A case of identifying an epicardial non–pulmonary vein premature atrial complex using intracardiac pace-match scoring

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Introduction

Pulmonary vein isolation is a well-established technique for catheter ablation of atrial fibrillation (AF). However, non–pulmonary vein premature atrial complexes (non-PV PACs) are also triggers of AF, and their ablation can potentially reduce recurrent AF following catheter ablation.[1] Nevertheless, mapping non-PV PACs is inducibility-dependent, and a single ectopic beat can trigger AF, making the identification and treatment of their origin challenging.

In ventricular arrhythmias, the conventional pace-mapping technique and pace-mapping software (PASO Module; Biosense Webster, Diamond Bar, CA) in the CARTO 3 mapping system (Biosense Webster) can display pace-match scores as a correlation map to search for origins.[2] However, identifying non-PV PAC origins with pace mapping of atrial arrhythmias is difficult. Therefore, we introduced a novel atrial pace-mapping technique called "intracardiac pattern-match scoring" (iPASO) mapping, which effectively identifies origins of non-PV PACs using the Intracardiac Pattern-Matching (ICPM; Biosense Webster) module.[3–5] The iPASO technique is particularly useful for identifying origins of single ectopic beat–triggered AF and is objective, reproducible, and simple.

However, there have been no reports on whether the atrial pace-mapping technique can identify epicardial origins. We present a case in which the iPASO technique identified a non-PV PAC with an epicardial origin in the left atrial (LA) roof area.

KEY TEACHING POINTS

- The intracardiac pattern-match scoring technique, using dual-chamber electrograms, effectively identifies the earliest activation site, even with a single ectopic beat.
- Increasing the output from low to high during the pace map with the intracardiac pattern-match scoring technique can identify the epicardial origin of non–pulmonary vein premature atrial complexes.
- Successful ablation of epicardial non–pulmonary vein premature atrial complexes using the intracardiac pattern-match scoring technique offers a reproducible and objective method, potentially improving the treatment outcomes for atrial fibrillation patients.

Case report

The patient was a 70-year-old woman with persistent AF complicated by heart failure. Transthoracic echocardiography revealed a left ventricular ejection fraction of 68% and LA dimension of 36 mm with no valvular or structural heart disease. The first catheter ablation was performed under general anesthesia using a contact force–sensing catheter equipped with microelectrodes (QDOT MICRO Catheter; Biosense Webster) and electroanatomic mapping system (CARTO 3). A 6F, 20-pole electrode catheter (BeeAT; Japan Lifeline, Tokyo, Japan) was inserted through the right jugular vein into the coronary sinus to obtain dual-chamber electrograms (EGMs) from the coronary sinus and right atrium. Upon admission, the rhythm was AF, which reverted to sinus rhythm with external cardioversion after the transeptal puncture.

After circumferential pulmonary vein isolation, frequent monomorphic PACs were induced by means of a continuous

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intravenous infusion of isoproterenol. The P-wave morphology on the 12-lead electrocardiogram suggested an origin near the ostium of the right superior PV, and the earliest activation site (EAS) was recorded in the high right atrium and distal coronary sinus (Figure 1A and 1B). This PAC did not induce AF, but because it was monomorphic and occurred frequently, we decided to estimate its origin and treat it. The intracardiac EGMs of the PAC were saved onto the ICPM module as a reference (Figure 1C). Using a multielectrode catheter (Octaray 3-3-3; Biosense Webster), the PAC was mapped in the LA, and the wavefront annotation indicated the EAS was located at 2 locations on the LA roof (points A and C; Figure 2). However, the ripple map revealed a breakout from point B located between the 2 EASs (Figure 2, Supplemental Video 1). The unipolar waveform of the local potentials revealed an R wave at points A and C, whereas point B displayed a QS pattern, which appeared to represent the breakout point on the ripple map. Subsequently, due to a significant decrease in the PAC frequency, the origin was identified using the iPASO technique. The iPASO mapping was performed as follows:

