

**Case Report**

## Spontaneous Localized Persistent Atrial Fibrillation with an Exit Block Mimicking Atrial Tachycardia at the Left Posterior Wall

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We describe a 37-year-old man with spontaneous localized atrial fibrillation (AF) with an exit block at the posterior wall of the left atrium (LA). The 12-lead ECG exhibited an atrial tachycardia-like pattern, with distinctive P waves and an isoelectric baseline between the P waves. The cycle length of the P waves ranged from 320 to 500 msec. While the fractionated and rapid deflections were recorded from the posterior wall of the LA, the rest of the atria and the coronary sinus exhibited discrete atrial potentials with irregular intervals. Radiofrequency energy applications to the surrounding tissue created complete isolation of the localized AF area, and the AF was terminated. Fibrillatory activation in the posterior wall of the LA can act as a driver as well as an initiator of atrial fibrillation.

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**Key words:** Atrial fibrillation, Exit block, Atrial tachycardia, Ablation

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### Introduction

Atrial fibrillation (AF) associated with consistent atrial activation sequences after prior ablation has been reported and emanates mostly from localized sources that can be mapped and ablated.<sup>1)</sup> This phenomenon occurs often in patients taking antiarrhythmic drugs but also occurs spontaneously. Spontaneous tachycardia with an exit block is usually transient and originates from the superior vena cava<sup>2-5)</sup> or pulmonary veins (PVs).<sup>2,6)</sup> We present here a patient with spontaneous localized AF at the posterior wall of the left atrium (LA) which was persistent for several years and mimicked an

irregular atrial tachycardia.

### Case Report

A 37-year-old man with drug-resistant persistent “atrial tachycardia” was admitted to our institution for catheter ablation. The tachycardia was first diagnosed 3 years prior to his presentation. He had failed treatment using antiarrhythmics, including amiodarone, and multiple direct-current cardioversions (early recurrences after the cardioversions). The 12-lead ECG exhibited an “atrial tachycardia” with distinctive P waves and an isoelectric baseline between the P waves. The cycle length of the P

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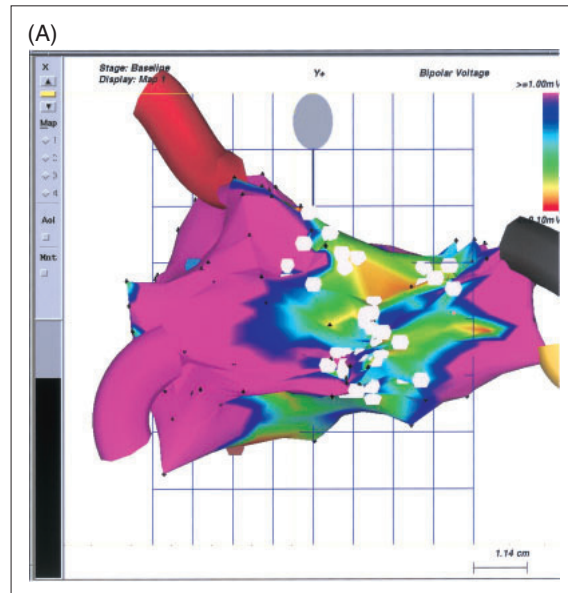
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**Figure 1** Intracardiac recordings during the tachycardia. While the coronary sinus exhibited discrete atrial potentials with irregular intervals, fractionated and rapid deflections with a mean cycle length of 88 msec were recorded from the posterior wall of the left atrium. A = discrete atrial potential; ABL = ablation catheter; CS = coronary sinus.

waves ranged from 320 to 500 msec, and the P wave polarity was positive in leads I, aVL, and V1, and biphasic (initially positive) in leads II, III, aVR, aVF, and V2-6.

