Isolation of left pulmonary veins by ethanol injection in the vein of Marshall

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Introduction

The vein of Marshall (VOM) is localized on the posterior part of the left atrium. It drains the blood from the left atrial endocardium to the coronary sinus. There are epicardial fibers that travel along the VOM. The endocardial radiofrequency ablation of these fibers, to achieve mitral isthmus isolation, has been challenging, because the fibers are surrounded and isolated by fatty tissue.[1] Ethanol infusion of the VOM has been proposed to solve this issue.

The VOM ethanol infusion serves a dual purpose: it targets potential epicardial fibers bridging the coronary sinus and left atrium, and it impacts the left atrial endocardium that drains into the VOM.[2–4] In our practice, the ethanol infusion of the VOM is pivotal for achieving durable block along the mitral line. In 700 patients it has shown a low rate of complications[5] and can be incorporated in the treatment of persistent atrial fibrillation and atrial tachycardia.[1,2,6–8]

In this report, we detail a case involving a patient whose left pulmonary veins had reconnected following a pulsed field ablation (PFA) procedure conducted 2 months earlier. Notably, these veins were successfully isolated after an ethanol application in the VOM without reablation. This case underscores the potential endocardial or epicardial influence of the VOM ethanol infusion.

Case report

The patient is a 64-year-old man presenting with exertional dyspnea and palpitations after having undergone a PFA procedure for paroxysmal atrial fibrillation 2 months prior. During the previous procedure the veins showed entrance and exit block and were therefore isolated. The current procedure was scheduled owing to the patient's symptoms and the electrocardiogram findings of atrial tachycardia with a fixed cycle length, likely to be perimitral flutter.

Besides the atrial tachycardia, the patient had arterial hypertension and obstructive sleep apnea. His current medication consisted of flecainide, nebivolol, and rivaroxaban.

The baseline electrocardiogram at the beginning of the procedure showed a supraventricular tachycardia (Figure 1a).

The procedure was performed under conscious sedation with the Rhythmia (Boston Scientific, Cambridge, MA) mapping system. A steerable sheath (Agilis NxT; Abbott, St Paul, MN) was inserted by right femoral access. A quadripolar catheter (Dynamic XT; Boston Scientific, Marlborough, MA) was positioned in the coronary sinus. Left atrial access was obtained by transseptal puncture with an intracardiac needle (BRK 1; St Jude Medical, St Paul, MN).

We performed a left atrial activation and voltage map of the tachycardia using a basket mapping catheter (IntellaMap Orion; Boston Scientific, Cambridge, MA). The activation map showed perimitral flutter at a cycle length of 230 ms with a 2:1 ventricular activation (Figure 1b). In addition to the perimitral flutter, the map showed collision of activation fronts on the posterior wall of the left atrium as well as reconnected left pulmonary veins (Figure 2a). The posterior and superior part of the left pulmonary veins showed a line of block, marked as the black line in Figure 2a, with an inferior gap. The ridge and left superior vein were also connected; however, they were activated through the left inferior

KEY TEACHING POINTS

- Vein of Marshall (VOM) ethanol infusion has effect on the epicardium as well as epicardial fibers.
- In our case a pulmonary vein, treated with pulsed field ablation in a previous procedure, was isolated using solely ethanol injection in the VOM.
- In our case the perimitral flutter cycle length was not affected by the administration of ethanol in the VOM. Endocardial ablation prolonged the cycle length, followed by termination and mitral line block during coronary sinus ablation.

