Lengthening the atrioventricular delay reduces large left atrial v waves and dyspnea after atrial fibrillation and tachycardia ablation

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Introduction

Pulmonary vein isolation (PVI) remains the cornerstone of therapy for patients with drug-refractory paroxysmal atrial fibrillation; and recent studies have suggested that even for patients with persistent atrial fibrillation, addition of linear lesions to PVI may not confer any advantage in efficacy.[1–3] Despite this, patients with significant left atrial fibrosis and chamber enlargement may have additional substrate for atrial fibrillation or tachycardia that remains after PVI because of nonpulmonary vein (PV) substrate requiring further ablation.[4–6] These additional lesions may have effects on left atrial conduction and physiology through a variety of mechanisms including alteration of left atrial compliance as suspected in stiff left atrial syndrome and alteration of left atrial and left ventricular synchrony.[7–11] Two cases are presented here in patients with previous permanent pacemakers undergoing PV and substrate ablation for atrial fibrillation and/or atrial tachycardia, leading to variable degrees of left atrial appendage (LAA) delay causing abnormal hemodynamics and symptoms, which were improved by lengthening the pacemaker atrioventricular delay (AVD).

Case reports

Patient 1

A 66-year-old male patient with obstructive sleep apnea, diabetes, hypertension, and preexisting complete heart block with a dual-chamber pacemaker underwent radiofrequency catheter ablation for symptomatic, drug-refractory (sotalol) atrial fibrillation and atrial tachycardia despite ventricular rate control due to heart block. A preprocedural echocardiogram showed a left ventricular ejection fraction of 60%, mild left ventricular hypertrophy, a left atrial volume index of 33 mL/m^2, no significant mitral regurgitation, and pulmonary artery systolic pressures of 35–40 mm Hg. High-density mapping of the left atrium and ablation were performed with PentaRay NAV and ThermoCool SmartTouch catheters (Biosense Webster Inc., Diamond Bar, CA). After PVI, 2 morphologies of atrial tachycardia were easily inducible with single atrial extrastimuli, which matched his clinical atrial tachycardia. Mapping and concealed entrainment revealed a roof mediated tachycardia and an anterior wall tachycardia, both of which were successfully ablated after creation of a linear roofline and lesions in the anterior left atrium. No further arrhythmias were inducible with triple atrial extrastimuli or burst pacing down to 180 ms. Hemodynamic tracings were recorded through the transseptal sheath upon initial transseptal puncture as well as throughout the case. The effects of varying the AVD were examined postablation. The observation of large left atrial v waves prompted further investigation of the left atrial conduction times and the effect of varying the AVD of the pacemaker.

Patient 2

Patient 2 had undergone ablation almost 8 months before patient 1. Three years before ablation, he had undergone implantation of a cardiac resynchronization therapy (CRT)–defibrillator for ischemic cardiomyopathy and a left ventricular ejection fraction of 30% with alternating bundle branch block and syncope. He subsequently developed symptomatic, drug-refractory (dofetilide) atrial fibrillation. A preprocedural echocardiogram showed a previous anteroapical infarction, a left ventricular ejection fraction of 40%, and a left atrial volume index of 42 mL/m^2. High-density mapping of the left atrium and ablation were performed with Lasso circular mapping and ThermoCool SmartTouch catheters (Biosense Webster Inc.). He also underwent roofline and focal anterior ablation for low-amplitude fractionated signals along the anterior left atrium, which were present in sinus rhythm, because of recurrent spontaneous atrial fibrillation despite cardioversion after PVI and PVI + roofline but not after delivery of the anterior lesions. At follow-up, he complained of dyspnea with activities of daily living and was effectively in New York Heart Association functional class II-III despite being in sinus rhythm with appropriate CRT pacing. He noted this to be much worse than when he had been cardioverted and held sinus rhythm for 1 month in the year before

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KEY TEACHING POINTS

- Left atrial appendage activation time in sinus rhythm should be assessed before and after atrial fibrillation ablation and compared to the PR interval.
- Placement of anterior lesions should take into consideration the effects on left atrial appendage activation.
- Patients with PR prolongation or heart block and a dual-chamber or cardiac resynchronization therapy–pacemaker who experience this phenomenon may be helped by reprogramming the atrioventricular delay beyond the measured activation time to the left atrial appendage.