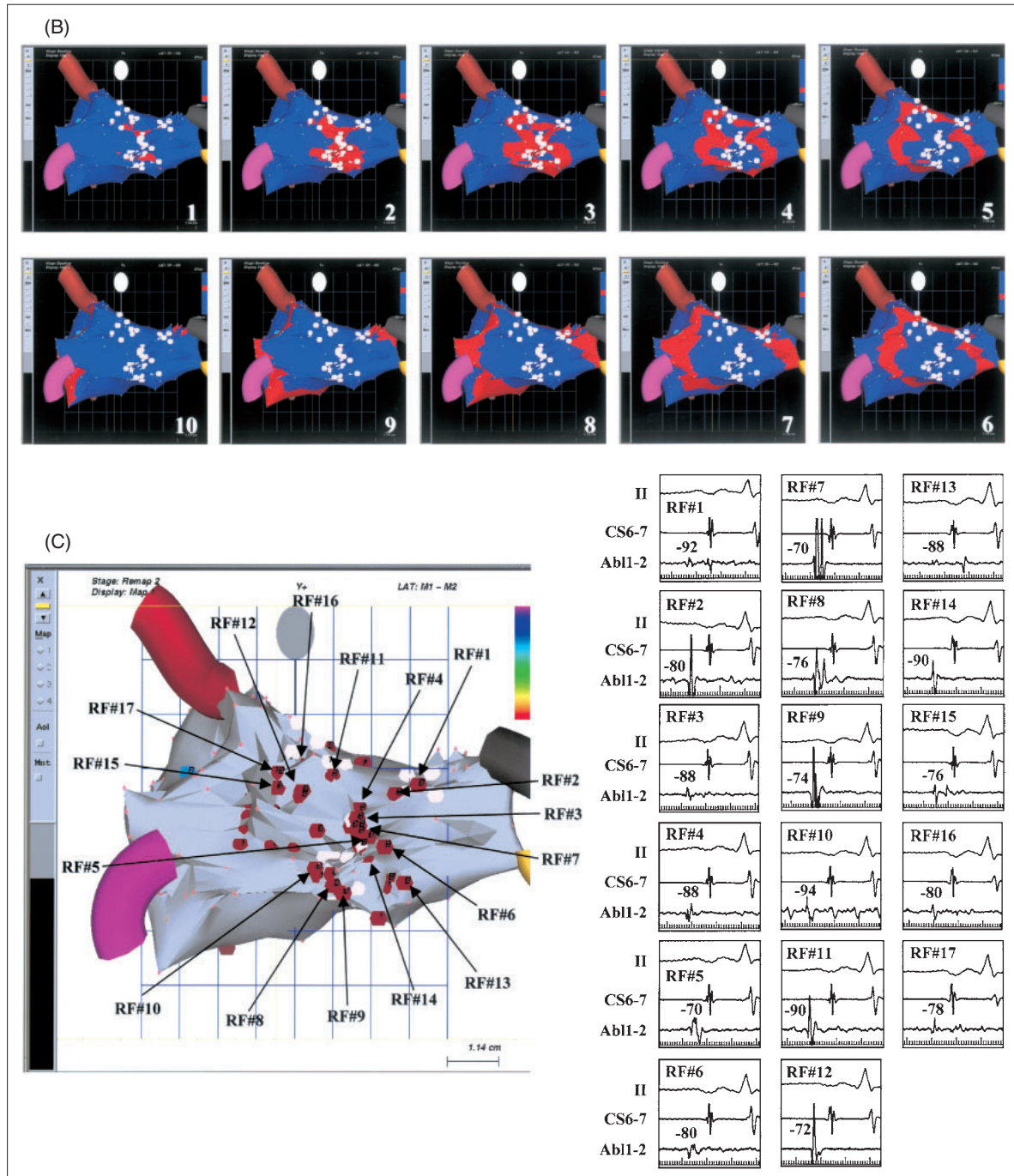
Endocardial mapping of the right and left atria was performed. The right atrium and coronary sinus (CS) exhibited discrete atrial potentials with irregular intervals. No rapid deflections were recorded within either PV. While the right atrium and CS exhibited discrete atrial potentials with irregular intervals, fractionated and rapid deflections with a mean cycle length of 88 msec were recorded from the posterior wall and roof of the LA (**Figure 1**). Electroanatomical mapping during the tachycardia revealed that low-amplitude rapid electrograms were recorded from the posterior wall and roof of the LA (**Figure 2A**). The electrograms recorded from the surrounding tissue had a high amplitude (>1.0 mV) and discrete activity. A large part of the LA other than the fibrillatory area was activated in a centrifugal manner (**Figure 2B**). However, several points around the low-voltage fibrillatory area first activated simultaneously. Radiofrequency (RF) ablation was performed targeting the early atrial activation around the fibrillatory area using a maximum power of 30 W and maximum electrode-tissue interface temperature of 55°C (**Figure 2C**). The RF energy applications were 30 seconds in duration. At the RF #1 site, a spiky pre-potential was recorded and this potential preceded the reference atrial electrogram at



