Assessing Epicardial Substrate Using Intracardiac Echocardiography During VT Ablation

Rupa Bala, MD; Jian-Fang Ren, MD; Mathew D. Hutchinson, MD; Benoit Desjardins, MD; Cory Tschabrunn, CEPS; Edward P. Gerstenfeld, MD; Rajat Deo, MD; Sanjay Dixit, MD; Fermin C. Garcia, MD; Joshua Cooper, MD; David Lin, MD; Michael P. Riley, MD; Wendy S. Tzou, MD; Ralph Verdino, MD; Andrew E. Epstein, MD; David J. Callans, MD; Francis E. Marchlinski, MD

Background—Intracardiac echocardiography (ICE) has played a limited role in defining the substrate for ventricular tachycardia (VT). The purpose of this study was to assess whether ICE could identify abnormal epicardial substrate in patients with nonischemic cardiomyopathy (NICM) and VT.

Methods and Results—We studied 18 patients with NICM and recurrent VT who had abnormal echogenicity identified on ICE imaging. Detailed left ventricular (LV) endocardial and epicardial electroanatomic mapping was performed in all patients. Low-voltage areas (<1.0 mV) in the epicardium were analyzed. ICE imaging in the NICM group was compared to a control group of 30 patients with structurally normal hearts who underwent ICE imaging for other ablation procedures. In 18 patients (age, 53±13 years; 17 men) with NICM (ejection fraction, 37±13%), increased echogenicity was identified in the lateral LV by ICE imaging. LV endocardial electroanatomic mapping identified normal voltage in 9 patients and at least 1 confluent low-voltage area (6.6 cm²; minimum-maximum, 2.1–31.7 cm²) in 9 patients (5 posterolateral LV, 4 perivalvular LV). Detailed epicardial mapping revealed areas of low voltage (39 cm²; minimum-maximum, 18.5–96.3 cm²) and abnormal, fractionated electrograms in all 18 patients (15 posterolateral LV, 3 lateral LV). In all patients, the epicardial scar identified by electroanatomic mapping correlated with the echogenic area identified on ICE imaging. ICE imaging identified no areas of increased echogenicity in the control group.

Conclusions—ICE imaging identified increased echogenicity in the lateral wall of the LV that correlated to abnormal epicardial substrate. These findings suggest that ICE imaging may be useful to identify epicardial substrate in NICM. (Circ Arrhythm Electrophysiol. 2011;4:667-673.)

Key Words: catheter ablation ■ imaging ■ echocardiography ■ tachycardia ventricular ■ epicardial mapping

Clinical Perspective on p 673

We present a unique series of patients with nonischemic cardiomyopathy (NICM) and recurrent VT in whom ICE imaging identified abnormal echogenicity in the lateral wall of the left ventricle (LV). We characterized this substrate by detailed endocardial and epicardial mapping; analysis of electrograms in low-voltage areas; and correlation with other imaging modalities, including MRI and CT angiography.

Methods

Study Population

The study population comprised 18 patients with NICM and recurrent VT who underwent radiofrequency ablation at our institution. These 18 patients had increased echogenicity in the lateral wall of the LV as identified by ICE imaging. We compared the ICE imaging in
the NICM group to a control group of 30 patients with structurally normal hearts who underwent ablation procedures for atrial fibrillation and premature ventricular contractions. These patients underwent detailed ICE imaging as part of their procedures. All patients gave written informed consent in accordance with the institutional guidelines of the University of Pennsylvania Health System.

**Imaging**

Imaging was performed using a Sequoia ultrasound system (Acuson Corporation; Mountain View, CA) with an 8- or 10-F phased-array ultrasound catheter (Acuson AcuNav Catheter). The ultrasound catheter has a changeable ultrasound frequency (5.5–10 MHz) and a 4-way steerable tip (anteroposterior or left-right direction). The imaging catheter was placed in the left-side femoral vein and advanced across the tricuspid annulus into the right ventricle. Two-dimensional slices were obtained with slight clockwise or counterclockwise rotation of the imaging catheter along its long axis.

ICE imaging was analyzed by an echocardiographer who was blinded to the mapping data obtained from the ablation procedure in the study population. The echocardiographer was blinded to the indication for the ablation procedure, patient and clinical characteristics, mapping data, and prior echocardiographic evaluations in the control group.

