# Atrial flutter with alternating tachycardia cycle length after atrial fibrillation ablation

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## Introduction

Atrial flutter (AFL) observed in the context of atrial fibrillation (AF) ablation is one of the most important proarrhythmic complications.[1] Previous studies have demonstrated the mechanism and the prevalence of AFL after AF ablation.[2,3] It is well known that gaps in pulmonary vein (PV) isolation or the liner ablation line and the low-voltage area (LVA) cause scars and PV-related AFL.[4,5] In addition, recent studies have also implicated the epicardial Marshall bundle as a substrate of AFL.[6]

Regular tachycardia cycle length (TCL) has always been considered as a regular characteristic of AFL. We herein present an unusual case of AFL with alternating TCL.

## Case report

A 77-year-old woman was diagnosed with persistent atrial tachycardia (AT) and frequent episodes of palpitation and dyspnea. The patient had undergone PV and superior vena cava isolation; liner ablations including the roof, bottom, mitral isthmus (MI), and cavotricuspid isthmus line; and left atrial (LA) anterior LVA ablation for persistent AF in 2 ablation procedures 6 years ago.

At the time of her third admission, 12-lead electrocardiogram showed AT with 2:1 conduction (Figure 1C). A transthoracic echocardiogram showed a normal left ventricular ejection fraction of 72.5% and an LA diameter of 38 mm. The patient was administered an oral anticoagulant drug as well as a beta blocker and a calcium channel blocker as antiarrhythmic drugs. The oral anticoagulant drug was skipped once and the antiarrhythmic drugs were discontinued for at least 5 half-lives prior to the ablation.

During the procedure, 2 mapping catheters with 10 electrodes were placed in the coronary sinus (CS) and around the tricuspid annulus (Figure 1A). The sequence of the electrogram in both catheters showed the proximal-to-distal sequence, and the TCL was 262 ms (Figure 1E). An Advisor HD Grid mapping catheter (Abbott, St Paul, MN) and the EnSite Precision 2.2 software (Abbott) were used to create voltage and activation maps during the AT. The voltage map during the AT revealed scarred area in the LA anterior and roof area, with an LVA around the scarred area (Figure 1B). All PVs were isolated, but the posterior wall was not isolated. The activation map showed a counterclockwise propagation of reentry in the LA anterior area (Figure 1D). In addition, the postpacing interval (PPI) measured at the LA anterior area was 265 ms (Figure 1E and F). The patient was definitively diagnosed with AFL. The PPI from the distal CS site was 372 ms.

### KEY TEACHING POINTS

- Atrial flutter (AFL) observed in the context of atrial fibrillation ablation has many variations, such as perimitral flutter, pulmonary vein gap reentry, and Marshall bundle reentry. Basically, the tachycardia cycle length (TCL) of AFL is regular.
- AFL with alternating TCL are thought to have dual-loop reentry circuits. The wavefront alternately conducts along endocardial and epicardial pathways.
- The entrainment pacing maneuver does not effectively identify details of AFL with alternating TCL because of its irregularity. Arranging the timing windows in a 3D mapping system according to each TCL is needed to determine the reentry form.

