Repeat Left Atrial Catheter Ablation
Cardiac Magnetic Resonance Prediction of Endocardial Voltage and Gaps in Ablation Lesion Sets

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Background—Studies have reported an inverse relationship between late gadolinium enhancement (LGE) cardiac magnetic resonance (CMR) signal intensity and left atrial (LA) endocardial voltage after LA ablation. However, there is controversy regarding the reproducibility of atrial LGE CMR and its ability to identify gaps in ablation lesions. Using systematic and objective techniques, this study examines the correlation between atrial CMR and endocardial voltage.

Methods and Results—Twenty patients who had previous ablation for atrial fibrillation and represented with paroxysmal atrial fibrillation or atrial tachycardia underwent preablation LGE CMR. During the ablation procedure, high-density point-by-point Carto voltage maps were acquired. Three-dimensional CMR reconstructions were registered with the Carto anatomies to allow comparison of voltage and LGE signal intensity. Signal intensities around the left and right pulmonary vein antra and along the LA roof and mitral lines on the CMR-segmented LA shells were extracted to examine differences between electrically isolated and reconnected lesions. There were a total of 6767 data points across the 20 patients. Only 119 (1.8%) of the points were ≤0.05 mV. There was only a weak inverse correlation between either unipolar (r=−0.18) or bipolar (r=−0.17) voltage and LGE CMR signal intensities with low voltage occurring across a large range of signal intensities. Signal intensities were not statistically different for electrically isolated and reconnected lesions.

Conclusions—This study demonstrates that there is only a weak point-by-point relationship between LGE CMR and endocardial voltage in patients undergoing repeat LA ablation. Using an objective method of assessing gaps in ablation lesions, LGE CMR is unable to reliably predict sites of electrical conduction.

Key Words: atrial fibrillation | atrium | cardiac magnetic resonance imaging | catheter ablation | voltage

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WHAT IS KNOWN

- Previous studies in patients undergoing repeat left atrial catheter ablation have suggested a qualitative and quantitative relationship between areas of high late gadolinium enhancement cardiac magnetic resonance signal intensity and areas of low endocardial voltage.
- There is controversy regarding the reproducibility and diagnostic ability of atrial late gadolinium enhancement cardiac magnetic resonance to reliably identify ablation lesions and to predict electrical reconnection by identifying gaps in ablation lesions.

WHAT THE STUDY ADDS

- This study demonstrates that there is only a weak point-by-point relationship between atrial late gadolinium enhancement cardiac magnetic resonance signal intensity and endocardial voltage and that low endocardial voltage can occur at sites of low and high cardiac magnetic resonance signal intensity.
- Using an objective method of assessing gaps in ablation lesions, late gadolinium enhancement cardiac magnetic resonance is unable to reliably predict sites of electrical conduction.

Given these conflicting findings, this study aimed to use systematic and objective techniques to investigate atrial LGE CMR to determine its relationship with atrial voltage recordings and also to investigate its potential as a predictor of electrical reconnection in patients undergoing repeat catheter ablation.

Methods

Patients

Twenty patients requiring a clinically indicated repeat left atrial (LA) catheter ablation werestudied. All had previously undergone LA catheter ablation for PAF, PsAF, or AT. The index procedure for patients with PAF was a wide-area PV encirclement alone. For patients with PsAF, the index procedure was a stepwise ablation approach incorporating wide-area PV encirclement, electrogram-guided ablation and linear lesions at the LA roof, mitral isthmus and cavo-tricuspid isthmus as deemed appropriate. Only those patients with recurrence of either PAF (and therefore in sinus rhythm) or AT were included, so as to optimize CMR image quality. Patients presenting with AF on the day of the repeat procedure or with any contraindication to CMR were excluded from the study. The study was approved by the local research ethics committee, and all subjects gave written informed consent to participate.