ablation. Further workup included cardiac computed tomography showing no PV stenosis, nuclear myocardial single-photon emission computed tomography imaging showing previous infarct with mild peri-infarct ischemia, cardiac catheterization revealing normal filling pressures and no significant stenosis aside from the previously occluded mid left anterior descending, fluoroscopy showing no phrenic nerve palsy, echocardiography showing no pulmonary hypertension or mitral regurgitation and a left ventricular ejection fraction of 40%, and normal pulmonary function test results. Based on knowledge gained from patient 1, sinus activation maps were reexamined pre- and postablation, based on which the AVD of his CRT-pacemaker was prolonged from 110 to 160 ms, which did not cause fusion or decreased CRT pacing.

Results

In patient 1, the left atrial hemodynamic waveform was normal at baseline with a mean pressure of 15 mm Hg and v waves to 21 mm Hg (Figure 1A); however, postablation it revealed a mean pressure of 27 mm Hg with large v waves up to 35–40 mm Hg (Figure 1B). Intracardiac echocardiography did not show any significant mitral regurgitation by color-flow or pulsed wave Doppler. Examination of LAA intracardiac electrograms revealed that activation which was before the QRS complex at baseline and 120 ms after the atrial pacing spike (Figure 1D) was now at 310 ms postablation and after the paced QRS (Figure 1E). Small left atrial P waves are even visible in the ST segment (Figure 1E, yellow oval).

