

Endocardial and epicardial activation maps for three-dimensional perimitral flutter using a three-dimensional mapping system: a case report

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Background

Perimitral flutter (PMF) is a macro-reentrant tachycardia, and mitral isthmus (MI) linear ablation is considered to be the preferable mode of treatment. Additionally, PMF can sometimes develop via epicardial connections, including coronary sinus and vein of Marshall. However, there are no reports of three-dimensional (3D) atrial tachycardia (AT) via the intramural tissue.

Case summary

A 78-year-old man underwent catheter ablation for paroxysmal atrial fibrillation and AT, including pulmonary vein isolation, left atrial posterior wall isolation, superior vena cava isolation, and MI linear ablation in a total of four procedures. However, AT reoccurred, and he underwent a 5th procedure for AT. Although the MI block line was complete in both the endocardial and epicardial voltage maps, AT indicated PMF. The total activation time did not cover all phases of tachycardia cycle length due to the conduction pathway through the intramural muscle/bundles that could not be mapped with the addition of epicardial mapping. The tachycardia was terminated by ablation at the mitral valve annulus in the 2 o'clock position, where the bundles might have been attached.

Discussion

Both endocardial and epicardial activation maps indicated 3D-PMF, whose circuit included the intramural muscle and bundles in a tachycardia circuit. It is necessary to recognize AT, which is involved via intramural tissues.

Keywords

Case report • Catheter ablation • Mitral isthmus line • Perimitral flutter • Septopulmonary bundle • Vein of Marshall

Learning points

- Some atrial tachycardia via epicardium have a circuit bypassing the intramural bundles that are scar areas on both endocardial and epicardial maps.
- Intramural bundles that cannot be mapped should also be considered; therefore, it is important to create both endocardial and epicardial maps to identify the circuit.

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Introduction

Perimitral flutter (PMF) is a macro-reentrant tachycardia that develops during or after catheter ablation, and mitral isthmus (MI) linear ablation is considered the preferred treatment approach. However, the success rate of achieving transmural lesion and bidirectional conduction blocks in MI is reportedly 76–92%.¹ Both endocardial and epicardial approaches, including the coronary sinus (CS) and vein of Marshall (VOM), may be required for ablation.^{1,2} A previous study reported that 38% of left atrial (LA) tachycardia (AT) cases involved a partial epicardial circuit.³ However, there are no reports regarding a three-dimensional (3D) perspective of AT (3D-AT) conducted via the intramural tissue similar to that in 3D ventricular tachycardia reported by Tung et al.⁴

Timeline

Time	Events
2009, January	Patient developed atrial fibrillation (AF).
2016, February	Patient underwent pulmonary vein (PV) isolation and underwent cavo-tricuspid isthmus ablation.
2017, January	Patient had AF recurrence.
2017, April	Patient underwent 2nd session of AF recurrence. Left atrial posterior wall isolation and superior vena cava isolation were performed. Additionally, left PV reconduction was recorded and was performed left PV re-isolation.
2018, December	Patient had atrial tachycardia (AT) recurrence and underwent 3rd session. Right superior PV reconduction was recorded and was performed right superior PV re-isolation.
2019, November	Patient had AT recurrence with palpitation.
2019, December	Patient underwent 4th session for AT recurrence and mitral isthmus linear ablation was performed.
2020, January	Patient underwent this session for AT recurrence.
2021, April	Patient was followed up, and no arrhythmia was reported.

Case presentation

A 78-year-old man with a history of hypertension and hyperuricaemia underwent catheter ablation to treat paroxysmal atrial fibrillation and AT, including pulmonary vein (PV) isolation, LA posterior wall isolation, and superior vena cava isolation as the 1st, 2nd, and 3rd sessions. One year after the 3rd procedure, the patient developed PMF. Thus, MI linear ablation and CS ablation were performed as the 4th procedure. However, 1 month after the 4th procedure, the patient

complained of palpitations caused by AT, refractory to antiarrhythmic drugs (bepridil and bisoprolol). A 12-lead surface electrocardiogram indicated AT with a cycle length (CL) of 240 ms and positive P waves in the inferior leads (Figure 1). Cardiac ultrasonography showed a LA diameter of 34.1 mm with a left ventricular ejection fraction of 73%. Written informed consent was obtained from the patient, and he underwent the 5th procedure for AT.

