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ORIGINAL RESEARCH

High-Resolution Mapping and Ablation of Atrial Tachycardias Involving the Lateral Left Atrium

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BACKGROUND: The lateral left atrium (LA) is often associated with atrial tachycardia (AT) because of its complex anatomy. We sought to characterize ATs associated with the lateral LA, including the posterolateral mitral isthmus (MI) and left atrial ridge.

METHODS AND RESULTS: Twenty-eight lateral LA-associated ATs were mapped with high-resolution mapping systems and entrainment pacing. The vein of Marshall was mapped with a 1.8-Fr mapping catheter when possible. ATs were associated with the posterolateral MI in 18 ATs (14 perimitral, 3 small reentry, and 1 focal AT). All patients had undergone MI area ablation, and all ATs were successfully eliminated. During 27.0 (interquartile range, 10.5–40.0) months of follow-up, all were free from any atrial tachyarrhythmias, with 3 patients on antiarrhythmics. Of 10 ATs involving the ridge or Marshall bundle, 3 were ridge related, 3 were Marshall bundle related based on vein of Marshall mapping, and 1 was a persistent left superior vena cava related AT. All 7 patients had undergone MI linear ablation. The critical isthmus was in the LA-ridge junction or the LA-Marshall bundle junction. Bidirectional conduction block between the LA and ridge or Marshall bundle was created. Two patients had the critical isthmus in the other area. The remaining patient had micro-reentry in the ridge. All 10 ATs were terminated during ablation at the critical isthmus. During 12.0 (5.2–31.7) months of follow-up, all were free from any atrial tachyarrhythmias, with 7 patients on antiarrhythmics.

CONCLUSIONS: Most ATs occurred after MI area ablation. An high resolution mapping-guided approach is highly effective for identifying the mechanism.

Key Words: atrial tachycardia ■ high-resolution mapping ■ Marshall bundle ■ perimitral atrial tachycardia ■ ridge

Catheter ablation of atrial fibrillation (AF) is an established treatment strategy. Atrial tachycardias (ATs) are often encountered in the context of AF ablation.[1] Catheter ablation is a reasonable therapeutic option for such ATs because they are difficult to manage with medical treatment. The mechanisms causing ATs are wide ranging. Although an understanding of the mechanisms underlying ATs is essential to successfully eliminating them, point-by-point mapping using a conventional 3-dimensional mapping system has limited the resolution of maps for clarifying the exact reentrant circuit.[2] The origin and substrate of ATs could be distributed in anywhere in either atrium. The lateral left atrium (LA), including the posterolateral mitral isthmus (MI) and the left atrial ridge between the left atrial appendage and left pulmonary veins (PVs), is a site that is frequently involved in ATs. This is presumably because (1) the area consists of complex anatomical structures, thick myocardial tissue, and epicardial fibers,[3] (2) ablation of the left atrial ridge from the PV side is always required for PV isolation, and (3) the posterolateral MI is often targeted for substrate modification in AF ablation.[4] Indeed, recent studies showed that ethanol infusion in the vein of Marshall (VOM), which could ablate this area, had favorable outcomes for rhythm control in AF ablation.[5] The recently introduced high-resolution

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CLINICAL PERSPECTIVE

What Is New?

- The vast majority of atrial tachycardias (ATs) involving the lateral left atrium occurred after previous mitral isthmus area ablation.
- Creating conduction block between the left atrium and Marshall bundle or left atrial ridge at the junction was an optimal procedural end point for typical Marshall bundle- or left atrial ridge–related ATs.
- The posterolateral mitral isthmus was involved in perimitral AT as well as small reentrant and focal ATs.

What Are the Clinical Implications?

- High-resolution mapping systems are highly useful for identifying the critical isthmus of ATs.
- Identification of the critical isthmus leads to the individual optimal ablation strategy, which results in a high success rate.

Nonstandard Abbreviations and Acronyms

AADs	antiarrhythmic drugs
ATs	atrial tachycardias
CS	coronary sinus
MB	Marshall bundle
MI	mitral isthmus
PLSVC	persistent left superior vena cava
PV	pulmonary vein
TCL	tachycardia cycle length
VOM	vein of Marshall

mapping systems using multipolar mapping catheters and algorithms[6,7] and specific catheters for VOM mapping enabled clarification of the detailed tachycardia circuit. The aim of this study was to characterize ATs in which the lateral LA was involved using currently available high-resolution mapping systems.

METHODS

Study Population

This study consisted of 28 patients who underwent mapping and ablation of ATs between November 2016 and March 2021 in which the lateral LA, including the posterolateral MI and the left atrial ridge between the left atrial appendage and left PVs, was involved in the mechanism according to high-resolution mapping systems. Patients who had a history of cardiac surgery were excluded. The study protocol was approved by the hospital's institutional review board. All patients gave their written informed consent. The study complied with the Helsinki Declaration as revised in 2013. The data that support the findings of this study are available from the corresponding author upon reasonable request.

Electrophysiological Study

All procedures were performed with an uninterrupted systemic anticoagulation regimen using heparin (target activated clotting time >300 seconds). Transesophageal echocardiography was performed before the procedure to exclude any atrial thrombi. The surface electrocardiogram (ECG) and bipolar intracardiac electrograms were continuously monitored and stored on a computer-based digital recording system (EP-WorkMate; Abbott, Chicago, IL, USA). Bipolar intracardiac electrograms were filtered at 30 to 500 Hz.

The procedure was performed without sedation or with minimal sedation to avoid reducing AT inducibility. A 7-Fr catheter with 20 poles for 3-site mapping (BeeAT; Japan Lifeline, Tokyo, Japan) was inserted into the coronary sinus (CS) through the right jugular vein for pacing, recording, and internal cardioversion. In some patients, CS venography was performed to identify the VOM. In patients with a visible VOM, a 1.8-Fr hexapolar catheter (EP star; Japan Lifeline) was inserted into the VOM as distally as possible via the inner lumen of a decapolar mapping catheter (EP star; Japan Lifeline) from the right jugular vein in exchange for the 7-Fr catheter with 20 poles for 3-site mapping. A single transseptal puncture was performed using a radiofrequency needle (Baylis Medical, Montreal, Canada) and a long sheath (SL-0; Abbott) to map the LA.

