Selective ethanol ablation targeting the distal vein of Marshall for a peri–left atrial appendage reentrant atrial tachycardia after completing anterior mitral isthmus conduction block

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Introduction
Some reentrant atrial tachycardias (ATs) after a percutaneous or surgical ablation of atrial fibrillation (AF) include the Marshall bundle (MB) in reentrant circuits, such as perimitral ATs, or ridge-related or MB-related reentrant ATs.1–11 An ethanol infusion into the vein of Marshall (VOM) can create an ablation lesion along the posterolateral left atrium (LA) and ridge between the LA appendage (LAA) and left pulmonary veins (PVs) and can be useful for terminating those MB-related ATs and creating conduction block along the mitral isthmus (MI) between the left PVs and posterolateral mitral annulus (MA). We present a case of a peri-LAA reentrant AT associated with the distal MB after a PV and posterior LA isolation for persistent AF and LA linear ablation for an induced perimitral AT. Owing to the presence of conduction block along the anterior MI between the anterior MA and LA roof, ethanol was infused into the VOM as distally as possible, which terminated the peri-LAA and MB-related AT without any remarkable change in the atrial voltage and conduction velocity along the posterolateral MA area and without an electrical isolation of the LAA.

Case report
A 73-year-old woman with a previous mitral valve replacement owing to mitral valve stenosis underwent a first radiofrequency catheter ablation of persistent AF. After a PV and posterior LA isolation was performed during sinus rhythm, a clockwise perimital AT was induced by atrial burst pacing. The perimital AT failed to be terminated by a posterolateral MI linear ablation, but succeeded in being terminated by an anterior MI linear ablation between the anterior MA and LA roof. Finally, bidirectional conduction block was achieved along the anterior MI, but was not achieved along the posterolateral MI. Four months after the first ablation procedure, she underwent a second ablation procedure for a recurrent AT with a tachycardia cycle length (TCL) of 494 ms (Figure 1A). During the second procedure,
Figure 1  
A: Twelve-lead electrocardiogram during the clinical atrial tachycardia (AT) with a tachycardia cycle length (TCL) of 494 ms. B: Isochronal activation maps during the AT exhibiting endocardial activation gaps from the base of the left atrial appendage (LAA) to the top of the ridge in the superoanterior view (left panel) and along the middle portion of the ridge in the left lateral view (right panel). The local endocardial electrograms were not recorded during phase 4 in the left panel and phases 7–8 in the right panel, which appeared to indicate the activation gaps during the AT. The numbers in the local electrograms indicate the intervals from the reference electrogram recorded in the coronary sinus to the local electrogram. C–H: High-resolution activation maps exhibiting a clockwise peri-LAA reentrant AT with partial epicardial conduction along the ridge. The activation wavefront (dark red) propagated (C–D) from the top to the middle portion of the ridge and (E–F) through the lateral left atrium (LA) toward the anterolateral mitral annulus (MA), and (G–H) returned to the top of the ridge passing between the LAA and previous anterior mitral isthmus ablation line (Supplemental Video 1). The white solid arrows and double lines represent the endocardial activation propagation and conduction block, respectively. The white dotted arrows from the base of the LAA to the top of the ridge (C, G, and H) and from the middle portion of the ridge to the lateral LA (D and E) represent a possible epicardial activation propagation via the Marshall bundle. The white tags and numbers on the maps indicate the entrainment pacing sites and post-pacing interval minus the TCL (ms), respectively. The confidence mask of the maps was set at 0.03 mV. LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein; VOM = vein of Marshall.
high-resolution activation mapping during the AT in the LA and proximal VOM using the Rhythmia system (Boston Scientific, Marlborough, MA) with a mini-basket catheter (IntellaMap Orion; Boston Scientific) and 1.6F multielectrode catheter (EPSkinny; FMD Co, Ltd, Tokyo, Japan) suggested that the clinical AT was a clockwise peri-LAA reentrant AT with partial epicardial conduction from the top to the middle portion of the ridge, which appeared to be associated with the distal MB (Figure 1B–1H, Supplemental Video 1). The post-pacing interval was almost similar to the TCL upon entrainment from the middle portion of the ridge and around the LAA, but was not upon entrainment from the posterolateral MA, proximal VOM, and LA septum (Figure 1C–1H). The activation map and entrainment pacing during the AT suggested the presence of conduction block along the anterior MI. Therefore, after selective VOM venography, an angioplasty over-the-wire balloon with a 2 mm nominal diameter and 8 mm length (Emarge; Boston Scientific) was advanced into the VOM as distally as possible, in order not to ablate the proximal VOM and posterolateral MA area and consequently not to isolate the LAA by an ethanol infusion into the VOM (Figure 2A). An infusion of 2.5 mL ethanol 98% into the VOM successfully terminated the AT (Figure 2B). After the ethanol infusion with a total of 10 mL, voltage and activation mapping exhibited no remarkable change in the atrial voltage and conduction velocity along the posterolateral MA area (Figure 3), and verified the presence of anterior MI conduction block (Supplemental Video 2). Thereafter, no ATs could be further induced by any programmed or rapid atrial stimulation.