We created an endocardial LA voltage and activation map for clinical AT with tachycardia CL (TCL) of 255 ms in an intracardiac electrocardiogram by inserting a 20-electrode mapping catheter (PENTARAY; Biosense Webster, Diamond Bar, CA, USA) and using a 3D mapping system (CARTO3; Biosense Webster). The voltage map indicated isolated PV and the posterior wall. The area around MI, which had been ablated before, was visualized as a wide range of low-voltage zones (LVZs) (scar cut-off value at <0.05 mV) (Figure 2A). The LA activation map identified a centrifugal pattern at the posteroinferior wall. The course of the tachycardia circuit followed a clockwise rotation around the mitral annulus (MA) that reached the basal side of the LA appendage (LAA), similar to PMF. The MI line appeared wholly blocked as the area of the previous MI line extended up to the scar region. Local activation time (LAT), which was 189 ms, did not match the TCL (Figure 2B, Video 1). We speculated that the course of the tachycardia circuit could be through the epicardium and added an epicardial map.

A steerable sheath was inserted via a subxiphoid approach, and epicardial LA activation and voltage maps during PMF were created using a 10-electrode mapping catheter (Deca Nav; Biosense Webster). The epicardial LA voltage map indicated the area around the MI, and the posterior wall was visualized like an LVZ in the endocardial voltage map. The epicardial activation map indicated a centrifugal pattern at the posteroinferior wall, similar to the endocardial activation map (Figure 2C and D). After combining the endocardial and epicardial activation maps, the combined activation map showed PMF, involving clockwise rotation around the MA with a total LAT of 219 ms (Video 2). The MI block line was complete in both the endocardial and epicardial voltage maps. Despite the addition of epicardial mapping, the total activation time did not cover all phases of the TCL.

On the epicardial side, the post-pacing interval (PPI) in the anterior side of the previous MI line and above it were more than the TCL by 20 ms [Figure 2C, yellow point (a)]. The PPI at the posteroinferior side of the previous MI line displayed a centrifugal pattern on the activation map that was lower than the TCL by 20 ms [Figure 2C and D; blue point (b)].

Additionally, we inserted a 1.8-Fr catheter (EP star, Japan Lifeline, Tokyo, Japan) in the VOM to evaluate if it was included in the tachycardia circuit. However, the VOM was short, and the catheter could only be inserted halfway despite best attempts and ethanol injection could not be performed (Figures 3). The PPI at the distal end of the VOM was equal to the TCL [Figure 2C and D; green point (c)]. The PPI at the epicardial centrifugal region was the site where PPI in the VOM was equal to the TCL. Therefore, we ablated the epicardial side of the VOM using a 3.5-mm open-irrigated-tip ablation catheter (ThermoCool SmartTouch® SF; Biosense Webster) (25 W, 30 s) [Figure 2C and D; green point (c)]. However, tachycardia continued despite ablating the endocardial side of the VOM where PPI was less