High-Resolution Activation Mapping and Entrainment Mapping

Detailed high-resolution maps of the ATs were created using (1) a 64-minielectrode basket catheter (Rhythmia, Lumipoint module; Boston Scientific, Natick, MA, USA)[6] or (2) a multielectrode mapping catheter (PentaRay, Coherent module, CARTO 3; Biosense Webster, Irvine, CA, USA).[7] The detailed settings of the systems are described elsewhere.[6,7] Both systems allow for automatic annotation of local activation time with high accuracy. The wavefront propagation was visualized by advancing a 10-ms window of activation along the timescale.

Entrainment mapping was performed to confirm the exact active reentrant circuit unless there were unstable ATs or ATs with a short tachycardia cycle length (TCL) (<200 ms). Sites were considered within or outside the active circuit if the difference between the postpacing interval and TCL was <20 or >40 ms, respectively. Low voltage area, scar, and dense scar

were defined as areas with bipolar cutoffs of 0.5, 0.1, and 0.03 mV, respectively.

Definition of ATs

We classified ATs involving the left lateral LA into 2 groups: (1) ATs in which the critical isthmus or the origin of the tachycardia was located in the posterolateral MI and (2) ATs in which the isthmus was not located in the posterolateral MI and the ridge or Marshall bundle (MB) was involved in the tachycardia circuit based on high-resolution activation mapping and entrainment pacing. In ridge- and MB-related macroreentrant ATs, the critical isthmus was located in the ridge or MB, and conduction block was confirmed in the ventricular part of the MI and inside the CS. Such ATs were not included as peri-mitral ATs.[8]

Catheter Ablation

The isthmus region was chosen and targeted based on propagation, ie, sites of wavefront narrowing or slowing. Ablation was performed with a 4-mm irrigated-tip radiofrequency catheter (FlexAbility, Abbott) with a power of 30–35 W. The procedural end point was no inducibility of any stable ATs with burst atrial pacing (up to 200 ms) from multiple sites in both atria without isoproterenol infusion. Focal AT and micro-reentry (localized AT) were defined according to previous studies.[2,7]

Follow-Up

Patients underwent continuous in-hospital ECG monitoring for 3 to 7 days following the procedure. The first outpatient clinic visit was 1 month after the procedure. Subsequent follow-up visits consisted of a clinical interview, ECGs, and/or 24-hour Holter monitoring every 3 months in our cardiology clinic. Recurrence was defined as any ATs lasting longer than 30 seconds beyond a 3-month blanking period.

Statistical Analysis

Continuous data are expressed as means±SD for normally distributed variables or as medians [interquartile range] for nonnormally distributed variables.

RESULTS

Type of ATs

A total of 141 patients underwent AT mapping, including patients with cavotricuspid isthmus–dependent ATs, during the study period. Among the 28 ATs whose mechanism involved the lateral LA, the posterolateral MI was identified as the AT origin or substrate in 18 (64.3%) patients. By contrast, the left atrial ridge or MB was involved in the remaining 10 (35.7%) patients. Mapping was performed with the Rhythmia and CARTO systems in 25 and 3 patients, respectively.

ATs Involving the Posterolateral MI

Among the 18 patients whose ATs involved the posterolateral MI, 14 (77.8%) had peri-mitral ATs with the critical isthmus identified in the MI area (Figure 1A), 3 (16.7%) had a small reentrant circuit in the MI area (Figure 2), and 1 (5.5%) had a focal AT originating from the MI area (Figure 1B).

All 14 patients with peri-mitral ATs (63.1±11.1 years, 10 men, 11 patients with non-paroxysmal AF, left atrial diameter 40.5±9.0 mm) had a history of posterolateral MI ablation (linear ablation in 12 patients and ablation targeting complex fractionated electrograms in 2 patients). For 6 patients, posterolateral MI ablation was the second procedure whereas it was the third procedure for 8 patients. Mean TCL was 257±39 ms. Activation maps of 10 410 (6868–12 015) points were acquired during the AT over 17.7 (11.9–23.8) minute of mapping time (Figure 1A). The activation map revealed 12 (85.7%) clockwise and 2 (14.3%) counter-clockwise peri-mitral ATs. Peri-mitral ATs were terminated during endocardial ablation in 13 patients, with a single application in 9 patients. One patient underwent linear ablation after restoration to sinus rhythm with cardioversion. Complete posterolateral MI line block was successfully achieved in all patients. A total of 5 concomitant ATs, including left atrial roof dependent AT in 2 patients, anterior left atrial wall AT in 1 patient, and a small reentrant AT in the CS and right atrium in 1 patient, were identified among 4 patients; all were successfully eliminated. During a median follow-up period of 23.5 (8.7–40.2) months, all were free from any atrial tachyarrhythmias. However, 3 patients remained on the same dose of antiarrhythmic drugs (AADs) as before the procedure to control AF (Table).

All 3 patients with a small reentrant AT (59.0±14.1 years, 2 men, 2 patients with non-paroxysmal AF, left atrial diameter 30.9±1.9 mm) had a history of posterolateral MI ablation; 1 patient had linear ablation and 2 patients had ablation targeting complex fractionated electrograms. For 2 patients, posterolateral MI ablation was the second procedure while it was the fourth procedure for the remaining patient. Mean TCL was 265±39 ms. Activation maps of 25 533 (9051–28 647) points were acquired during the AT over 23.9 (17.8–28.5) minutes of mapping time (Figure 2). The activation map revealed 1 clockwise and 2 counter-clockwise reentry. All ATs were terminated by a single application at the critical isthmus below the left inferior PV. A total of 4 concomitant ATs, including peri-mitral AT in 2 patients, left atrial roof dependent AT in 1 patient, and common flutter in 1 patient, were identified among 2 patients; all were successfully eliminated. During a median follow-up period of 13.0