Discussion
This is a case report demonstrating that selectively infusing ethanol into the distal VOM resulted in the termination of a peri-LAA reentrant AT via the distal MB and preservation of the atrial voltage and conduction along the posterolateral MI area in the patient with conduction block along the anterior MI ablation line.

The MB can be involved in post–AF ablation reentrant ATs. An ethanol infusion into the VOM, as well as endocardial and epicardial radiofrequency ablation, is effective for treating localized and macroreentrant ATs associated with the MB. During a VOM ethanol infusion procedure, an angioplasty balloon is generally inflated within the proximal VOM, and ethanol is infused from a proximal site of the VOM. That is because the MB often has multiple connections with the LA. Infusing ethanol from a more proximal site of the VOM can eliminate more MB-LA connections and create a more extensive ablation lesion along the VOM, which would be beneficial especially for achieving conduction block along the posterolateral MI ablation line.

On the other hand, minimizing the effect of an ethanol infusion in the proximal VOM and posterolateral MI area is extremely important in cases with anterior MI conduction
block. If conduction block is not achieved along the anterior MI, there is no risk of electrically isolating the LAA by a VOM ethanol infusion. However, if conduction block is achieved along the anterior MI, as in the present case, there is a risk of electrically isolating the LAA by a VOM ethanol infusion. Therefore, after confirmation that the proximal VOM was outside the AT reentrant circuit based on the entrainment mapping, an angioplasty balloon was advanced to the distal site of the VOM, where the balloon could not be advanced any further, and was inflated to the nominal pressure. Thereafter, ethanol was infused through the balloon. Wedging the balloon into the distal VOM may have minimized the backflow of the ethanol infused into the VOM. However, even if an angioplasty balloon is placed in a more distal site of the VOM, the ablated area by the ethanol infusion may be more extensive than expected. Thus, understanding the location and proximity of the VOM and MA is necessary to predict the ablated area by a VOM ethanol infusion to a certain extent. The VOM in the present case was located relatively far from the MA, where a bioprosthetic mitral valve was placed. That may also be one of the reasons why the atrial conduction along the posterolateral MI was mostly preserved after the VOM ethanol infusion. Further, the mitral valve prosthesis may have prevented the creation of conduction block near the posterolateral MA. The difficulty of achieving a sufficient ablation lesion formation near the posterolateral MI.

Figure 3  A: Bipolar voltage maps before the vein of Marshall (VOM) ethanol infusion during the atrial tachycardia (AT) (left panel) and after the VOM ethanol infusion during left atrial appendage (LAA) pacing (right panel). The VOM ethanol infusion eliminated the electrograms along the ridge, but the electrograms from the lateral to posterolateral mitral isthmus (MI) area did not significantly change before and after the VOM ethanol infusion. The low-voltage and scar areas were defined as areas with bipolar electrogram amplitudes of <0.5 mV and <0.1 mV, respectively. B: Isochronal activation maps before the VOM ethanol infusion during the AT (left panel) and after the VOM ethanol infusion during LAA pacing (right panel). Each color between red and purple represents a 10 ms activation time. The distance between the 2 black tags in the lateral and posterolateral MI area (white dotted bidirectional arrows) is the same between the 2 isochronal maps and is 30.5 mm. The mean conduction velocity between the 2 black tags is 0.68 mm/ms before the VOM ethanol infusion and 0.64 mm/ms after, respectively. Abbreviations are as in Figure 1.
MA owing to the valve prosthesis was likely to have contributed to the preservation of the atrial conduction along the posterolateral MA after the radiofrequency ablation and VOM ethanol infusion.

