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Persistence and Distortion of Electrical Activity in the LAA 5 Years After Endovascular Occlusion

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ABSTRACT

This is the first report demonstrating persistence and distortion of electrical activity in the left atrial appendage 5 years after endovascular occlusion with a Watchman device. Electrical conduction is impaired providing an arrhythmogenic substrate for atrial tachyarrhythmias. Localized inflammation may result in structural and electrical remodeling in these patients. **(Level of Difficulty: Intermediate.)** (J Am Coll Cardiol Case Rep 2020;2:583–7) © 2020 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

The left atrial appendage (LAA) is characterized by an extensive network of pectinate muscles with high variability in fiber orientation. This complex anatomy may facilitate intra-atrial conduction disorders and asynchronous activation of the endo- and epicardial layers, thereby providing an arrhythmogenic substrate for atrial fibrillation (AF) (1). Several mapping studies suggested that the LAA plays an important role in the pathophysiology of AF by demonstrating focal activity or re-entry circuits in the LAA (2,3).

LEARNING OBJECTIVES

- To recognize potential arrhythmogenic effects of endovascular LAA occlusion.
- To understand differences between epicardial exclusion and endovascular occlusion of the LAA on electrical conduction.
- Mechanical occlusion of the LAA is not similar to electrical isolation of the LAA.

Most intracardiac thrombi in patients with AF arise from the LAA. Besides oral anticoagulants, percutaneous/surgical LAA exclusion is an alternative strategy to reduce stroke risk in patients with AF. LAA exclusion devices, such as the AtriClip (AtriCure, West Chester Township, Ohio) and LARIAT (SentreHEART, Redwood City, California) result in both mechanical isolation and electrical isolation (EI) of the LAA (4,5). However, there are no data on the impact of endovascular LAA occlusion devices on atrial conduction.

HISTORY OF PRESENTATION

A 63-year-old obese woman presented with progressive paroxysmal AF. Despite pharmacological therapy and multiple electrical cardioversions, she became progressively symptomatic. Physical examination was unremarkable. The electrocardiogram showed AF with a ventricular response of 120 beats/min.

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ABBREVIATIONS AND ACRONYMS

AF = atrial fibrillation

EI-LAA = electrical isolation of the left atrial appendage

LAA = left atrial appendage

PAST MEDICAL HISTORY

The patient had a history of paroxysmal AF for 11 years, no structural heart disease, and was afflicted with multiple ischemic and hemorrhagic strokes while using oral anticoagulants. On this indication she was treated with an endovascular LAA occlusion device, Watchman (Boston Scientific, Natick, Massachusetts), 5 years ago. Complete LAA occlusion was achieved and strokes did not occur ever since.

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DIFFERENTIAL DIAGNOSIS

The patient had progressive AF symptoms lasting <48 h.