The ICE survey was analyzed for the presence of increased echogenicity compared with the adjacent structures. If increased echogenicity was present, the gain functions on the Sequoia ultrasound system were systematically decreased to exclude background noise artifacts in the LV cavity to assess this finding. The full thickness of the myocardium was assessed in both real-time and still ICE images. The myocardium was divided into 3 layers and qualitatively assessed for echogenicity. The layer adjacent to the LV cavity was defined as endocardial, the middle layer as midmyocardial, and the distal layer as epicardial. Epicardial echogenicity appears as a linear structure, and we applied this criterion to the definition as well. The following criteria were applied to the finding of increased echogenicity: (1) location by LV segment, (2) LV layer (endocardial, midmyocardial, epicardial), and (3) degree of extension (base only, base to middle, base to apex).

Twelve patients underwent MRI or CT angiography before the ablation procedure. The MRI was performed with a 1.5 Tesla system (Avanto, Siemens Medical Solutions, Malvern, PA). Patients with ICDs were imaged with a 16-channel anterior and 4-channel posterior array, and patients without ICDs were imaged with a similar setup. ECG-gated cardiac CT acquisition was performed on a 64-slice dual source scanner (Somatom Definition DS, Siemens Medical Solutions, Malvern, PA). Patients with (endocardial, midmyocardial, epicardial), and (3) degree of extension (base only, base to middle, base to apex).

Twelve patients underwent MRI or CT angiography before the ablation procedure. The MRI was performed with a 1.5 Tesla system (Avanto, Siemens Medical Solutions, Malvern, PA). Patients with ICDs were imaged with a 16-channel anterior and 4-channel posterior array, and patients without ICDs were imaged with a similar setup. ECG-gated cardiac CT acquisition was performed on a 64-slice dual source scanner (Somatom Definition DS, Siemens Medical Solutions, Malvern, PA) with IV iodinated contrast. MRI and CT angiography studies were analyzed off-line by an experienced radiologist who was blinded to the ICE and mapping data obtained from the ablation procedure.

**Electrophysiology Study and Ablation**

Programmed stimulation was performed from right ventricular and LV endocardial sites. The stimulation protocol included the delivery of up to three extrastimuli at ≥2 ventricular sites at ≥2 drive cycle lengths. Each VT that was induced was analyzed for 12-lead electrocardiographic characteristics suggestive of an epicardial origin. If the induced VT was stable and hemodynamically tolerated, either endocardial or epicardial activation mapping (or both) and entrainment mapping were used. If the induced VT was not hemodynamically stable or reproducibly initiated, detailed characterization of the arrhythmia substrate was performed and all sites demonstrating distinct late potentials were tagged. Pace mapping was used in the area of abnormal substrate to approximate the exit site of the VT circuit or to identify locations manifesting delayed conduction (long stimulus to QRS interval during pace mapping) that matched the clinical VT. A substrate-based ablation strategy targeted the abnormal substrate by incorporating the best pace map sites and areas of abnormal electrograms.

We elected to perform endocardial ablation before epicardial ablation if pace mapping in the abnormal endocardial substrate was similar to the inducible VTs or if we were able to define outer loop sites on the LV endocardium that suggested proximity to the circuit. If these patients remained inducible after endocardial ablation, then epicardial ablation was performed.

**Statistical Analysis**

Continuous variables are expressed as mean±SD or median (minimum to maximum [min-max]). A Fisher exact test was used to assess the significance of abnormal echogenicity identified by ICE imaging in the NICM group and in the control group.

**Results**

**Patient Characteristics**

The study population comprised 18 patients (age, 53±13 years; 17 men) with NICM and recurrent VT in whom ICE imaging identified abnormal echogenicity. All 18 patients presented with recurrent VT documented by implantable cardioverter-defibrillator (ICD)-stored data (n=16) or ECG documentation (n=2) that was refractory to medical therapy with antiarrhythmic medications (n=17) or β-blocker therapy (n=1). Eleven patients underwent unsuccessful LV endocardial mapping and ablation before the initial endocardial and epicardial ablation procedure. Echocardiography performed before the procedure revealed an LV ejection fraction of 37±13%.

