EDITORIAL COMMENT

Mapping of Complex Atrial Tachycardia Circuits by 3-Dimensional Body Surface Mapping

The First Step in the Dawn of a New Era*

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The 12-lead electrocardiogram (ECG) has been the gold standard for diagnosis and localization of atrial arrhythmias (1). Since the initial description of the string galvanometer by Willem Einthoven more than a century ago, there has been little modification to the basic electrode location and information recorded by this 9-electrode body surface system. Meanwhile, the last 2 decades have witnessed the explosive growth of sophisticated intracardiac mapping systems that have given us great insight into the mechanism of atrial tachycardias (AT). These systems allow for 3-dimensional (3D) activation mapping using point-by-point reconstruction of endocardial geometry and activation time. We have learned that AT may be focal (true point source or micro re-entrant) or macro-re-entrant (2). Focal tachycardias emanate from stereotypical regions of either the right or left atrium, whereas macro-re-entrant circuits have a predilection for the tricuspid or mitral valve annulus or may involve gaps in ablation liners or scars, which commonly occur after aggressive ablation for atrial fibrillation (3). Display of these 3D maps requires a stable rhythm that allows acquisition of multiple points sequentially throughout the cardiac chamber(s) of interest.

In this issue of the Journal, Shah et al. (4) describe a novel approach using a 252-electrode body surface mapping system to project epicardial activation times onto cardiac anatomy acquired noninvasively from a cardiac computed tomography scan. The technique is derived from the work of the Ruddy lab to solve the inverse solution using multiple body surface electrocardiographic signals to estimate epicardial activation. Specifically, local unipolar epicardial electrograms are derived from the inverse solution of multiple body surface electrocardiographic potentials, allowing construction of a 3D epicardial activation map from a single beat (5,6).

As outlined by Ashok et al., the results are nothing short of spectacular. For example, the system correctly diagnosed 85% of the re-entrant arrhythmias, and 100% of the centrifugal tachycardias. Four of 5 perimetal AT showed no diagnostic pattern because of 2:1 conduction with superimposition of the P waves and the prior QRS complex or T waves—a situation that may easily be mitigated by use of adenosine. Remarkably, even though the system is only set up to record from the epicardium, a septal focus was correctly inferred from a pattern of epicardial exit observed at the anterior interatrial groove. Furthermore, review of the 12-lead ECG in the figures shows that localization of the tachycardia based on the standard ECG would have been difficult because of extensive previous ablation.

Because we have long experience using 3D endocardial mapping for defining AT circuits, it is important to apply these lessons to this novel 3D recording technique. For example, macro-re-entrant circuits within or around scars will frequently reveal channels showing long-duration low-amplitude signals that may be the critical isthmus of the re-entrant circuit. In the laboratory, one can integrate the recordings of these low-duration potentials with respect to the surrounding mapped points. In addition, we can confirm the impression of the site of a tachycardia isthmus by use of entrainment pacing, which should reveal a pattern of “concealed entrainment” from the true isthmus. If a great deal or most of the circuit is taken up by delayed activation through the circuit, the emerging wave front may appear as a focal breakthrough. It is yet to be proven how well the novel 3D surface mapping technique can properly record and integrate these signals and formulate the correct diagnoses.

Moreover, our experience with intraisthmus re-entry (7) amplifies an additional important lesson. A true focal arrhythmia may have unidirectional block with spread of electrical activity over a “circuit” that may perfectly mimic a re-entrant circuit (electrical activity covering >90% of the tachycardia circuit). The correct diagnoses, however, are made by use of entrainment mapping; to wit, by proving that areas remote from the tachycardia source are out of the circuit.

Can 3D ECG imaging replace these sophisticated techniques for localizing the origin and mechanism of AT? There are certainly many hurdles that remain to be overcome. A prospective blinded comparison is critical to determining how well the system can perform outside of a carefully controlled study from an experienced lab. Endocardial signals in diseased or ablated atrium are often characterized by widely split potentials. Whether these low-amplitude split potentials can be reliably estimated using signals recorded from the body surface remains to be

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determined. For example, we are told that “extensive ablation” was performed in many of the patients, but in Figures 3 and 4 of Shah et al. (4), it is not clear that whether the pulmonary vein antra are electrically isolated or whether there are any barriers present from previous linear ablation. Although a substantial number of patients in the investigators’ series had undergone previous ablation, the identified macro-re-entrant tachycardias, mitral annular flutter, left atrial roof-dependent flutter, and isthmus-dependent right atrial flutter are circuits that activate a large portion of their respective chamber with relatively uniform conduction. Whether the system would be able to localize tachycardias emanating from partially reconnected pulmonary veins is not reported. Many of us experienced the limitations of noncontact endocardial basket mapping, when electrograms estimated from endocavitary signals recorded >4 cm from the cardiac surface were poorly reproduced. Such systems worked well in normal hearts with uniform conduction, but less accurately in diseased and scarred atrium. Although the investigators report outcome in patients with extensive previous ablation, whether such results will hold up to prospective analysis remains to be determined. Moreover, localization of arrhythmias within the atrial septal region may prove to be a limitation of this system. Finally, it is unknown how the system will perform localizing ventricular arrhythmias.

Some might argue that the system is of intellectual curiosity only, as one will always need to perform invasive mapping and entrainment to confirm the tachycardia mechanism prior to ablation. However, there are significant advantages to using such a system if it proves to be as effective as this initial report. Counseling a patient on whether septal access is required may be very helpful prior to the procedure. In addition, often a significant amount of time is taken to construct a detailed activation map of both atria and the thoracic veins. Focusing on the chamber and region of interest would significantly decrease the time needed for mapping and ablation procedures. It would also facilitate mapping arrhythmias that terminate or change during mapping. The ability to perform “single beat” mapping is a clear advance compared with present technology.

This work by Shah et al. (4) summarizes experience from 3 laboratories well experienced in the use of the newer technology. The results reported are truly incredible especially because almost one-half of the AT were obtained from patients who had undergone prior atrial fibrillation ablation. It remains to be seen whether these incredible results can be matched by the rest of the electrophysiologic community. In summary, the investigators are to be congratulated on the launching of an important new endeavor. It is said that the hardest part of any important journey is the first step, and we are indebted to these investigators for this great leap forward.

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