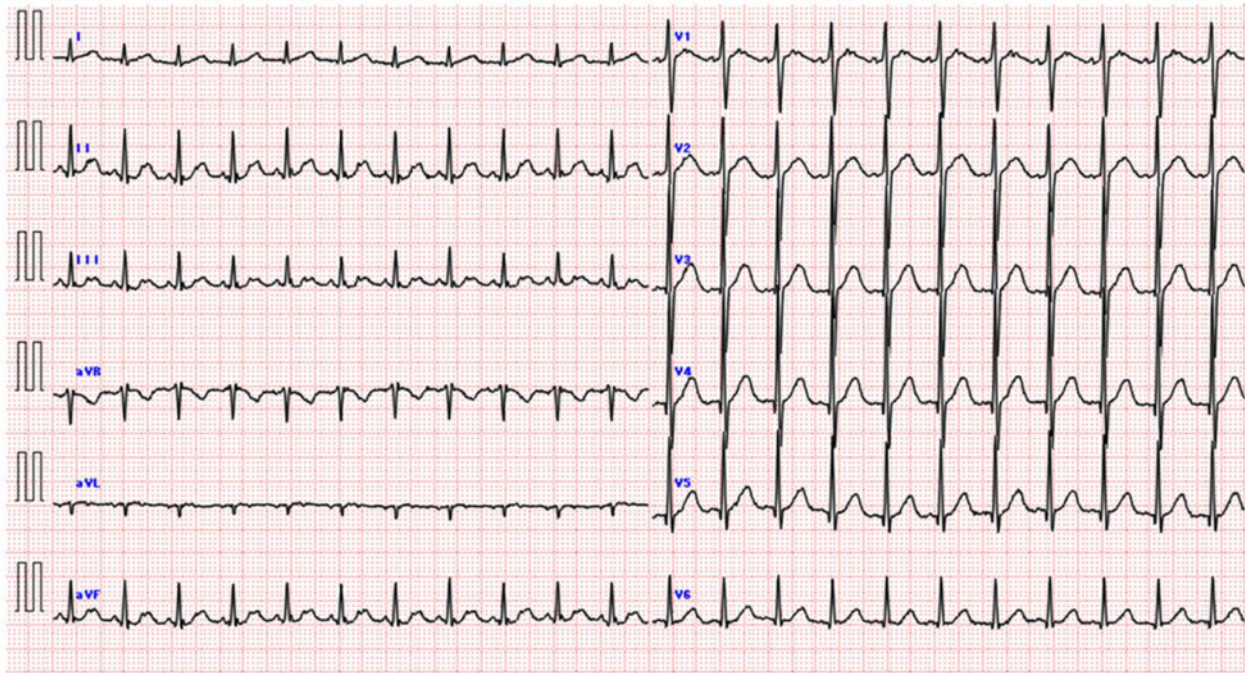


Figure 1 The 12-lead surface electrocardiogram indicates atrial tachycardia with a cycle length of 240 ms and positive P waves in the inferior leads.

than the TCL by 20 ms using the same irrigated ablation catheter (40 W, 30 s) [Figure 2A and B; green point (d)].

On the endocardial side, PPIs in the ridge of the left PV and anterior side of the previous MI line were less than the TCL by 20 ms [Figure 2B; blue points (e)], while the PPI in the posterior side of the previous MI line was more than the TCL by 20 ms [Figure 2A and B; yellow point (f)]. The fragmental potential was found at the base of the LAA, where the PPI was equal to the TCL. Finally, the tachycardia was terminated by ablation of the MA at the 2 o'clock position where the fragmental potential was found (40 W, 30 s) [Figure 2A and B; pink point (g)]. The procedure was terminated after confirming a bidirectional conduction block at the MI through differential sites pacing with ablation catheter in LAA and distal CS and creating activation map during CS ostium pacing. Postoperatively, the patient remained in sinus rhythm during the 12-month follow-up period.

Discussion

PMF cannot be terminated in some cases despite ablating the inner side of the CS and endocardial side of the VOM. In this case, both the endocardial and epicardial maps indicated that the MI block was complete because the MI line was visualized like an LVZ on both maps. We used an epicardial approach and ablated the epicardial side of the VOM where the PPI was less than the TCL by 20 ms. However, tachycardia could not be terminated. Additionally, we could not perform the ethanol injection for VOM because VOM was short. Moreover, both endocardial and epicardial activation maps did not

cover all phases of tachycardia. We speculated that the missing TCL was a conduction path through the intramural tissue.

It has been reported that the heat sinks due to the presence of the CS, coronary artery blood flow, and thickness of the muscular layer influence the factors that make it challenging to create the MI block line.⁵ In this case, the potential remained in the intramural tissue alone in the MI line due to the heat sink. In addition, a study reported that the base of the LAA and ridge are also sites through which multiple bundles pass, such as the VOM and septopulmonary and septoatrial bundles.^{6,7} We believe that these bundles running in the intramural tissue around the LAA might be involved in a part of the tachycardia circuit (Figure 4). Atrial tachycardia via the epicardium has been reported. In the present case, although both endocardial and epicardial MI sites were scarred, MI was considered to be part of the tachycardia circuit. Moreover, both endocardial and epicardial activation maps did not cover all phases of tachycardia. From these findings, when assuming a tachycardia circuit via epicardium in AT, we recommend that both maps are created, and if the epicardial side is scarred, the involvement of intramural bundles in the tachycardia circuit should be suspected. Although the partial epicardial circuit of the AT was identified by both the endocardial and epicardial maps,³ no report has created a map on both sides indicating the involvement of the intramural muscle or bundles in the circuit.

