Rotors during AF: drivers or bystanders?

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This editorial refers to ‘Epicardial wave mapping in human long-lasting persistent atrial fibrillation: transient rotational circuits, complex wavefronts, and disorganized activity’\textsuperscript{1}, by G. Lee et al., on page 86–97

In the study of Lee and co-workers,\textsuperscript{1} in 18 patients undergoing open heart surgery for mitral or aortic valve disease, high-density mapping of the atria was performed (128 electrodes/6.75 cm\textsuperscript{2}). The mapping plaque was positioned consecutively on the left and right atrial appendages, the posterior wall of the left atrium, and the right superior pulmonary vein (RSPV)–left atrial (LA) junction. In the majority of maps, activation patterns were highly heterogeneous, with multiple unstable activation patterns transitioning from one to another during each recording. The most common patterns seen were multiple wavefronts (56.2 ± 32%) and disorganized activity (24.2 ± 30.3%). Focal activations accounted for 11.3 ± 14.2% of activations and were all short lived (≤ 2 beats), with no site demonstrating sustained focal activity. Two of 36 maps (5.5%) showed a stable activation pattern, consisting of consecutive planar wavefronts propagating in the same direction. Only three transient rotational circuits were observed [two in the posterior left atrium and one on the anterior surface of the left atrial appendage (LAA)]. Two of these circuits lasted for only 5–7 revolutions, whereas the third broke up within < 5 s. On the basis of these observations, the authors conclude that: ‘Human long-lasting persistent AF is characterized by heterogeneous and unstable patterns of activation including multiple wavefronts, transient rotational circuits, and disorganized activity’.

The investigators have to be complimented on their efforts carefully to visualize and dissect the complex activation patterns during long-standing human atrial fibrillation (AF). Studies like these are quite scarce, because attempts to diagnose the underlying substrate of AF are very time-consuming and usually considered less attractive than evaluating the rate of success of various treatment modalities. Although at first sight, the above conclusion of Lee et al. seems rather general and not very specific, the importance of their study lies in what they did not observe. (i) Although focal activations were commonly seen, generally they only occurred as one or two isolated beats and sustained focal activity was not observed. (ii) In the majority of patients (15/18) a rotational circuit could not be detected. (iii) If occasionally maps were suggestive for the presence of a rotational circuit, these were always transient and usually broke up within 5–7 revolutions. Stable rotors were not observed.

We especially welcome this publication because it largely supports the results of our own high-density mapping studies in patients with valvular heart disease and long-standing AF.\textsuperscript{2,3} In our opinion, such support is important to counterbalance the growing belief that persistent AF in humans is due to the presence of a single rotor (Figure 1A) that drives and sustains AF. This growing belief is based on recent low-density computational mapping of the atria using two basket catheters (eight splines of eight electrodes; Constellation, Boston Scientific, Natick, MA, USA).\textsuperscript{5,6} Depending on the size of the basket, the 64 intracavitary electrodes record from a total endocardial surface as large as 30–60 cm\textsuperscript{2}. After filtering and digital processing of the intracavitary signals, a special algorithm (Topera Inc.) was applied that produces a video of the computed activation process of the right and left atria during AF. In 98 of 101 patients with sustained AF, this video-algorithm exhibited an electrical rotor somewhere in the right or left atrium.\textsuperscript{5} Radiofrequency energy delivered at the centre of rotation terminated AF in 31 of 36 patients (86%). Rotor ablation, in addition to conventional ablation, resulted in an almost doubling of the long-term success rate. After a median of 273 days, 82.4% vs. 44.9% of patients were reported to be free from AF.\textsuperscript{5} Another emerging novel technique, aiming to increase the efficacy of radiofrequency ablation of persistent AF, is the reconstruction of the activation of fibrillating atria by body surface mapping.\textsuperscript{7} A computed tomography (CT) scan served to obtain the biaxial geometry and relative positions of the 252 body surface electrodes. A specific signal analysis process, combining filtering, wavelet transform, and phase mapping, was applied to transform the signals from the thorax into a colour movie of atrial activation (CardioInsight Inc., Cleveland, OH, USA). One patient with paroxysmal AF and one patient with persistent AF were mapped a few hours before the ablation procedure was started. In the patient with persistent AF, a drifting rotor was identified in the LA wall that was not stationary for more than two rotations. Ablation at the rotor locations abruptly converted AF into atrial tachycardia after 10 min of radiofrequency application.
VIPs or fibs?