High-resolution mapping is useful for establishing an accurate diagnosis of the mechanisms of postablation complex ATs, but in the present case could not visualize the entire circuit of the AT along the ridge. The AT activation map exhibited endocardial activation gaps from the base of the LAA to the top of the ridge and along the middle portion of the ridge. Endocardial entrainment pacing near the top of the ridge revealed a long postpacing interval minus the TCL of 43 ms, which suggested that epicardial tissue in this area was included in the AT circuit but endocardial tissue was not. Endocardial entrainment pacing from the middle portion of the ridge with the endocardial activation gap was not performed because local potentials in that area could not be captured by pacing. Further, epicardial mapping was not performed along the ridge because a 1.6F mapping catheter could not be advanced into the distal VOM. However, the termination and noninducibility of the AT by the VOM ethanol infusion may have suggested that a part of the AT circuit along the ridge, which could not be visualized on the endocardial map, was associated with the distal MB.

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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.2022.06.006

References
1. Takigawa M, Derval N, Martin CA, et al. Mechanism of recurrence of atrial tachycardia: comparison between first versus redo procedures in a high-resolution mapping system. Circ Arrhythm Electrophysiol 2020;13:e007273.
2. Kitamura T, Takigawa M, Derval N, et al. Atrial tachycardia circuits include low voltage area from index atrial fibrillation ablation relationship between RF ablation lesion and AT. J Cardiovasc Electrophysiol 2020;31:1640-1648.
3. Vlachos K, Denis A, Takigawa M, et al. The role of Marshall bundle epicardial connections in atrial tachycardias after atrial fibrillation ablation. Heart Rhythm 2019;16:1341-1347.
4. Briceño DF, Valderrábano M. Recurrent perimital flutter due to vein of Marshall epicardial connections bypassing the mitral isthmus: response to ethanol infusion. Circ Arrhythm Electrophysiol 2014;7:988-989.
5. Kitamura T, Vlachos K, Denis A, et al. Ethanol infusion for Marshall bundle epicardial connections in Marshall bundle-related atrial tachycardias following atrial fibrillation ablation: the accessibility and success rate of ethanol infusion by using a femoral approach. J Cardiovasc Electrophysiol 2019;30:1443-1451.
6. Takigawa M, Vlachos K, Martin CA, et al. Acute and mid-term outcome of ethanol infusion of vein of Marshall for the treatment of perimital flutter. Europace 2020;22:1252–1260.
7. Margato R, Tampakis K, Albenque JP, Combes S. Illuminating the Marshall: novel techniques highlighted in an atrial tachycardia case report. Eur Heart J Case Rep 2020;4:1–5.
8. Chugh A, Gurr HS, Krishnasamy K, et al. Spectrum of atrial arrhythmias using the ligament of Marshall in patients with atrial fibrillation. Heart Rhythm 2018;15:17–24.
9. Hayashi T, Fukamizu S, Mitouhashi T, et al. Peri-mitral atrial tachycardia using the Marshall bundle epicardial connections. JACC Clin Electrophysiol 2016;2:27–35.
10. Chik WW, Chan JK, Ross DL, et al. Atrial tachycardias utilizing the Ligament of Marshall region following single ring pulmonary vein isolation for atrial fibrillation. Pacing Clin Electrophysiol 2014;37:1149–1158.
11. Sasaki W, Nakamura K, Minami K, Sasaki T, Take Y, Naito S. Left atrial roof-dependent atrial tachycardia via the Marshall bundle. HeartRhythm Case Rep 2021;7:566–570.
12. Hwang C, Chen PS. Ligament of Marshall: why it is important for atrial fibrillation ablation. Heart Rhythm 2009;6:S35–S40.
13. Kim DT, Lai AC, Hwang C, et al. The ligament of Marshall: a structural analysis in human hearts with implications for atrial arrhythmias. J Am Coll Cardiol 2000;36:1324–1327.
14. Báez-Escudero JL, Morales PF, Dave AS, et al. Ethanol infusion in the vein of Marshall facilitates mitral isthmus ablation. Heart Rhythm 2012;9:1207–1215.