In conclusion, the endocardial and epicardial activation maps delineated the 3D-PMF pathway, including the intramural muscles and bundles, creating a tachycardia circuit. Thus, the circuit of the macro-reentrant AT via MI might include intramural tissues, muscular tissues, and bundles.

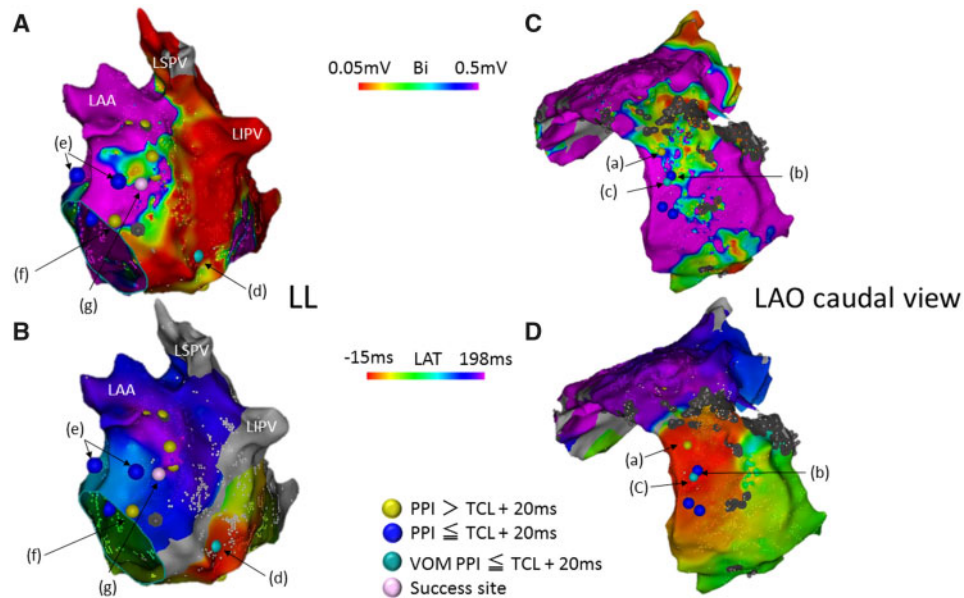
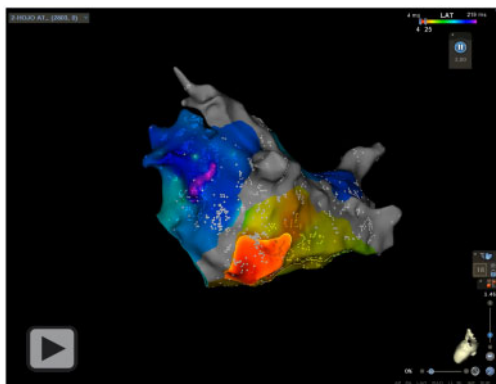
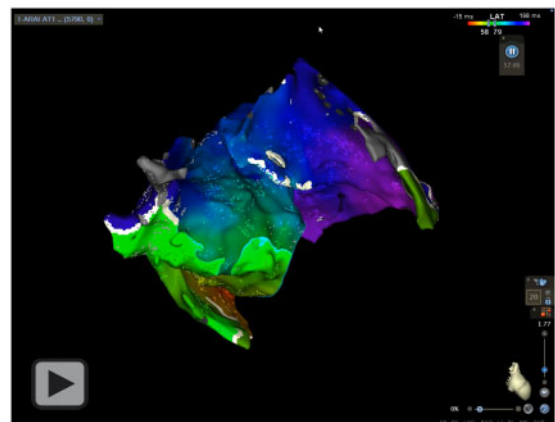