The control group comprised 30 patients (age, 55±14 years; 23 men) with structurally normal hearts (LV ejection fraction, 62±6%) who underwent ablations for atrial fibrillation and premature ventricular contractions. These patients underwent detailed ICE imaging as part of their procedures.
Imaging
Increased echogenicity was identified in the lateral wall of the LV by ICE imaging in all 18 patients with NICM and in 0 patients in the control group (P<0.0001). The echogenicity was located in the following segments: posterolateral LV (n=11) posterolateral/lateral LV (n=2), and posterolateral/inferior LV (n=5). The abnormal echogenicity was identified in both the midmyocardium and the epicardium in 10 of the 18 patients (Figures 1 through 4) and only the epicardium in 8 of the 18 patients (Figure 5). In 15 patients, the echogenicity extended from basal to mid LV, and in 3 patients, it extended from the base to near the apex (Table).

Eight patients underwent MRI before the ablation procedure. In 2 patients, there was significant artifact from the ICD generator, which was placed in the left pectoral area, making it difficult to assess for delayed enhancement on the lateral wall. In the remaining 6 patients, there were areas of delayed enhancement on the lateral, anterolateral, and inferolateral walls (Figures 2 and 3, Table). Six patients underwent CT angiography, which revealed thinning in the lateral, posterolateral, and inferolateral walls and hypokinesis in the lateral and inferolateral walls (Table).

Electroanatomic Mapping
Detailed LV endocardial (248 points; min-max, 95–617 points) and epicardial (615 points; min-max, 342–1243 points) electroanatomic mapping was performed in all 18 patients. All 18 demonstrated normal or small LV endocardial voltage abnormalities and more extensive epicardial late potentials on Abl D (arrows). Abl D indicates ablator distal; Abl P, ablator proximal; LA, left atrium; LV, left ventricle; mv, mitral valve; RVA, right ventricular apex.
dial substrate. LV endocardial voltage mapping identified completely normal voltage in 9 patients and small areas of low voltage (<1.5 mV, 6.6 cm²; min-max, 2.1–31.7 cm²) in the posterolateral LV (n=5) and perivalvular LV (n=4). These smaller areas of low voltage on the LV endocardium were opposite to larger areas of epicardial low voltage.

The areas of low voltage (<1.0 mV) on the LV epicardium (39 cm²; min-max, 18.5–96.3 cm²) were localized to the following areas: posterolateral LV (n=12), posterolateral/inferolateral LV (n=3), and lateral LV (n=3). The epicardial scar was homogeneous in 14 patients and heterogeneous in 4; these 4 patients had patchy areas of low voltage on the lateral wall of the LV epicardium interspersed with areas of normal voltage (>1.0 mV) but displayed abnormal, fractionated electrograms with split and late potentials (Figure 4). The low-voltage areas on the epicardium were located at the basal LV in 7 patients and basal to mid LV in 11 patients.

Correlation Among ICE Imaging, Electroanatomic Mapping, and MRI

In all 18 patients, the abnormal echogenicity identified on ICE imaging correlated to the areas of low voltage documented on the LV epicardium. During epicardial mapping of the low-voltage areas, the catheter tip was tagged and located on ICE imaging (Figure 3). In all cases, the catheter tip location over abnormal epicardial substrate was adjacent to the ICE echogenicity.

**Figure 3.** A, Intracardiac echocardiography (ICE) image with increased echogenicity in epicardium (white arrows) and midmyocardium (blue arrows) identified on the posterolateral wall. Pericardium is marked by the red arrow. The ablation catheter in pericardial space also is marked by a red arrow. B, ICE image with ablation catheter (arrows) in pericardial space with catheter tip adjacent to area of midmyocardial/epicardial echogenicity. C, LV epicardial voltage map with area of low voltage on the posterolateral wall. The star marks the location of the catheter tip on the epicardial voltage map that is seen on the ICE image in B. Late potentials are denoted by black tags. D, MRI image (long axis) with extensive areas of DE in epicardium (white arrow) and midmyocardium (blue arrow). Normal endocardium also is noted. Abbreviations as in Figures 1 and 2.

