

# Diagnosing pseudo-conduction block across an anteromedial mitral ablation line: Limitations of bidirectional and differential pacing



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## Introduction

Macroreentrant or scar-related reentrant atrial tachycardias, including perimitral atrial flutter, are often seen in patients with a history of atrial fibrillation ablation.<sup>1</sup> Multiple strategies for ablating this arrhythmia have been proposed, but traditionally the lateral mitral isthmus line (mitral annulus to the left inferior pulmonary vein) has been the preferred approach. Achieving a line of block across the isthmus can prove difficult secondary to challenging anatomy, including epicardial connections, convective cooling from coronary sinus (CS) blood pool, and myocardial thickness. Pseudo-line of block across the mitral isthmus line is well described and is associated with an increased risk of recurrent arrhythmia.<sup>2</sup> An alternative ablative approach for perimitral flutter is to create an anteromedial line (from the septal mitral annulus to the right superior pulmonary vein [RSPV]). This line, although longer, avoids the issue of epicardial bridges in the CS and vein of Marshall (VoM); however, this ablative approach introduces other pitfalls that may complicate the interpretation of standard criteria for confirming trans-ablation line block. We describe herein an example of pseudo-line of block across an anteromedial line, despite findings of putative block as confirmed by bidirectional and differential pacing. The correct diagnosis was achieved with reinduction of the arrhythmia and demonstrating propagation

of very slow conduction through a circumscribed gap at 1 end of the ablation line with an ultra-high-density mapping catheter. This case highlights the importance of performing bidirectional and differential pacing at several sites along the course of a relatively long ablation line, since conduction across the line may be masked if testing is performed at a single trans-ablation site, particularly if it is several centimeters from the area of conduction breakthrough.

## Case report

An 85-year-old man underwent pulmonary vein isolation and radiofrequency (RF) ablation of the cavotricuspid isthmus line in 2015 for atrial fibrillation and atrial flutter, respectively. He did well for several years before presenting with atypical atrial flutter and undergoing repeat ablation. A persistent line of block across the cavotricuspid isthmus was confirmed, as was electrical isolation of all 4 pulmonary veins. The clinical tachycardia had a cycle length of 260 ms with counterclockwise activation recorded in the CS catheter. Activation mapping with an ultra-high-density multielectrode catheter (Orion; Boston Scientific, Cambridge, MA) was also consistent with counterclockwise perimitral atrial flutter as well as with a secondary loop around the right pulmonary veins. Proximal and distal sites along the CS catheter were entrained and showed postpacing intervals – tachycardia cycle length < 10 ms. RF ablation was performed (IntellaNav OI; Boston Scientific, Minneapolis, MN) to create an anteromedial mitral line. During ablation, the tachycardia first slowed and then terminated when ablating near the RSPV.

The anteromedial line was completed and a line of block was putatively confirmed based on prolonged conduction during pacing from either side of the ablation line (151 ms immediately inferior to the line and 175 ms superior to the line) (Figure 1A). CS activation also reversed when pacing above and below the line, respectively. Differential pacing above the ablation line also confirmed an apparent absence of slow conduction across the line (Figure 1B). Despite these

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## KEY TEACHING POINTS

- Achieving a line of block across the lateral mitral isthmus can prove difficult secondary to epicardial connections and convective cooling from the coronary sinus. Pseudo-block may result from sparing of epicardial structures.
- An alternative approach for ablating perimitral flutter is to create an anteromedial line (from the septal mitral annulus to the right superior pulmonary vein), which avoids the challenges of epicardial bridges in the coronary sinus and the vein of Marshall. However, pseudo-line of block may also occur across the relatively long anteromedial line. This can be detected by pacing at several sites along the ablation line. Conduction breakthrough may be masked if testing is evaluated at just a single site, particularly if it is relatively distant from the conduction gap.
- Activation mapping with an ultra-high-density mapping array along the mitral ablation line is the most definitive method for differentiating pseudo-line of block from complete line of block, and for precisely identifying the critical site of conduction breakthrough.