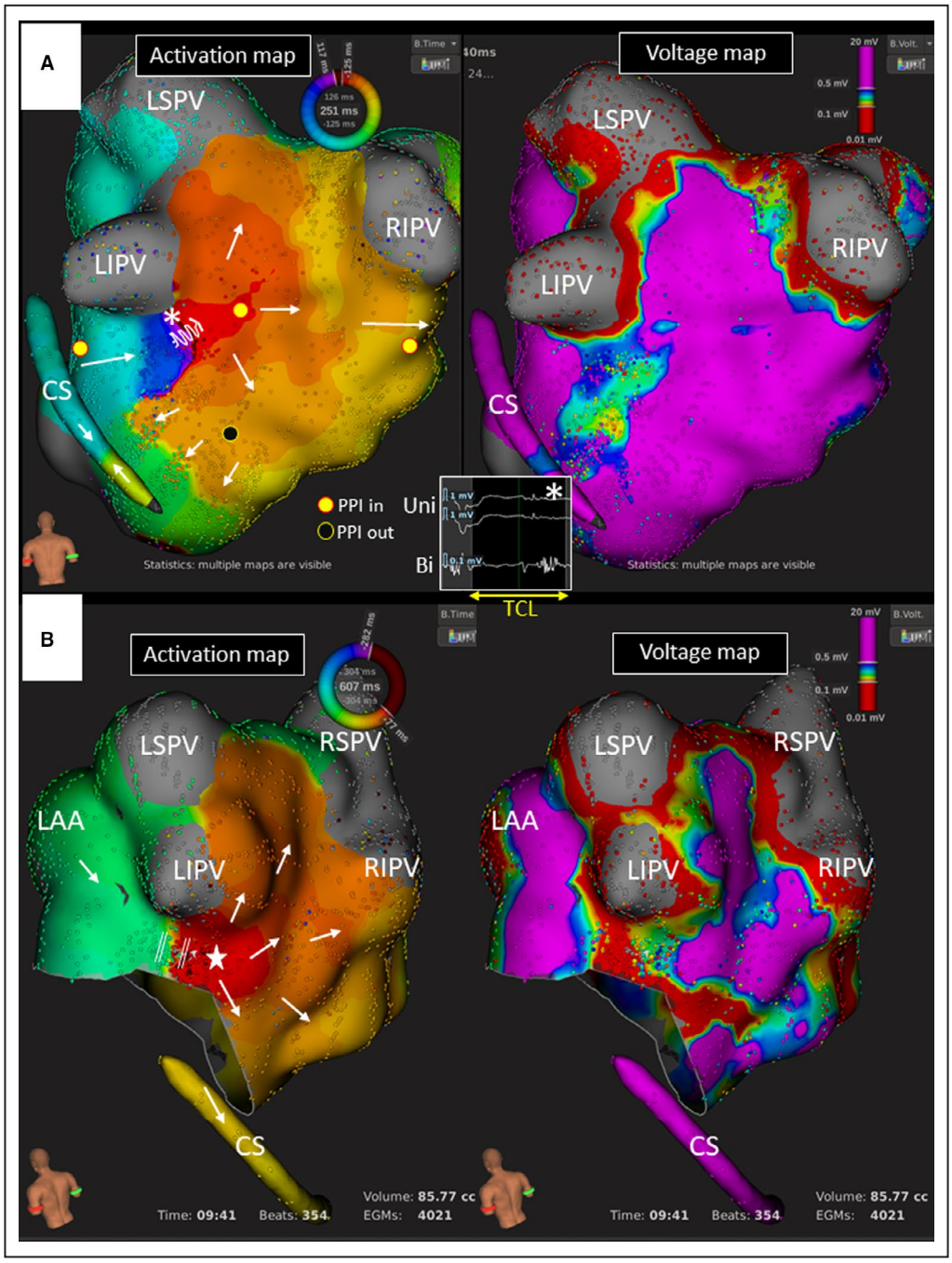

Figure 1. A representative case of peri-mitral AT (A) and focal AT originating from the posterolateral MI (B).

A, An activation map revealed a clockwise peri-mitral AT with an endocardial conduction gap located below LIPV (asterisk) in the prior MI linear lesion. The AT was terminated by a single radiofrequency application in the gap. A voltage map showed a low-voltage area in the previous MI linear lesion. **B**, An activation map revealed a focal AT originating from the posterolateral MI (white star) behind the previously created complete MI block line. Left atrial activation time accounted for only 30% of the TCL. The AT was terminated by focal ablation at the earliest local activation site (white star). The white arrows show the propagation of the activation. AT indicates atrial tachycardia; Bi, bipolar; CS, coronary sinus; LIPV, left inferior pulmonary vein; LSPV, left superior pulmonary vein; LAA, left atrial appendage; MI, mitral isthmus; PPI, post pacing interval; RIPV, right inferior pulmonary vein; RSPV, right superior pulmonary vein; TCL, tachycardia cycle length; and Uni, unipolar.

