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Editorial

Propagation velocity at atrial fibrillation sources: Go with the flow

Albert J. Rogers, Neal K. Bhatia, Chad R. Brodt, Sanjiv M. Narayan *

Stanford University, United States of America

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1. Introduction

Although rotation and focal source ablation for AF often produces positive clinical results [1,2], the basic mechanism(s), determinants, and contribution to AF of these sites are not well understood. In particular, their relationship to fibrosis or scar may be important by causing block, reentry, or potentially wavebreak ('fibrillatory conduction') from drivers. In principle, voltage and conduction velocity could be used as functional surrogates of structural abnormalities that may impact our understanding of AF mechanisms and help plan ablation [3].

2. Electrographic flow mapping

In their report in this edition of the *Journal*, Bellman and colleagues focus on sources with net centrifugal action, termed "sources of excitation". They apply a novel electrographic flow (EGF) mapping method, which has been shown to correlate with AF sources by other approaches [4], to evaluate temporal and spatial source stability in relation to propagation velocity on EGF. Temporal stability was measured as the percentage of time the source was present during the one-minute record. Spatial variability was reported as the percentage of the mapped area covered by 80% of the detected sources for 1 min. Velocity was calculated in mean electrode distances per second averaged over 2 s of a time segment. The index procedure was guided by clinically-indicated Focal Impulse and rotor mapping (FIRM), and EGF analysis was completed retrospectively. EGF mapping identified 40/50 (80%) of sites detected by FIRM, in agreement with other recent studies [5]. Intuitively, the authors found that sources with the highest temporal stability also had the lowest spatial variability. Notably, highly stable sites showed higher propagation velocity than those with lesser temporal and spatial stability. Finally, ablation at sources of excitation led to decreased velocity and increased spatial variability.

3. Interpretation and limitations

The investigation approaches important questions regarding mechanisms of AF sources, but whether this approach could solve key clinical questions such as separating primary from secondary sources and improve treatment outcomes are not yet known. While conduction slowing has been reported at AF sources [6], conduction properties have not yet been shown to separate primary from secondary sites. Such a study may be clinically difficult, as it would require enough confidence to test the impact of leaving 'secondary' sites unablated. However, it could be studied in computational models, or potentially in animal models of AF.

Mechanistically, the authors should be congratulated on focusing on propagation velocities, source stability and ablation. Since faster propagation is more likely to break down into fibrillatory conduction [7], their findings are surprising but could potentially be reconciled. First, stable sources near sites of lower fiber anisotropy may show higher propagation velocity. Second, stable sources remote from areas of fibrosis would also show higher propagation velocity, although this is contrary to some prior studies [3,8]. Third, it would be useful to learn if stable sources were more often solitary, since concurrent sources may decrease the domain, stability, and conduction velocities of the index source [9]. Finally, some cases were clearly stable with high velocities and some clearly unstable with low velocities. It is unclear if intermediate cases represent a midpoint between these spectra, or represent another orthogonal variable or phenotype that describes this group.

There are some limitations of the study which must be considered. The range of propagation velocities in this study are lower than expected. Based on maximum and minimum electrode spacing (9-13 mm), the reported propagation velocity range (6–25 electrode distances per second) translate to 0.54 m/s-0.325 m/s which is below those previously reported in surgical mapping [10]. Although the authors measured propagation only along the spline (to control for variable spline spacing), this will not correct for variable propagation vectors of noncoherent wave fronts. Results could also be affected by averaging multidirectional waves in fibrillation (over 2 s), which will reduce magnitude of the resultant vector (measured here as propagation velocity) as the authors acknowledged in prior work [4]. 3D analyses of propagation velocities may add useful data by allowing correction for absolute electrode distances and calculation of propagation vectors in all directions. Finally, it would have been helpful to validate the

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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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importance of sources in this study by examining those where ablation terminated AF.

4. Conclusions

We would like to congratulate Bellman and colleagues on the application of EGF mapping to interrogate AF sources in relation to physiologic properties of the atria. Their studies on the relationship between source stability, propagation velocities and response to ablation should be further enhanced by studies on tissue structure, 3D analyses of wavefront vectors and comparison to other mapping methods. Ultimately, we hope that better understanding of these mechanistic determinants of AF sources will improve identification of primary versus secondary sites, and hence improve ablation guidance and patient outcomes.

Conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

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