Vein of Marshall Cannulation for the Analysis of Electrical Activity in Patients With Focal Atrial Fibrillation

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Background—Whether or not the muscle bundle within the ligament of Marshall (LOM) can serve as the origin of focal atrial fibrillation (AF) is unknown.

Methods and Results—A total of 28 consecutive patients with paroxysmal AF underwent balloon-occlusion coronary sinus angiograms to identify the vein of Marshall (VOM). Attempts were then made to advance a 1.5-French electrophysiological catheter into the VOM via the coronary sinus orifice. In 17 of the 28 patients (10 of 17 were men aged 38±15 years), cannulation was successful. Double potentials were registered in 8 of these 17 patients. The first potential corresponded with local left atrial activation. The second potential was shorter and narrower than the first. The sequence of activation in the second potential in the VOM was proximal to distal. In 6 patients with direct VOM recordings, we documented that the origin of AF was in the muscle bundle within the LOM. Radiofrequency catheter ablation aimed at the insertion site of the VOM successfully terminated AF in 4 of these 6 patients.

Conclusions—(1) It is possible to cannulate and to record electrical potentials from the VOM. (2) The characteristics of the double potentials within the VOM suggest that the second potential is from the muscle bundle (Marshall bundle) within the LOM. (3) The Marshall bundle may be the origin of focal AF in some patients. **(*Circulation*. 2000;101:1503-1505.)**

Key Words: arrhythmias ■ electrophysiology ■ catecholamines ■ catheter ablation ■ potentials

Recently, we demonstrated that isoproterenol infusion in the isolated canine left atrium induced automatic activity from the ligament of Marshall (LOM, a fold of the pericardium from a developmental vestige of the left primitive veins).[1–3] In left atria from 2 dogs that underwent rapid atrial pacing, the automatic activity from the LOM induced rapid and irregular activity that simulated AF.[1] In a separate study of human patients with paroxysmal AF,[4] we recorded double potentials from the left superior pulmonary vein that were similar to those registered in the LOM in canines.[1,3] We hypothesized that the LOM may serve as a source of activation in paroxysmal focal AF.[5] However, because we did not directly record from the vein of Marshall (VOM), we were not able to confirm that the Marshall bundle was, in fact, the origin of activation during AF. We have since developed techniques to directly record from within the VOM. The purpose of the present study was to test the hypothesis that the Marshall bundle within the LOM is a source of focal AF in humans.

Methods

All patients included in the study had failed ≥3 antiarrhythmic drugs, had recurrent symptomatic AF, and had no structural heart disease. The electrophysiological study and the radiofrequency ablation were performed in the same session using standard procedures.[6] Written, informed consent was obtained before the study. All antiarrhythmic drugs were discontinued for ≥5 half-lives. No patients were treated with amiodarone. Fentanyl and Versed (midazolam) were used for sedation. Three quadripolar catheters were inserted via the femoral veins and positioned in the high right atrium, atrioventricular junction, and right ventricle. A luminal decapolar catheter was inserted via the right internal jugular vein into the coronary sinus and advanced to the most distal position. Via a transeptal approach, a standard ablation catheter was inserted into the left atrium. The patients were anticoagulated with heparin as soon as the catheters were placed. The activation times were determined with the usual criteria.[7]

If atrial tachyarrhythmias did not occur spontaneously, we attempted to induce them with an isoproterenol (1 to 5 μg/min) infusion and, if necessary, by withdrawing sedatives. The left atrium was mapped to determine the earliest activation site at the onset of the atrial tachyarrhythmia. Radiofrequency ablation was done with a target temperature of 55°C delivered to the site of earliest activation. Multiple applications were performed until all atrial arrhythmias were eliminated.

VOM Recordings

In 28 consecutive patients, we performed balloon-occlusion angiograms to identify the VOM. We then attempted to cannulate the VOM using a 1.5-French quadripolar catheter (Cardima). Four

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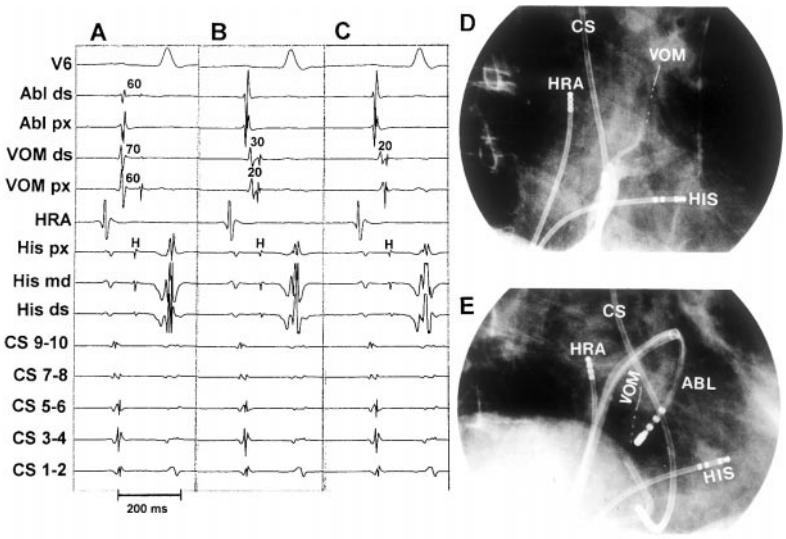