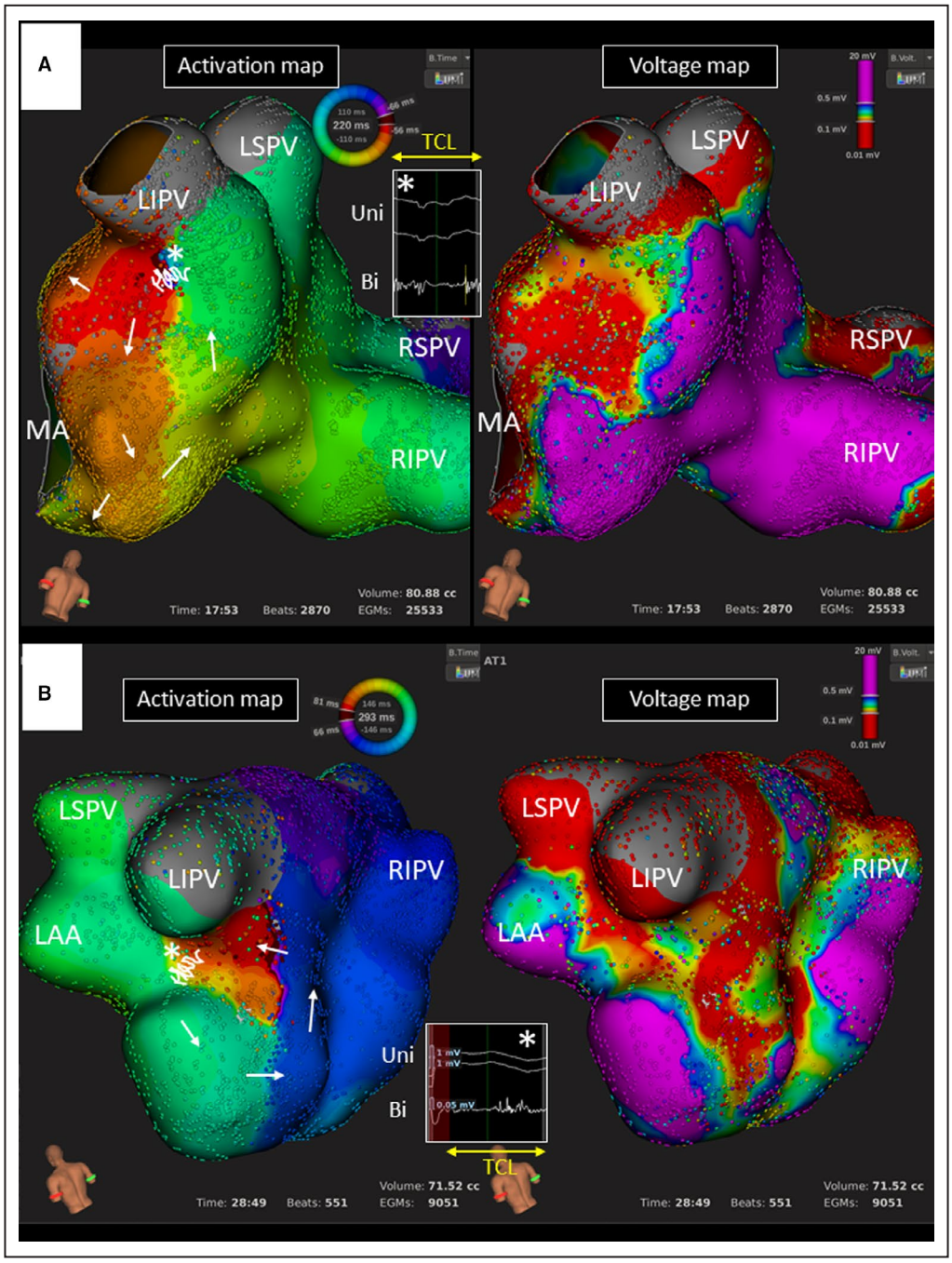

Figure 2. Two representative cases (A and B) of small reentrant AT in the posterolateral MI. Activation maps revealed a small reentrant circuit in a counter-clockwise fashion in the posterolateral MI. A macroreentrant AT was ruled out by entrainment pacing. An area with slow conduction and low-amplitude fractionated signals was identified below the LIPV (asterisks). ATs were terminated by a single application at the site. Both patients had a history of previous MI area ablation and low-voltage areas were identified on the voltage maps. AT indicates atrial tachycardia; Bi, bipolar; LIPV, left inferior pulmonary vein; LSPV, left superior pulmonary vein; LAA, left atrial appendage; MA, mitral annulus; MI, mitral isthmus; RIPV, right inferior pulmonary vein; RSPV, right superior pulmonary vein; TCL, tachycardia cycle length; and Uni, unipolar.

(10.0–36.0) months, all were free from any atrial tachyarrhythmias without AAD therapy.

The remaining patient with focal AT (64 years, woman, non-paroxysmal AF, left atrial diameter 32.9 mm) had a history of posterolateral MI linear ablation.[9] TCL was 607 ms. Activation maps of 4021 points were acquired during the AT over 9.6 minutes of mapping time (Figure 1B). The activation map showed a

Table. Type of ATs and Clinical Outcome After the Procedure

Type of ATs	No.	FU period (mo)	AT/AF freedom, n	AADs
ATs involving the posterolateral MI	18			
Peri-mitral ATs	14	23.5 (8.7–40.2)	100%	3 on, 11 off
Small reentrant AT	3	13.0 (10.0–36.0)	100%	Off
Focal AT	1	40	100%	Off
ATs involving the LA ridge or MB	10			
Typical LA ridge- or MB-related AT	6	31.0 (5.5–33.0)	100%	On
PLSVC-related AT	1			
Atypical MB- or LA ridge-related ATs	2	7.5 (3.0–12.0)	100%	Off
Micro-reentry within the LA ridge	1	12	100%	Off

AAD indicates antiarrhythmic drug; AF, atrial fibrillation; AT, atrial tachycardia; FU, follow-up; LA, left atrial; MB, Marshall bundle; MI, mitral isthmus; n, number; and PLSVC, persistent left superior vena cava.

focal pattern and left atrial activation accounted for 30% of TCL. Focal ablation at the earliest local activation site terminated the AT. A concomitant small reentrant AT inside the CS was also eliminated. During a follow-up period of 40 months, she was free from any atrial tachyarrhythmias without AAD therapy.

ATs Involving the Left Atrial Ridge and Marshall Bundle

Among the 10 patients whose ATs involve the left atrial ridge or MB, the left atrial ridge or MB junction was the critical isthmus in 7 patients (Figures 3A, 4, 5A, and 6). The other area was the critical isthmus in 2 patients (Figure 5B). The remaining patient had micro-reentry (localized reentry) in the left atrial ridge (Figure 3B).

Six patients had typical left atrial ridge- or MB-related AT (74.2±9.6 years, 3 men, 6 patients with non-paroxysmal AF, LA diameter 42.2±4.6 mm) (Figures 3A, 4, and 5A).[10,11] One patient (79 years, woman, paroxysmal AF, LA diameter 41.0 mm) had persistent left superior vena cava (PLSVC)-related AT (Figure 6).[12] VOM mapping was performed in 3 patients in the former group (Figures 4 and 5A). The procedure for AT mapping was the second procedure in 4 patients, third procedure in 1 patient, fourth procedure in 1 patient, and fifth procedure in 1 patient. All 7 patients had a history of previous MI linear ablation. Mean TCL was 303±124 ms. Activation maps of 9657 (6078–13 726) points were acquired during the AT over 19.0 (14.1–25.8) minutes of mapping time. The activation ascended on the left atrial ridge or MB in 4 ATs, and descended in the remaining 3 ATs. Activation and entrainment mapping revealed that the critical isthmus was located in the LA-left atrial ridge junction or LA-MB junction and the LA-PLSVC junction. Focal ablation of the critical isthmus successfully terminated ATs in all patients. Bi-directional conduction block between the LA and left atrial ridge or MB at the junction (Figure 4B and 4C) or between the LA and PLSVC at the junction (Figure 6B and 6C) was confirmed in all patients. MI linear block was also confirmed. During a median follow-up period of 31.0 (5.5–33.0) months, which excluded 2 patients with ≤3 months of follow-up, all were free from any atrial tachyarrhythmias on AAD therapy.