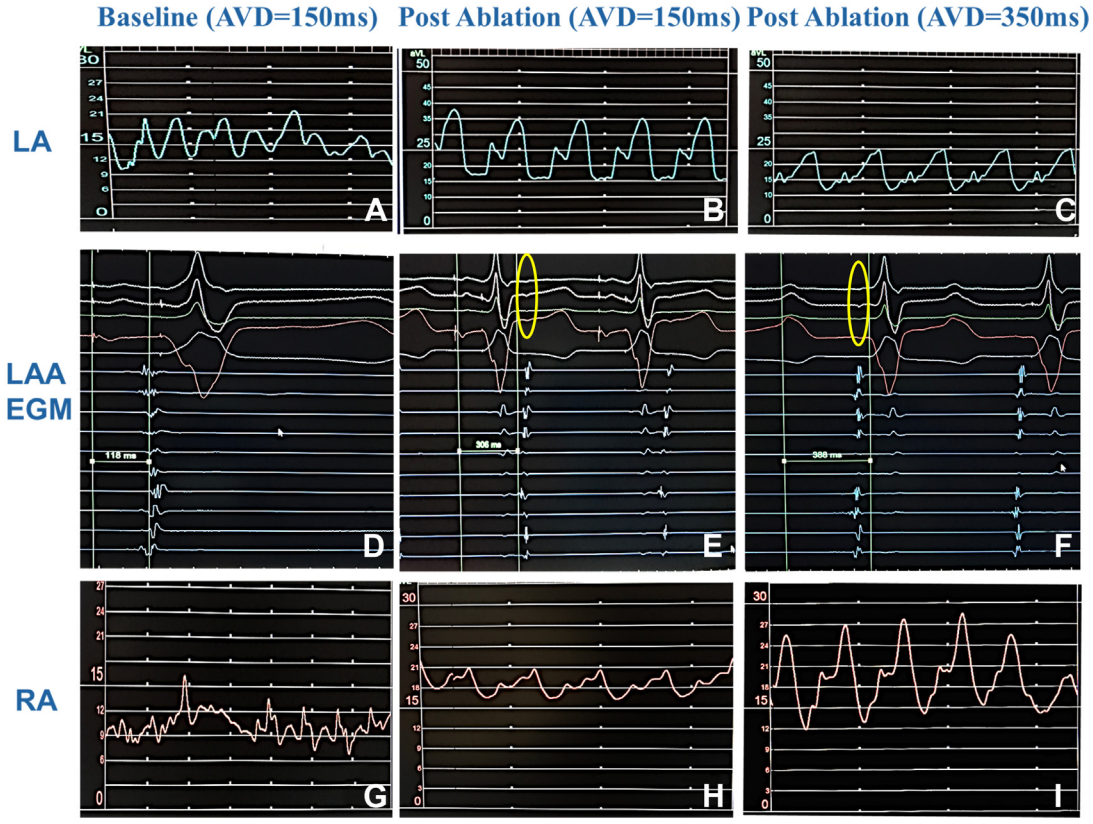

Figure 1 Patient 1: Atrioventricular delay (AVD) effects on left and right atrial pressures with respect to intracardiac left atrial appendage (LAA) electrograms (EGMs). **Column 1 Baseline AVD 150 ms:** (**A**) left atrial (LA) pressures @ 30-mm scale, (**D**) LAA electrograms occurring at 118 ms after the atrial pacing spike, and (**G**) right atrial (RA) pressure @ 30-mm scale at an AVD of 150 ms. **Column 2 Postablation AVD 150 ms:** (**B**) LA pressures @ 50-mm scale with large v waves, (**E**) LAA electrograms now delayed to 306 ms after the QRS with the delayed left atrial P wave in the ST segment (*yellow oval*), and (**H**) RA pressure. **Column 3 Postablation AVD 350 ms:** (**C**) reduction of LA v waves @ 50-mm scale, (**F**) LA electrograms and delayed LA P wave (*yellow oval*) now occurring before the QRS, and (**I**) elevation of RA a waves @ 30-mm scale

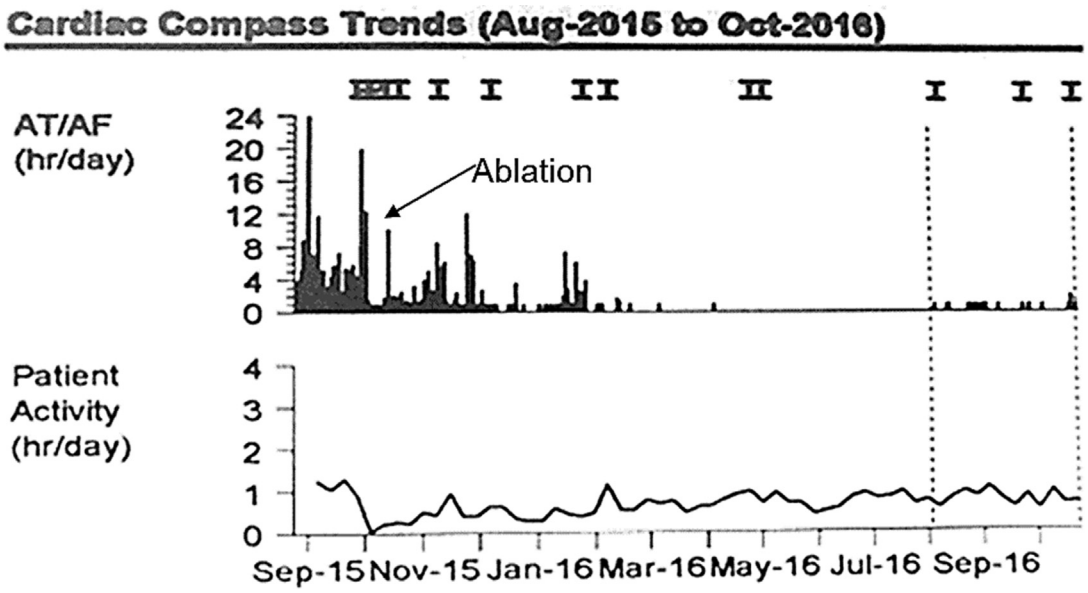

Figure 2 Patient 1: Pacemaker log showing reduction of atrial tachycardia/atrial fibrillation (AT/AF) burden postablation.