**KEYWORDS** Atrial flutter; Atrial tachycardia; Atrial fibrillation; Alternating tachycardia cycle length; Dual-loop reentry
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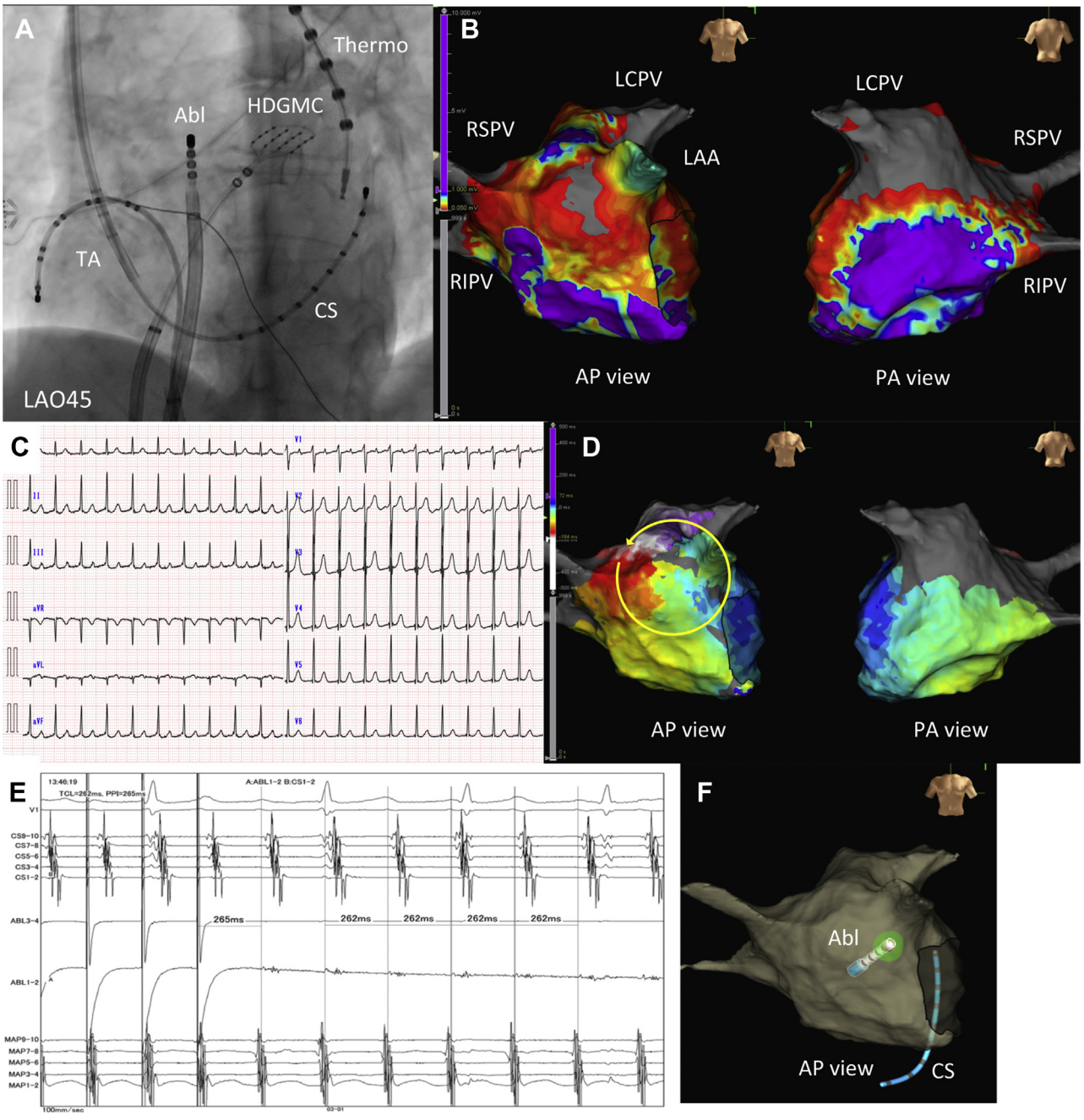


**Figure 1** **A:** Cine angiography shows the ablation catheter (Abl); the mapping catheters, including the Advisor HD Grid mapping catheter (HDGMC; Abbott, St Paul, MN); and thermometer (Thermo). The 2 mapping catheters with 10 electrodes are placed in the coronary sinus (CS) and around the tricuspid annulus (TA). LAO = left anterior oblique. **B:** The bipolar voltage map obtained during the initial atrial flutter (AFL) is shown. The low-voltage and scarred areas are defined as 0.05−1.0 mV and <0.05 mV, respectively. The scarred area is observed in the left atrial (LA) anterior and roof areas, and the low-voltage areas around the scarred area are identified in the LA anterior, anteroseptal, and lateral areas. All pulmonary veins are completely isolated. The mitral isthmus area shows low voltage, and the posterior wall is not isolated. AP = anteroposterior; LAA = left atrium appendage; LCPV = left common pulmonary vein; PA = posteroanterior; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein. **C:** The 12-lead electrocardiogram of the initial AFL shows 2:1 atrioventricular conduction. The P waves in leads II, III, aVF, and $V_1$ show positive polarity. **D:** The activation map of initial AFL shows a counterclockwise propagation around the scarred area. The timing window is set from -184 ms to 72 ms. **E, F:** The electrogram of the pacing response from the ablation catheter shows that the postpacing interval is 265 ms, which corresponds to the atrial tachycardia cycle length. The pacing site is marked by the pink tag.