findings, programmed stimulation once again induced perimitral atrial flutter, which had a similar cycle length and identical intracardiac activation sequence as the clinical arrhythmia. Attempts at entrainment resulted in reproducible termination of tachycardia. During mapping of the tachycardia, the arrhythmia terminated with a catheter “bump” near the RSPV and could not be reinduced. Therefore, total mapping time was <30 s before termination. The limited activation map showed localized wavefront breakthrough in the anteromedial line near the RSPV, where a region of prolonged, highly fractionated potentials was observed (Figure 2). This site correlated to the site of catheter contact termination. The line was reinforced with additional RF lesions, beginning at the termination site and extending to the RSPV, eliminating any remaining atrial potentials. Testing for line of block was again performed and showed more marked conduction prolongation: 220 ms when pacing immediately inferior to the ablation line and 240 ms with pacing just superior to it (Figure 3). This suggested that initial prolongation of bidirectional conduction on either side of the ablation line (Figure 1A) was caused by slow conduction (pseudo-block) rather than by a true line of block.

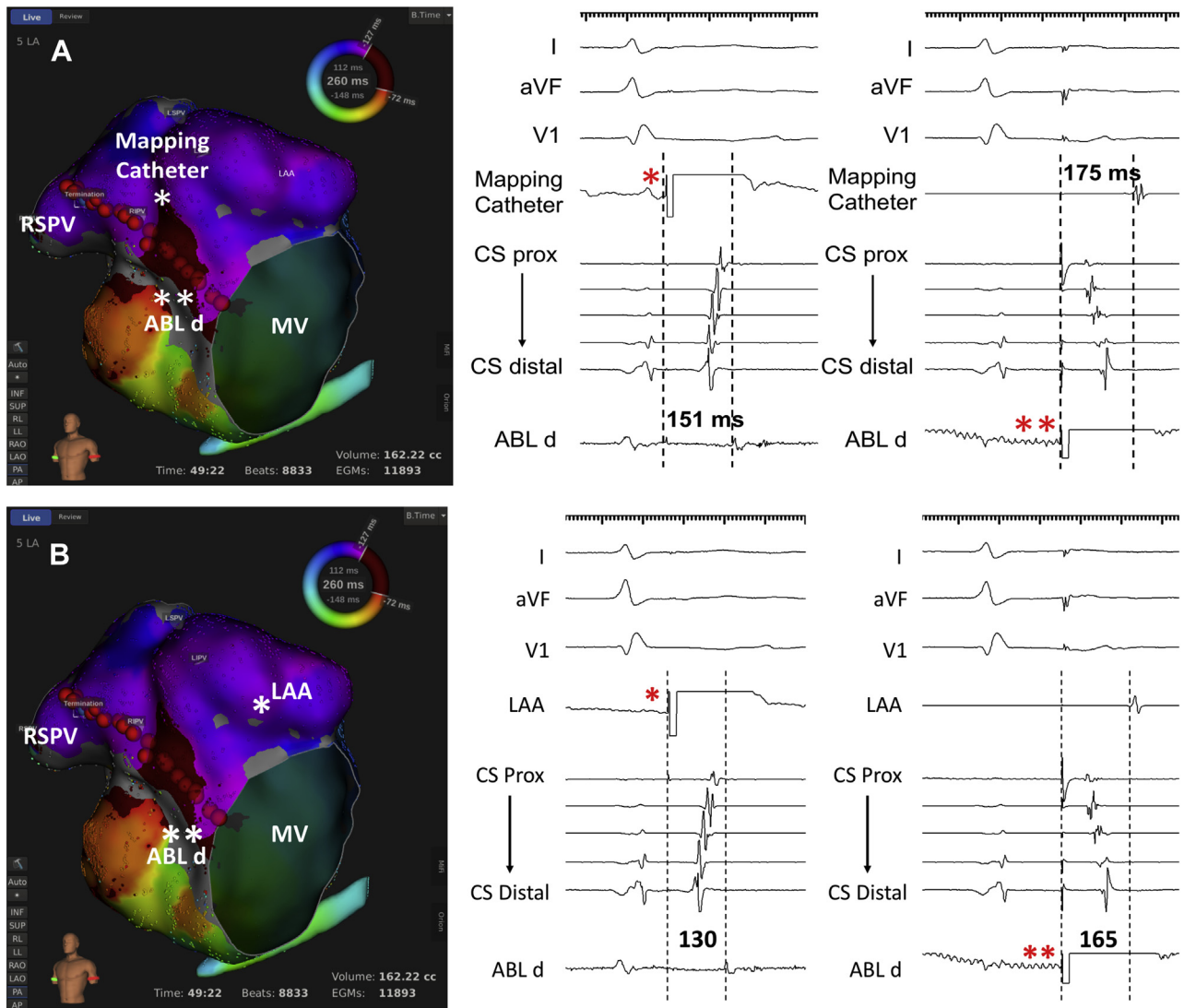
## Discussion

The principal finding of this study is that pseudo-line of block can occur following ablation of perimitral atrial flutter with

an anteromedial approach and that activation mapping with an ultra-high-density mapping array along the mitral ablation line is a definitive method for differentiating pseudo-line of block from complete line of block. The anteromedial approach avoids the confounding presence of epicardial bridging connections (ie, the VoM and its associated muscle bundles as well as CS muscular sleeves), the predominant cause of pseudo-block with the lateral approach. A less common cause of pseudo-block is persistence of slow endocardial conduction across the ablation line. Differential pacing maneuvers, with rare exceptions, are thought to reliably rule out slow conduction across an ablation line, but proved to be misleading in our study (Figure 1A and B). The diagnosis of pseudo-block across the ablation line was made by deploying an ultra-high-density mapping catheter along the ablation line and identifying a localized area of low-amplitude, fractionated potentials that slowly