# Expecting the Expected
## Electrocardiographic Identification for Ablation Targets

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An invariable hallmark of an experienced invasive electrophysiologist is his or her ability to thoroughly examine preprocedural data so as to anticipate the likely site of successful ablation. Indeed, teaching rounds for electrophysiology at any institution is likely replete with examples of how careful analysis of the ECG and structural imaging data are used to anticipate not only where to ablate but also the specific anatomic hurdles that may be encountered, facilitating planning to avoid complications. Although advanced imaging has become increasingly useful to understand substrate abnormalities, the mainstay for preprocedural prediction of successful ablation remains the ECG.

**See Article by Namdar et al**

### ECG and the Electrophysiologist: Best Friends Forever

From the outset, the field of electrophysiology has relied heavily on the ECG for clinical and invasive diagnoses. Every student's knowledge base includes ECG localization for accessory pathways and detailed ECG vector analysis to diagnose the origin of monomorphic ventricular tachycardia and atrial tachycardia.[1] In this installment of Teaching Rounds in Cardiac Electrophysiology, Namdar et al[2] add to this legacy instructing us on an advanced multielectrode surface ECG analysis system that allowed a surprisingly high degree of spatial resolution in identifying a coronary venous epicardial ventricular tachycardia focus. The authors elegantly instruct us on the use of the new system, but, more importantly, provide a perspective on analyzing the different kinds of information—ECG, structural integration with the refined electrocardiographic data, and knowledge of the relevant anatomy when mapping epicardially and within the venous system. We also need to understand, however, the limitations inherent in the ECG even despite with sophisticated processing and image integration.

### Arrhythmia Mechanism

Astute analysis of the ECG tells us the pattern or vectors of wave front activation in the heart.

For focal source tachycardia, the site of earliest activation deduced from the ECG is indeed likely to approximate the tissue of origin and, thus, the target for ablation. However, in diseased hearts, ventricular tachycardias are usually caused by reentry, for which the ECG at best defines the so-called exit of the tachycardia circuit, an approximation of the interface between abnormal tissue housing the reentrant circuit and normal myocardium from which the wavefront proceeds to activate the rest of the heart. High-density body surface mapping, like the ECG, is limited in its ability to indicate the exact mechanism of tachycardia and, thus, provides only a limited indication of where to ablate.

### Inverse Solution and Focal Arrhythmia

Assessing cardiac activation from body surface recordings by solving the inverse solution has generally had the limitation imposed by inability to distinguish endocardial from epicardial activation. Namdar et al provide evidence from a system that attempts to address this failing[2]; however, challenges still remain, even with discrete tachycardia sources.

### Cul-de-Sacs of the Heart

Supravalvar myocardial extensions, pulmonary venous sleeves, and myocardium within the coronary venous system may have such small amounts of electrically active tissue that a depolarization of a sufficiently large mass of myocardium to be detected on the body surface occurs from an exit region distant from the true origin of arrhythmia. In the electrophysiology laboratory, small, closely spaced electrodes with accurate knowledge of contact are often required to identify small prepotentials at the tachycardia origin and avoid ablating relatively far away exit sites.

### Conduction System Origin

Automatic tachycardias may arise either within or close to the cardiac conduction system. As a result, there may be multiple myocardial exits at varying distances from the site of origin. As such, the electrocardiographic vector represents a composite of individual vectors from each exit wherein straightforward analysis from the surface ECG's inverse solution may not be possible. For example, even with a large number of surface or intracardiac basket electrodes and sophisticated image integration, it would be difficult to identify the actual pattern of ventricular myocardial activation for a normal sinus beat in a normal heart.

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### Endocavitary Structures

The so-called fourth dimension of the heart (beyond the endocardium, midmyocardium, and epicardium) includes endocavitary structures such as the papillary muscles, prominent trabeculations, false tendons, and the moderator band.[3] Distinguishing between a papillary muscle, a bridge between 2 papillary muscles, and the endocardial surface with or without the included Purkinje network within a papillary muscles is a tough ask for even the most advanced electrocardiographic system using an inverse solution principle.

## Inverse Solution and Reentry

Perhaps the greatest strides made with novel investigational uses of electrocardiographic data to identify arrhythmia origins relate to reentry. Beyond the vector identification of tachycardia exit, analyzing activation in the diastolic period and changes in the primary electrocardiographic vector from the wave front's negotiation of scar and slow-conducting areas may provide remarkable value.

### Inverting the Inverse Solution

A detailed high-density invasive map of the endocardium, epicardium, and endocavitary structures should, when reconstructed, approximate closely a high-density ECG. As a result, a real-time comparison of the reconstructed ECG from invasive data and the raw (nonprocessed) electrocardiographic data should help us identify missing data in our activation and substrate maps and thus guide us to where further points need to be taken or where existence of midmyocardial substrate may be present.

## Registration and Artifacts

In addition to opening our eyes to the potential that exists with advanced ECG-based surface mapping, Namdar et al also point out potential difficulties with registration and far-field artifacts.[2] Cardiac size both as a whole and relative sizes of compressible and pulsatile structures like the veins vary during arrhythmia, with volume status, and when using anesthetic and cardiostimulatory drugs. The far-field artifact that gives us the impression of a separate origin in the outflow tract can be challenging, particularly when the actual arrhythmia may be arising from a smaller focus somewhere near the outflow tract with a distant exit in the outflow tract.[4]

## Anticipating the Future

Great teaching gives us practical, useful tips on performing today's procedures safely and effectively. A unique and valuable aspect of Namdar et al's[2] teaching segment is the linking of a complex, present procedure with familiar historical ECG analysis and preparing us for what may well be the norm in the future—surface mapping and spatially distinct inverse solutions. With parallel strides in noninvasive ablation[5] that necessarily require accurate noninvasive mapping to provide a truly noninvasive solution for arrhythmia management, we may well have the future of cardiac ablation.

## Disclosures

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