In the other 2 patients (78.5±6.4 years, 2 women, 1 patient with non-paroxysmal AF, LA diameter 50.7±3.7 mm) with atypical MB- or left atrial ridge–related ATs, the procedure for AT mapping was the first or second procedure. Neither patient had a history of posterolateral MI ablation. Mean TCL was 239±3.5 ms. Activation maps of 11 873 (8556–15 189) points were acquired during the AT over 25.5 (20.5–30.5) minute of mapping time. VOM mapping was performed in 1 patient. Activation and entrainment mapping revealed left atrial ridge–related ATs. However, the critical isthmus was identified within a scar at the left atrial roof (Figure 5B) or anterior left atrial wall. Both ATs were terminated by a single application at the critical isthmus. Two concomitant ATs, ie, left atrial roof dependent AT and anterior left atrial wall AT, were observed in 1 patient each. Creation of a linear lesion successfully eliminated both ATs. During a median follow-up period of 7.5 (3.0–12.0) months, both patients were free from any atrial tachyarrhythmias without AAD therapy.

The remaining patient (75 years, woman, paroxysmal AF, LA diameter 35.0 mm) had persistent AT and paroxysmal AF. She did not have a history of any previous procedures. TCL was 297 ms. An activation map of 25 282 points was acquired during the AT over 23.2 minutes of mapping time (Figure 3B). The activation map showed localized reentry within the left atrial ridge, and the entire TCL was covered within the small area. A single application terminated the AT, which was followed by cryoballoon PV isolation. During a follow-up period of 12 months, she was free from any atrial tachyarrhythmias without AAD therapy.

DISCUSSION

The present study characterized the various types of ATs whose mechanism involved the lateral LA using currently available high-resolution mapping systems.

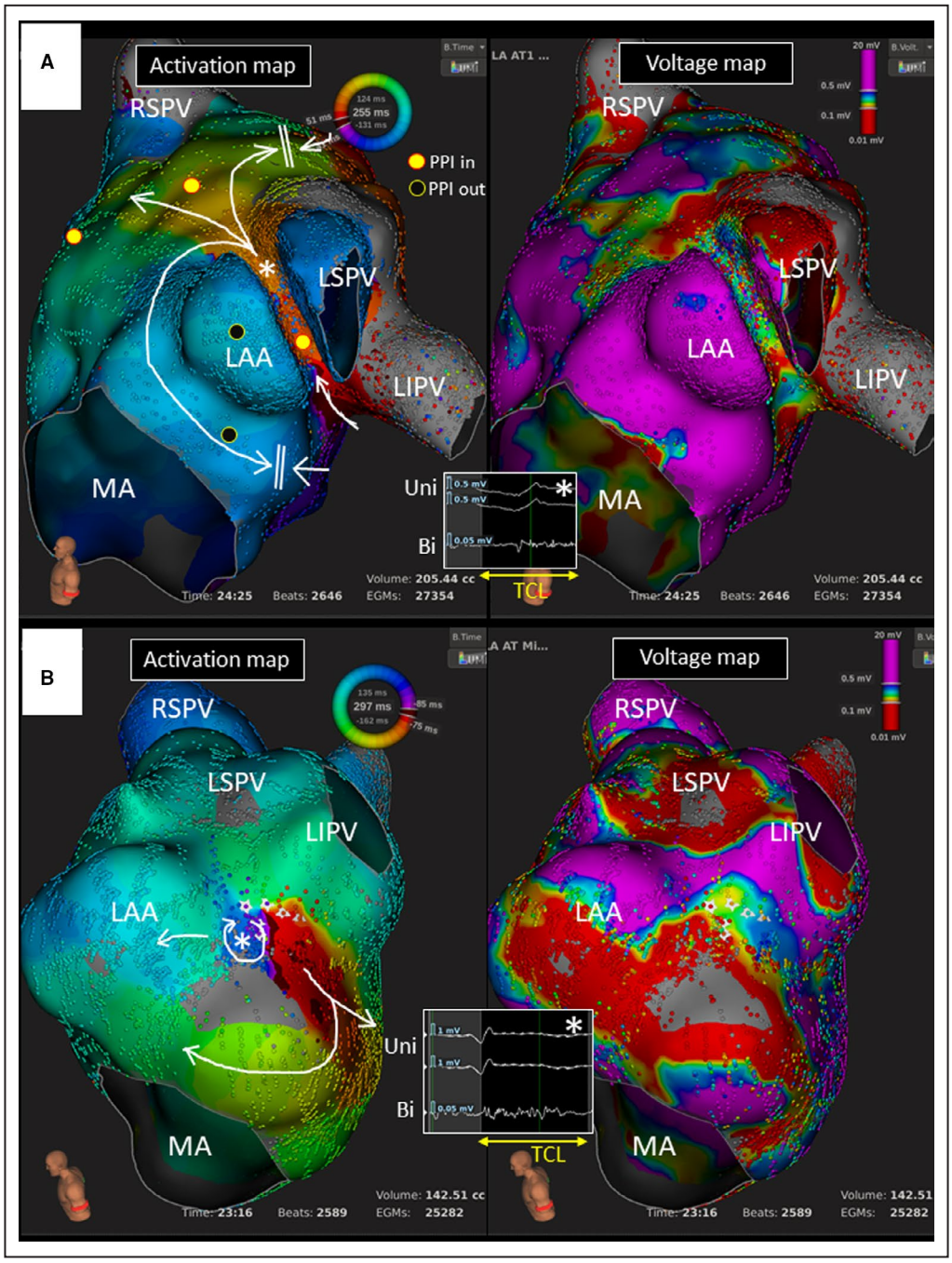

Figure 3. A representative case of left atrial ridge–related AT (A) and micro-reentry in the left atrial ridge (B).
A, An activation map revealed left atrial ridge–related AT with upward activation in the ridge. The patient had a history of MI linear ablation. The critical isthmus was identified at the ridge-LA junction (exit from the ridge) (asterisk), and a single application terminated the AT. Subsequently, ridge-LA conduction block was confirmed. **B**, An activation map revealed micro-reentry (localized reentry) within the left atrial ridge, and activation of a small area (asterisk) accounted for the entire TCL. The patient had no history of ablation. A single application terminated the AT. Bi indicates bipolar; LAA, left atrial appendage; LIPV, left inferior pulmonary vein; LSPV, left superior pulmonary vein; MA, mitral annulus; PPI, post pacing interval; RSPV, right superior pulmonary vein; TCL, tachycardia cycle length; and Uni, unipolar.

