EDITORIAL COMMENTARY

From trailer parks to the human atria: The meandering path of rotors

Mohammed Saeed, MD, PhD, Hakan Oral, MD, FHRS

From the Cardiac Arrhythmia Service, Division of Cardiovascular Medicine, University of Michigan, Ann Arbor, Michigan.

I pass with relief from the tossing sea of Cause and Theory to the firm ground of Result and Fact.
—Winston S. Churchill, 1897

The urban myth that tornadoes target vulnerable trailer parks is often whispered whenever a nightly newscast features a story about a trailer park ravaged by a powerful tornado. Interestingly, recent observational data provide a plausible explanation as to why tornadoes seem to be attracted to trailer parks. Tornadoes more frequently touch down near geographic/topographic transition zones (eg, urban structures transitioning to relatively sparsely populated suburbs and rural areas, dense forests transitioning to flat plains). Trailer parks tend to be located in such transition zones, thus feeding the myth about tornadoes and trailer parks.

Complex and chaotic activation patterns during atrial fibrillation (AF) are perhaps the electrophysiological correlate of a storm and tornado in the atria. Similar to tornadoes, rotors have a rotational vortex owing to functional reentry, with a curved wave front and tail meeting each other at a singularity point (phase singularity, PS) where the tissue is not refractory. Spatiotemporal stability of the rotors, preferential sites of prevalence, and the potential causal role they play in the genesis and perpetuation of AF are the subjects of ongoing debate and remain to be further clarified.

In the current issue of Heart Rhythm Journal, Salinet et al report an observational study from 8 patients with persistent AF who underwent de novo catheter ablation. Offline analyses of electrograms recorded with a noncontact multielectrode array were performed before and after pulmonary vein (PV) isolation (PVI) in the phase and frequency domains. Prior studies on phase mapping of AF in humans utilized direct-contact basket catheters with 64 electrodes, along with spatial interpolation techniques, or a noninvasive approach using an array of body-surface electrodes embedded on a vest and a computer system to record unipolar surface potentials via inverse solution–based algorithms. However, far-field electrogram interference, suboptimal tissue–electrode contact, and accuracy of inverse modeling and interpolation solutions can introduce noise and bias into phase analysis. An important source of uncertainty in phase analysis of AF is the far-field ventricular signals, which require QRS subtraction, with inadvertent removal of concomitant atrial activation.

In simplest terms, phase analysis of signals involves identifying the relationship of a signal with the “delayed” version of itself to characterize electrogram periodicity. The computed phase can be seen as the estimated degree of delay (between $-\pi$ and $+\pi$ radians). The authors utilized an in-house algorithm based on the Hilbert transformation. The PSs appeared to meander throughout the left atrium, but had preferential sites near the PVs and left atrial roof. In 8 patients, 2854 ± 737 PSs were observed. The mean duration of PSs was 188 ± 63 ms, which persisted for only 17% ± 6% of the time. In studies that utilized body-surface mapping, the number of driver regions (rotational sources or focal discharges) was ~40 per patient, and the mean duration of rotational activity was ~400 ms. However, during focal impulse and rotor modulation–guided mapping there were far fewer focal sources, 2.8 ± 1.4 per patient, which were remarkably stable and stationary within an area of 2-3 cm$^2$ over multiple epochs. Rotors often meandered when panoramic mapping was employed. It may be that panoramic vs regional mapping, extent of interpolation, and proprietary signal processing and phase transformation algorithms used, along with the patient characteristics and duration of AF, account for the reported discrepancies among these techniques. Left atrial size also affects the density of mapping, and sparse sampling may lead to aliasing.

A novel finding of this study was that PS points typically appeared in pairs, consistent with prior 3-dimensional (3D) computational studies on rotor dynamics. Representation of rotors in 2 dimensions, as has been reported in clinical studies, may be overly simplistic, considering that both atrial cavities as well as the endocardium and epicardium have a complex 3D structure. Although the current studies only explored estimated endocardial or epicardial atrial activation, a complex 3D interplay between subendocardial and...
subepicardial atrial tissue, potentially setting up intramural reentrant local drivers of AF, has been recently proposed.\(^3\) Successful elimination of drivers will require better understanding of the 3D behavior of AF in humans and the subsequent design of ablation strategies tailored for each patient.

In this study, 62% of the PSs co-localized with sites of highest dominant frequency (HDF), consistent with prior experimental studies. Otherwise PSs were observed in close proximity to HDF. Although HDF were stable, PSs meandered. Spectral mapping to identify sites with HDF has been investigated to guide substrate-based ablation in AF. However, the role of DF mapping and ablation to eliminate AF has been equivocal in clinical studies. As in phase mapping, spectral mapping is sensitive to far-field electrogram contamination, particularly near the left atrial appendage and ventricles, and requires high-fidelity, high-density mapping. A primary trigger or driver of AF may not have to be the one with the HDF. A primary driver may initiate secondary drivers/perpetuators with a higher dominant frequency at a site where refractoriness is conducive.\(^4\)

The authors report that the prevalence of rotors decreased by \(\sim 79\%\) in and around the PVs, and by 37% at extra-PV sites after PVI. It would have been helpful to determine the relationship between HDF and PSs after the PVI, as the probability of PSs depends on the HDF, and ablation scars after PVI may have affected PSs. Voltage maps to characterize the preablation and postablation scar burden would have been helpful to determine the relationship between scar location and the spatiotemporal behavior of the PSs.

PSs are primarily driven by the activation rate and the electroanatomical characteristics of the underlying/adjacent tissue, including conduction velocity, refractoriness, anisotropy, and extent of fibrosis. Therefore PSs are more likely to be observed in and around sites of HDF where the electrophysiological characteristics of the surrounding atrial myocardium are suitable for development of PSs, similar to frequent occurrence of tornadoes at sites of topographic changes, as observed around trailer parks.

It is plausible that PSs are passive phenomena observed close to primary drivers/triggers of AF that have a high activation rate facilitating functional refractoriness, and are rather focal. This is consistent with the observations that PSs are brief and are observed only during a fraction of the AF duration even with panoramic mapping, and therefore are unlikely to be able to perpetuate AF by themselves for extended periods of time. It has also been recognized that primary drivers of AF, such as PV tachycardias, are intermittent and that there is a dynamic interplay between a variety of drivers. Therefore, as the driver of AF at a point in time switches from one to the other, PSs are likely to be brief and shift from one site to the other. PSs do not necessarily have to occur around every driver with a high dominant frequency, as long as the atrium can propagate high activation rates without reaching refractoriness.

The conflicting reports on clinical efficacy of mapping and ablating PSs may simply reflect whether an adjacent primary driver was successfully ablated. Ablation of PSs, as described in prior reports, usually involved large areas with prolonged applications of radiofrequency energy, suggesting that PSs by themselves may not have been the primary target. Therefore the question that remains is not so much whether or not PSs exist during AF, but whether PSs are causally responsible for the initiation and perpetuation of AF.

The authors should be commended for providing incremental information on simultaneous phase and spectral mapping of human AF using an endocardial, multielectrode, noncontact mapping catheter. They described the spatiotemporal characteristics of PSs and demonstrated a spatial relationship with HDF. Real-time, simultaneous phase and spectral mapping of both atria in 3D to guide ablation of drivers of AF remain critically needed steps to better understand the mechanisms of AF.

References