propagated across a circumscribed gap in the ablation line (Figure 2). A single ablation lesion at this site resulted in permanent conduction block across the ablation line. This case illustrates the potential limitations of differential pacing following anteromedial ablation for perimitral atrial flutter, and highlights the fundamental importance of noninducibility as an unambiguous means for distinguishing genuine bidirectional conduction block from pseudo-block.

Criteria for establishing lateral mitral isthmus conduction block secondary to ablation include the presence of widely spaced double potentials during CS pacing, changes in CS activation when pacing septal or lateral to the line, and bidirectional block. The response to differential pacing is thought to further distinguish between slow conduction across the ablation line and complete conduction block.<sup>3</sup> However, because propagation near the lateral mitral isthmus can also conduct through epicardial bridges, these criteria are not always reliable for assessing line of block in the mitral isthmus with a lateral ablative approach.<sup>4</sup> These bridges can take 1 of 2 forms: either epicardial CS muscular sleeves that are electrically coupled to the left atrial endocardium, and which allow for perpetuation of perimitral atrial flutter<sup>2,5,6</sup>; or muscle bundles associated with the VoM, and which provide a bridge for epicardial conduction across the mitral isthmus and participation in macroreentrant left atrial flutter.<sup>2,7,8</sup>

To circumvent the ablative challenges presented by these epicardial bridges, alternative approaches to lateral mitral isthmus ablation have been developed. An anterolateral approach (anterior mitral annulus to left superior vein, coursing anterior to the left atrial appendage [LAA]), obviates ablating within the CS but often increases interatrial conduction time (>50 ms), and may result in unintentional LAA isolation.<sup>9</sup> A superolateral mitral isthmus line (anterior mitral annulus to the left-sided pulmonary veins, coursing posterior to the LAA) is likely to avoid some epicardial bridges because ablation is performed along the distal course of the CS where muscle sleeves are less likely to extend, but this approach does not entirely eliminate the need to ablate VoM bridging muscle bundles. The superolateral approach is associated with a higher incidence of cardiac perforation,



