Electrocardiographic imaging (ECGI), a novel diagnostic modality used for mapping of focal left ventricular tachycardia in a young athlete

Anselma Intini, MD,a Robert N Goldstein, MD,a Ping Jia, PhD,c Charulatha Ramanathan, PhD,c Kyungmoo Ryu, PhD,c Bartolomeo Giannattasio, MD, PhD,a Robert Gilkeson, MD,b Bruce S. Stambler, MD,a Pedro Brugada, MD,d William G. Stevenson, MD,e Yoram Rudy, PhD,c,f Albert L. Waldo, MDa

aFrom the Department of Medicine (Cardiology), bRadiology, and cBiomedical Engineering, Case Western Reserve University/University Hospitals of Cleveland, Cleveland, Ohio, the dCardiovascular Center Aalst/OLV Hospital, Aalst, Belgium, and the eDepartment of Medicine (Cardiology) Brigham and Women’s Hospital, Boston, Massachusetts. fPresent affiliation: Cardiac Bioelectricity and Arrhythmia Center, Washington University in St. Louis, St. Louis, Missouri.

We report the first clinical application of electrocardiographic imaging (ECGI), a new, noninvasive imaging modality for arrhythmias, in an athlete with focal ventricular tachycardia (VT) originating from a left ventricular (LV) diverticulum. A reconstructed map of the epicardial activation sequence during a single premature ventricular complex (PVC) of an identical QRS morphology to the clinical VT, generated from 224-electrode body surface ECGs and a chest CT (ECGI), localized the PVC to the site of the diverticulum. This correlated with subsequent maps obtained using standard techniques. We describe the first case that used ECGI to guide diagnosis and therapy of a clinical tachyarrhythmia.

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As summarized recently,1,2 electrocardiographic imaging (ECGI) is a new, noninvasive imaging modality for arrhythmias, in an athlete with focal ventricular tachycardia (VT) originating from a left ventricular (LV) diverticulum. A reconstructed map of the epicardial activation sequence during a single premature ventricular complex (PVC) of an identical QRS morphology to the clinical VT, generated from 224-electrode body surface ECGs and a chest CT (ECGI), localized the PVC to the site of the diverticulum. This correlated with subsequent maps obtained using standard techniques. We describe the first case that used ECGI to guide diagnosis and therapy of a clinical tachyarrhythmia.

ECGI represents an inverse technique to determine noninvasively and with high resolution the electrical activity of
the heart from electrical data recorded on the body surface together with cardiac CT images. These data are used to reconstruct epicardial potential maps, epicardial electrograms, and maps of epicardial activation and repolarization. This technique has been studied extensively and has been tested and validated experimentally in a torso-tank model, in the normal and abnormal canine heart, and, recently, in humans. In particular, it has successfully provided images of cardiac activation and repolarization during normal excitation, during ventricular pacing, in the presence of conduction disturbances, and during atrial flutter.

ECGI has been recently evaluated through comparison with intraoperative mapping in patients undergoing cardiac surgery and has demonstrated accuracy better than 10 mm in locating focal activity initiated by pacing. But to date ECGI has not been used as a primary clinical tool for arrhythmia diagnosis and management.

Here we describe the first case in which ECGI was used in a patient to assist in the diagnosis and management of a tachyarrhythmia, an unusual ventricular tachycardia (VT). In particular, ECGI was applied to guide the catheter ablation of a focal VT in a young athlete. This case further validates the ECGI technique, but in a real clinical setting.

Case report

A 29-year-old athlete with no significant past medical history was referred to our institution for EP consultation. He had asymptomatic premature ventricular complexes (PVCs) on a routine, otherwise normal ECG and was subsequently shown to develop a spontaneous, sustained, wide QRS complex tachycardia (WCT) during an exercise treadmill test (Figure 1). On presentation to our hospital, the physical exam was normal and the baseline ECG was normal. Further diagnostic testing was performed. An echocardiogram was normal apart from a small left ventricular (LV) apical diverticulum, which was confirmed on magnetic resonance image and chest computed tomography (CT) (Figure 2). Previously, he had a normal standard cardiac catheterization, including normal coronary angiography. Because of suspicion that the WCT was emanating from the ventricular diverticulum and the fact that the patient continued to have PVCs of a morphology similar, if not identical, to the WCT, an ECGI was performed to characterize the ventricular activation sequence of the PVCs. Our intention was to provide objective data to confirm our suspicion.

Methods and results

Before an invasive EP study, isolated PVCs of an identical morphology to the WCT were recorded using a body-surface 224-electrode vest covering the circumference of the thorax, along with a chest CT anatomic scan (the ECGI procedure). The noninvasively reconstructed epicardial electroanatomic ECGI map localized the origin of the PVCs to the LV apex (Figure 3A), precisely to the area of the diverticulum. Importantly, the QS wave pattern of the reconstructed electrogram from the site of origin of the PVCs indicated that those beats were emanating from the ventricular epicardium at that site (Figure 3A).

An invasive EP study was then performed, guided by the noninvasive ECGI images. The tachycardia was sustained but could only be induced in the presence of isoproterenol (1–2 μg/minute). Intracardiac activation mapping during tachycardia demonstrated earliest activation in the apical LV diverticulum. During sinus rhythm, pace mapping at this area produced a QRS complex morphology remarkably similar to the VT morphology in 12/12 ECG leads. Electroanatomic three-dimensional mapping (CARTO) of the LV chamber performed during PVCs (Figure 3B) again identified the earliest site of activation at the LV apex in the region of the diverticulum, consistent with a focal origin of the tachycardia. Analysis of the CARTO maps and transient resolution of the VT during several radiofrequency applica-

![Figure 1](image1.png)  
Twelve-lead ECG of the patient’s clinical tachycardia. Rhythm strip is shown in the **bottom panel** is lead II. Paper speed = 25 mm/second.

![Figure 2](image2.png)  
Cardiac CT scan of a cross section of the LV. Note the small LV apical diverticulum (**arrow**).
therapy of a patient with a tachyarrhythmia, in this case a VT. Noninvasively, and before any invasive EP study, ECGI generated a map of the epicardial activation sequence during a single PVC. This map guided the following invasive EP studies by indicating the origin of the tachycardia. Furthermore, the map generated by the ECGI correlated precisely with the invasive epicardial maps created using standard EP mapping techniques. Thus, this example describes the first case in which ECGI provided important information regarding the origin of a patient’s clinical arrhythmia before embarking on an invasive EP study and attempting therapeutic ablation. It serves also as a harbinger of the possible use of this technique to assist in the management of both simple and complex cardiac arrhythmias.

Discussion

We describe the first case in which ECGI has been used in a real clinical scenario to assist in the diagnosis and

Figure 3  Comparison of three different modalities of mapping in this patient with focal VT. A Reconstructed activation map of a single PVC using noninvasive ECGI. A reconstructed epicardial electrogram from the PVC site of origin (asterisk) is also shown. B Endocardial activation map of the LV chamber using three-dimensional electroanatomic (CARTO) mapping of PVCs—right anterior oblique view. Ablation sites are shown (arrow). C Because of the infrequent PVCs, the epicardial map was obtained during sinus rhythm. All sites with a bipolar electrogram amplitude >1.45 mV are shown as purple. A low-amplitude region is present at the apex, including regions that are electrically unexcitable to pacing at 10 mA (2-ms pulse width). The gray circles are sites where pacing captured the phrenic nerve. Pace mapping in the low-voltage region produced a QRS morphology similar to that of the PVCs (not shown). Respective time scales are shown in panels A and B. Voltage scale is shown in panel C.

References