INVESTIGATIONS

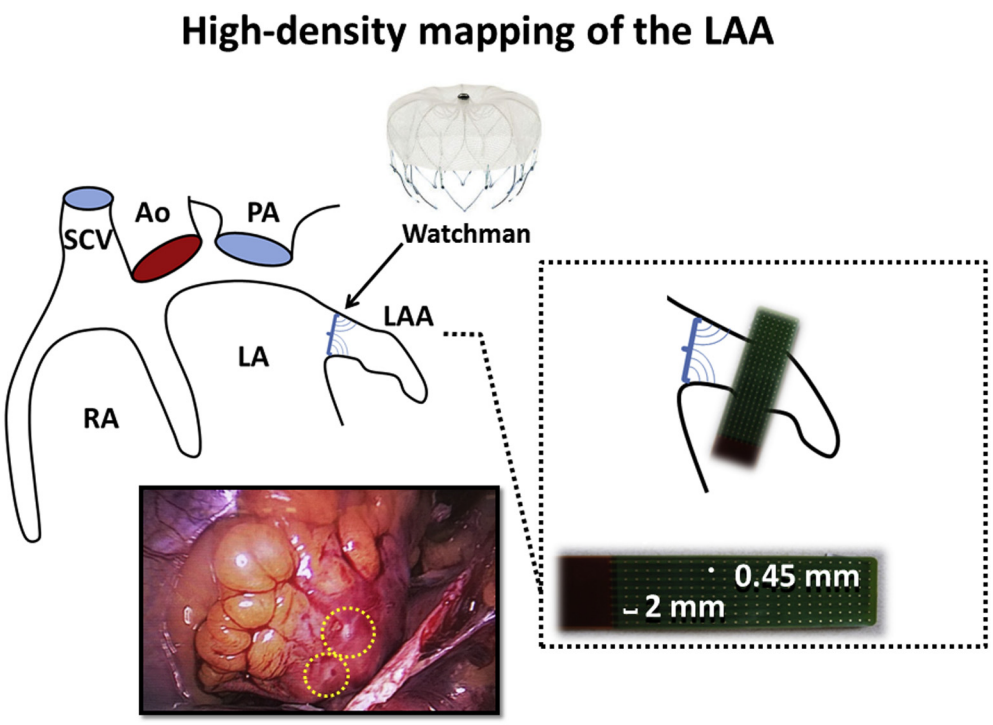
The patient was accepted for surgical AF ablation. As demonstrated in **Figure 1**, metal struts with anchor hooks of the Watchman device were visible beneath the epicardial layer and characterized by white epicardial scarring lesions. As the LAA was occluded with the Watchman device, LAA clipping was not required. In addition, there was no lesion created from the box toward the LAA to avoid dislodgement or damage to the Watchman.

After opening the pericardium from a left thoracoscopic view, the mapping array was introduced through an incision and positioned transversely on the LAA (**Figure 1**). High-density epicardial mapping of the LAA was then performed (n = 192, diameter = 0.4 mm, interelectrode distance: 2 mm). Recordings were sampled with a rate of 1 kHz, amplified (gain 1,000), filtered (bandwidth 0.5 to 400 Hz), analog-to-digital converted (16 bits).

Local activation times were determined by marking the steepest negative slope of atrial potentials (**Figure 2**). Conduction delay and block were defined as time differences of respectively ≥7 ms and ≥12 ms between neighboring electrodes (6).

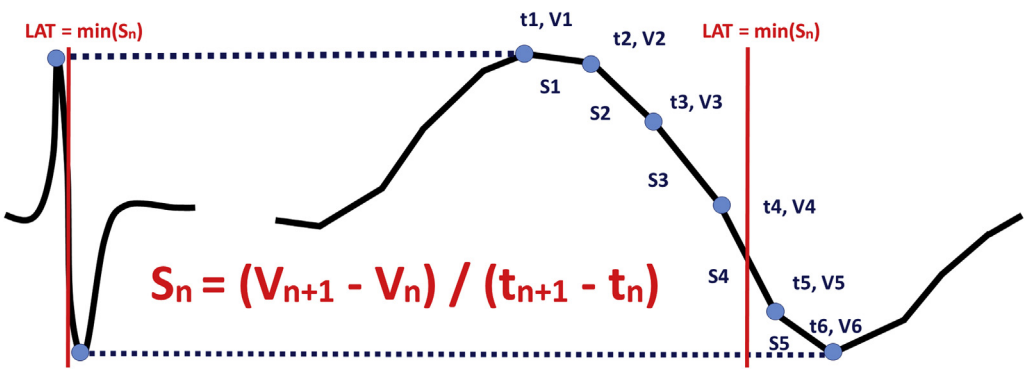
Epicardial mapping of the LAA was performed during 10 s of sinus rhythm. The recording included 13 beats with a median cycle length of 725 ms (interquartile range: 723 to 726 ms). Consecutive LAA activation maps show a consistent pattern of activation

FIGURE 1 Schematic View of Anatomy and Positioning of the Array



Operative view shows white epicardial scarring lesions near the metal struts of the Watchman **(circled in yellow).** Ao = aorta; LA = left atrium; LAA = left atrial appendage; PA = pulmonary artery; RA = right atrium; SCV = superior caval vein.