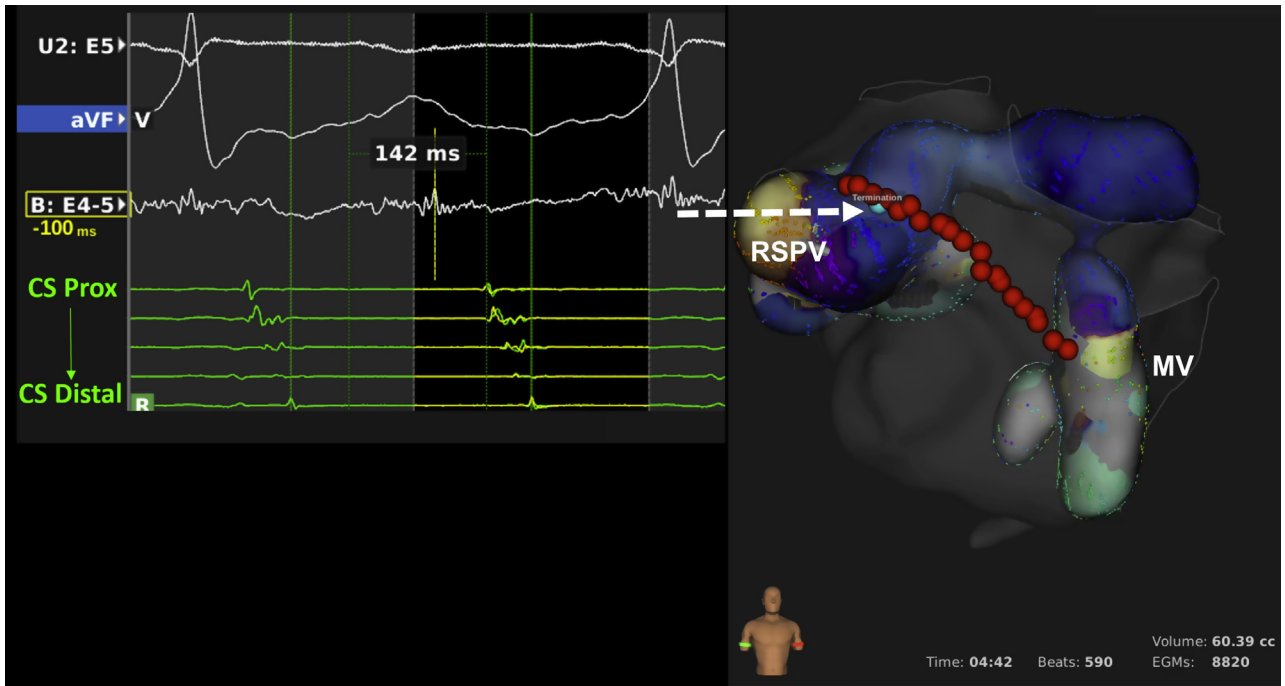
**Figure 1** **A:** Pseudo-block with differential pacing after initial anteromedial ablation lesion set. Pacing on opposite sides of the ablation line shows prolonged conduction times. Surface leads I, aVF, and V<sub>1</sub> are shown, as well as intracardiac electrograms from the coronary sinus (CS), distal electrodes from the ablation catheter (ABL d), and a selective recording from an ultra-high-density multielectrode mapping catheter. Pacing was performed at a cycle length of 500 ms in this figure and those that follow. MV = mitral valve; RSPV = right superior pulmonary vein; \* = site of pacing from the mapping catheter; \*\* = site of pacing from the distal ablation catheter. **B:** Differential pacing after initial ablation lesion set. Pacing after repositioning the mapping catheter to the left atrial appendage (LAA) results in shorter activation times compared with panel A (mapping catheter adjacent and superior to the ablation line), and is suggestive of line of block. Other abbreviations as previously described. \* = site of pacing from the left atrial appendage; \*\* = site of pacing from the distal ablation catheter.

likely secondary to thinner myocardium in this region.<sup>10</sup> The anteromedial mitral ablation line employed in our patient avoids discrete epicardial structures along its course. Its disadvantages include its long ablation lesion set and potential delay of left atrial activation.