CMR Acquisition

CMR was performed 2 to 3 weeks before repeat catheter ablation on a 1.5-Tesla MR system (Achieva; Philips Medical Systems, Best, The Netherlands) with a 32-channel cardiac phased array coil. First, survey and sensitivity encoding reference scans were obtained followed by a 2D multicardiac phase cine scan acquired in a 4-chamber orientation. From this scan, the trigger delay was determined for all subsequent scans to minimize artefacts from atrial wall motion.

This was then followed by a three-dimensional (3D) balanced steady state free precession acquisition in a sagittal orientation with whole-heart coverage and 2-mm isotropic resolution, reconstructed to 1.3-mm resolution, with T2 preparation, using the respiratory navigator to minimize motion artefacts. The length of the acquisition window was set to maximum of 150 ms to minimize motion artefacts.

Twenty minutes after the administration of 0.2 mL/kg Gadovist (Bayer HealthCare Pharmaceuticals, Berlin, Germany), 3D LGE imaging was performed with a respiratory-navigated, ECG-triggered inversion recovery turbo field echo acquisition. The spatial resolution was 1.3×1.3×4 mm³, which was reconstructed to 0.6×0.6×2 mm³, using an echo time/repetition time of 3.0/6.2 ms and a flip angle of 25°. The inversion time was determined using a preceding Look-Locker sequence, to achieve optimal suppression of ventricular myocardium. The scan was acquired in an axial orientation, typically with 30 to 40 slices to achieve complete coverage of the LA. The length of the acquisition window was set to maximum of 150 ms to minimize motion artefacts. Examples of LGE images are shown in Figure 1.

Ablation Procedure

Class I and III antiarrhythmic medications, with the exception of amiodarone, were discontinued ≥5 half-lives before the procedure. Patients taking warfarin discontinued this 5 days before the procedure and were administered with subcutaneous low-molecular weight heparin for 3 days before ablation. In 2 patients, the procedure was performed on uninterrupted warfarin. In patients with an original diagnosis of PsAF or a CHADS2Vasc score of ≥2, transesophageal echocardiography was performed before the procedure to exclude intracardiac thrombus.

At the start of the ablation procedure, a 6F decapolar catheter was placed in the coronary sinus to provide a reference for electroanatomical mapping. For procedures performed under general anesthesia, transesophageal echocardiography was used to guide transseptal puncture. For those performed under conscious sedation, fluoroscopy alone was used to guide a single transseptal puncture through which 2 Schwartz right 0 sheaths were passed to the LA. Immediately after needle access to the LA, intravenous heparin was administered to achieve an activated clotting time of ≥300 seconds. The Schwartz right 0 sheaths were used to place a 3.5-mm tip irrigated ablation catheter (Thermocool; Biosense Webster, Diamond Bar, CA) and a 20-pole circumferential mapping catheter (Lasso; Biosense Webster) in the LA.

A 3D geometry of the LA was created using the Carto 3 electroanatomical mapping system (Biosense Webster). A high-density preablation voltage map was constructed using points acquired with

Figure 1. Transverse slices from 3-dimensional left atrial late gadolinium enhancement cardiac magnetic resonance scans from 5 different patients.
the ablation catheter to give coverage of the entire LA. The voltage map was constructed in either sinus rhythm or AT, according to the presenting arrhythmia.

The circumferential mapping catheter was positioned and manipulated at each PV ostium to assess for PV–LA reconnection. If electrical silence was documented at the ostium, PV isolation was confirmed. If any potential was recorded on the circumferential catheter, pacing techniques were used to confirm its origin as either from the PV or the atrium. In sinus rhythm, conduction across the LA roof and left lower PV-mitra! line was also assessed using previously validated pacing techniques.20,21

For patients with an original diagnosis of PAF and in sinus rhythm, the procedural end point was the reisolation of the PVs, achieved by targeting the site(s) of PV reconnection(s) and verification of conduction block at the sites of any previous linear lesion. For patients presenting with AT, the procedural end point was the termination of tachycardia, reisolation of incompletely isolated PVs, and achievement of conduction block across linear lesions.