FIGURE 2 Local Activation Times

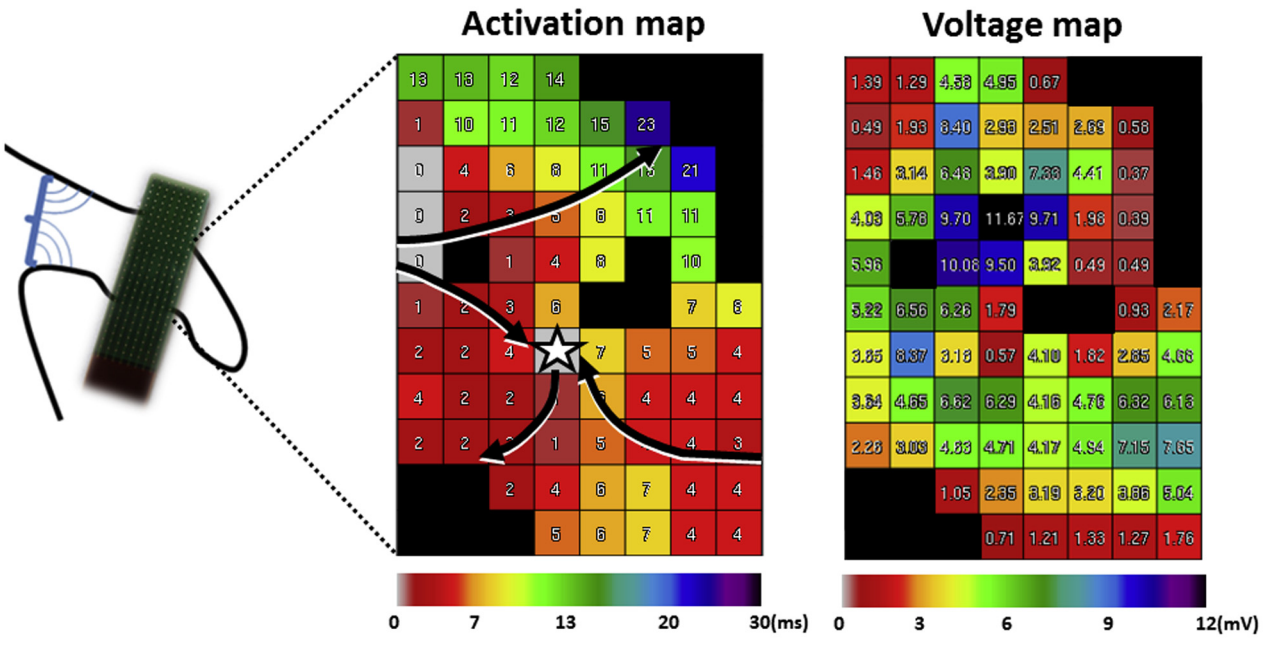


Local activation times (LAT) were determined by annotating the steepest part of the negative deflections of atrial potentials on every electrode. S = slope (mV/ms); t = time (ms); V = voltage (mV).

consisting of 2 colliding wave fronts in addition to an epicardial breakthrough wave expanding to a limited area (**Figure 3**, Video 1).

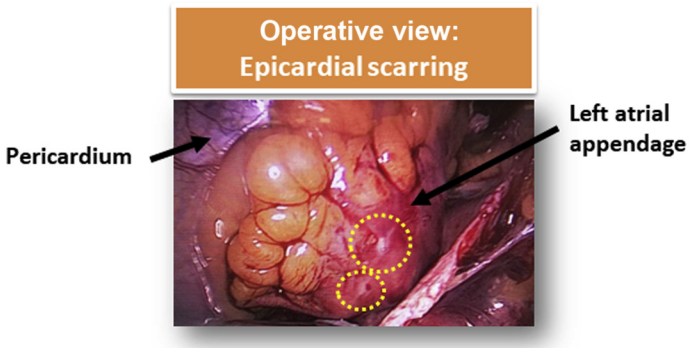
Areas of conduction delay and block were present in 5% of the mapping area (median length conduction delay/block lines: 2 mm). Overall median signal amplitude was 3.86 mV ranging from 0.4 to 11.7 mV. Fixed rate pacing at the LAA resulted in atrial capture, confirming persistence of electrical activity.