We found that (1) high-resolution mapping systems are useful for identifying the critical isthmus of ATs; (2) the vast majority of ATs involving the lateral LA occurred after previous MI area ablation; (3) the posterolateral MI was involved in peri-mitral AT as well as small reentrant and focal ATs; (4) creating conduction block between

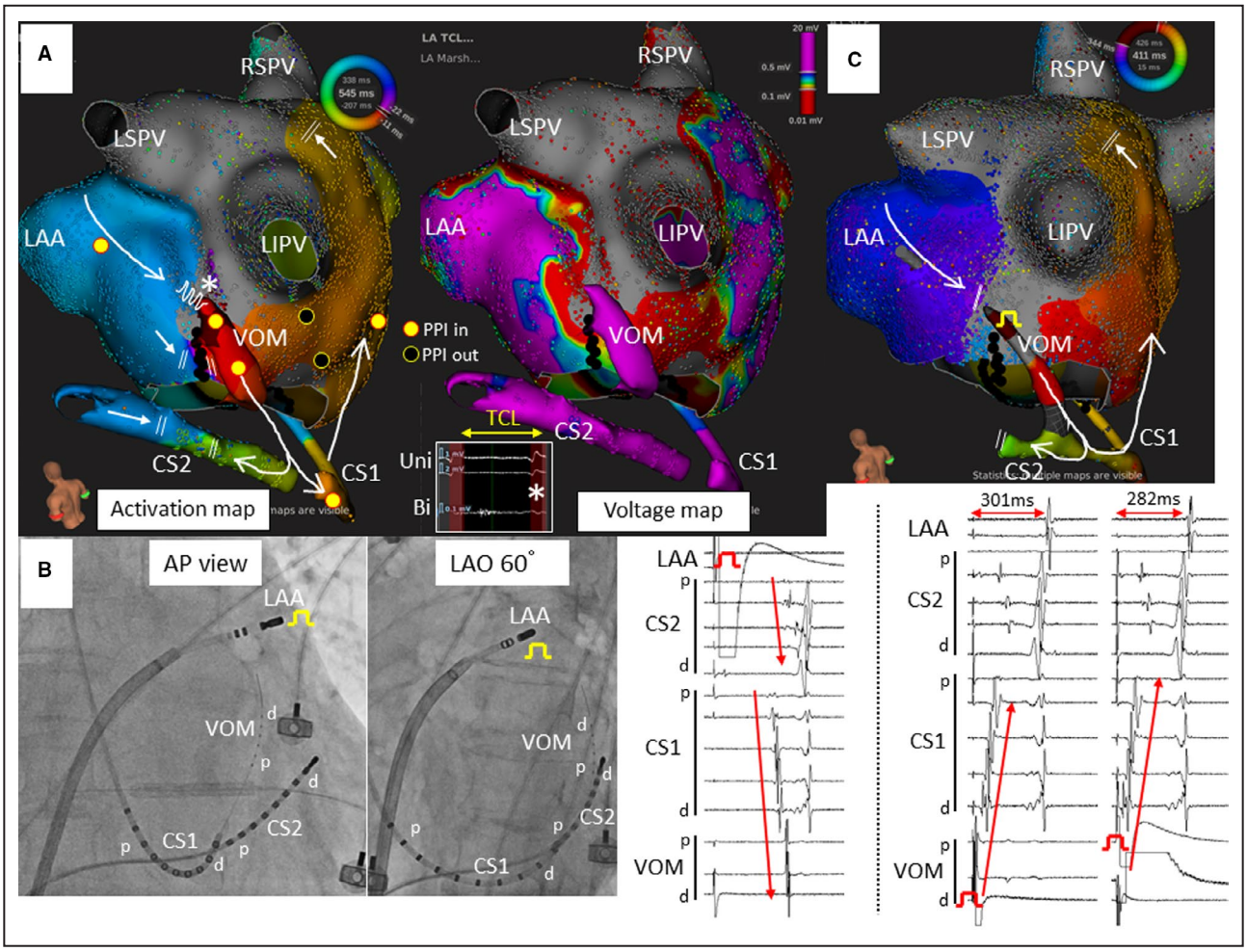

Figure 4. A representative case of MB-related AT.
A, An activation map revealed MB-related AT with downward activation in the VOM. Slow conduction (asterisk) was identified at the LA-MB junction (entrance of MB), and a single application at the site terminated the AT. **B**, Following venography, a 1.8-Fr hexapolar catheter was inserted into the VOM to validate the MB-related AT (**left panel**). Pacing from the LAA (ablation catheter) showed proximal-to-distal VOM activation (**right panel**), suggesting LA-MB conduction block. **C**, Differential pacing from the proximal and distal VOM electrodes showed longer LAA delay during distal VOM pacing, suggesting MB-LA conduction block (**lower panel**). An activation map during pacing from the VOM showed MB-LA conduction block, indicating bidirectional MB-LA conduction block at the junction (**upper panel**). AP, antero-posterior; Bi, bipolar; CS, coronary sinus; d, distal; LAA, left atrial appendage; LAO, left anterior oblique; LIPV, left inferior pulmonary vein; LSPV, left superior pulmonary vein; p, proximal; PPI, post pacing interval; RSPV, right superior pulmonary vein; TCL, tachycardia cycle length; Uni, unipolar; and VOM, vein of Marshall.

the LA and MB or left atrial ridge at the junction was an optimal procedural end point for typical MB- or left atrial ridge–related ATs, and (5) the left atrial ridge was involved in multiple types of ATs, and the critical isthmus needs to be identified individually for successful treatment.