Radiofrequency (RF) pulses (30 s, 30 W) were delivered to the LA anterior area in which the PPIs corresponded to the TCL (Figure 1F). During the RF applications, the TCL was prolonged without termination and alternated between 271 and 379 ms (Figure 2C and D). The alternating TCL was stable and sustainable, and continued for 90 minutes until termination by RF pulses. Despite the alternating atrial TCL, the R-R interval in the 12-lead electrocardiogram was regular (Figure 2A).

The new activation map showed that the wavefront turned downwards in the LA anteroseptal area from the LA roof

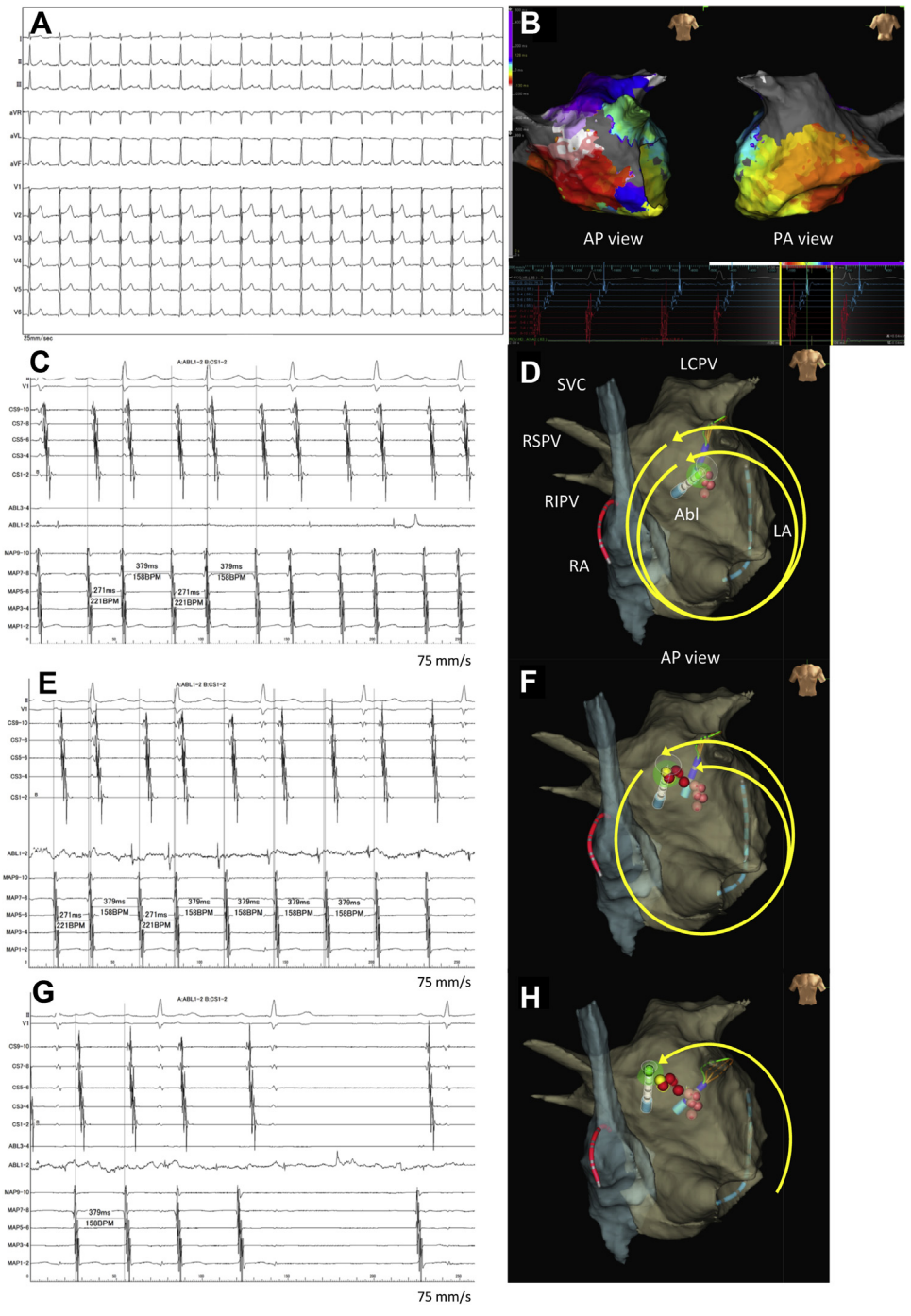


**Figure 2** **A:** The 12-lead electrocardiogram of atrial flutter (AFL) with alternating tachycardia cycle length (TCL) shows positive polarity in leads II, III, and aVF and positive-negative polarity in lead $V_1$. Despite the irregular atrial TCL, the R-R interval is regular. **B:** The activation map shows counterclockwise rotation in the left atrium. The anterior area appears to be obstacles in the tachycardia. The timing window (*yellow line*) is set at 256 ms (−130 to 126 ms). AP = anteroposterior; PA = posteroanterior. **C:** The electrogram shows that TCL alternates between 271 and 379 ms. In both mapping catheters with 10 electrodes, the activation sequence shows proximal-to-distal conduction. **D:** The 3D map shows both the atriums and 2 mapping catheters placed in the coronary sinus and around the tricuspid annulus. Pink tags indicate the ablation sites for the initial AFL. Yellow arrows indicate the dual-loop reentry circuits. Abl = ablation catheter; LA = left atrium; LCPV = left common pulmonary vein; RA = right atrium; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein; SVC = superior vena cava. **E, F:** The electrogram shows the conversion to regular AFL. AFL with the short TCL terminates during the radiofrequency (RF) application for the yellow tag; thus, AFL with the long TCL remains. **G, H:** The electrogram shows the termination of AFL with the long TCL during RF application for the green tag.

(Figure 2B). Subsequently, the wavefront turned around the LA posterior area and returned to the LA roof through the LA ridge. The tachycardia was considered to reflect reentry, but the pacing maneuver to measure PPI − TCL was not clearly detailed because the atrial TCL was not regular. We tried to insert a 2F electrode guidewire (EP Starfix; Japan