**Figure 2** Electroanatomical mapping during the tachycardia.

(A) Voltage map. Low-amplitude rapid electrograms were recorded from the posterior wall and roof of the left atrium (white dots). The electrograms recorded from the surrounding tissue had a high amplitude (>1.0 mV) and discrete activity. (Continued)

CS6-7 by 92 msec. After RF #1, the pre-potential disappeared and early atrial activation was searched in the adjacent area. During RF #17, the tachycardia



**Figure 2** (Cont'd)

(B) Propagation map. A large part of the left atrium, except for the fibrillatory area, was activated in a centrifugal manner. However, several points around the low-voltage fibrillatory area first activated simultaneously. The electrogram recorded from the CS6-7 was used as a reference. (C) Ablation sites and the intracardiac electrograms. The red dots indicate the ablation sites. The tachycardia was terminated during the radiofrequency energy application at the RF#17 site. CS = coronary sinus; RF = radiofrequency.

slowed and terminated (Figure 3). Figure 4 shows the 12-lead ECGs and the intracardiac electrograms from the ablation sites. After 16 RF energy applications to the sites with the early atrial activation

around the fibrillatory area, the morphologies of the surface P-waves were slightly changed. (Figure 4B) From the RF #17 site, a spiky pre-potential was recorded from electrodes 1–2. There was a signifi-



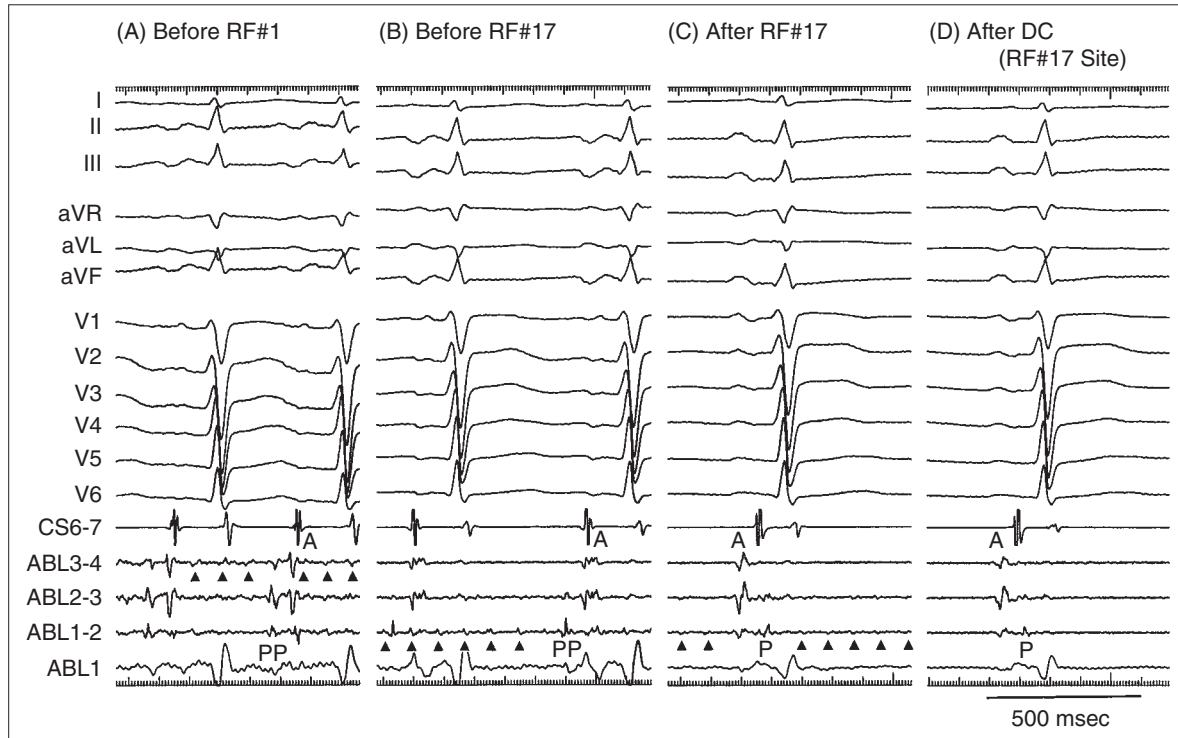
**Figure 3** Termination of the tachycardia during the RF energy application. During RF energy application #17 the tachycardia slowed and terminated.