CMR Image Processing

CMR images were not analyzed before repeat ablation, and the procedure was performed without any of the CMR anatomic or LGE information available to the operator.

CMR images were processed according to previously described methods.22 In summary, an automatic 3D segmentation of the LA was created from the balanced steady state free precession whole-heart acquisition. The LGE acquisition was registered to the 3D balanced steady state free precession acquisition and projected on to the 3D LA shell using a maximum intensity projection technique, whereby the maximum signal intensity within 3 mm of the 3D surface was selected. Signal intensities were then displayed on the 3D LA shell as a number of standard deviations from the mean signal intensity of the LA blood pool to avoid the need for thresholding.

Comparison of CMR Signal Intensity and Endocardial Voltage

Voltage maps were exported from Carto 3 and imported into software custom-written with Matlab (The Mathworks, Matick), so that the voltage maps and data collection points could be reconstructed for off-line analysis. The CMR-segmented LA shells were registered to the Carto 3 LA shells using a 2-step process: (1) landmark registration (using the PV ostia and LA appendage as landmarks) and (2) iterative closest point registration. This allowed the LGE CMR signal intensities to be projected on to the Carto 3 LA anatomy using point correspondence between the 2 LA shells (Figure 2). Thereafter, for each Carto 3 sampling point, the unipolar and bipolar endocardial voltage and LGE CMR signal intensity were extracted to allow a point-by-point comparison. To allow for potential registration errors, the same analysis was repeated twice using the mean LGE CMR signal intensity within a radius of 2.5 mm and 5 mm from each voltage point.

CMR Analysis of Gaps in Ablation Lesions

The presence or absence of gaps in ablation lesions on 3D CMR reconstructions depends on the signal intensity threshold chosen to denote scar. A lower threshold will result in more scar, whereas a higher threshold will result in more gaps. To avoid selecting an arbitrary threshold, it was necessary to develop an objective user-independent method for detecting gaps.

Using custom-written software, a path was traced around the left and right PV antra, the LA roof line, and the mitral line on the CMR-segmented LA shell (Figure 3). Where a PV encirclement, roof line or mitral line were clearly visible on the LGE CMR reconstruction, the path was traced through the center of the enhancement. For each vertex along these paths, the mean signal intensity within a radius of 10 mm was extracted and assigned to the vertex. Minimum (LGEmin) and mean (LGEmean) signal intensities were then extracted for each path. The LGErecording on these paths represents the weakest link in the chain and the most likely site for reconnection, on the basis of LGE information. The LGE of the path gives an assessment of the total scar burden along the path. The values of LGEmin and LGEmean were compared with the electrical integrity of the lesion sets (assessed at the time of repeat catheter ablation).

In RVs with a single discrete electrical gap along the estimated trajectory of the previous wide-area encirclement, the site of PV reisolation was annotated on the Carto 3 LA geometry. The signal intensity at the corresponding anatomic location on the CMR-segmented LA shell was extracted and compared with LGEmax.

Statistical Analysis

Statistical analysis was performed using Prism 6 (GraphPad Software Inc, La Jolla, CA). Continuous variables are expressed throughout as mean±SD, except where stated otherwise, and were compared using Student t test for paired and unpaired data. Correlation coefficients, r, were determined using Pearson product-moment analysis and a weighted mean±SD calculated using Fisher z transformation. A significance level of P<0.05 was considered statistically significant.

Results

Patients

Twenty patients (17 men; 3 women; mean age, 59±7 years) completed the study. Patient characteristics are shown in
The median number of previous ablation procedures was 2 (range, 1–5). The original diagnosis was PAF in 11 patients, PsAF in 8 patients, and AT in 1 patient. The arrhythmia of recurrence was PAF in 10 patients and AT in 10 patients. Of the 10 ATs, 2 patients had perimitral AT, 3 had localized re-entrant AT, 2 had typical cavotricuspid isthmus–dependent flutter, 1 had a right PV focal AT, 1 had roof-dependent AT, and 1 patient had multiple ATs.