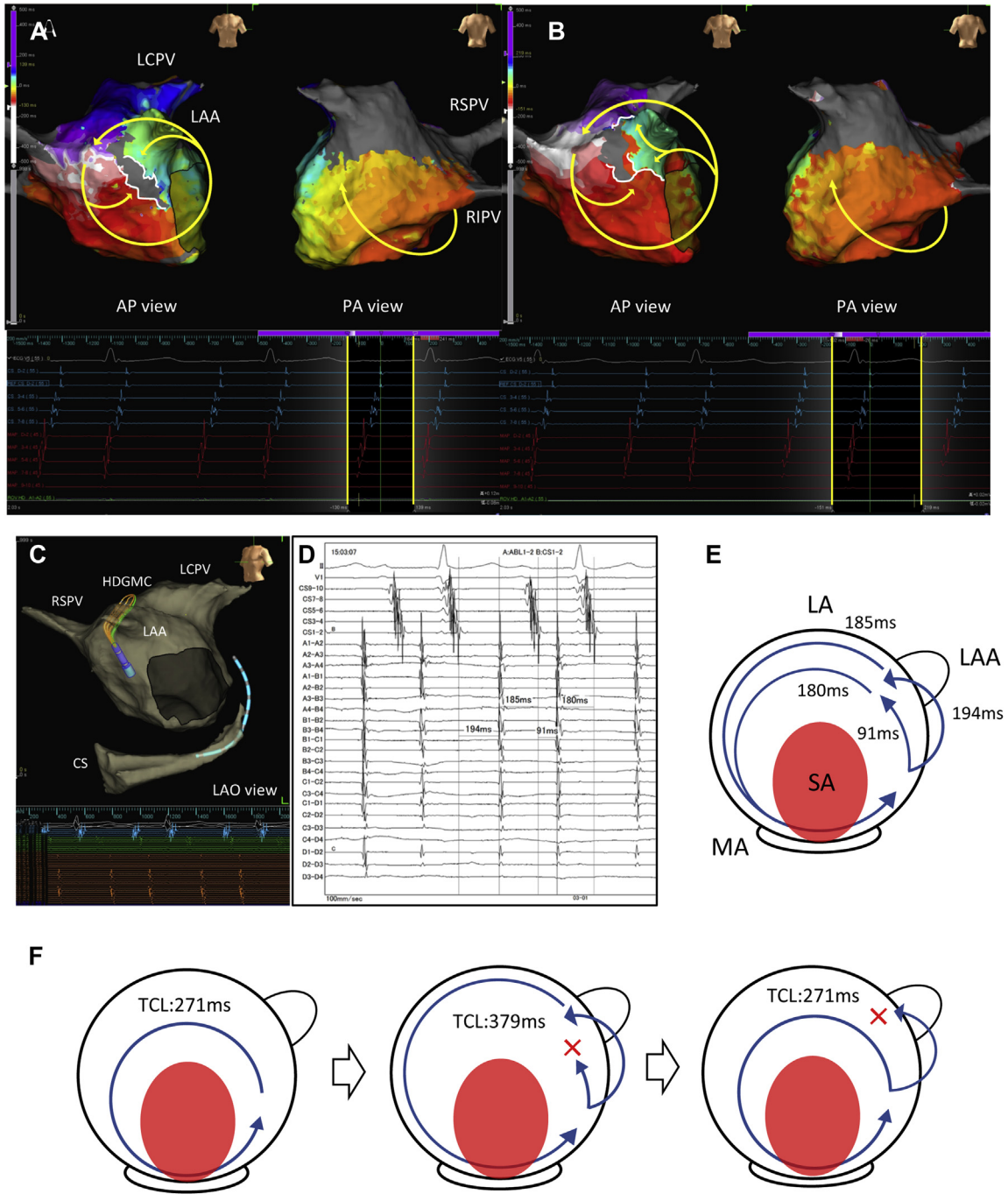


**Figure 3** **A:** The activation map is reanalyzed after the procedure and the timing window (*yellow line*) is arranged to cover the short tachycardia cycle length (TCL) and set at 269 ms (−130 to 139 ms) The map shows counterclockwise rotation (*yellow arrow*). The wavefront turns downward in the left atrial (LA) anteroseptal area, is blocked in the anterior area (*white line*), and turns around the mitral annulus through the LA posterior area. The wavefront that has turned back to the anterior area is also blocked (*white line*) and goes up to the roof area through the LA ridge. AP = anteroposterior; LAA = left atrium appendage; LCPV = left common pulmonary vein; PA = posteroanterior; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein. **B:** The activation map is reanalyzed and the timing window (*yellow line*) is arranged to cover the long TCL and set at 370 ms (−151 to 219 ms). The map shows counterclockwise rotation (*yellow arrow*) as well as the atrial flutter (AFL) with the short TCL, but the wavefront going upward is blocked at the LA ridge (*white line*) and jumps to the roof line. The map is devoid of the blue-colored area. **C, D:** The Advisor HD Grid mapping catheter (HDGMC; Abbott, St Paul, MN) and the mapping catheter with 10 electrodes are placed in the roof area and coronary sinus (CS), respectively. The electrogram shows that the conduction times from the CS distal site to the HDGMC during the AFLs with the short and long TCLs are 91 and 194 ms, respectively. The conduction times from the HDGMC to the CS distal site during the AFLs with the short and long TCLs are 180 and 185 ms, respectively. LAO = left anterior oblique. **E:** The schema demonstrates the dual-loop reentry circuits and each conduction time. LA = left atrium; MA = mitral annulus; SA = scarred area. **F:** The mechanism by which the wavefront alternately turns around dual-loops is considered to involve the following: The wavefront turns around in the LA once, and has a TCL of 271 ms. It is blocked at the LA ridge because of its long refractory period. The wavefront conducts to the LA roof through the epicardial connection and turns around at the LA. The TCL is 379 ms. The wavefront that turns back to the CS distal site reflects endocardial and epicardial conduction pathways. However, the epicardial conduction is blocked at the roof area because of the refractory period by the endocardial conduction.

Lifeline, Tokyo, Japan) to the Marshall vein to obtain epicardial electrical information. However, the crooked shape of the Marshall vein made this maneuver impossible.