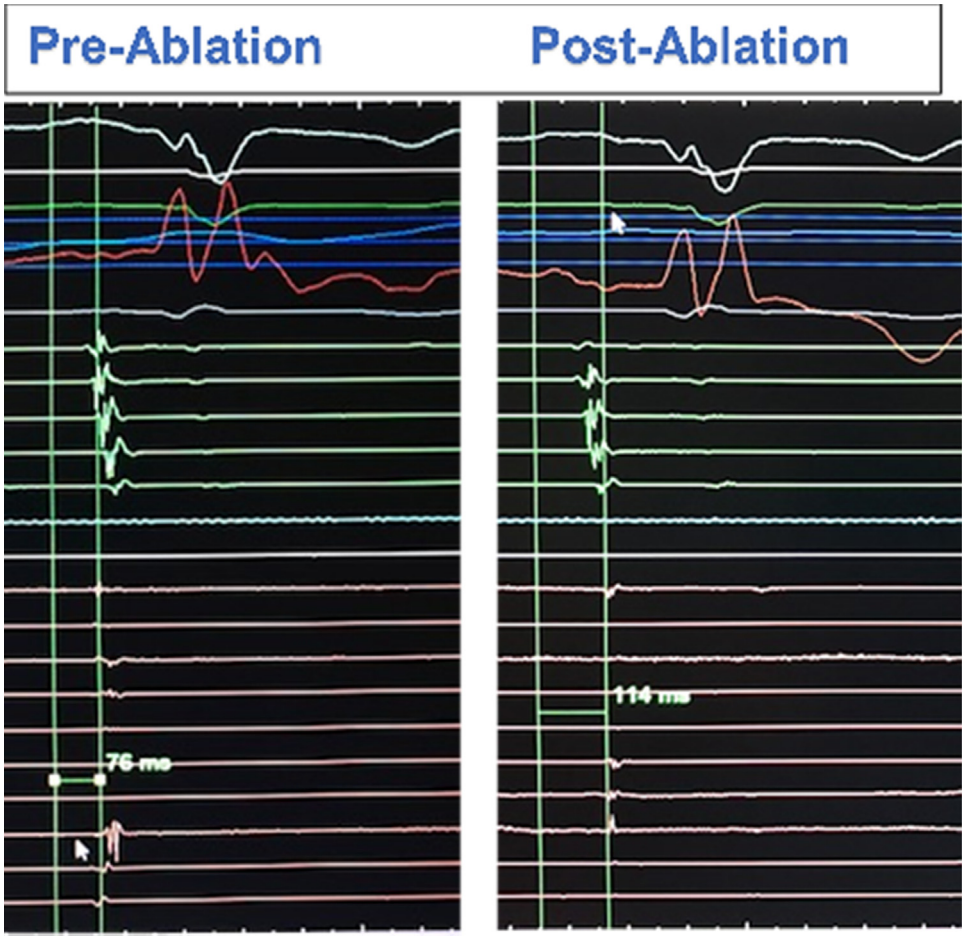

Figure 3 Patient 2: Activation time to left atrial appendage preablation (75 ms) and postablation (114 ms). Note: Cardiac resynchronization therapy pacing was turned off during measurements but was initially set at 110 ms, and upon reviewing these data, it was reprogrammed to 160 ms with resolution of symptoms of dyspnea.

It was hypothesized that large v waves may in part be due to delayed LAA occurring during left ventricular systole, and therefore, extending the AVD beyond the LAA conduction time of 310 ms might allow LAA activation and contraction to occur before ventricular systole.

Figure 1C shows that increasing the AVD to 350 ms markedly reduced the mean left atrial pressure to 18 mm Hg and v waves decreased to 25 mm Hg. After prolongation of the AVD to 350 ms, LAA activation occurs before the paced QRS (Figure 1F) and the delayed left atrial P wave now occurs before the paced QRS (Figure 1F, yellow oval). The effects of these maneuvers on the right atrial waveform are shown in Figures 1G–1I. The baseline mean right atrial pressure was 10 mm Hg. Postablation at an AVD of 150 ms, the right atrial waveform appears normal with a mean pressure of 19 mm Hg (Figure 1H). The improvement in left atrial hemodynamics seen with an AVD of 350 ms, however, is associated with large a waves to 28 mm Hg with a mean right atrial pressure of 21 mm Hg because of right atrial activation occurring near the end of right ventricular systole (Figure 1I). It was felt that the patient would still do better with lower mean left atrial pressures and v waves; thus, the AVD was left at 350 ms. In addition, some of the increase from baseline of the mean right and left atrial pressures was from the fluid given during the procedure (net 2 L positive), and a postprocedure administration of diuretics was expected to decrease the mean right and left atrial pressures. The effect of increased volume, however, would not explain the marked decrease in v waves seen with prolonging the AVD. The patient has done well from a symptomatic standpoint postablation and has had a marked decrease in atrial fibrillation postablation, as shown in Figure 2.