KEYWORDS Atrial fibrillation; AF mechanisms; Vein of Marshall; Ethanol infusion; Pulsed field ablation
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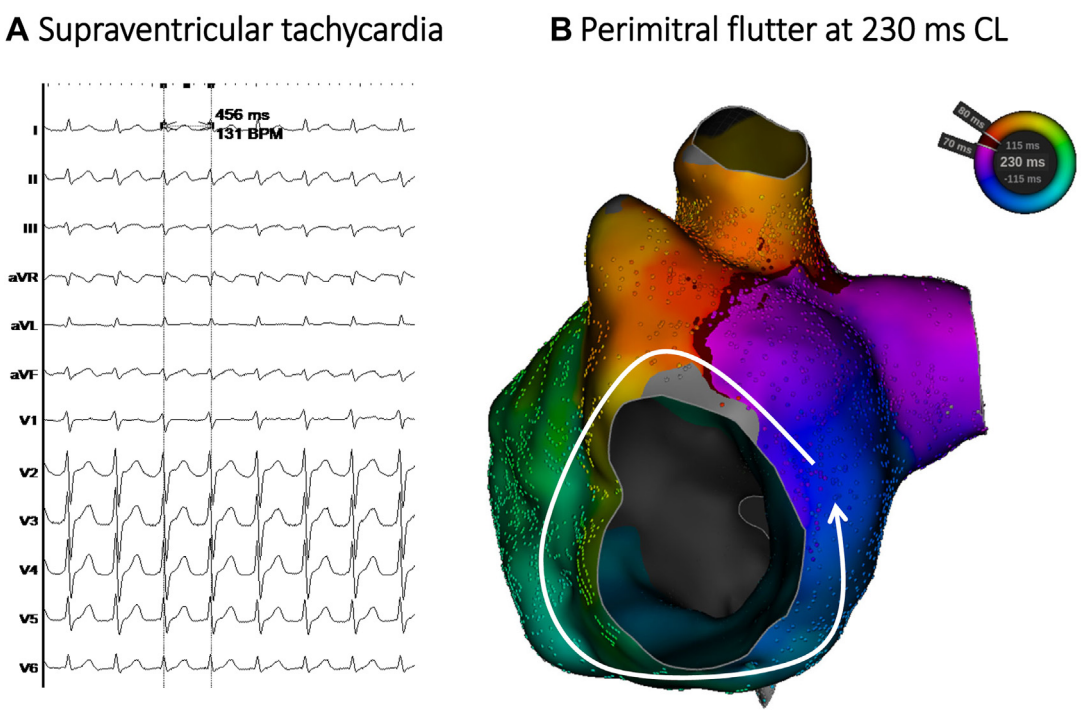

Figure 1 Depiction of the situation at the beginning of the procedure. **a:** Twelve-lead electrocardiogram of a supraventricular tachycardia at the beginning of the procedure. **b:** The corresponding left atrial activation map in a left lateral view. A perimitral flutter with a cycle length of 230 ms was observed, as well as reconnected left pulmonary veins.

pulmonary vein. The corresponding voltage map showed a reconnection and high-voltage signals in the left pulmonary veins. The remaining atrium showed no relevant low-voltage zones, with the exception of the circular lesions surrounding the pulmonary veins owing to the previous PFA.

Pulmonary vein reconnection being rare after PFA in our center, we hypothesized that this reconnection was possibly due to epicardial connections along the VOM. The ethanol infusion also required for the perimitral flutter ablation was therefore performed.