The new map also showed that the area between the LA anterior wall and roof line was the critical isthmus of reentry and that the MI block line was still incomplete. We chose to ablate the critical isthmus area first because it was possible that the reentry circuit included the epicardial conduction pathway and that MI ablation may not effectively terminate the tachycardia.

RF pulses were delivered carefully point by point from the LA anterior area to the roof line. During the RF applications, the alternating TCL changed to a regular TCL of 379 ms (Figure 2E and F). Moreover, after 1 more RF pulse, the tachycardia was terminated completely (Figure 2G and H). Finally, no ATs/AFLs were induced by burst pacing with isoproterenol administration. The potentials of the LA appendage (LAA) remained and were not delayed compared to the start of the procedure.

After the procedure, we reanalyzed the electrical information in activation maps during the tachycardia. The timing window was arranged to cover each of the TCLs, and new activation maps, which were created for each TCL, clearly showed both reentry propagations. In the case of short-TCL (271 ms) AFL, the wavefront that turned downwards in the LA anteroseptal area was blocked at the anterior area, but turned around at the mitral annulus through the posterior area and went up to the LA roof through the LA ridge (Figure 3A). The long-TCL (379 ms) AFL was nearly identical to the short-TCL AFL, but the wavefront was blocked at the LA ridge and jumped to the LA roof (Figure 3B).

An external loop recorder that was performed for 7 days 3 months after the procedure did not show AF or AT/AFL recurrence.

## Discussion

In this case, it was very difficult to determine the tachycardia form because of its eccentric TCL. Even though the activation map showed that the tachycardia was AFL with alternating TCL, the mechanism of the alternating TCL remained unidentified until the end of the session. In addition, we emphasize that the entrainment pacing maneuver was not available because the atrial TCL was not regular. We had to choose the ablation site without information obtained from the pacing response.

We first considered that the reentry circuit possibly included an epicardial conduction pathway because of the long interval of 379 ms. We estimated that MI ablation was ineffective in terminating the AFL. Moreover, we considered that we could not ablate the critical isthmus area after completing the MI block line because it would be risky to isolate the LAA. Therefore, we chose to initially ablate the critical isthmus area. We refrained from ablating the LA ridge because of the risk involved in isolating the LAA.

First, the RF pulses delivered to the critical isthmus area changed the alternating TCL to a regular TCL (Figure 2E and F); the subsequent RF pulses terminated the regular tachycardia (Figure 2G and H). These responses suggested the presence of 2 reentry circuits (Figure 2D). We estimated that the series of RF pulses terminated dual-loop reentry circuits one by one.

The activation maps recreated after the procedure revealed that all reentry circuits of the short-TCL (271 ms) AFL were in the endocardium (Figure 3A). However, in the long-TCL (379 ms) AFL, the wavefront was blocked at the LA ridge and jumped to the roof region. The lack of blue-colored area in the map indicated that part of the reentry circuit was in the epicardium (Figure 3B). These findings also suggested that the AFL with alternating TCL had dual loops and that the wavefront turned around both the reentry circuits.

The potentials of the HD Grid mapping catheter placed at the LA roof during the AFL revealed that the difference in length between the 2 TCLs depended on the conduction time from the CS to the LA roof (Figure 3C–E). Therefore, we concluded that the Marshall bundle was the epicardial conduction pathway and that the AFL with alternating TCL included Marshall bundle reentry.

The mechanism underlying the alternating turn-arounds in both reentry circuits is considered to involve the following (Figure 3F): (1) The wavefront turns around once in the LA endocardium. (2) The refractory period of the LA ridge is longer than the short TCL (271 ms), so the wavefront is blocked at that location. (3) The wavefront conducts to the roof area through the epicardial conduction pathway and turns around in the LA endocardium again. (4) The wavefront turning back to the CS reflects both conduction pathways, but the epicardial conduction is blocked at the LA roof by the refractory period of the endocardial conduction.

Alternating conduction of the wavefront by 2 conduction pathways is very rare. We are not aware of another report of AFL with alternating TCL.

## Conclusion

We experienced a patient who exhibited AFL with endocardial and epicardial conduction pathways that caused alternating TCL.

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