FIGURE 3 Activation and Voltage Maps



Operative findings, including color-coded activation and voltage maps.

FIGURE 4 Operative View of Epicardial Scarring



Operative view demonstrating white epicardial scarring **(circled in yellow),** near the anchors of the Watchman device, that is macroscopically suggestive for fibrous tissue.

FOLLOW-UP

Post-operative AF did not occur and the patient was discharged with sinus rhythm at sixth day after cardiac surgery.

DISCUSSION

This is the first report on epicardial mapping of the LAA 5 years after endovascular occlusion with a Watchman device. Despite mechanical occlusion and in contrast to epicardial clipping or ligation of the LAA, there is no EI-LAA. Yet, there is a distorted pattern of activation that is clearly different from activation patterns of the LAA observed in prior mapping studies.

Several studies have demonstrated the importance of EI-LAA in improving outcomes of ablative AF therapy. In 987 patients (6% with paroxysmal AF) undergoing redo catheter ablation for AF, 27% had triggers originating from the LAA and in 9% the LAA was the only source (3). Patients with (long-standing) persistent AF undergoing catheter ablation in combination with EI-LAA showed lower AF recurrence rates (3,7–9). Lakkireddy et al. (8) demonstrated, in a cohort of 69 patients undergoing ablative therapy for AF and LAA ligation matched with 69 patients undergoing ablative therapy only, that patients with LAA ligation, hence EI-LAA, had a higher freedom from AF at 1 year (65% vs. 39%, $p = 0.002$). Panikker et al. (9) showed that concomitant EI-LAA by catheter ablation followed by endovascular LAA exclusion with a Watchman device is safe and may improve AF-free survival in patients with (long-standing) persistent AF.

LAA occlusion results in localized inflammation and subsequent endothelialization and fibrosis of not only the nearby area of the device but also in more distant parts of the LAA (10). In our case, white epicardial scarring lesions, macroscopically suggestive for fibrous tissue, were observed at the LAA near the anchors of the Watchman device (**Figure 4**). The anchors in the LAA (corpus alienum effect) in combination with mechanical stress and localized inflammation may lead to structural and electrical remodeling, which affects electrical conduction. As a consequence, normal pattern of activation is disrupted resulting in collision of parts of sinus rhythm wave fronts propagating in different directions and repetitive epicardial breakthrough waves. Hence, persistence of LAA electrical activity and structural remodeling provide an arrhythmogenic substrate for atrial tachyarrhythmias in these patients.

Besides eliminating potential AF triggers, epicardial exclusion of the LAA may also down-regulate the renin-angiotensin-aldosterone system. Lakkireddy et al. (11) compared the effect of epicardial clipping and endocardial occlusion on renin-angiotensin-aldosterone system after a follow-up period of 3 months and found that systolic blood pressure and glucose levels were lower and insulin levels were higher after clipping.

CONCLUSIONS

Our case demonstrates that impaired electrical activation of the LAA persists even 5 years after endovascular LAA occlusion. This is a point of interest when performing lone endovascular LAA occlusion, especially in patients with persistent AF in whom rhythm control is endeavored. In current European and American guidelines, recommendations of either percutaneous or surgical LAA occlusion and/or exclusion, EI-LAA is not taken into consideration. Targeting the LAA is more challenging in the presence of an endovascular device, hence epicardial clipping or endovascular occlusion with concomitant EI-LAA might be suitable alternatives. Future studies focusing on electrical activity in the LAA after endovascular exclusion are required to further unravel the arrhythmogenic substrate.

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KEY WORDS ablation, atrial fibrillation, electrophysiology

APPENDIX For a supplemental video, please see the online version of this paper.