1. Dual-chamber EGMs obtained from 6 electrodes in the right atrium and 4 electrodes in the coronary sinus during triggered beats were recorded as a reference waveform using the ICPM module.
2. A new local activation map was created with a window of interest from −100 to 0 milliseconds.
3. We conducted detailed pace mapping starting from the vicinity where the reference signal exhibited the earliest onset. Pace mapping was performed at each site using a contact force–sensing catheter with a minimal output (monophasic: 5 V at 0.5 milliseconds) at a pacing cycle length of 600 milliseconds. Moreover, the matching pattern window of interest was adjusted to avoid any stimulus artifact.
4. The weighted average value of the matching score of the paced signals and reference EGMs was automatically calculated using the ICPM software. The pace-match score was displayed between 0 and 1.0, representing the ratio of the match. However, unlike the PASO Module, there was no method to display the score on the 3-dimensional map. To express this as a percentage on the 3-dimensional map, we first multiply it by 100. To designate the highest score as the earliest site, we further multiplied it by −1 millisecond.
5. The annotation timing was manually moved to a value equal to the obtained score regardless of the presence of a potential. By creating a local activation time map from −100 to 0 milliseconds, a matching score of 0.70, for instance, would be displayed as −70 milliseconds with a window of interest from −100 to 0 milliseconds, and a matching score of 0.82 would be displayed as −82 milliseconds. This conversion allowed the display to indicate the points with a higher matching rate as having earlier activation. In summary, the highest score indicated the