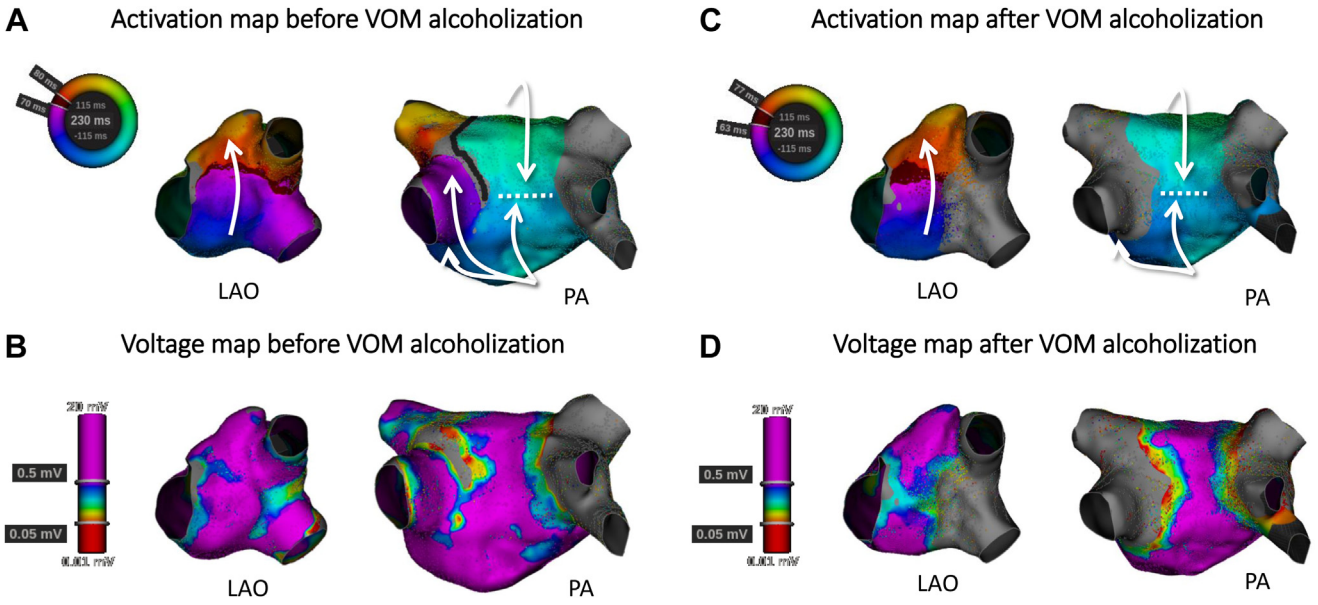

Figure 2 Activation and voltage maps of the left atrium (left anterior oblique and posterior view) before and after the vein of Marshall (VOM) ethanol infusion. **a:** The initial activation map, on which the perimitral flutter was observed. The left pulmonary veins are reconnected, even though a line of block can be identified in the superior/posterior part (*black line*). On the posterior wall, collision of a superior and an inferior activation front can be observed. **b:** Voltage map before the VOM ethanol infusion with high voltage in the left pulmonary veins. **c:** Activation map after the VOM ethanol infusion, with a persisting perimitral flutter and the collision of activation on the posterior wall, but the left veins are now isolated. **d:** Voltage map after the VOM ethanol infusion.

