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Editorial

Structurally-based electrical predictors of atrial arrhythmias

Albert J. Rogers, Nosheen F. Moosvi, Chad R. Brodt, Sanjiv M. Narayan *

Stanford University, United States of America

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1. Introduction

Mechanisms of atrial tachycardias (AT) can be defined as reentrant (micro- or macro-) or focal (automatic or triggered) once the rhythm is expressed. Indeed, in the absence of typical atrial flutter or inducible arrhythmia, it is difficult to predict the location or substrates for atrial arrhythmias. This is distinct from VT, in which low voltage channels in sinus rhythm and other predictors may help in identification [1]. ATs do provide some hints, however, such as the fact that up to 40% of ATs after prior ablation for atrial fibrillation (AF) are due to microreentry [2], which requires areas of slow conduction which, in turn, often arise at sites of atrial scar [3]. However, not all sites of slow conduction create a nidus for arrhythmia. Conduction slowing may also be dynamic, uncovered by rate acceleration [4] leading to reentry, which may be influenced by the direction of muscle fibers or localized ultrastructural changes. Better understanding of atrial conduction slowing and determinants of atrial arrhythmias are likely key to advancing their treatment and prevention.

2. Functional and structural dynamics

In this issue of the Journal, Honarbakhsh et al. [5] address the important question of whether zones of structural remodeling identified by low voltage may reflect sites of rate dependent conduction slowing and of atrial tachycardia maintenance. High-density bipolar voltage maps were collected to define structural remodeling as atrial scar (very-LVZ, <0.2 mV), border zone (LVZ, 0.2–0.5 mV), and healthy tissue (non-LVZ, >0.5 mV). They performed a thorough pacing protocol to interrogate conduction velocity restitution across the atrium across various activation vectors using a 64-pole basket catheter in sinus rhythm. Atrial tachycardias and mechanisms were determined by conventional mapping, entrainment, and response to ablation. They report that sites of rate-dependent conduction slowing predominantly localize to border zone (74%) and, for the first time, define a signature of conduction restitution for prediction of localized atrial tachycardias (found in 82% of cases).

Honarbakhsh et al. [5] should be congratulated for developing an approach to predict sites of potential atrial tachycardia formation, which could be used in patients who may be non-inducible in the electrophysiology laboratory or in whom multiple tachycardia circuits may exist. Healthier tissue shows conduction slowing only at rapid rates/short cycle lengths (steep restitution), whereas unhealthy tissue exhibits conduction slowing even at slow rates (broad restitution) [4]. This functional assessment contrasts with studies that simply quantify tissue viability via voltage mapping or imaging [6]. The high sensitivity of conduction slowing for sites of microreentrant AT suggests that conduction slowing is mechanistically implicated in the initiation of these circuits.

Our group and others have shown that mechanisms sustaining localized atrial tachycardias and AF, in patients with both arrhythmias, may be shared [7,8]. Of note, 16 out of the 18 patients (89%) in this study had previously undergone AF ablation (wide area circumferential pulmonary vein ablation with or without other ablation). It would have been intriguing to investigate whether any of these sites displayed localized rotational or focal drivers on AF mapping at initial electrophysiology study and ablation for AF.

3. Limitations

Several limitations are noted in review of this well-done investigation. First, the analysis is focused on microreentrant AT, and may not apply to automatic, triggered, or other mechanisms. Second, patients in this study had largely undergone prior AF ablation, which may reduce generalizability of these results to patients without prior ablation or with different prior lesion sets. Prior ablation lesions may themselves produce areas of slow conduction if not transmural or contiguous. Third, it may be expected that a change in conduction velocity would be seen by altering the activation vector (pacing site) relative to atrial fiber orientation, yet was not observed in this study. Fiber orientation is difficult to directly measure in patients, but diffusion tensor MRI

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* Corresponding author at: Stanford University, Suites CJ250F, CV01-09, 780 Welch Road, Stanford, CA 94305, United States of America.
E-mail address: sanjiv1@stanford.edu (S.M. Narayan).

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or other novel techniques may enable further insights into atrial conduction velocity. Fourth, basket catheters have imperfect contact, although the authors have also shown that the current basket is superior to older designs [9]. Finally, the robustness of using low voltage to assess the presence of atrial scar is debated although it has been correlated to late gadolinium enhancement regions on MRI in AF and sinus rhythm. Nevertheless, the absence of low voltage does not indicate absence of border zone, as fibrosis below the resolution of clinical MRI or even voltage mapping has been shown to be critical to reentry in human atrial arrhythmias including AF [10].

4. Conclusions

The authors provide one of the first studies to link structural abnormalities with the mechanism for localized atrial tachycardia via rate-dependent slow conduction. The method could be used in sinus rhythm to predict sites where localized atrial tachycardia sites may arise, and also provide mechanistic insight into atrial tachycardia and perhaps its relationship with AF. In time, this may lead to prospective targeting of these sites and reduce AT occurrence after AF ablation.

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