Response to Letter From Bisbal et al Regarding, “Repeat Left Atrial Catheter Ablation: Cardiac Magnetic Resonance Prediction of Endocardial Voltage and Gaps in Ablation Lesion Sets”

We thank Bisbal et al for their interest in our study and for raising several important questions.

First, and importantly, we would like to clarify that pulmonary vein reconnection and conduction across the mitral and roof lines were assessed using electrophysiological techniques and not voltage mapping. We agree that using point-by-point voltage mapping to identify gaps in ablation lesions is unreliable. The number of voltage points is therefore not relevant to our finding that late gadolinium enhancement (LGE) cardiac magnetic resonance was unable to reliably predict sites of electric conduction.

However, our study did use voltage mapping to determine the relationship between LGE cardiac magnetic resonance signal intensity and endocardial voltage. The number of points in our study was greater than in 3 previous studies comparing these 2 parameters, and the weak correlation seen in all 20 patients (including 12 with over 300 points) makes insufficient point density an improbable cause for the negative result.

Regarding the image acquisition parameters, increasing the slice thickness of the LGE cardiac magnetic resonance acquisition from 2.5 mm to 4 mm greatly increases the signal to noise ratio of each voxel, increasing the likelihood of detecting LGE. Furthermore, the gaps seen in the study by Bisbal et al were 13.30±5.78 mm, well within the resolution of our sequence.

We acknowledge that 11 out of 20 patients had undergone >1 previous left atrial ablation procedure. We specifically recruited patients where one would expect an unequivocal demonstration of LGE to allow a rigorous assessment of the relationship between LGE signal intensity and endocardial voltage. We do not agree that this is a limitation of our study. The reduction in LGE seen at ≥2 years postablation compared with 3 months postablation, to which Bisbal et al refer, has not been published in manuscript format and potentially contradicts findings from the same group showing no reduction in LGE from 3 to 6 and 6 to 9 months postablation. It is unlikely that there is a pathological difference in ablation-induced atrial scar in the patients in our study compared with the study by Bisbal et al, in which the median time from the first procedure was 15 (range 3–95) months.

The LGE cardiac magnetic resonance acquisition used in our study has been histopathologically validated in an animal study, and no superiority over other methodologies is claimed. We agree that the representation of atrial LGE data on a 3D shell is challenging, and there is no perfect or validated method. We have used a method published by our group and used by other groups.

It is our opinion that the presence of LGE is specific for the presence of chronic ablation-induced scar, but that the technique is not sufficiently sensitive—the absence of LGE does not necessarily indicate a gap in an ablation lesion. We look forward to future developments in this exciting field.

Disclosures

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


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