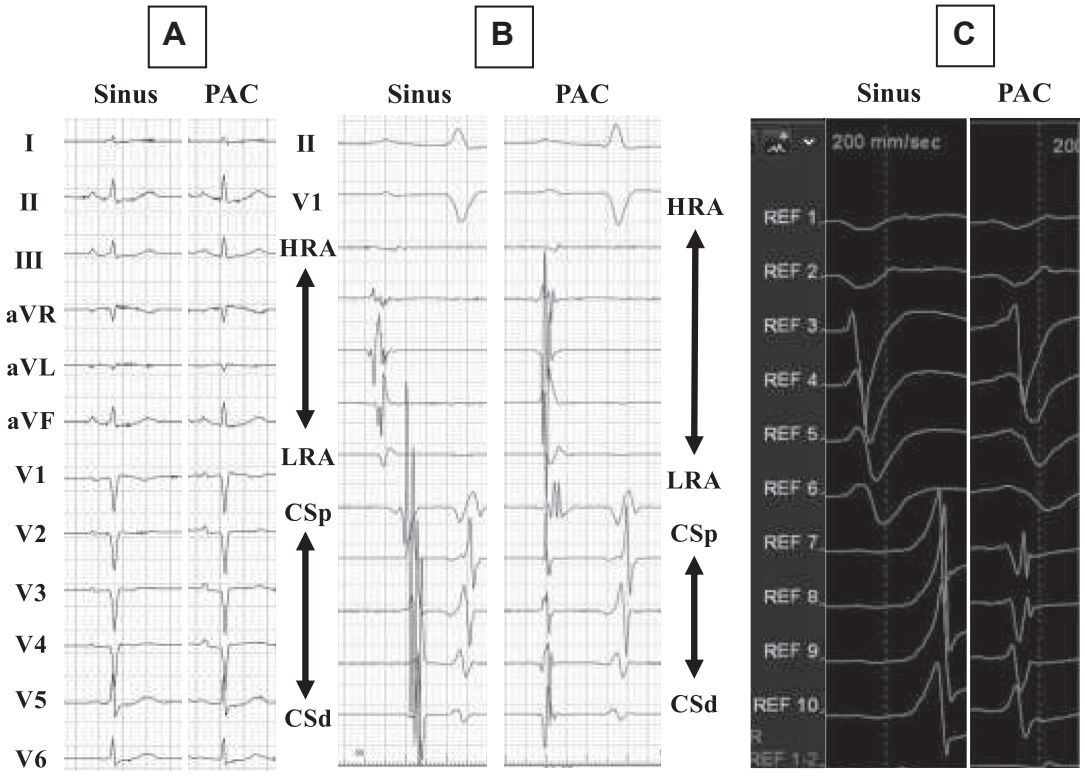

Figure 1 **A:** A 12-lead electrocardiogram showing sinus rhythm and the target premature atrial complex (PAC). **B:** Intracardiac recordings showing sinus rhythm and the target PAC. **C:** Unipolar waveform on the intracardiac pattern-matching module of sinus rhythm and the target PAC recorded from the coronary sinus (CS) and right atrium (RA). d = distal; H = high; L = low; p = proximal.