As pointed out above, a clear and important discrepancy exists between the results of direct high-resolution mapping of AF such as in the study of Lee et al.1–3 and computed maps based on low resolution intracavitary or body surface recordings.5–7 Whereas a so-called ‘panoramic’ view exhibits a large rotor that drives the rest of the atria in an irregular way, high-resolution mapping reveals a much higher degree of complexity, with rotors happening only occasionally or not at all. Although this discrepancy might partly be explained by a difference in patient population in the different studies, the main question is which of the two is representing the true nature of long-standing persistent AF in patients with valvular heart disease. Is the high complexity during AF missed by low-density mapping and is the algorithm for computing the activation process fabricating rotors in the case of lack of sufficient data? Or is high-density mapping missing rotors because it is not looking everywhere and is focusing too much on details?

The problem with computational mapping is that there is no way to check the validity of the movie-generating software. The Topera device uses software based on an undisclosed algorithm which makes the methodology difficult to evaluate. In addition, the signals that are used to make the movies are usually not shown and mainly consist of far-field potentials. Because local activation times are not indicated on the signals, it is not possible to trace-back the colours in the movie to the actual moments of activation in the recordings. It is also impossible to know to what extent the movies are the result of extrapolation in the case of paucity of data.

On the other hand, high-density mapping also has its pitfalls. In the case of fractionated electrograms, it is not always easy to decide which components are far-field potentials generated by neighbouring wavefronts, and which component actually represents the true local activation. Since high-resolution epicardial plaques are covering only part of the atrial surface, a rotor could be missed when it is anchored somewhere outside the mapped areas, or, in cases where it is very large, only part of the rotor is propagating under the mapping array. In this respect, it is interesting that in the study of Lee et al., in 2 of 36 cases a stable activation pattern was found in the LAA. During a 10 s recording, the area under the mapping electrode (6.75 cm²) was activated consecutively by a broad planar wavefront propagating in the same direction.1 This leaves the possibility open that the LAA was part of a large mother wave circulating in the left atrium. In other words, whereas computed maps largely count on our instinct that ‘seeing is believing’, high-resolution mapping may prevent us from ‘seeing the wood for the trees’.

The double layer hypothesis

Because at this moment it is not possible to answer the question of whether rotors should be considered as drivers or bystanders in patients with long-standing persistent AF, we would like briefly to discuss an alternative mechanism for perpetuation of AF. The new
concept proposes that the substrate of persistent AF is the result of progressive endo-epicardial dissociation, transforming the atria into an electrical double layer of dissociated waves that constantly ‘feed’ each other. Figure 1 gives a diagrammatic presentation of the double layer hypothesis. In Figure 1A and B, a rotor is compared with an epicardial breakthrough. Whereas a rotor at best can maintain itself, endo-epicardial breakthroughs act as multiplication sites of fibrillation waves. Figure 1C illustrates how endo-epicardial (and epi-endocardial) breakthroughs can perpetuate AF. We calculated that in patients with long-standing AF, endo-epicardial breakthroughs can generate >400 fibrillation waves per second. Also in the present study of Lee et al., > 35% of the fibrillation waves arose from a focal point on the epicardium, distributed over the entire atrial surface. Simultaneous endo-epicardial mapping is required to answer the question of whether long-standing persistent AF is due to a rotor or to endo-epicardial dissociation.

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References