**Table 1.** Patient Demographics

<p>| | |</p>
<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Age, y</td>
<td>59±7</td>
</tr>
<tr>
<td>Sex, n</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>17 (85%)</td>
</tr>
<tr>
<td>Female</td>
<td>3 (15%)</td>
</tr>
<tr>
<td>Original diagnosis, n</td>
<td></td>
</tr>
<tr>
<td>PAF</td>
<td>11 (55%)</td>
</tr>
<tr>
<td>PsAF</td>
<td>8 (40%)</td>
</tr>
<tr>
<td>AT</td>
<td>1 (5%)</td>
</tr>
<tr>
<td>Recurrent arrhythmia, n</td>
<td></td>
</tr>
<tr>
<td>PAF</td>
<td>10 (50%)</td>
</tr>
<tr>
<td>AT</td>
<td>10 (50%)</td>
</tr>
<tr>
<td>Coronary artery disease, n</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Smoking, n</td>
<td>1 (5%)</td>
</tr>
<tr>
<td>Valve disease, n</td>
<td>2 (10%)</td>
</tr>
<tr>
<td>Hypertension, n</td>
<td>2 (10%)</td>
</tr>
<tr>
<td>Diabetes mellitus, n</td>
<td>2 (10%)</td>
</tr>
<tr>
<td>CVA/TIA, n</td>
<td>2 (10%)</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>53±3</td>
</tr>
<tr>
<td>LVEF &lt;40%, n</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>CHADS2,Vasc, n</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>14 (70%)</td>
</tr>
<tr>
<td>1</td>
<td>2 (10%)</td>
</tr>
<tr>
<td>2</td>
<td>3 (15%)</td>
</tr>
<tr>
<td>3</td>
<td>1 (5%)</td>
</tr>
<tr>
<td>&gt;3</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Number of previous LA ablations, n</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>9 (45%)</td>
</tr>
<tr>
<td>2</td>
<td>6 (30%)</td>
</tr>
<tr>
<td>3</td>
<td>3 (15%)</td>
</tr>
<tr>
<td>&gt;3</td>
<td>2 (10%)</td>
</tr>
</tbody>
</table>

AT indicates atrial tachycardia; CVA, cerebrovascular accident; LA, left atrial; LVEF, left ventricular ejection fraction; PAF, paroxysmal atrial fibrillation; PsAF, persistent atrial fibrillation; and TIA, transient ischemic attack.

**Comparison of CMR Signal Intensity and Endocardial Voltage**

There were a total of 6767 voltage measurements in the 20 patients (mean number of points per patient was 338±210). One-hundred nineteen (1.8%) of the points were ≤0.05 mV (bipolar), whereas 1819 (27%) of the points were ≤0.3 mV (Figure 4). The mean LA unipolar and bipolar voltages were significantly lower in patients with AT at recurrence than in those with PAF (unipolar: 1.59±0.37 versus 2.14±0.57 mV; P=0.02 and bipolar: 0.74±0.31 versus 1.48±0.44 mV; P=0.0004).

To demonstrate a qualitative comparison between the 2 datasets, voltage maps thresholded to ≤0.05 mV and ≤0.3 mV are shown in Figure 5, alongside corresponding nonthresholded 3D CMR reconstructions.
Examples of coregistered voltage maps and CMR signal intensity maps are shown in Figure 2. For each patient, the registration error was calculated as the mean distance from each vertex on the Carto shell to the nearest vertex on the registered CMR shell. The overall error for all patients was 0.5±0.2 mm.

