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Editorial Comment

Functional Block in the Posterior Left Atrium

Another Piece in the Puzzle of Atrial Fibrillation Initiation*

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Our understanding of the initiation and maintenance of atrial fibrillation (AF) has grown tremendously over the past decade. However, there is much about the initiation of AF that we still do not understand. There is conflicting evidence regarding whether re-entry (1,2), triggered activity (3,4), or enhanced automaticity (5) plays a dominant role in pulmonary vein (PV) ectopy (6). It is also unclear how PV ectopic beats initiate AF. It is evident that atrial premature depolarizations (APDs) originating from the PVs are much more likely to initiate AF than APDs from other sites. Yet, the reason why the posterior left atrium (LA) is so arrhythmogenic remains uncertain. Is it the tight coupling interval that occurs because of short refractory periods in the PV (7)? Is there some inherent anatomic feature of the posterior LA that favors re-entry? Typically, the explanation of AF initiation involves much hand-waving and showing of pictures demonstrating the complex insertion of PV myocardial sleeves into the anisotropic LA myocardium. However, there are little data to support the assertion that this complex fiber arrangement plays a role in AF initiation.

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A welcome contribution comes from Roberts-Thomson et al. (8), who studied wave front propagation in the posterior LA in humans undergoing cardiac surgery. The patients were divided into 4 groups: those with normal left ventricular function undergoing coronary artery bypass graft surgery, those with severe left ventricular dysfunction, those with severe mitral regurgitation, and those with severe aortic stenosis. Before coronary bypass was initiated, high-density epicardial mapping of the posterior LA was performed during sinus rhythm and during atrial pacing from the corners of the plaque at 2 cycle lengths. During pacing from the plaque corners, the investigators observed a line of incomplete or complete functional block, extending from the LA roof inferiorly between the superior and inferior PVs. There was marked anisotropic conduction, with slower conduction perpendicular to the line of functional block than parallel to it. The extent of conduction slowing and block was more pronounced in patients with left ventricular dysfunction or mitral regurgitation compared with patients with structurally normal hearts or aortic stenosis. Electrogram recordings in this area of slow conduction were either fractionated or characterized by double potentials.

These findings are supported by a prior noncontact mapping study performed by Markides et al. (9). Using noncontact mapping of the LA during sinus rhythm, atrial pacing, and PV ectopy, Markides et al. (9) also found a line of functional conduction block in the posterior LA running craniocaudally between the PVs. The APDs originating from the PVs typically developed wavebreak around these areas, leading to the development of AF. The data from Roberts-Thomson et al. (8) confirm these observations using contact mapping and find that pathology that contributes to LA remodeling such as mitral regurgitation and congestive heart failure exacerbates the conduction slowing present along this line.

There are several limitations to the study. The investigators only mapped the posterior LA; therefore, we do not know whether areas of functional block also develop in other regions of the LA that may predispose to re-entry. The investigators used only continuous pacing from the corners of the plaque to assess conduction and did not investigate the patterns of wave front propagation occurring with programmed atrial stimulation from the PVs—a more realistic picture of AF initiation. In addition, pacing was performed at 600 and 400 ms, relatively slow rates compared with the rate of activation during AF. Finally, none of the patients in the study had a history of clinical AF, suggesting that this line of functional block is ubiquitous to the posterior LA rather than a unique feature in patients with AF.

Despite these limitations, these data add an important piece to the puzzle of AF initiation. One can certainly envision a wave front exiting a PV and then encountering a line of partial functional block in the posterior LA that causes wave rotation and breakup into daughter wavelets

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initiating AF. Such a situation is certainly not unique to the posterior LA. Friedman et al. (10) found that a functional line of block in the posterior right atrium is often the posterior boundary of typical RA flutter. Tai et al. (11) have shown that an incomplete line of functional block along the crista terminals can lead to re-entrant atrial tachycardias. It may be the complex interplay between the PVs, the tightly coupled APDs, and the development of areas of functional block in the posterior LA that makes this region so arrhythmogenic. More information is still needed, particularly during the onset of AF in humans with clinical AF.

Another interesting finding is that electrograms recorded along this line of block frequently were noted to be highly fractionated or to contain double potentials. Such complex atrial fractionated electrograms (CAFE), recorded during AF, have been sought out as targets for AF ablation (12). The results from the present study suggest that rather than representing AF drivers, another possible mechanism for such electrogram fractionation is passive wavebreak around areas of functional block. It should be noted, however, that this region between the posterior PVs is not commonly a site of CAFE during AF. We need to learn more about the mechanisms of CAFE and, in particular, which are important for AF maintenance, before empiric ablation of all CAFE electrograms can be recommended during AF ablation.

One might be tempted to use the information in the study by Roberts-Thomson et al. (8) to develop a new or modified ablation strategy, perhaps converting this area of functional block into a line of complete block by ablating from the roof of the LA inferiorly between the PVs. However, it is premature to consider such a strategy for several reasons. First, constructing such a line of complete electrical block is not always easy, and an incomplete line may be more arrhythmogenic and lead to an enhanced propensity for macro–re-entry or focal re-entrant atrial tachycardias (13). Second, this area frequently overlies the esophagus, and extensive ablation with radiofrequency energy in the posterior LA overlying the esophagus should be avoided until we can be assured of the safety of the technique. Finally, simple PV isolation, by eliminating the triggers of AF, may also prevent the initiation of posterior LA re-entry without directly targeting this area.

In summary, Roberts-Thomson et al. (8) have described an area of functional block in the posterior LA with marked anisotropic conduction that is exacerbated in the setting of LA disease, namely congestive heart failure and mitral regurgitation. This information may help to explain why APDs originating from the PVs tend to lead to AF. However, more information, particularly in patients with clinical AF, is needed before we can best determine how to use this new information in our therapeutic armamentarium.

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