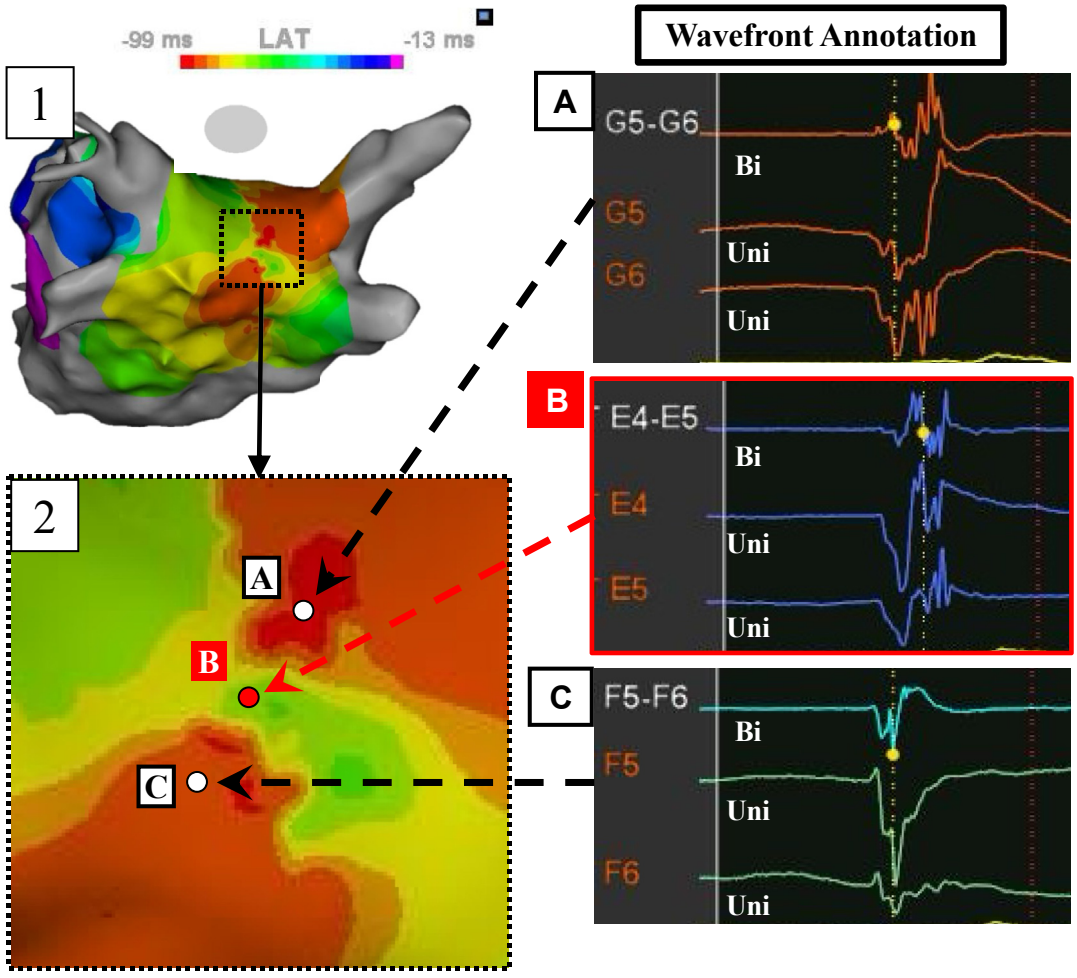

Figure 2 **1:** Local activation time map recorded by the mapping catheter of the target premature atrial complex. **2:** Expanded view around the earliest activation sites. *Points A and C*. The earliest activation sites indicated by the wavefront annotation on the roof of the left atrium. In the unipolar waveform acquired by the mapping catheter, R waves are present. *Point B:* The breakout point determined by the ripple map. In the unipolar waveform, a QS waveform is recorded by the mapping catheter. Bi = bipolar, Uni = unipolar.

EAS. The matching scores at points A and C were 0.50 and 0.37, respectively. The highest-scoring site was point B, the breakout point on the ripple map with a score of 0.71. After pace mapping was performed at 7 points, point B was surrounded by lower points, showing a centrifugal pattern on the iPASO isochronal map (Figure 3). However, point B's score was 0.71, which was not a high matching score, and the ICPM module's unipolar waveform showed the time phase of the coronary sinus potential was slightly ahead. Increasing the pacing output at point B from 5 V at 0.4 milliseconds to 20 V at 0.4 milliseconds raised the score to 0.86 (Supplemental Video 2). Despite the larger pacing spike in the ICPM module, the unipolar waveform remained consistent, and the LA time phase matched better than the low output. Similarly, pacing at a high output at points A and C increased the scores compared with a low output, but the highest scoring point with a high output remained at point B. The increase in the scores with the high output compared with the low-output iPASO technique suggested that the origin of this PAC was on the epicardial side of point B. Radiofrequency applications were delivered at point B with 35 W and a target ablation index of 450. Before delivering the radiofrequency applications, the frequency of the target PAC had already decreased, so its disappearance during the ablation was not evident. However, after the ablation at point B, the target PAC no longer occurred. In addition, point B was located exactly in the middle of the ablation lines for the left and right pulmonary vein isolations. To prevent a roof-dependent atrial tachycardia, we created a roof line. Isoproterenol was administered again, and atrial burst pacing was repeated; however, no atrial tachyarrhythmias were observed. The patient was discharged without complications, and no atrial arrhythmia recurrences were observed during the 6-month follow-up.

Discussion

The iPASO technique, previously reported by Yamashita and colleagues,[3] enables the identification of arrhythmia origins through atrial pace mapping, similar to the PASO Module for ventricular arrhythmias, even when only a single ectopic

