentry mechanism.

The findings from Pak et al. further confirmed the important role of non-PV triggers in the initiation of persistent AF, and the atrial substrate located in the left atrial roof is important for the maintenance of AF.[7] Therefore, identification of atrial arrhythmogenesis beyond the PVs is important for understanding the mechanisms and ablation techniques for the treatment of AF.

Mechanisms Versus Ablation Techniques for Curing AF—Which Is Important?

AF has a quite variable clinical presentation, from lone AF to severe organic heart disease, and from no symptoms to frequent symptomatic attacks. AF is also the most common sustained arrhythmia, and might also be the most complex arrhythmia. We should consider several important issues before we choose the optimal ablation technique. First, have the mechanisms of AF in the animal model been proven in human AF? Second, are the AF mechanisms between paroxysmal and persistent AF similar? Are they similar between young and older patients? Are they similar between idiopathic AF and patients with structural heart disease? Third, does every AF patient have only one AF mechanism or several AF mechanisms? Fourth, does every AF patient need only one ablation technique or a combination of several ablation techniques to cure the AF? It is clear that further investigation into these issues is necessary before we can obtain the right answers. Although some ablation techniques have been established after carrying out several clinical trials in large volume centers, the presentation of a high success rate of AF ablation in the future should not be the golden rule for the

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electrophysiologists worldwide who are devoted to curing AF. How to identify the clinical mechanisms of AF, and how to tailor the ablation techniques for both paroxysmal and persistent AF are the most important challenges we face.

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