cant activation gradient between electrodes 1–2 and 3–4. The spiky pre-potentials were followed by atrial potentials with fragmentation. During sinus rhythm, the sequence of atrial electrograms at the ablation site was reversed. The atrial potentials were followed by spiky potentials. While sinus rhythm was obtained after RF applications, low-amplitude fibrillatory electrograms were still observed at the posterior wall (**Figure 4C**). This suggested the complete isolation of the localized AF. To terminate the fibrillatory activity itself, a total of 40 additional RF applications were delivered to the sites with fractionated and rapid deflections. However, we could not terminate the localized AF and direct-current cardioversion was carried out at the end of the session. **Figure 4D** shows the intracardiac electrogram at the RF #17 site after a direct-current cardioversion. Fibrillatory electrograms disappeared and localized AF at the posterior wall was successfully defibrillated. The patient has been maintained in sinus rhythm for 10 months without any antiarrhythmic drugs.

## Discussion

AF associated with consistent atrial activation

sequences after prior ablation has been reported and emanates mostly from localized sources that can be mapped and ablated.<sup>1)</sup> During such organized AF, the ECG exhibits irregular “atrial tachycardia” with discrete monomorphic P waves. Spontaneous tachycardia with an exit block is usually transient, and originates from the superior vena cava<sup>2–5)</sup> or PVs.<sup>2,6)</sup> In the previous case reports, intermittent conduction from the source to the right or left atrium resulted in an irregular monomorphic atrial tachycardia and the mean cycle lengths of the P waves were 240 msec to 300 msec. In the present patient, the tachycardia was observed without any antiarrhythmic drugs and was persistent for several years. The cycle length of the atrial tachycardia ranged from 320 to 500 msec and the isoelectric baselines between the P waves were clearly observed. The source of the AF was the posterior wall of the LA, where fractionated and rapid deflections with a mean cycle length of 88 msec were recorded. During the tachycardia, the surrounding tissue was not activated in a 1:1 fashion but was responsible for the exit block and irregular activation of the rest of the atria. To the best of our knowledge, this kind of spontaneous localized AF in the posterior LA with an exit block has not been



**Figure 4** Twelve-lead ECGs and intracardiac electrograms from the ablation sites. (A) From the RF #1 site, fibrillatory electrograms (arrowheads), a spiky pre-potential (PP), and the following discrete atrial potential were recorded. (B) After 16 RF energy applications to the sites with the early atrial activation around the low-amplitude fibrillatory area, the morphologies of the surface P-waves were slightly changed. From the RF #17 site, a spiky pre-potential was recorded from electrodes 1–2. There was a significant activation gradient between electrodes 1–2 and 3–4. The spiky pre-potentials were followed by atrial potentials with fragmentation. (C) During sinus rhythm after RF #17, the sequence of atrial electrograms at the ablation site was reversed. The atrial potentials were followed by spiky potentials (P). However, low-amplitude fibrillatory electrograms (arrowheads) were still observed. (D) After a direct-current cardioversion at the end of the session, fibrillatory activities disappeared. The ablation catheter was repositioned at RF #17 site. A = atrial potential; PP = pre-potential.

reported. The ablation of a larger extent of the circumference was required to obtain the complete isolation of the localized AF area, suggesting multiple breakthroughs to the surrounding tissue of the LA. From several RF energy application sites including the final isolation site (RF #17), spiky pre-potentials were recorded. After RF applications, these potentials disappeared or occurred after the discrete atrial activation. We hypothesized that these spiky potentials represent the activation of the isthmus at the breakthrough points to the surrounding tissue of the LA. This explains why a spiky potential disappeared or appeared after the atrial potential following RF energy application.

Interestingly, the area with the low-amplitude rapid electrograms in this patient was quite similar to silent areas in macroreentrant left atrial tachycardia. Jais et al. reported that nearly half of all silent areas in the patients with persistent macroreentrant left atrial tachycardia were posteriorly located.<sup>7)</sup> There is a possibility that spontaneous localized AF in the

posterior LA will have a macroreentrant tachycardia when the area with the low-amplitude rapid electrograms becomes silent, because the surrounding tissue also has a property of slow conduction and block.

We conclude that this rare tachycardia is a spontaneous localized AF in the posterior wall of the LA with an exit block. Fibrillatory activation in the posterior wall of the LA can act as a driver as well as an initiator of atrial fibrillation.

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