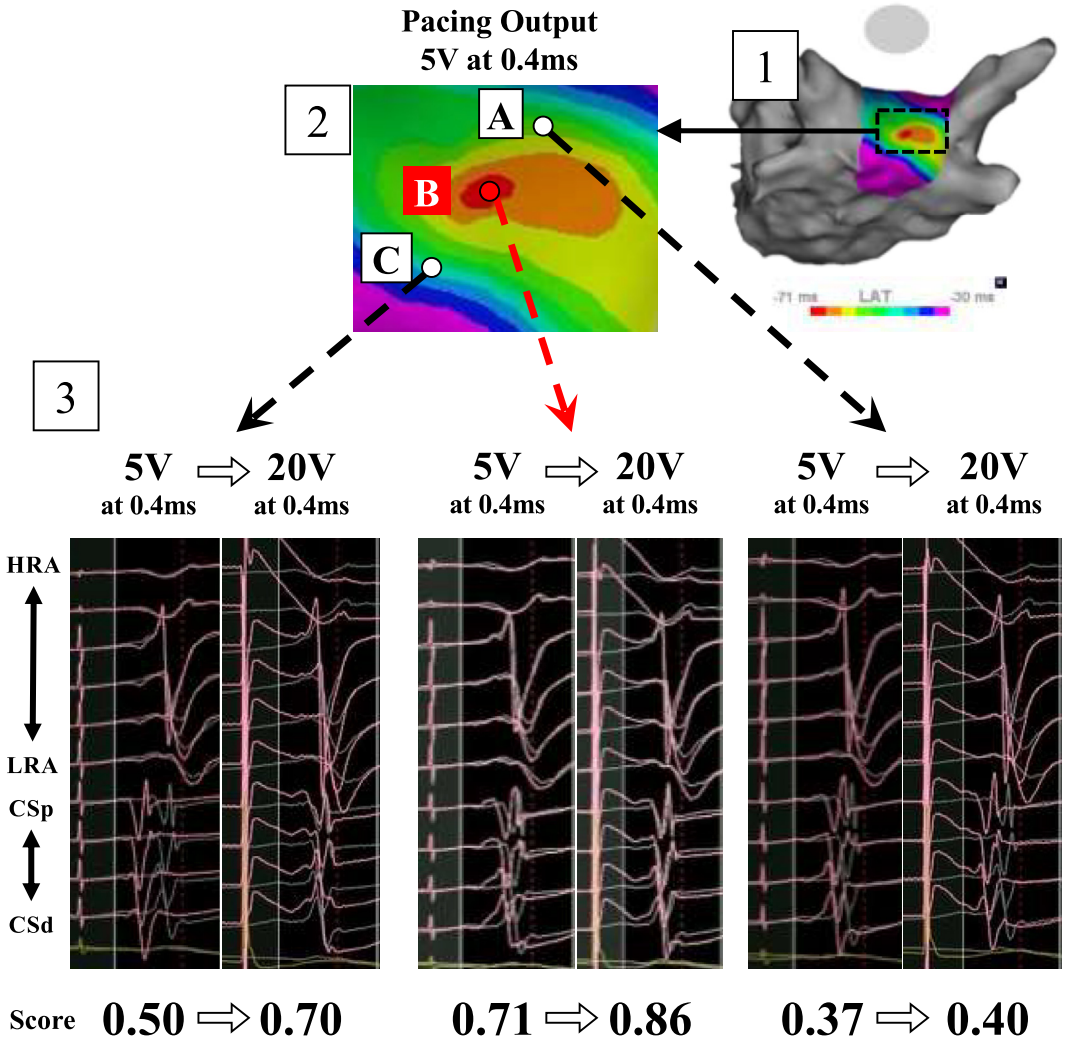

Figure 3 **1:** Intracardiac pace-match scoring (iPASO) map with 5 V at 0.4 milliseconds. **2:** The expanded view around the origin of the target premature atrial complex showing that *point B* is the highest scored site. **3:** Unipolar waveform on the intracardiac pattern-matching module with the iPASO technique. When the pacing output was increased from 5 V to 20 V, the matching score for *point A* increased from 0.5 to 0.70, *point B* from 0.71 to 0.86, and *point C* from 0.37 to 0.40. This led us to conclude that the epicardial side of *point B* was the origin of the target premature atrial complex. CSd = distal coronary sinus; CSp = proximal coronary sinus; HRA = high right atrium.

beat is recorded. This technique holds promise as a new standard treatment option for multiple focal atrial tachycardias and immediate recurrences of AF. In our study, we reported on 26 arrhythmias treated in 22 patients using the iPASO technique. The median of the highest matching score in 22 cases with 26 triggers using iPASO technique was 0.85 (interquartile range, 0.73–0.92). Of these, 84.6% had a highest matching score >0.80. Although the definitive cutoff value for the matching score is still unclear, based on these results, a score of 0.80 is often used as a reference for a high score when using the iPASO technique. In this case, the highest matching score using the iPASO technique at point B with 5 V at 0.4 milliseconds was 0.71, which was <0.80. However, the matching score with 20 V at 0.4 milliseconds was 0.86, which was >0.80. In addition, we also demonstrated the impact of the pacing output on the pace-match score.[4] When the pacing cycle length was set to 600 milliseconds and the pacing output was increased from 5 V to 20 V at 0.4 milliseconds, the matching score using the dual-chamber ICPM decreased from 0.88 to 0.75. This reduction occurs because higher pacing outputs capture a wider area of the myocardium. However, in this case, the matching score at point B increased with higher pacing outputs. That finding suggested that point B may not have been the highest scoring site, but the iPASO map's centrifugal excitation pattern with 5 V at 0.4 milliseconds indicated that, at least on the endocardial side, point B was the target PAC's origin. Furthermore, the ICPM waveform from the pattern bank displayed a clearly larger pacing spike with higher outputs, yet the score increased, and the timing and polarity of the unipolar

