Noninvasive Mapping of Ventricular Arrhythmias

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KEYWORDS

• Noninvasive mapping • Ventricular arrhythmias • Ventricual premature beat
• Ventricular tachycardia • Electrical scar • Arrhythmogenic substrate

KEY POINTS

- As validated by electrophysiologic studies, noninvasive electrocardiographic mapping can be undertaken preprocedurally and periprocedurally to accurately diagnose the mechanism of ventricular arrhythmia (focal vs reentrant), identify the chamber of interest, and localize the site of focal arrhythmia.
- Such prior information, which is superior to that obtained from the conventional 12-lead ECG, provides necessary guidance to facilitate catheter ablation in index and previously failed procedures.
- Noninvasive mapping has demonstrated the ability to image arrhythmogenic ventricular substrate having electrophysiologic characteristics of low voltage, altered sinus rhythm activation, electrogram fragmentation, and presence of late potentials in postinfarction myocardium, which correlated well with the anatomic location of scar as depicted by MRI and radionuclide scan.
- Although spatial accuracy of noninvasive imaging has been cited as a possible limitation, the most intriguing aspect of ventricular imaging involves the variable contributions of the endocardium and epicardium to the arrhythmic activity and its inability to directly visualize the interventricular septum.

INTRODUCTION

For more than 100 years, 12-lead electrocardiography (ECG) has been the standard-of-care tool, which involves measuring electrical potentials from limited sites on the body surface to diagnose cardiac disorder, its possible mechanism, and the likely site of origin. Several decades of research has led to the development of a 252-lead ECG-based three-dimensional imaging modality to refine noninvasive diagnosis and improve the management of heart rhythm disorders.[1] This article reviews the clinical potential of this noninvasive mapping technique in identifying the sources of electrical disorders and guiding the catheter ablation of ventricular arrhythmias (premature ventricular beats and ventricular tachycardia [VT]). We also briefly refer to the noninvasive electrical imaging of the arrhythmogenic ventricular substrate based on the electrophysiologic characteristics of postinfarction ventricular myocardium.

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MAPPING TECHNIQUE

The signal acquisition from the patient and subsequent computational methods used in the reconstruction of noninvasive maps using multiple torso electrodes have been previously described.[1] Briefly, a 252-electrode vest is applied to the patient's torso and connected to the noninvasive imaging system and surface potentials are recorded. It is followed by a noncontrast thoracic computed tomography (CT) scan to obtain high-resolution images of the heart and the vest electrodes. The three-dimensional epicardial biventricular geometry is reconstructed from segmental CT images. The relative positions of body surface electrodes can be visualized on the torso geometry. The system reconstructs epicardial potentials, unipolar electrograms, and activation maps from torso potentials during each beat/cycle using mathematical reconstruction algorithms. Details of the mathematical methods have been provided in detail elsewhere.[2–6]

VENTRICULAR PREMATURE COMPLEX AND TACHYCARDIA

Premature ventricular complexes (PVCs) or outflow tract (OT) VTs are commonly encountered in clinical practice.[7] Localizing the origin of these arrhythmias can be challenging and it may be further complicated in cases where they occur infrequently or last transiently (ie, few beats). Noninvasive mapping is particularly useful in diagnosing the mechanism of arrhythmia (focal vs reentrant), identifying the chamber of interest, and localizing the site of focal arrhythmia.[7,8] In animal models, the cardiac activation sequences during single or multipoint pacing and VT are accurately depicted by noninvasive mapping.[9] The origin of focal firing and activation sequence of polymorphic VT, such as torsades de pointes, can also be identified noninvasively.[10] The clinical use of noninvasive mapping in reentrant and focal ventricular arrhythmias including the PVCs has been validated by invasive electrophysiologic studies (EPS) and ablation.[7,11]

Intini and colleagues[12] used the electrocardiomapping (ECM) technique for the first time in a clinical setting to guide diagnosis and therapy for a focal VT in a young athlete. Isolated ventricular ectopic beats of an identical morphology to the sustained tachycardia were mapped before the invasive procedure and their origin was localized to the left ventricular (LV) apical diverticulum. Importantly, the QS wave pattern of the reconstructed electrogram from the site of origin of the ectopies indicated that those beats were emanating from the ventricular epicardium at that site. **Fig. 1** shows a clinical example of such a ventricular beat. In the electrophysiology laboratory, the sustained tachycardia was induced in the presence of isoproterenol. Intracardiac activation mapping during tachycardia demonstrated earliest activation in the apical LV diverticulum. During sinus rhythm, pace map at that site was remarkably similar to the tachycardia morphology in 12/12 ECG leads. Electroanatomic three-dimensional mapping of the LV chamber performed during the ectopy also identified the earliest site of activation at the LV apex in the region of the diverticulum, consistent with a focal origin of the tachycardia. Later, transient

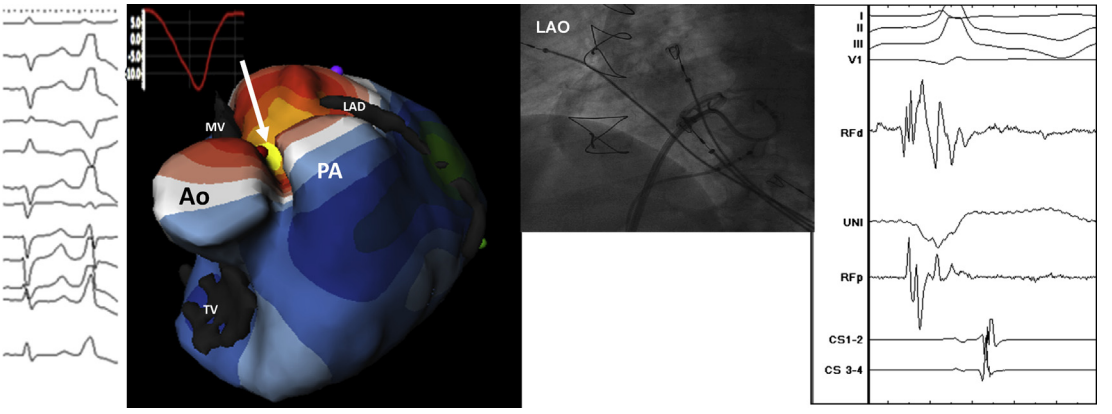

Fig. 1. Biventricular isopotential map (*yellow* denotes the earliest activation) during premature ventricular complex (12-lead ECG). Inserted is an epicardial virtual electrogram (QS morphology) from the earliest site. Also shown are the local intracardiac electrograms at the same site before the start of successful ablation. Ao, aortic root; LAD, left anterior descending artery; LAO, left anterior oblique; MV, mitral valve; PA, pulmonary artery; TV, tricuspid valve.