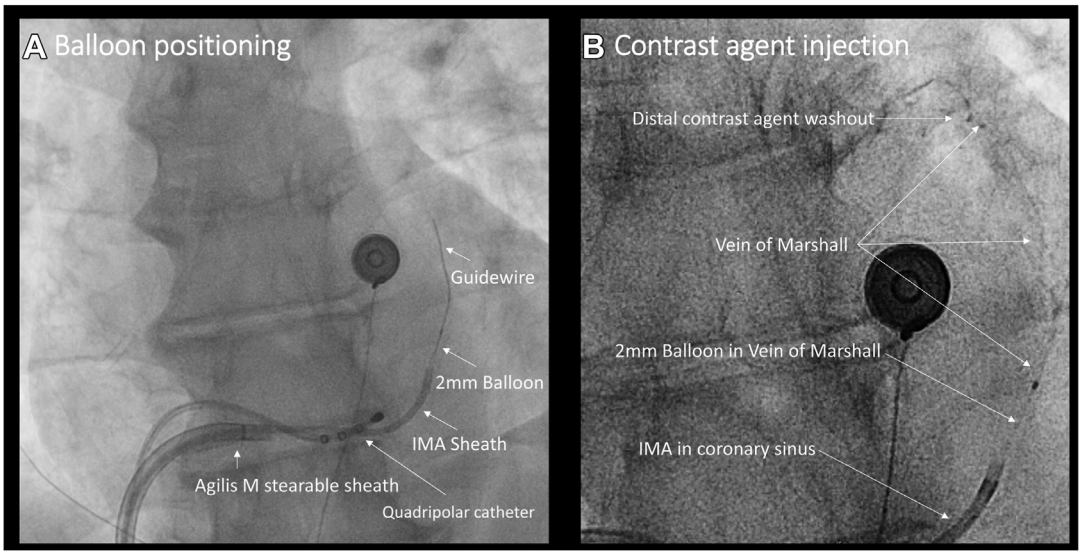

Figure 3 Fluoroscopic images of the heart acquired in anterior-posterior view during the ethanol infusion of the vein of Marshall (VOM). **a:** Balloon placement. The Agilis steerable sheath (Abbott, St Paul, MN) is inserted in the coronary sinus and an internal mammary artery (IMA) sheath is advanced to the orifice of the VOM. Once the orifice is identified a guidewire is placed inside the VOM, along which the balloon can be positioned at the proximal part of the VOM. The quadripolar catheter was used to record coronary sinus electrograms during the procedure and is not essential to the ethanol infusion. **b:** Enlarged image of a contrast agent injection. The contrast agent injections are performed to confirm the complete occlusion of the VOM. The contrast agent shows the VOM with the distal end of the vein and some contrast agent washout toward the left atrium.

For the ethanol infusion of the VOM, the coronary sinus was cannulated with the steerable sheath (Agilis NxT; Abbott, St Paul, MN). An IMA sheath (5F left internal mammary artery; Medtronic, Minneapolis, MN) was positioned proximally inside the coronary sinus lumen, and contrast (iodine) was injected to localize the VOM ostium. Subsequently, an angioplasty wire (Whisper 0.014; Abbott) was advanced inside the VOM lumen and used to position a preloaded over-the-wire balloon (MINI TREK, 1.5–3 mm diameter and 6–15 mm length; Abbott) within the first 15 mm of its proximal portion (Figure 3a). After inflation at 2 atm and wire removal, a selective angiography was performed through the wire port to confirm balloon occlusion and visualize VOM arborization. Three successive injections of 3 mL of absolute ethanol (96%) were slowly administered over 3 minutes, with selective VOM angiogram repeated each time to confirm balloon stability and occlusion (Figure 3b). Owing to washout of contrast agent at the distal part of the VOM, an additional ethanol injection of 3 mL was administered.

The atrial tachycardia was not impacted by ethanolization of the VOM, either on cycle length or on the coronary sinus activation, but we decided to map the left veins before any ablation at the mitral isthmus, to evaluate the effect of the VOM in this unusual case of left pulmonary vein reconnection after PFA. The activation map showed the collision on the roof, similar to the first map, and the unchanged perimitral flutter at 230 ms cycle length (Figure 2c). However, the left pulmonary veins were disconnected. The corresponding voltage map showed an increase of the blocked area in the lower part of the left inferior pulmonary vein and a small area along the mitral line, with no relevant increase of low-voltage tissue seen in the rest of the atrium (Figure 2d).

We then proceeded with mitral isthmus ablation on the endocardial side with a radiofrequency ablation catheter (IntellaNav Stablepoint; Boston Scientific, Cambridge, MA). During this ablation, a cycle length increase of 10 ms, to 240 ms, was observed for the tachycardia. Following this, ablation in the coronary sinus led to tachycardia termination and restoration of the sinus rhythm with complete bidirectional posterior mitral isthmus line block.

Discussion

From our point of view there are 2 possible mechanisms that could have caused the isolation of the pulmonary vein by VOM ethanol infusion.

The first entails epicardial connections, following the ligament of Marshall, which are connected to the coronary sinus and the left atrium and can conduct either from endocardial to endocardial, from coronary sinus to coronary sinus or between the coronary sinus and the endocardium,[7] thereby forming multiple possible conduction bridges behind already isolated endocardial surface and permitting connection despite an intact endocardial line of block. The ethanol infusion of these fibers would then cause the isolation of the pulmonary vein.

The second possible mechanism is that an endocardial gap existed, which was affected by the ethanol infusion of the VOM and completed the circumferential line of block.

A combination of both mechanisms is possible, since both are affected by the ethanol infusion of the VOM.[9,10]

Conclusion

In our case, an ethanol infusion, without additional ablation, isolated the left pulmonary veins, which had previously

reconnected after PFA. This emphasizes the potential role of epicardial connections from the VOM for left pulmonary veins resistant to PFA.

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