Figure 1. VOM recordings. A through C show recordings with VOM catheter in most distal, middle, and most proximal (near the coronary sinus) positions, respectively, during sinus rhythm. Only 1 beat was shown because recordings were stable from beat to beat. Ablation catheter was in left superior pulmonary vein in A and near Marshall bundle insertion sites in B and C. D shows angiogram of coronary sinus and VOM, with a 1.5-French electrode catheter inserted. E shows ablation catheter when moved to insertion of VOM. Abl indicates ablation; CS, coronary sinus; ds, distal; HRA, high right atrium; His, His bundle; md, middle; and px, proximal.

recording electrodes spaced 2 mm apart were built on the catheter to register 2 bipolar electrograms. Electrograms were acquired during sinus rhythm and during spontaneous or induced atrial tachyarrhythmias. Filter and gain settings for VOM recordings were the same as those for recordings from the coronary sinus and pulmonary veins.

Data Analyses

The data were presented as mean±SD. Student's *t* tests were used to compare the means. $P \leq 0.05$ was considered significant.

Results

The patients included 18 men and 10 women with a mean age of 36±11 years. The left ventricular ejection fraction was 0.61±0.04, and the left atrial size averaged 30±2 mm by echocardiographic examinations. On average, 3.0±1.2 antiarrhythmic drugs were used per patient before the ablation procedures.

VOM Recordings

In 19 of the 28 patients, the VOM was visualized by balloon-occlusion coronary sinus angiography. The VOM is the first atrial branch of the coronary sinus, and it runs obliquely toward the left superior pulmonary vein. The VOM was successfully cannulated with the recording catheter in 17 of the 19 patients. On average, 21±9 minutes were needed for successful cannulation. These 17 patients were 38±15 years of age and included 10 men and 7 women. In comparison, the other 11 patients included 7 men and 4 women who had an average age of 40±2 years (P=NS).

In 8 of the 17 patients in whom the VOM was successfully cannulated, double potentials were registered both inside the left superior pulmonary vein and inside the VOM (Figure 1). The first potential corresponded to local left atrial activation, and the second was shorter and narrower than the first. During sinus rhythm, the activation