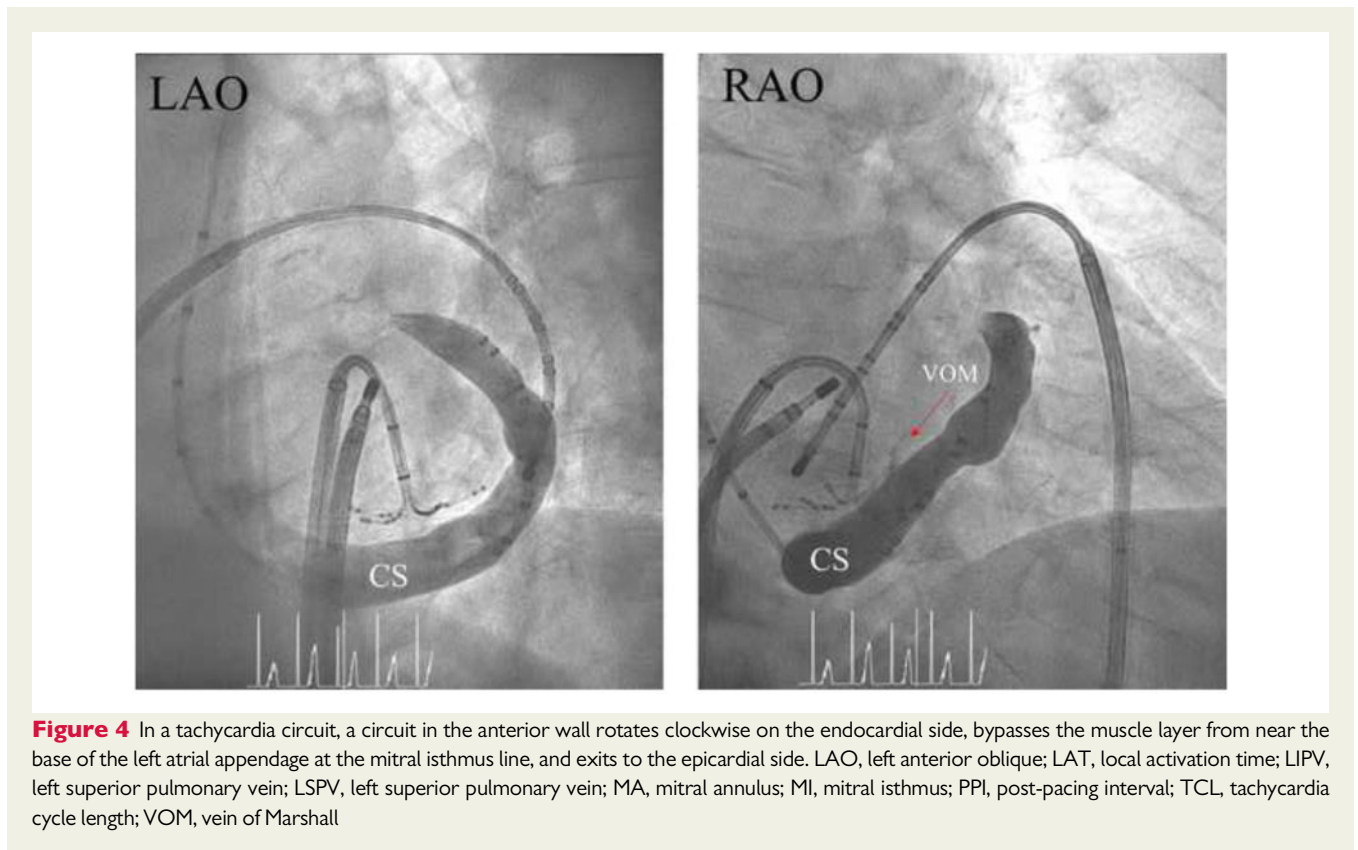
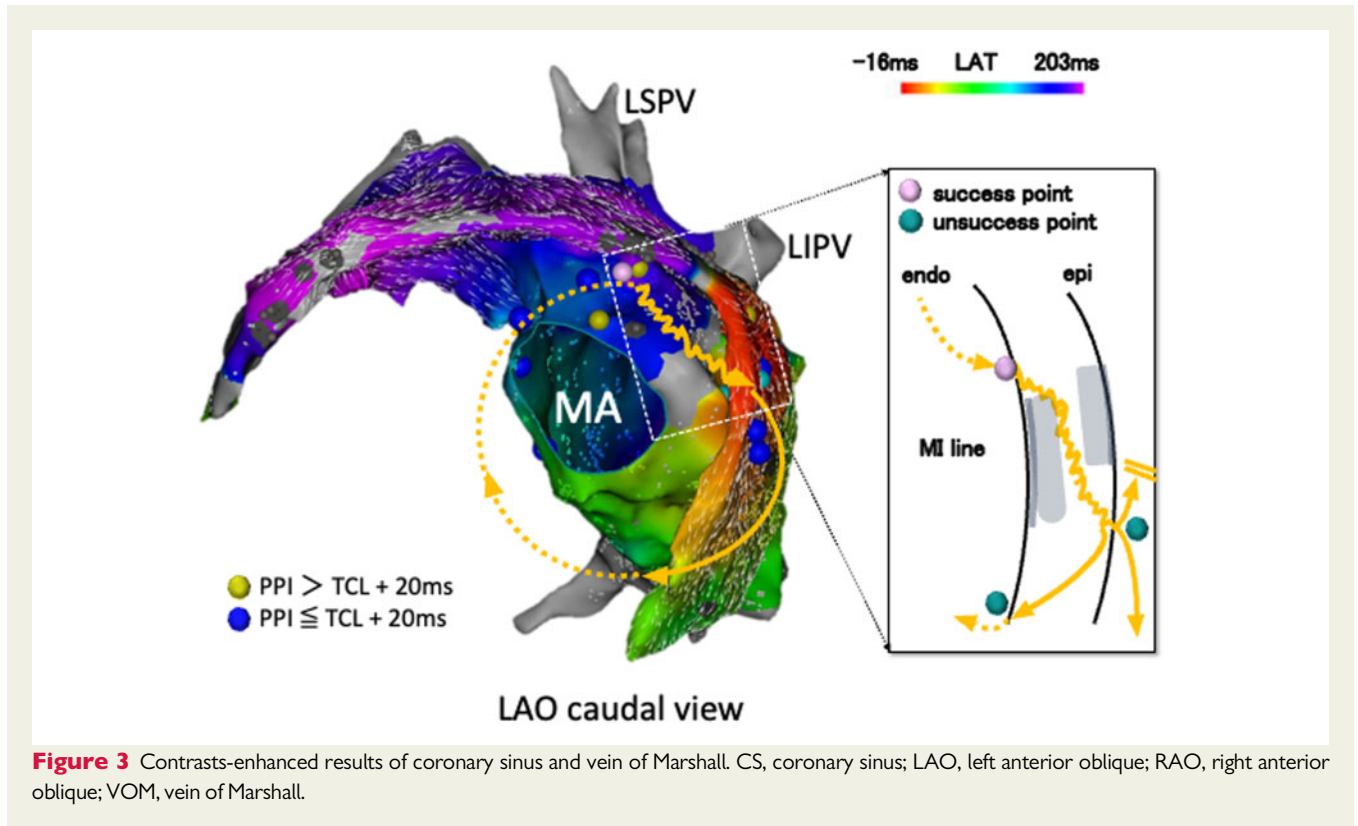
Figure 2 (A) An endocardial voltage map indicates complete pulmonary vein isolation and posterior wall isolation. Moreover, the area around the mitral isthmus ablated earlier was visualized as a wide range of low-voltage zones. The post-pacing intervals that are less than the tachycardia cycle length by 20 ms are shown as blue points, and post-pacing intervals that are more than the tachycardia cycle length by 20 ms are displayed as yellow points. The green points indicate the distal vein of Marshall, where the post-pacing interval was equal to the tachycardia cycle length. Tachycardia was terminated by ablation at the base of the left atrial appendage. (B) The endocardial activation map indicates a centrifugal pattern that is recognized at the posteroinferior wall, and the tachycardia circuit involves a clockwise rotation around the mitral annulus that converges on the base of the left atrial appendage. (C) The epicardial voltage map shows that the area around the mitral isthmus and posterior wall is visualized as a low-voltage zone. (D) The epicardial activation map shows a centrifugal pattern in the posteroinferior wall. (a) On the epicardial side, the post-pacing interval on the anterior side of the previous mitral isthmus line and above it was more than the tachycardia cycle length by 20 ms. (b) The post-pacing interval at the posteroinferior side of the previous mitral isthmus line, which displayed a centrifugal pattern on the epicardial activation map, was 20 ms lower than the tachycardia cycle length. (c) The post-pacing interval at the distal end of the vein of Marshall was equal to the tachycardia cycle length. (d) The endocardial side of the vein of Marshall where post-pacing interval was less than the tachycardia cycle length by 20 ms. (e) On the endocardial side, post-pacing interval in the ridge of the left pulmonary vein and anterior wall side of the previous mitral isthmus line was less than the tachycardia cycle length by 20 ms. (f) The post-pacing interval in the posterior side of the previous mitral isthmus line was more than the tachycardia cycle length by 20 ms. (g) The tachycardia was terminated by ablation of the mitral annulus at the 2 o'clock position. Bi, bipolar; LAA, left atrial appendage; LAO, left anterior oblique; LAT, local activation time; LIPV, left superior pulmonary vein; LL, left lateral view; LSPV, left superior pulmonary vein; PPI, post-pacing interval; TCL, tachycardia cycle length; VOM, vein of Marshall.



