Case Presentation
A 68-year-old man with hypertension, dyslipidemia, obstructive sleep apnea, chronic obstructive pulmonary disease, sick sinus syndrome status post dual chamber pacemaker, and a history of 2 pulmonary vein isolation ablations for persistent atrial fibrillation was evaluated in our institution for recurrent atrial flutter.

He underwent a complete electrophysiology study. A duodecapolar catheter was used, so that the distal 10 poles were inside the coronary sinus (CS) and the proximal were in the right atrium. The underlying rhythm was atrial flutter with a cycle length of 350 ms and ostial-to-distal activations in the CS. Responses to entrainment were consistent with a left atrial origin. After a trans-septal puncture, a 3-dimensional map of the left atrium was performed using the NavX system (St Jude Medical, St Paul, MN) and a circular duodecapolar catheter, which was consistent with counterclockwise perimital flutter (PMF). However, the entire cycle length was not present in the mitral annulus (Figure A and B). An extensive scar was present in the previously ablated mitral isthmus, where no endocardial signals were detectable. Sites neighboring the scar (1–5) in Figure C and D were either early (1–2) or late (3–4–5) relative to a reference (Ref) signal (Figure D).

To ascertain the role of the vein of Marshall (VOM) in providing with epicardial connections bypassing the endocardial mitral isthmus scar, we cannulated the VOM via the CS. A sheath was inserted in the CS through the right internal jugular vein. A LIMA (left internal mammary artery) angioplasty guide catheter was inserted in the CS with its tip facing posteriorly and superiorly in the right anterior oblique projection (Figure E). Contrast injection identified the VOM, which was subsequently cannulated with a 0.014″ angioplasty wire and 2×6 mm angioplasty balloon (Medtronic, Minneapolis, MN; Figure E and F). VOM signals were obtained by connecting the angioplasty wire in a unipolar fashion using alligator clips with the indifferent electrode clamped to the skin. By slightly exposing the wire beyond the balloon, only the exposed wire tip (∼3 mm) would serve as an electrode, which could be located by the NavX system, and from which unipolar signals could be sequentially recorded at different depths of the VOM. Local activation times from different levels of the VOM were added to the NavX map (Figure C and D) and were shown to complete the reentrant activation sequence with the portion of the cycle length that was missing in the endocardial map.

Up to 4 injections of 98% ethanol were injected via the angioplasty balloon at different depths of the VOM. The first injection led to termination of the PMF (Figure G and H). Bidirectional perimital block was subsequently proven by differential pacing. No radiofrequency ablation was required.

Discussion
PMF is a macroreentrant tachycardia that typically occurs after left atrial ablation for atrial fibrillation. PMF is commonly treated by ablation in the so-called mitral isthmus, although this has been consistently reported as suboptimal. Failures can occur due to poor catheter contact, motion, or the thickness of the lateral ridge of the left atrium, as well as epicardial cooling of CS or VOM flow.1,2 It has also been hypothesized to occur due to remaining epicardial connections that bypass the endocardially ablated line3; however, definitive proof of such connections and their anatomic substrate has been lacking.

We have previously demonstrated that the VOM is part of the PMF circuit and that ethanol infusion in this vein assists in achieving mitral isthmus bidirectional block.4 Herein, we report the first proof a mechanistic role of VOM in recurrent PMF; by showing epicardial signals from the VOM in areas endocardially ablated, and the successful treatment by ethanol infusion.

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Disclosures
None.

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References


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Figure. Vein of Marshall (VOM) and perimetal flutter. A and B, 3-dimensional map of the left atrium illustrating counterclockwise perimetal flutter with extensive scar in the previously ablated mitral isthmus. The full reentrant cycle length is complete only after adding VOM activation times in C and D. Sites neighboring the scar (1–5) were either early (1–2) or late (3–4–5) relative to a reference (Ref) signal. Local activation times from different levels of the VOM were shown to complete the reentrant activation sequence with the portion of the cycle length that was missing in the endocardial map (VOMp and VOMd). E–G, Contrast injection identified the VOM, which was subsequently cannulated with a 0.014" angioplasty wire and 2×6 mm angioplasty balloon. H, Perimetal flutter terminates with VOM ethanol infusion. CSd indicates coronary sinus distal; CSp, coronary sinus proximal; RAd, right atrial distal; and RAp, right atrial proximal.
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