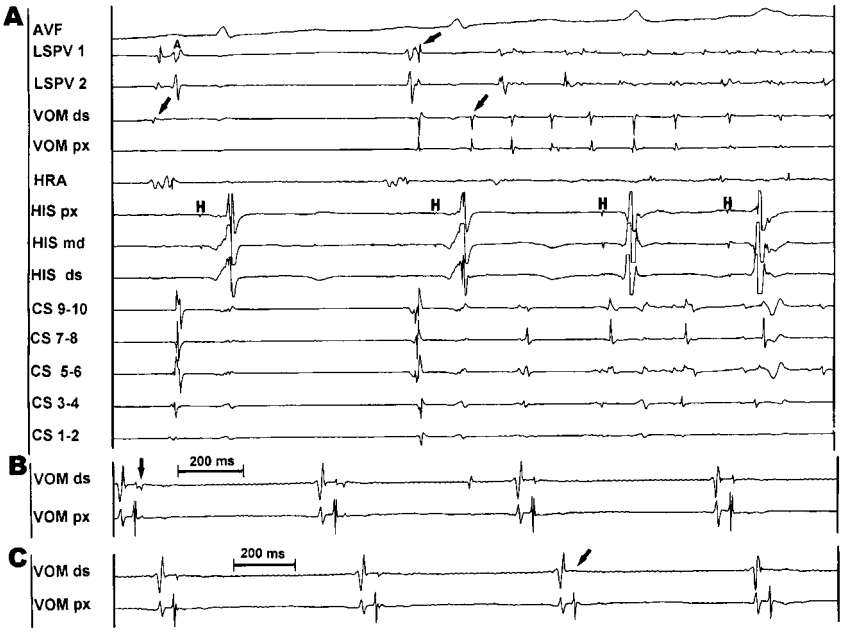

Figure 2. VOM recording at onset of AF and during radiofrequency ablation. A, At onset, catheter within VOM was pushed to most distal position and registered only activation of Marshall bundle. Ablation catheter was in left superior pulmonary vein (LSPV). First beat is a fusion beat between sinus rhythm and a premature contraction from the LOM (first arrow). Second beat shows sinus rhythm, with double potentials in left superior pulmonary vein (second arrow). At onset of AF, Marshall bundle electrogram was first to activate (third arrow), and it continued to activate at a high rate, with 2:1 conduction to atria. B, At beginning of radiofrequency energy application, Marshall bundle electrogram split into 2 potentials (arrow). In third sinus beat, second of these potentials moved earlier but did not conduct its impulse to atria or proximal Marshall bundle. C, A recording was also made 30 s later, during same radiofrequency energy application. Remaining electrogram at distal VOM was eliminated (arrow). A indicates atrial electrogram; H, His bundle potential. Other abbreviations as in Figure 1.

sequence of the second potential within the VOM was proximal to distal. The timing of the second potential in the left superior pulmonary vein was similar to that registered in the distal VOM (Figure 1A). The interval between the first and the second potential was 64.3±9.2 ms in the pulmonary vein and 73.0±8.5 ms in the distal VOM (P=NS). The temporal separation between the local atrial electrograms and the VOM electrograms gradually shortened as the catheter pulled back from the most distal to the most proximal position in the VOM (Figures 1A to 1C).

Fusion of the 2 electrograms (Figure 1C) indicated that the catheter was at the site where the Marshall bundle inserts into the muscle tract surrounding the coronary sinus or the left atrium. A total of 8 episodes of spontaneous AF occurred in 6 of these 8 patients. Rapid activation of the Marshall bundle preceded the onset of AF (Figure 2). Radiofrequency catheter ablation (Figure 2), guided by the VOM catheter (Figure 1E), successfully terminated atrial tachyarrhythmias and prevented its reinduction in 4 of these 6 patients. No radiofrequency applications were needed in the pulmonary veins. During a follow-up of 3.9±2.6 months, none of these 4 patients had recurrent AF.

In 2 of the 17 patients, the VOM recording showed double potentials during sinus rhythm. However, AF originated from the right superior pulmonary vein. The VOM catheter failed to register double potentials in the remaining 7 patients, and AF did not originate in this area.

Among the 28 patients who underwent VOM angiographic studies, 2 had a dissection of the VOM and 1 had a dissection of coronary sinus. These complications did not result in pericardial effusion, tamponade, or hemodynamic compromises. No long-term complications occurred in patients in the study.

Discussion

The major findings of this study are as follows. (1) It is possible to cannulate and to record electrical potentials from the VOM. (2) The characteristics of the double potentials within the VOM suggest that the second potential is from the muscle bundle within the LOM (Marshall bundle). (3) The Marshall bundle may serve as the origin of focal AF in some patients.

LOM as a Source of Focal AF

In humans, the sinus node is not the only pacemaker. Boineau et al[8] demonstrated widely distributed atrial pacemaker complexes in the human heart. In the isolated, perfused canine right atrium, ectopic pacemaker activity was most often found near the sinus node or the crista terminalis.[9] These pacemakers may exhibit different responses to norepinephrine and acetylcholine. Scherlag et al[3] reported that sympathetic stimulation could also induce left atrial ectopic activity. To study the source of these ectopic activities, we performed a computerized mapping study in the isolated-perfused canine left atrium.[1] We found that isoproterenol can cause automatic rhythm with this preparation. On the basis of these findings, we hypothesized that the Marshall bundle may serve as a source of focal AF in humans.

In the present study, we successfully cannulated the VOM and recorded sharp potentials directly from the catheter within the VOM. Because the VOM is an epicardial structure and the recording site was not close to the pulmonary veins, it is unlikely that these sharp second potentials originated from the extension of the atrial musculature into the pulmonary veins.[10] Rapid activation of the Marshall bundle might serve as a trigger of atrial arrhythmias, including AF. Finally, we were able to use the VOM catheter as a guide for radiofrequency ablation. A radiofrequency lesion placed in the posterolateral left atrium between the Marshall bundle insertion and the ostium of the left inferior pulmonary vein resulted in successful treatment of the focal AF. This finding suggests that the trigger of the focal AF episodes resides not within the pulmonary veins, but in the Marshall bundle.

Limitations of the Study

One limitation of the present study is that successful cannulation of the VOM was not achieved in all patients. The potential complications related to the dissection of the veins are other limitations. Furthermore, in some patients, double potentials were not registered within the VOM. This procedure (VOM cannulation and recording) may not be applicable to all patients. However, these limitations do not invalidate our conclusion that in some patients, the Marshall bundle is a source of focal AF.

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