Patient 2 did not have full hemodynamic tracings from the case available for review; however, Figure 3 shows that preablation the LAA activation time was 75 ms but increased to 114 ms postablation. The patient's AVD had been programmed to 110 ms from the initial implantation. The AVD was reprogrammed to 160 ms to allow LAA activation to occur before left ventricular activation (left ventricular activation 40 ms earlier than right ventricular activation), which still allowed biventricular capture. The patient was ambulated in the office immediately after reprogramming and noted a marked improvement in dyspnea and returned to New York Heart Association functional class I.

Discussion

Based on a literature search, this is the first article demonstrating improvement in both the left atrial pressure waveform and symptoms of dyspnea by considering the effects of LAA activation time pre- and postablation and using this information to reprogram the AVD in patients with a pacemaker undergoing ablation for persistent atrial fibrillation. The large v waves seen in patient 1 are reminiscent of stiff left atrial syndrome, which has been attributed to decreased left atrial compliance related to scarring of the left atrium postablation and as associated with dyspnea post atrial fibrillation ablation despite sinus rhythm.[9–11] These cases demonstrate that in select patients who underwent anterior left atrial ablation, another possible cause of large left atrial v waves may be delayed LAA activation, leading to left atrioventricular dyssynchrony from left atrial contraction after the QRS during ventricular systole. In this case, large v waves are more likely left atrial cannon a waves superimposed on the v wave, like that seen in pacemaker syndrome. In patient 1, the presence of heart block and a dual-chamber pacemaker confirmed this mechanism. This may account for unexplained dyspnea in some patients post left atrial ablation despite remaining in sinus rhythm. Even a mild to moderate degree of LAA activation delay as seen in patient 2 may result in similar physiology depending on the PR interval or programmed AVD in a patient with a pacemaker. The marked clinical improvement in the second patient achieved by lengthening of the AVD beyond the measured LAA activation time strongly supports, but does not definitely prove, this hypothesis. Delayed conduction to the LAA may not be apparent on the surface electrocardiogram in all cases, since the delayed left atrial P wave may be buried in the QRS complex. The administration of intravenous adenosine may unmask delayed LAA activation on the surface electrocardiogram. In patients with such physiology without heart block or a pacemaker, options to mitigate the symptoms might include administration of medications that prolong the PR interval such as diltiazem or digoxin. In the cases presented here, the conduction delay to the LAA occurred in the left atrium since no right atrial lesions were delivered. Extensive right to left atrial conduction delay due to extensive right atrial or septal ablation may create a similar delay and has been reported in up to 11% of patients depending on the ablation approach.[7,8,10,11] Avoiding ablation in the region of Bachmann's bundle insertion along the superior anterior aspect of the left atrium would be important to avoid activation delay to the LAA. Up to 1.5% of patients may present with unexplained dyspnea post atrial fibrillation ablation.[11] These patients may warrant an electrophysiology study with electrical and hemodynamic measurements to see whether LAA conduction is delayed with respect to the QRS. If so, left atrial pacing directly or through the distal coronary sinus may decrease the large v wave as compared with sinus or right atrial pacing. Further studies are required to determine the frequency of this mechanism contributing to dyspnea postablation.

Conclusion

Delay of LAA activation due to left atrial anterior lesions during radiofrequency catheter ablation may result in elevation of left atrial pressures with large v waves. While the hemodynamic and symptomatic consequences may be mitigated in patients with preexisting pacemakers (for atrioventricular block or PR prolongation) by lengthening the AVD, options for patients with normal PR conduction may be limited. This may be the mechanism of unexplained dyspnea in some patients post atrial fibrillation ablation, particularly in the absence of mitral regurgitation or PV stenosis. In patients undergoing repeat atrial

fibrillation ablation procedures, the presence of large v waves on transseptal puncture should prompt assessment of LAA activation and conduction during sinus rhythm.

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