In the present case, the patient had inducible tachycardia despite fulfilling criteria for conduction block during differential pacing. This likely occurred because the ablation line functionally behaved as if it were blocked, at least within the limits of spatial resolution of the recording electrodes and catheter proximity to the ablation line. Therefore, when pacing superior to (above) the ablation line, conduction proceeds more rapidly around the ablation line than across it; that is, the antidromic pacing wavefront collides with the orthodromic wavefront in close proximity to the ablation

line, thus appearing as if there were conduction block across the line (Figure 1A). In this scenario, the results of differential pacing would also be consistent with an apparent line of block (Figure 1B).

Other pacing maneuvers could have also conceivably identified slow conduction across the ablation line. Specifically, when encountering a long ablation line (eg, anteromedial line), bidirectional pacing should be performed close to the line, directly across from a second catheter deployed on the opposite side of the line. Ideally, pacing at the proximal, mid, and distal ends of the ablation line should also be performed. In the present case, we paced at the mid portion and proximal end of the ablation line, sites remote from the breakthrough site near the RSPV (distal end of ablation line). Because of the very long conduction time (175 ms), it was thought that

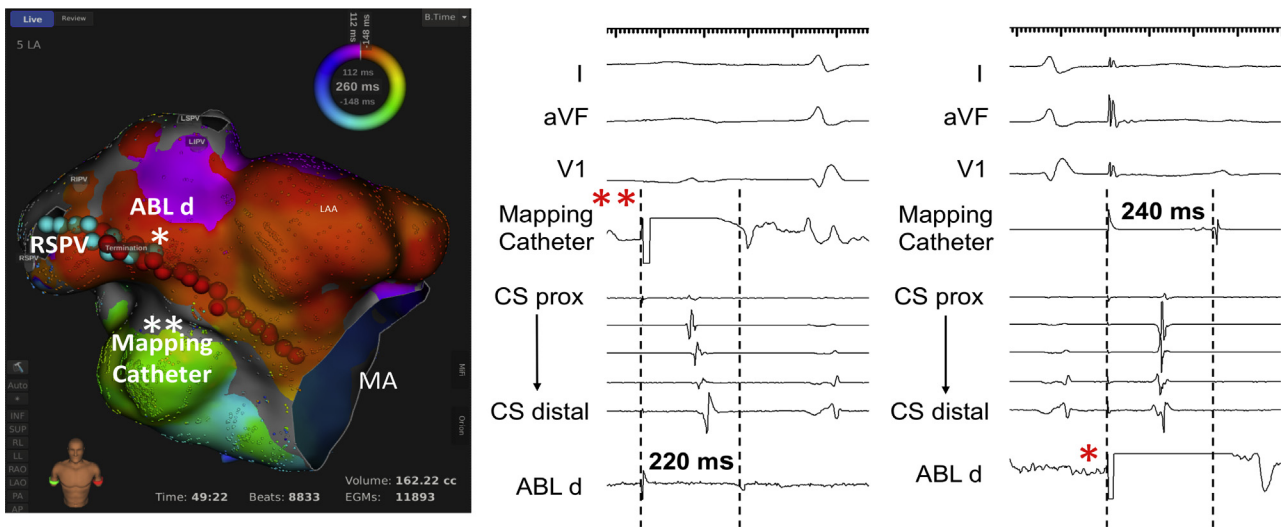


**Figure 2** Limited activation map of induced perimitral atrial flutter after the initial ablation line was completed. Activation map shows slow conduction and propagation through a localized gap in the ablation line (aqua circle) adjacent to the right superior pulmonary vein (RSPV). The local electrogram at the site of the gap demonstrates low amplitude and highly fractionated potentials, consistent with slow conduction. Ablation lesions are represented as having a diameter of 2 mm.

true conduction block was present. The distal aspect of the ablation line was therefore not tested, but likely would have disclosed conduction breakthrough near the RSPV.