waveform better aligned (Figure 3). Those observations, along with the increased scores from low to high outputs at points A and C, implied that the origin of the target PAC was on the epicardial side rather than the endocardial side.

In our iPASO technique, we used an ablation catheter to perform atrial pace mapping. By using a multielectrode catheter, it might be possible to perform pace mapping with a greater density and estimate the origin of non-PV PACs in a shorter time. However, it would be challenging to assess whether there was adequate catheter contact with the myocardium, and the stability of the ablation catheter should be better. Therefore, to identify the origin of non-PV PACs within a narrower range, we used an ablation catheter when applying the iPASO technique.

Furthermore, the roof region is a challenging area for catheter stability and the contact force fluctuates widely due to respirations. In this case, the catheter remained at point B, while increasing the pacing output from 5 V to 20 V at 0.4 milliseconds, and despite the respiratory fluctuations, the contact force remained relatively stable around 15*g*, as shown in Supplemental Video 2. Minimizing respiratory fluctuations and stabilizing the contact force are crucial factors for a successful iPASO technique, and performing the procedure under general anesthesia can help achieve this stability. The impact of contact force on the matching score of the pace map has not been clarified and warrants further investigation. In addition, this roof area is distant from both reference electrodes, the right atrium and coronary sinus. Consequently, the presence of the Bachmann bundle, intra-atrial tract, or fibrotic tissue between the pacing site and these 2 electrodes, which could potentially affect the matching score. To determine the optimal matching score and demonstrate the reproducibility and reliability of the procedure, it should be necessary to accumulate sufficient clinical data.

Although our previous reports have identified PAC origins in the LA roof region,[4] no cases have been identified as originating from the epicardium, as in this instance. To the best of our knowledge, this is the first report where an epicardial PAC was identified using pace mapping from the endocardial side. Although performing pace mapping directly on the epicardium would be the most definitive way to confirm its origin, that approach is impractical. On the mitral annulus, comparing pace maps from the endocardial side and coronary sinus side might provide insight into how the ICPM module waveforms change between the epicardium and endocardium, which warrants further investigation. Although the endocardial origin of the non-PV PACs can be demonstrated using the iPASO map with a centrifugal pattern, as in the present case, when the matching score is relatively low and an epicardial origin is suspected, the iPASO technique with high output pacing may be useful. However, in this case, due to the decrease in the frequency of the target PAC prior to treatment, it cannot be conclusively determined that the PAC was eliminated completely by applications. It is desirable to have a reproducible method for inducing non-PV foci, but because such a method does not exist, this represents a limitation of pace mapping–guided treatment. The iPASO technique we proposed is a novel method for identifying the origins of non-PV PACs, demonstrating superiority regarding objectivity, reproducibility, and simplicity. Although there are still few reports, this technique has the potential to improve the treatment outcomes and prognosis for non-PV PACs with further applications and various studies.

Conclusion

This case report demonstrated the successful identification and treatment of an epicardial PAC using the iPASO technique, marking the first instance of such a finding via endocardial pace mapping. The iPASO technique offers a reliable, reproducible, and straightforward method for identifying non-PV PAC origins. Further studies are needed to explore its broader applications and efficacy in improving the treatment outcomes for non-PV PACs.

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Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2024.09.001

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