For each patient, the correlation coefficients comparing unipolar or bipolar voltage with LGE CMR signal intensities are shown in Table 2. The mean correlation coefficients for unipolar and bipolar voltages were −0.18 (95% confidence interval [CI], −0.24 to −0.12) and −0.17 (95% CI, −0.24 to −0.09), respectively. When signal intensities within a radius of 2.5 mm of each voltage point were included, the mean correlation coefficients were −0.19 (95% CI, −0.26 to −0.12) and −0.18 (95% CI, −0.26 to −0.10), respectively. When signal intensities within a radius of 5 mm of each voltage point were included, the mean

Figure 5. For all 20 patients, endocardial voltage maps are shown at 2 different thresholds (where blue indicates values greater than the threshold and red indicates values less than the threshold). Corresponding 3-dimensional cardiac magnetic resonance (CMR) reconstructions are shown without thresholding each with their own individual color scale.
correlation coefficients were \(-0.21\) (95% CI, \(-0.28\) to \(-0.13\)) and \(-0.19\) (95% CI, \(-0.27\) to \(-0.11\)) respectively.

**CMR Analysis of Gaps in Ablation Lesions**

All patients had previously undergone PV isolation. Electrophysiological assessment demonstrated right upper PV reconnection in 8 patients, right lower PV reconnection in 12 patients, left upper PV reconnection in 10 patients, and left lower PV reconnection in 10 patients. When assessed as pairs, there was right PV reconnection in 13 patients and left PV reconnection in 12 patients. Six patients had previously undergone LA roof line ablation, but only 4 of these remained blocked. Five patients had previously undergone mitral line ablation, but only 3 of these remained blocked.

The values for LGE\(_{min}\) and LGE\(_{mean}\) for electrically reconnected/unblocked and isolated/blocked lesions are summarized in Table 3. There were no statistically significant differences. There were a total of 13 PVs in 9 of the 20 patients with single discrete electrical gaps. The LGE CMR signal intensity at the site of PV reisolation was equal to LGE\(_{min}\) in only 1 of 13 PVs and significantly greater than LGE\(_{min}\) overall (3.08\(\pm\)2.63 versus 0.76\(\pm\)1.12; \(P=0.002\)).

**Discussion**

This study was designed to investigate the relationship between LGE CMR signal intensity and LA endocardial voltage in patients undergoing repeat catheter ablation for LA arrhythmias and to define an objective user-independent method for assessing the ability of LGE CMR to predict electrical isolation or reconnection of LA ablation lesions.

The principal findings are summarized as follows: (1) there is only a weak inverse point-by-point relationship between mean endocardial voltage and CMR signal intensity, (2) low endocardial voltage can occur at sites of low and high CMR signal intensity, and (3) low CMR signal intensities (LGE\(_{min}\)) occur at the site of both electrically isolated and connected ablation lesions, indicating that there is no clinically useful threshold to determine the electrical integrity of LA ablation lesions.

**Correlation Between LGE CMR and LA Endocardial Voltage**

In a retrospective analysis of 24 patients presenting to a single center for repeat catheter ablation of AF, 13 patients had voltage maps with >100 points and interpretable LGE CMR...
scans. This study demonstrated a qualitative correlation between regions of LGE and low voltage in all patients and a quantitative relationship ($R^2=0.57$) by subdividing the LA into 18 regions and scoring the extent of LGE and low voltage on a scale from 0 to 3.

A study of 10 patients, by different authors, demonstrated initial experience with coregistration of LGE CMR signal intensities with endocardial voltage mapping. In this study, authors used manual segmentation to distinguish between areas of visual nonenhancement and enhancement in the LA wall to create a binary (scar versus normal) 3D LGE CMR reconstruction. It was then determined whether each voltage point (mean number of points 90±24, total of 893 points) fell within an area of coregistered scar or not. This study demonstrated that the mean voltage within areas of CMR scar was lower than the mean voltage within normal areas on CMR. However, although the mean voltages were statistically different, there was almost complete overlap of the 2 populations, indicating that defining LGE CMR on the basis of visual assessment is neither sensitive nor specific for predicting endocardial voltage.