ATs Involving the Posterolateral MI

Peri-mitral AT is the most common macroreentrant tachycardia occurring after substrate-based ablation for AF.[1] Generally, the substrate for peri-mitral AT is a significant conduction delay in the posterolateral MI or the anterior left atrial or septal wall. The former is often created by prior ablation procedures. The latter is mostly due to the natural progression of underlying atrial disease. This study included the former population. All patients had a history of previous MI area ablation. In addition, this study demonstrated small reentrant and focal ATs within this area. All patients with small reentrant ATs had a low-voltage area, presumably due to previous ablation in this area, suggesting iatrogenic AT. The critical isthmus was identified below the left inferior PV, where a conduction gap often remains after MI linear ablation.[13] These peri-mitral ATs and small reentrant ATs were iatrogenic ATs, which highlights the importance of creating a durable MI linear conduction block once MI area ablation has been performed. Of

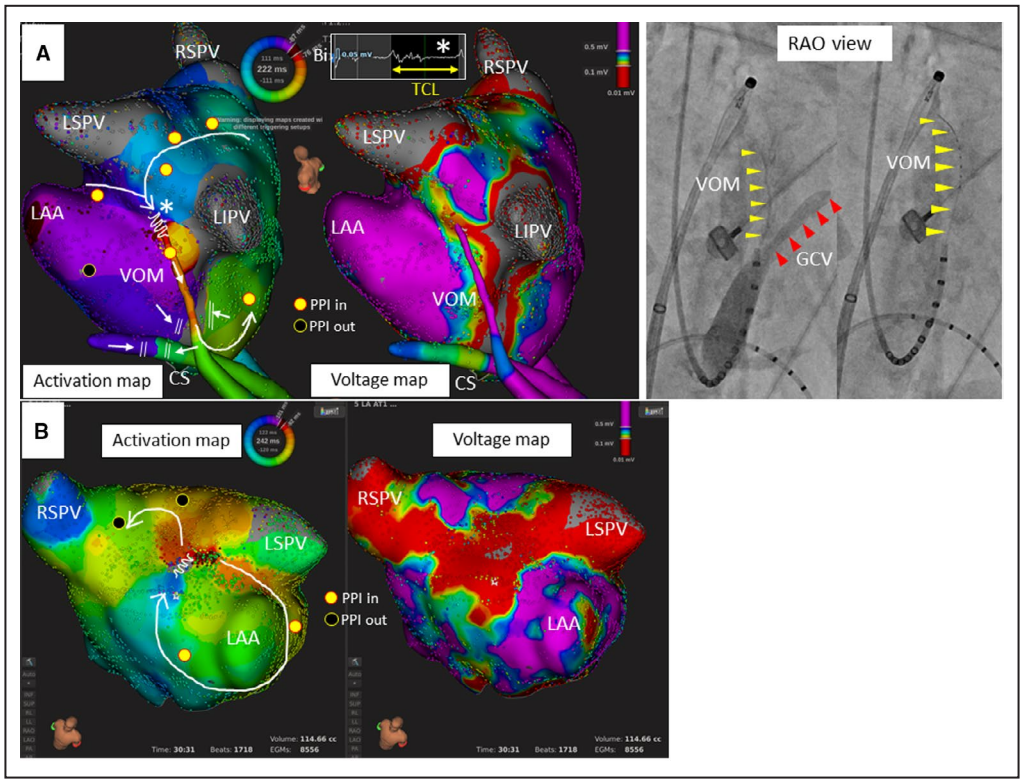

Figure 5. A representative case of MB-related AT with the critical isthmus in the left atrial ridge (A) and a left atrial ridge–related AT where the critical isthmus was not in the left atrial ridge (B). **A**, An activation map revealed dual-loop MB-related AT with downward activation in the MB. The common isthmus (asterisk) was identified at the LA-MB junction (entrance of MB), and ablation at the site terminated the AT (**left panels**). Following venography, a 1.8-Fr hexapolar catheter was inserted into the VOM to validate the MB-related AT (**right panels**). Yellow and red arrowheads indicate the VOM and great cardiac vein, respectively. **B**, An activation map revealed left atrial ridge–related AT; however, the critical isthmus was identified at the roof of the left atrium, inside a scar. A single application at the isthmus terminated the AT. AT indicates atrial tachycardia; Bi, bipolar; CS, coronary sinus; GCV, great cardiac vein; LAA, left atrial appendage; LIPV, left inferior pulmonary vein; LSPV, left superior pulmonary vein; MB, Marshall bundle; PPI, post pacing interval; RAO, right anterior oblique view; RSPV, right superior pulmonary vein; and VOM, vein of Marshall.

note, focal AT, which does not seem to be iatrogenic, could also occur from this area.

ATs Involving the Left Atrial Ridge or MB

Takatsuki et al initially reported 5 cases of left atrial ridge–related AT in 2013.[8] They showed that left atrial ridge–related AT could develop after PV isolation and MI linear ablation because the surviving myocardial tissue in the ridge is a critical pathway. Since conduction in the ventricular part of the MI and the CS was completely blocked, ridge-related AT seemed to be an entity distinct from peri-mitral AT. However, the role of the epicardial connection was not mentioned. VOM is a remnant of the left venous horn. It runs between the left PVs and the left atrial appendage, merging with the CS. The number of connections between the MB and the myocardium is variable. Most patients have ≥2 connections between the MB and the myocardium of the CS, LA, or PVs, as confirmed in histological[14] and clinical electrophysiological studies.[15] Hayashi et al described 4 patients with MB-related ATs based on VOM mapping.[16] In that study, the LA-MB junction was initially targeted. However, a block in the CS-MB junction was alternatively created in half of the patients due to failure of the initial strategy. Interestingly, incomplete MI linear block remained in patients in whom a CS-MB junction block was created.

In this study, we successfully terminated the AT and created a conduction block in the left atrial ridge or MB junction with a few applications in all cases. No ablation at the MB-CS junction was required, and MI linear block was confirmed subsequently. Interestingly, we also observed 1 PLSVC-related AT.[11] Embryologically, the PLSVC regresses to become a ligament; this AT was also terminated by focal ablation at the LA-PLSVC junction, as well as MB-related ATs. Since there might be multiple connections between the MB and CS or