**Figure 4.** A, Intracardiac echocardiographic image with increased echogenicity in epicardium (white arrows) and midmyocardium (blue arrows) identified on the posterolateral wall. Pericardium is marked by the red arrow. B, LV endocardial voltage map with normal voltage. C, LV epicardial voltage map with an area of low voltage on the posterolateral wall. Late potentials are denoted by black tags. The epicardial scar calculation includes the area of late potentials in normal voltage (>1 mV) and the area of low voltage more distally. Electrograms of representative late potentials are shown. Abbreviations as in Figures 1 and 2.
As stated previously, in 10 of the 18 patients, there was increased echogenicity in both the midmyocardium and the epicardium. Five of these patients underwent MRI, which revealed areas of delayed enhancement in the lateral wall that extended from the myocardium to the epicardium. In 3 of these patients, we were able to identify low-voltage areas on the epicardium, but they were not as extensive as the MRI findings (Figure 2).

**Electrophysiology Study and Ablation**

A median of 3.5 VTs (min-max, 1–6 VTs) were induced by programmed stimulation. Seven of 18 patients underwent initial LV endocardial ablation targeting abnormal substrate guided by pace mapping (n=5) or targeting outer loop sites based on entrainment mapping (n=2) before proceeding to LV epicardial mapping. A substrate-based ablation strategy on the LV epicardium was performed in 17 patients given the hemodynamic instability of the targeted VTs. Ablation was performed by targeting the abnormal epicardial substrate and incorporating sites with the best pace maps and areas of abnormal electrograms. In total, 12 of the 18 patients underwent combined endocardial and epicardial ablation.

**Table. ICE (Echogenicity), Electroanatomic Mapping, MRI, and CTA Data**

<table>
<thead>
<tr>
<th>Pt</th>
<th>ICE Location</th>
<th>ICE Level</th>
<th>ICE Extension</th>
<th>LV Endo VA/Area, cm²</th>
<th>LV Epi VA/Area, cm²</th>
<th>MRI (DE)</th>
<th>CTA</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>PL</td>
<td>M-myo, Epi</td>
<td>Base to mid</td>
<td>Normal</td>
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<td>2</td>
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<td>Epi</td>
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<td>Lat, AL</td>
<td>Lat hypokinesis</td>
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<tr>
<td>3</td>
<td>PL</td>
<td>Epi</td>
<td>Base to mid</td>
<td>Normal</td>
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<tr>
<td>4</td>
<td>PL</td>
<td>Epi</td>
<td>Base to near apex</td>
<td>PL/19.7</td>
<td>PL/96.3</td>
<td>Lat thinning</td>
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<tr>
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<td>Epi</td>
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<tr>
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<td>PL/49.1</td>
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<td>Base to mid</td>
<td>Normal</td>
<td>PL, IL/30</td>
<td>IL</td>
<td></td>
</tr>
</tbody>
</table>

AL, anterolateral; CTA, CT angiography; DE, delayed enhancement; Endo, endocardium; Epi, epicardium; ICE, intracardiac echocardiography; IL, inferolateral; Inf, inferior; Lat, lateral; M-myo, Midmyocardium; PL, posterolateral; VA, voltage abnormality.
Discussion
In this series of patients with NICM undergoing VT ablation, we demonstrated that ICE imaging was useful in identifying regions of abnormal epicardial substrate. In all 18 patients, ICE imaging identified abnormal echogenicity in the lateral wall of the LV that correlated anatomicall to electroanatomic findings of low voltage and abnormal electrograms manifested by split and late potentials. The majority of patients underwent MRI or CT imaging that confirmed abnormal substrate in the lateral wall by either delayed enhancement or thinning in the lateral wall, correlating to the ICE-defined abnormality.

The findings suggest that ICE imaging plays an important role in identifying abnormal substrate and, thereby, facilitating VT ablation. Increased echogenicity on ICE imaging coupled with normal or small areas of low voltage on LV endocardial electroanatomic mapping suggest the need for detailed epicardial mapping/ablation. Electroanatomic mapping currently is the gold standard for identification of abnormal epicardial substrate, and the strength of this study is that it validates ICE imaging in identifying abnormal substrate. MRI imaging in patients with ICDs is not uniformly performed, and there remains the issue of ICD generator artifact when interpreting MRIs in these patients. In our study, 2 patients had ICD generator artifact on the lateral wall, limiting interpretation.

