

# Influence of underlying substrate on atrial tachyarrhythmias after pulmonary vein isolation



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**BACKGROUND** Recurrent atrial tachyarrhythmias occur as a result of residual atrial arrhythmogenic substrates after atrial fibrillation (AF) ablation. In patients with AF, electrograms with reduced amplitudes indicate diseased myocardium.

**OBJECTIVE** The purpose of this study was to investigate the association between the distribution of low-voltage areas and the type of induced atrial tachyarrhythmias.

**METHODS** Our prospective observational study enrolled 152 consecutive AF patients scheduled for an initial ablation (46% persistent AF). After pulmonary vein isolation, voltage mapping was performed during sinus rhythm, and regions with reduced electrogram amplitudes ( $<0.5$  mV) were defined as low-voltage areas. Burst pacing was performed to investigate the inducibility of atrial tachyarrhythmias.

**RESULTS** Low-voltage areas were more frequently observed in patients with persistent AF than paroxysmal AF (50% vs 34%,  $P = .048$ ). A higher proportion of patients with low-voltage areas presented with inducibility of atrial tachyarrhythmias than those

without, as follows: AF 70% vs 16% ( $P = .0001$ ); perimitral macroreentrant atrial tachycardia (AT) 18% vs 0% ( $P = .0001$ ); and roof-dependent macroreentrant AT 13% vs 0% ( $P = .01$ ). Investigation into the regional distribution of low-voltage areas revealed that patients with perimitral macroreentrant AT more frequently coincided with low-voltage areas than those without in the septal (100% vs 18%,  $P < .0001$ ) and anterior regions (55% vs 11%,  $P = .001$ ), and those with roof-dependent AT in the roof (75% vs 15%,  $P < .0001$ ) and posterior regions (75% vs 15%,  $P = .0001$ ).

**CONCLUSION** Low-voltage areas are associated with high inducibility of atrial tachyarrhythmias after pulmonary vein isolation. In addition, the distribution of low-voltage areas is specific for each type of macroreentrant AT.

**KEYWORDS** Atrial fibrillation; Atrial tachycardia; Inducibility; Substrate; Low-voltage area

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## Introduction

Catheter ablation is commonly used to treat atrial fibrillation (AF). However, a number of patients experience recurrence of atrial tachyarrhythmias, including AF and other types of atrial tachycardias (ATs). Advanced atrial remodeling or prior ablation lesions result in the generation of residual atrial arrhythmogenic substrates and the development of atrial tachyarrhythmias after AF ablation. Although the relatively frequent recurrence of atrial tachyarrhythmias after AF ablation in patients with advanced atrial remodeling has been demonstrated, the association between the distribution of diseased atrial myocardium and the type of recurrent atrial tachyarrhythmias remains unclear.

Electrograms with reduced amplitude on atrial endocardial voltage maps correlate well with atrial scarring as detected by delayed enhancement magnetic resonance

imaging.<sup>1,2</sup> The presence of low-voltage areas in the left atrium after pulmonary vein isolation (PVI) is an accurate predictor of the recurrence of atrial tachyarrhythmias, and the addition of low-voltage guided ablation improves procedural outcomes compared to PVI alone.<sup>3,4</sup>

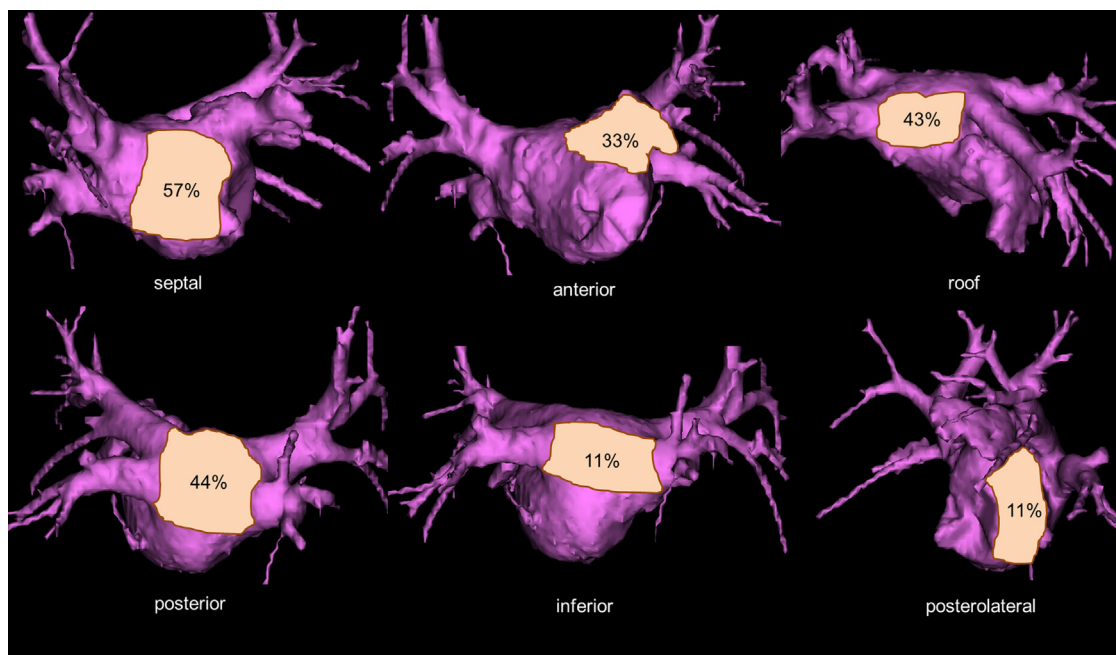
To our knowledge, however, no study has examined the differences in the distribution of low-voltage areas between the different types of atrial tachyarrhythmias after PVI. In this study, we investigated the association between the distribution of low-voltage areas and the type of induced atrial tachyarrhythmias.