More recently, a study of 11 patients undergoing repeat LA catheter ablation suggested a statistical difference between the mean unipolar and bipolar endocardial voltages corresponding to LGE CMR signal intensity thresholds of 3 to 5 SD above the mean signal intensity of the atrial blood pool. No statistical difference was seen above 5 SD, and the authors concluded that signal intensities above this might represent fully scarred atrial myocardium. However, this would not explain the lack of statistical significance at 0 to 3 SD. More importantly, although there may be a trend toward lower mean voltage with increased CMR signal intensity between 3 and 5 SD, the significant overlap between the populations means that determining a clinically useful threshold from these data is not possible.

### Atrial Endocardial Voltage Thresholds

In current clinical practice, atrial scar is most commonly identified by a bipolar voltage of ≤0.05 mV. This threshold originates from the baseline noise in early electroanatomical mapping systems and has been propagated through the literature and clinical practice without published pathological validation. A recent animal study from our own group has challenged this threshold with the finding that the mean bipolar voltage at the center of a dense scar was 0.3 mV. Another study has demonstrated that a bipolar voltage of <0.15 mV best predicted sites without pace capture in patients undergoing redo PV isolation. When a threshold of ≤0.05 mV was applied in this study (in patients with previous ablation), only 1.8% of the points were below this threshold. This further suggests that using a threshold of ≤0.05 mV to define atrial scar could significantly underestimate the extent of previous ablation injury. Although applying a threshold of ≤0.3 mV demonstrated a better visual correlation with CMR in some patients, this was not true for most patients (Figure 5). The absence of low-voltage points in areas of LGE on the CMR reconstructions raises the possibility that CMR cannot distinguish between complete and incomplete scar and that the 2 techniques may be complementary, rather than interchangeable.

### LGE CMR Prediction of Electrical Reconnection and Isolation

In one of the aforementioned studies, the authors reported that all patients with incomplete ablation sets marked by identifiable gap lesions had recovery of electrical activity on repeat electrophysiological study, suggesting that LGE CMR was able to predict electrical reconnection in all 13 patients. However, in another, the authors found no appreciable relationship between sites of PV reconnection and gaps on the LGE CMR images. Complete circumferential enhancement on LGE CMR was seen in only 2 of 37 PVs, and PV reconnection sites were frequently seen in the regions of CMR-defined scar.

A more recent study reported electrical reconnection in 49 PVs in 15 patients undergoing repeat catheter ablation for AF. The authors reported LGE CMR gaps in 46 of these, with 3 PVs showing a complete absence of LGE, concluding a strong concordance between the presence of a gap on CMR and PV reconnection. However, there is an important limitation to this approach; the high prevalence of PV reconnection in this study (49 of 56 PVs) does not allow a sufficient assessment of false positives (a gap on CMR, but an electrically isolated PV). In fact for isolated PVs, CMR was predictive only 4 of 7 times.

As low signal intensities on LGE CMR would be the most likely sites of reconnection and low signal intensities were seen in all lesions (whether electrically isolated or not) in this

### Table 3. Summary LGE\textsubscript{min} and LGE\textsubscript{mean} Values for Reconnected/Unblocked and Isolated/Blocked Lesions