ICE imaging identified both midmyocardial and epicardial echogenicity in 10 of the 18 patients, and these findings were confirmed in 5 of these patients with MRI, which revealed extensive areas of delayed enhancement. In 3 of these patients, we were able to identify low-voltage areas on the epicardium, but they were not as extensive as the midmyocardial MRI findings. Thus, one must hypothesize that these patients had larger areas of midmyocardial scar that were identified by ICE imaging and MRI, with a smaller epicardial component that was confirmed with electroanatomic mapping (Figure 2). This finding documents the limitation of bipolar mapping to identify midmyocardial scar and the need to explore other modalities, such as unipolar substrate mapping, in conjunction with ICE imaging and MRI to identify abnormal, myocardial substrate.15

In most cases, the epicardial low-voltage areas were homogenous with abnormal, fractionated electrograms. In 4 patients, there was heterogeneous areas of low voltage on the lateral wall of the LV epicardium interspersed with areas of normal voltage (>1.0 mV), but they were displaying abnormal, fractionated electrograms with split and late potentials. This patchy epicardial scar may be related to undersampling, a cutoff value of 1 mV for identifying low voltage on the epicardium, or sampling of midmyocardial substrate because 2 of these patients had midmyocardial and epicardial substrate identified by ICE imaging (Figure 4).

Limitations
In this study, MRI was performed in 8 of 18 patients. In 2 of these patients, there was significant artifact from the ICD generator in the left pectoral area, and it was difficult to assess for delayed enhancement on the lateral wall. Four of the 8 patients did not have ICDs at the time of MRI; thus, it may be difficult to acquire quality MRI images because of artifact, which may limit our ability to judge the extent of abnormal substrate in the lateral wall.

This study included 18 patients with increased echogenicity in the lateral wall defined on the initial ICE survey. Other regions in the LV were not studied, and thus, our findings are limited to a site-specific region. The sensitivity and specificity of this finding in NICM are unknown. Further studies are required to assess this finding as well as to identify abnormal substrate in other regions of the LV.

The ICE imaging was interpreted by an echocardiographer with extensive experience in ICE acquisition and interpretation. Operators less experienced with ICE interpretation may encounter difficulty in analysis.

Conclusions
We present a unique series of patients with NICM and recurrent VT who demonstrated predominantly normal LV endocardial voltage and abnormal epicardial substrate identified by ICE imaging. ICE imaging identified abnormal echogenicity in the lateral wall of the LV that correlated to epicardial low-voltage areas identified by electroanatomic mapping. This epicardial scar had abnormal electrograms with split and late potentials. In a subset of patients, MRI confirmed abnormal substrate in the lateral wall, correlating to the ICE-defined abnormality.

Disclosures
Drs Bala, Hutchinson, Gerstenfeld, Dixit, Garcia, Cooper, Lin, Riley, Callans, and Marchlinski have all participated in clinical research protocols on endocardial VT ablation in patients with coronary artery disease sponsored by Biosense Webster, but their work is unrelated to the content of this article. They also have received honoraria for educational lectures. Drs Hutchinson and Callans have received honoraria from Acuson Corporation for educational lectures.

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**CLINICAL PERSPECTIVE**

Intracardiac echocardiography (ICE) is routinely used during catheter ablation for atrial fibrillation, but has not been routinely used during catheter ablation for ventricular tachycardia. There is limited information on the ability of ICE to assess for abnormal substrate during ventricular tachycardia ablation. We present a unique series of patients with nonischemic cardiomyopathy and recurrent ventricular tachycardia in whom ICE imaging identified abnormal echogenicity in the lateral wall of the left ventricle. We characterized this substrate by detailed endocardial and epicardial mapping; analysis of electrograms in low-voltage areas; and correlation with other imaging modalities, including MRI and CT angiography. We demonstrated that ICE imaging was useful in identifying regions of abnormal midmyocardial and epicardial substrate. In all 18 patients, ICE imaging identified abnormal echogenicity in the lateral wall of the left ventricle that correlated anatomically with electroanatomic findings of low voltage and abnormal electrograms manifested by split and late potentials. In the majority of patients, MRI or CT imaging confirmed abnormal substrate in the lateral wall. The findings suggest that ICE imaging plays an important role in identifying abnormal substrate and, thereby, in facilitating ventricular tachycardia ablation. Increased echogenicity on ICE imaging coupled with normal or small areas of low voltage on left ventricular endocardial electroanatomic mapping suggest the need for detailed epicardial mapping and ablation.
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