Video 1 The left atrial activation map indicates the centrifugal pattern recognized in the posteroinferior wall. The course of the tachycardia circuit involves clockwise rotation around the mitral annulus that converges on the basal portion of the left atrial appendage, similar to perimitral flutter.



Video 2 Both endocardial and epicardial activation maps were combined, and the combined activation map showed perimitral flutter, involving clockwise rotation around the mitral annulus.



Lead author biography



Tomoyuki Arai was born in Tokyo, Japan, in 1989. He received MD degree from Yamagata University School of Medicine, Yamagata, Japan, in 2015 and completed 2 years of Japanese general residency training at National Center for Global Health and Medicine. Since 2017–2020, Dr Arai has worked as an internal medicine resident at Tokyo Metropolitan Hiroo Hospital and now he has been

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Supplementary material

[Supplementary material](#) is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

Consent: The authors confirm that written consent for the submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

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References

1. Fujisawa T, Kimura T, Nakajima K, Nishiyama T, Katsumata Y, Aizawa Y et al. Importance of the vein of Marshall involvement in mitral isthmus ablation. *Pacing Clin Electrophysiol* 2019;**42**:617–624.
2. Hojo R, Fukamizu S, Kitamura T, Maeno K, Hiraoka M, Sakurada H. Pseudo-conduction block at the mitral isthmus in a patient with epicardial impulse propagation through the vein of Marshall. *HeartRhythm Case Rep* 2015;**1**:232–237.
3. Nayak HM, Aziz ZA, Kwasnik A, Lee E, Shatz DY, Tenorio R et al. Indirect and direct evidence for 3-D activation during left atrial flutter: anatomy of epicardial bridging. *JACC Clin Electrophysiol* 2020;**6**:1812–1823.
4. Tung R, Raiman M, Liao H, Zhan X, Chung FP, Nagel R et al. Simultaneous endocardial and epicardial delineation of 3D reentrant ventricular tachycardia. *J Am Coll Cardiol* 2020;**75**:884–897.
5. Hamoud NS, Abrich VA, Shen WK, Mulpuru SK, Srivathsan K. Achieving durable mitral isthmus block: challenges, pitfalls, and methods of assessment. *J Cardiovasc Electrophysiol* 2019;**30**:1679–1687.
6. Cabrera JA, Ho SY, Climent V, Sánchez-Quintana D. The architecture of the left lateral atrial wall: a particular anatomic region with implications for ablation of atrial fibrillation. *Eur Heart J* 2008;**29**:356–362.
7. Pambrun T, Duchateau J, Delgove A, Denis A, Constantin M, Ramirez FD et al. Epicardial course of the septopulmonary bundle: anatomical considerations and clinical implications for roof line completion. *Heart Rhythm* 2021;**18**:349–357.