Epicardial recordings of Bachmann bundle activation during refractory mitral flutter with endocardial block

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Introduction

The left atrium is typically perceived as a thin-walled chamber without anatomic impediments to creating transmural ablation lesions. However, nontransmural lesions are a common cause of recurrent mitral annular atrial flutter (MAFL), representing ablation gaps in 3-D (depth). Percutaneous epicardial mapping in the region of the Bachmann bundle (BB) may provide mechanistic evidence for the difficulties associated with creating durable anterior block for MAFL. We report a case of MAFL with epicardial bridging over a previously blocked endocardial anterior linear lesion set.

Case report

A 67-year-old man presented with atrial flutter recurrence despite 3 prior procedures of pulmonary vein isolation with linear ablations. Spontaneous MAFL was detected in the previous procedure with failed mitral isthmus (MI) line ablation and finally terminated with additional ablation along the anterior wall. Only a unidirectional block of an inferolateral MI line was confirmed (proximal-to-distal coronary sinus activation with lateral pacing). However, atrial flutter recurred within 2 weeks.

Owing to multiple failed endocardial attempts, a minimally invasive maze procedure was offered, but the patient preferred an adjunctive percutaneous epicardial approach. Our approach to epicardial mapping for refractory atrial fibrillation has been previously described. Briefly, epicardial access was obtained prior to systemic heparinization and a steerable sheath was used to facilitate mapping of the posterior wall and sulcus between the left atrial appendage and left superior pulmonary vein. Activation mapping was performed of the clinical atrial flutter (tachycardia cycle length 360 ms), which indicated clockwise MAFL.

KEYWORDS Ablation; Atrial flutter; Bachmann bundle; Epicardial; Mitral isthmus; Transmurality

However, the prior endocardial anterior line appeared blocked with extensive low-voltage scar (<0.1 mV) and low-voltage zone (<0.5 mV) without discrete electrograms (EGM). A discontinuous activation pattern of endocardial activation was observed, with an incomplete leak at the roof and focal activation distal to the anterior line with propagation back towards it (Figure 1). Therefore, an epicardial breakthrough was suspected. Of note, there were split potentials within the coronary sinus and the epicardial lateral isthmus over a previously performed inferior mitral line. Additional advancement of a decapolar catheter (DecaNav, Biosense Webster, Diamond Bar, CA) through the epicardial aspect of the left-sided ridge resulted in direct recordings of the roof and anterior wall over the region of the BB. High-density mapping (4338 points) exhibited a long, local, fractionated EGM (103 ms) recorded over the epicardial anterior wall on the distal decapolar catheter, which bridged the flutter propagation during the endocardial activation gap (Figure 2, also shown in Supplementary Video).

Overdrive pacing was performed from the fractionated EGM on decapolar 3,4 was captured by pacing with 350 ms (1 mA and 1.0 ms), which demonstrated entrainment with concealed intracardiac fusion, proving that the epicardial BB was “in” the flutter circuit (Figure 2). Ablation targeting the fragmented epicardial EGM prolonged the cycle length.
immediately, with subsequent flutter termination to sinus rhythm lateral to this site (40 W, 17–20 mL/min). Additional endocardial ablation was performed directly across these successful sites to optimize transmurality. The patient was noninducible for atrial flutter as the procedural endpoint. At 2-month follow-up, the patient has remained free of recurrence.

Discussion
The functional relevance of epicardial structures like the vein of Marshall and septopulmonary bundle have been recently described in refractory left atrial flutter. MAFL is commonly encountered after atrial fibrillation ablation and terminated by creating linear lesion sets across the MI, although epicardial structures like the vein of Marshall and coronary sinus may serve as a barrier for MI ablation transmurality. While an anterior line is predominantly performed targeting thinner atrial myocardium, the thickness of epicardial structures in the region of BB is a barrier to achieving completely transmural lesions.

BB is a primary interatrial connection that spans from the superior vena cava to the left atrial appendage along the epicardial left atrial superior anterior wall. Piorkowski and colleagues demonstrated the requirement for epicardial ablation to achieve anterior block in 78% of cases as well as localized epicardial reentry within abnormal BB fibers. However, simultaneous epi-endo mapping with nonuniform activation between the 2 surfaces during macroreentry was not reported. The creation of biatrial reentry over BB with endocardial block has been previously described. Miyazawa and colleagues reported an MAFL across the endocardial anterior scar, while with indirect evidence indicating a detouring circuit conducting at the epicardial anterior wall. In our case, the refractory MAFL in the presence of endocardial anterior line block with focal distal breakthrough led to a suspected epicardial breakthrough. Direct epicardial recordings over the Bachmann bundle region exhibited a fragmented EGM with concealed entrainment, indicating a slowly conducting activation bridge over the BB region during MAFL. In this regard, the case highlights the difficulties associated with creating durable anterior block across the MI, and highlights complex 3-D activation in refractory cases of atrial flutter bridged by epicardial structures. We can only conclude that the recorded EGM is in the region of the BB, as precise correlation would require direct visualization and/or pathologic confirmation.

Conclusions
The BB consists of epicardial fibers that serve as an important barrier to achieving transmural conduction block across the anterior wall and is fully accessible via a percutaneous epicardial approach. Activation and entrainment mapping were consistent with critical participation of epicardial activation over the region of the BB to sustain refractory MAFL. Discontinuous endocardial activation patterns signify the presence of epicardial bridging, which highlights the need to consider 3-D activation in refractory cases of atrial flutter with prior endocardial block. To the best of our knowledge,
this is the first report of simultaneous epicardial and endocardial mapping during refractory left atrial macroreentry that provides direct evidence of epicardial bridging across the BB region as the mechanism of discontinuous endocardial activation.

Appendix A
Supplementary data
Supplementary data to this article can be found online at https://doi.org/10.1016/j.hrcr.2020.03.001.

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