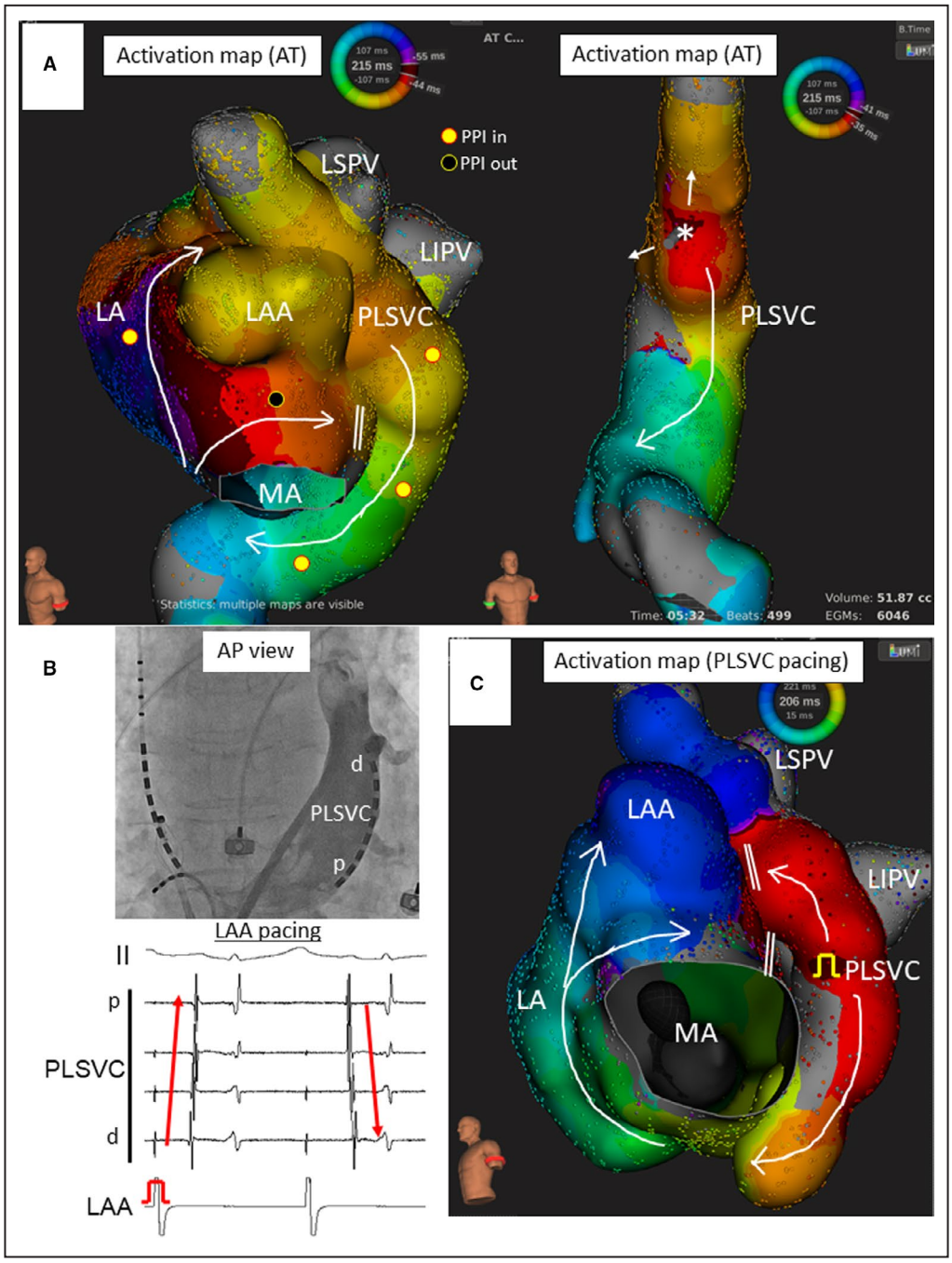

Figure 6. A representative case of PLSVC-related AT.
A, An activation map revealed PLSVC-related AT with downward activation in the PLSVC (**left panel**). Activation of PLSVC alone showed a focal pattern (asterisk) from the LA-PLSVC junction (**right panel**). Focal ablation at the site from the PLSVC side terminated the AT. **B**, Venography showed the PLSVC on fluoroscopy. The activation pattern inside the PLSVC during LAA pacing abruptly changed from distal-to-proximal to proximal-to-distal with additional ablation at the LA-PLSVC junction, suggesting LA-PLSVC conduction block at the junction. **C**, Subsequently, PLSVC pacing showed PLSVC-LA conduction block, indicating bidirectional conduction block at the LA-PLSVC junction. AP, antero-posterior; AT, atrial tachycardia; d, distal; LA, left atrium; LAA, left atrial appendage; LIPV, left inferior pulmonary vein; LSPV, left superior pulmonary vein; MA, mitral annulus; p, proximal; PLSVC, persistent left superior vena cava; and PPI, post pacing interval.

inferior LA, we believe that creating a left atrial ridge or MB block at the junction is a reasonable procedural end point for these types of ATs. We also observed 2 patients with left atrial ridge–related ATs where the critical isthmus was identified in another area. Both ATs were successfully eliminated by ablation targeting

the critical isthmus, and the left atrial ridge was not targeted because of the absence of conduction delay. This is presumably because these patients did not have a history of MI linear ablation. These results suggest that (1) previous MI linear ablation might create the substrate for typical left atrial ridge– or MB-related AT, and (2) the identification of the critical isthmus is essential for the treatment of left atrial ridge– or MB-related ATs. A single patient without any previous ablation had micro-reentry (localized reentry) in the left atrial ridge. This case highlighted that conduction disturbance could naturally progress in this area. These findings might partly explain the favorable impact of VOM ethanol infusion in the context of AF/AT ablation reported recently.[5]

Utility of High-Resolution Mapping Systems

The high-resolution mapping systems using multipolar catheters with small electrodes were extremely useful for exploring the mechanisms of ATs involving the lateral LA. Entrainment pacing further confirmed the active AT circuit and the mechanisms underlying the ATs. In patients with left atrial ridge–related ATs, direct VOM mapping was helpful for confirming the participation of the epicardial connection, even though the insertion of the mapping catheter was not always easy due to the absence or small size of the VOM or other technical reasons. Although AAD therapy was continued in patients with left atrial ridge– or MB-related ATs to suppress concomitant AF, the high rate of freedom from arrhythmia in this study supports the utility of high-resolution mapping systems and the reasonableness of the procedural end point. The usefulness of the present system was also supported by the capability of understanding the entire tachycardia circuit, and the precise identification of the critical isthmus of the ATs (this was supported by the AT termination with RF applications).

Study Limitations

The study population was relatively small. A limited number of patients underwent VOM mapping.

CONCLUSIONS

The vast majority of ATs involving the lateral LA occurred after MI area ablation. High-resolution mapping systems are very useful for identifying the critical isthmus of ATs. Direct VOM mapping is useful for validating the association between the mechanism for the tachycardia and an epicardial connection.

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