## Methods

### Patients

From December 2014 to August 2015, consecutive patients who underwent an initial ablation for AF at Kansai Rosai Hospital were enrolled. Patients who could not maintain sinus rhythm even after electrical cardioversion followed by PVI were excluded. Other exclusion criteria were age  $<20$

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**Figure 1** Distribution of low-voltage areas in each region. Low-voltage areas were more frequently observed in the septal, anterior, roof, and posterior regions than in the inferior and posterolateral regions.

years, prior cardiac surgery, prior catheter ablation, pacemaker implantation, or severe mitral valve disease.

This study complied with the principles of the Declaration of Helsinki. Written informed consent for the ablation and participation in the study was obtained from all patients, and the protocol was approved by our institutional review board.

### Radiofrequency catheter ablation procedure

Electrophysiologic studies and catheter ablation were performed with patients under intravenous sedation with dexmedetomidine. The latter procedures performed by two experienced operators. A 6Fr decapolar electrode was inserted into the coronary sinus, and another 6Fr decapolar electrode was placed in the right atrium. After a transeptal puncture at the fossa ovalis, three long sheaths were introduced into the left atrium using a single transeptal puncture technique. Two 20-pole circular catheters were placed at the ipsilateral superior and inferior pulmonary veins via long sheaths. Electrical cardioversion was performed in cases with persistent AF. Mapping and ablation were then performed under guidance from an electroanatomic mapping system (CARTO3, Biosense Webster, Diamond Bar, CA). Circumferential ablation around both ipsilateral pulmonary veins was performed using a dragging technique.

An open-irrigated ablation catheter with a 3.5-mm tip (ThermoCool SmartTouch catheter, Biosense Webster) via an Agilis or SL0 sheath (St. Jude Medical, Minnetonka, MN) was used. Radiofrequency energy was applied for 30 seconds at each site using a maximum temperature of 42°C, maximum power of 35 W, and flow rate of 17 mL/min. At the posterior wall near the esophagus, the duration of radiofrequency energy delivery was limited to 15 seconds.

Operators attempted to maintain contact force values between 10g and 30g to ensure that the degree of contact between the catheter and myocardium was appropriate. PVI was considered complete when the circular catheters no longer recorded any pulmonary vein potential.

### Voltage mapping

After PVI, detailed voltage mapping using a bipolar 3.5-mm tip catheter (ThermoCool SmartTouch, Biosense Webster) was performed during sinus rhythm in accordance with a previous study.<sup>4</sup> Mapping points were acquired to fill all color gaps on the voltage map using CARTO3 with an interpolation threshold of 15 mm for the fill threshold and 23 mm for the color threshold. In addition, high-density mapping was added at sites where low-voltage areas were recorded to exactly delineate the extent of the low-voltage area. Adequate endocardial contact was confirmed by stable electrograms, the distance to the geometry surface, and increased contact force values  $\geq 5g$ . The bandpass filter was set at 30 to 500 Hz. Each acquired point was classified according to the peak-to-peak electrogram as follows:  $>0.5$  mV, healthy; 0.2–0.5 mV, diseased; and  $<0.2$  mV, scarred. Low-voltage areas were defined as sites of  $\geq 3$  adjacent low-voltage ( $<0.5$  mV) points, which were  $<5$  mm apart from each other. The left atrium was divided into six regions: septal, anterior, roof, posterior, inferior, and posterolateral (Figure 1).

### Induction of atrial tachyarrhythmias after PVI

After voltage mapping, the inducibility of atrial tachyarrhythmias was sequentially examined with burst pacing from the electrodes placed in the right atrial appendage. Constant burst pacing was performed for 5 seconds at each cycle length, starting with 300 ms with a subsequent decrement of

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