We could not be certain that tachycardia induced after the initial ablation lesion set was identical to preablation perimitral atrial flutter. Although we believe that both preablation and postablation atrial flutter were due to perimitral flutter (similar intracardiac electrogram activation and similar cycle

length), we acknowledge that the latter tachycardia could have been due to roof-dependent flutter around the right-sided pulmonary veins. Extensive mapping was precluded because mechanical catheter termination of flutter near the RSPV rendered it noninducible. Nonetheless, the precise mechanism of the second tachycardia has little bearing on the relevance of our findings regarding pseudo-block, since both arrhythmias were dependent on conduction through



**Figure 3** Complete line of block demonstrated with pacing on each side of the ablation line after the localized gap was ablated. Conduction intervals are considerably longer compared with earlier assessment (Figure 1A), suggesting that previous apparent block was actually pseudo-block. Aqua dots represent additional ablation lesions after the initial anteromedial line was made. Abbreviations as previously described. \* = site of pacing from the distal ablation catheter; \*\* = site of pacing from the mapping catheter.

the anteromedial region and were only abolished when the true block was achieved.

High-resolution mapping adjacent to the ablation line is key to confirming the presence of pseudo-line of block during ablation of perimitral atrial flutter, either during pacing or during reinduction of the arrhythmia. An ultra-high-density mapping catheter can provide precise visualization of slow propagation through the ablation line, thus allowing for the identification of gaps, which may also be detected with differential pacing from several sites along the ablation line.

## References

1. Chugh A, Oral H, Lemola K, et al. Prevalence, mechanisms, and clinical significance of macroreentrant atrial tachycardia during and following left atrial ablation for atrial fibrillation. *Heart Rhythm* 2005;2:464–471.
2. Barkagan M, Shapira-Daniels A, Leshem E, Shen C, Anter E. Pseudoblock of the posterior mitral line with epicardial bridging connections is a frequent cause of complex perimitral tachycardias. *Circ Arrhythm Electrophysiol* 2019;12:e006933.
3. Jais P, Hocini M, Hsu LF, et al. Technique and results of linear ablation at the mitral isthmus. *Circulation* 2004;110:2996–3002.
4. Shah AJ, Pascale P, Miyazaki S, et al. Prevalence and types of pitfall in the assessment of mitral isthmus linear conduction block. *Circ Arrhythm Electrophysiol* 2012;5:957–967.
5. Antz M, Otomo K, Arruda M, et al. Electrical conduction between the right atrium and the left atrium via the musculature of the coronary sinus. *Circulation* 1998;98:1790–1795.
6. Chauvin M, Shah DC, Haissaguerre M, Marcellin L, Brechenmacher C. The anatomic basis of connections between the coronary sinus musculature and the left atrium in humans. *Circulation* 2000;101:647–652.
7. Wakabayashi Y, Hayashi T, Matsuda J, et al. Mitral isthmus ablation: is the conduction block completed? The importance of the Marshall bundle epicardial connections. *Circ Arrhythm Electrophysiol* 2016;9:e003049.
8. Hayashi T, Fukamizu S, Mitsuhashi T, et al. Peri-mitral atrial tachycardia using the Marshall bundle epicardial connections. *JACC Clin Electrophysiol* 2016;2:27–35.
9. Huemer M, Wutzler A, Parwani AS, et al. Comparison of the anterior and posterior mitral isthmus ablation lines in patients with perimitral annulus flutter or persistent atrial fibrillation. *J Interv Card Electrophysiol* 2015;44:119–129.
10. Maurer T, Metzner A, Ho SY, et al. Catheter ablation of the superolateral mitral isthmus line: a novel approach to reduce the need for epicardial ablation. *Circ Arrhythm Electrophysiol* 2017;10:e005191.