<table>
<thead>
<tr>
<th>Lesions</th>
<th>LGE\textsubscript{min}</th>
<th>PValue</th>
<th>LGE\textsubscript{mean}</th>
<th>PValue</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right PVS Reconnected (n=13)</td>
<td>0.79±1.11</td>
<td>0.13</td>
<td>3.25±1.88</td>
<td>0.19</td>
</tr>
<tr>
<td>Isolated (n=7)</td>
<td>0.11±0.15</td>
<td></td>
<td>2.20±1.07</td>
<td></td>
</tr>
<tr>
<td>Left PVS Reconnected (n=12)</td>
<td>0.23±0.41</td>
<td>0.67</td>
<td>1.83±0.75</td>
<td>0.43</td>
</tr>
<tr>
<td>Isolated (n=8)</td>
<td>0.15±0.25</td>
<td></td>
<td>2.19±1.22</td>
<td></td>
</tr>
<tr>
<td>Roof line Unblocked (n=16)</td>
<td>1.20±2.05</td>
<td>0.73</td>
<td>2.44±2.73</td>
<td>0.60</td>
</tr>
<tr>
<td>Blocked (n=4)</td>
<td>0.74±1.38</td>
<td></td>
<td>1.68±1.25</td>
<td></td>
</tr>
<tr>
<td>Mitral line Unblocked (n=17)</td>
<td>1.26±2.00</td>
<td>0.42</td>
<td>2.44±1.67</td>
<td>0.13</td>
</tr>
<tr>
<td>Blocked (n=3)</td>
<td>1.50±0.52</td>
<td></td>
<td>4.02±0.22</td>
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</tr>
</tbody>
</table>

The units for LGE\textsubscript{min} and LGE\textsubscript{mean} are signal intensity, expressed as the number of SD above the mean signal intensity of the atrial blood pool. LGE indicates late gadolinium enhancement; and PV, pulmonary vein.
study, this would suggest that LGE CMR cannot currently predict electrical integrity with a clinically useful sensitivity or specificity. However, this finding is unsurprising because it is known that PV isolation can occur with an incompletely circumferential ablation lesion and equally, electrical reconnection may be because of small strands of intact atrial myocytes, well beyond the resolution of any noninvasive imaging technique or even invasive endocardial voltage mapping.26,27

Limitations
1. Unlike computed tomography, with conventional LGE imaging sequences, signal intensity is expressed on an arbitrary scale that differs from one imaging study to another and is not necessarily suitable for quantification between patients. Image contrast is generated by the difference in signal intensity between normal and abnormal myocardium and is dependent on surface coil proximity, sequence parameters (particularly the inversion time), body mass index, hematocrit, renal function, and field strength. In this study, the inversion time was chosen to null the ventricular16,19 rather than LA28 myocardium because the thin atrial wall is highly susceptible to partial volume and motion artefacts on the Look-Locker sequence. However, gadolinium contrast wash-in and wash-out kinetics for LA and ventricular myocardium are different, and if the normal LA myocardium were not correctly nulled, this would affect the assessment of LGE signal intensities. Furthermore, there is currently no consensus on the optimum timing of atrial LGE CMR after contrast administration or on the choice and dose of contrast agent, which also affect signal intensity. As the time from contrast administration to image acquisition increases, the signal intensity of the atrial blood pool progressively reduces, whereas that of scarred atrial myocardium increases, altering the ratio between the 2 intensities dramatically. In this study, these parameters were chosen based on previously published studies of atrial CMR and clinical experience.
2. Invasive atrial endocardial voltage recordings depend on tissue contact (contact force assessment was not available for this study), catheter tip size, atrial wall thickness, and far-field electrograms. In this study, these limitations were mitigated as far as possible by the collection of a large number of voltage points by an experienced operator using a point-by-point technique.
3. Point-by-point comparison of the LGE CMR signal intensities and endocardial voltage requires the image registration of 2 differently acquired LA shells. The error in this registration was minimized (to 0.5±0.2 mm) by using both landmark and iterative closest point registration.
4. The use of a maximum intensity projection to display LA LGE signal intensities on a 3D LA shell can skew the intensities in favor of the atrial blood pool where the myocardial signal intensity is less than the blood pool.

Conclusions
Although previous studies have suggested a relationship between LGE CMR signal intensity and atrial endocardial voltage,10,15,16 this study demonstrates that there is only a weak point-by-point relationship between the 2 parameters and that low endocardial voltage can occur at sites of low and high CMR signal intensity. Furthermore, using an objective method of assessing gaps in ablation lesions, LGE CMR is unable to reliably predict the sites of electrical conduction.

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Disclosures
M. O’Neill has received speaker honoraria from Biosense Webster. The other authors report no conflicts.

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