

## Case Report

# Left Atrial-Pulmonary Vein Reentrant Tachycardia Following Pulmonary Vein Isolation

DIONYSSIOS LEFThERiOTIS, FEIFAN OUYANG, KARL-HEINZ KUCK

*II. Med. Abteilung, Asklepios Klinik St. Georg, Hamburg, Germany*

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Pulmonary vein isolation has become the cornerstone of ablation for atrial fibrillation. However, it is sometimes followed by the development of left atrial macro-reentrant tachycardia. There is evidence to suggest that the myocardial sleeves within the pulmonary veins may be implicated in the mechanism of the tachycardia. Here we present a case of a post-ablation macro-reentrant tachycardia, in which the pulmonary veins and left atrial myocardium were involved.

Since pulmonary veins (PVs) have been described as an important source of atrial fibrillation (AFib), pulmonary vein isolation (PVI) has become the cornerstone of AFib ablation in clinical practice.<sup>1,2</sup> Recent studies have shown that, in patients with paroxysmal or short standing persistent AFib, circumferential ablation in a wide area around the PV antrum yields better outcomes than ablation in small areas around the PV ostia.<sup>3</sup>

Although complete PVI following circular lesions has been associated with a better clinical outcome, it has also been related with the development of left atrial (LA) macro-reentrant tachycardia, especially if combined with linear lesions.<sup>4-8</sup> Following PVI, atrial tachycardia with a stable cycle length has been observed in a considerable proportion of the cases.<sup>7,9,10</sup> Previous studies suggest that, in these cases, the myocardial sleeves located within the PVs are involved in the mechanism of the tachycardia.<sup>8,11,12</sup> In this report, we present a case of a LA-PV macro-reentrant tachycardia, in which both PV and LA myocardium were involved. This tachycardia was related with the presence of a double gap in the circumferen-

tial lesion deployed during a previous PVI procedure.

## Case presentation

A 52-year-old hypertensive and hyperlipidemic man was admitted to our department for catheter ablation of symptomatic atrial tachycardia. Six months earlier, the patient had undergone PVI for persistent AFib. At that time, transthoracic echocardiography showed that the left atrial diameter was 44 mm. Left ventricular dimensions and function were within the normal range (ejection fraction: 55%).

During the initial procedure, two continuous circumferential antral lesions were performed in sinus rhythm: one around the right PVs and another around the left PVs. A double transseptal, single Lasso technique was followed, as described later. The end point of the procedure was the absence or dissociation of all PV potentials for at least 30 minutes after ablation, as documented by a circular (Lasso) catheter sequentially positioned in each PV. Following successful PVI, no complications were observed and the patient remained in sinus rhythm.

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*Address:*  
Dionyssios I.  
Leftheriotis

16 Kountouriotou St.  
152 33 Halandri  
Athens, Greece  
e-mail: [dleftheriotis@gmail.com](mailto:dleftheriotis@gmail.com)

He was discharged from hospital with anticoagulant (phenprocoumon) and antiarrhythmic treatment (flecainide, 200 mg per day).

During a scheduled follow-up examination three months later, the patient reported episodes of palpitations and fatigue during the previous week. Atrial tachycardia was recorded on a 12-lead surface electrocardiogram (ECG) and bisoprolol was added to the patient's treatment, without significant improvement in his symptoms. After a further three months, the patient was referred to the hospital for atrial tachycardia ablation. Three days prior to the procedure, phenprocoumon was discontinued and low-molecular weight heparin was administered as a bridge-treatment until ablation. Transthoracic echocardiography performed one week before admission to the hospital did not show any significant difference compared to that performed prior to the first ablation. On the day of admission the patient was in sinus rhythm. Transesophageal echocardiography was performed the day before ablation in order to rule out left atrial thrombi.

The second ablation procedure started with sedation of the patient using a continuous intravenous infusion of propofol. An octapolar deflectable-tip 7 Fr catheter (Webster D-type, Biosense-Webster, USA) was introduced into the coronary sinus (CS) via the left subclavian vein. Another octapolar deflectable-tip 6 Fr catheter (Webster D-type, Biosense-Webster, USA) was advanced to the His bundle region via the right femoral vein. Subsequently, a double transeptal puncture was performed and heparin was infused to maintain an activated clotting time of 250 to 300 seconds throughout the procedure. Additionally, heparinized normal saline was continuously infused through the transeptal sheaths, in order to avoid thrombus formation or air embolism. An open irrigated, 3.5-mm deflectable-tip catheter (NaviStar Thermocool™, Biosense-Webster) was introduced into the left atrium for atrial three-dimensional electroanatomical reconstruction and mapping. A decapolar circular mapping catheter of 15 mm diameter (Lasso, Biosense-Webster) was also advanced to the left atrium for evaluation of PV conduction. Bipolar electrograms were recorded at a bandpass of 30 to 500 Hz (EPMedSystems, West Berlin, New Jersey).

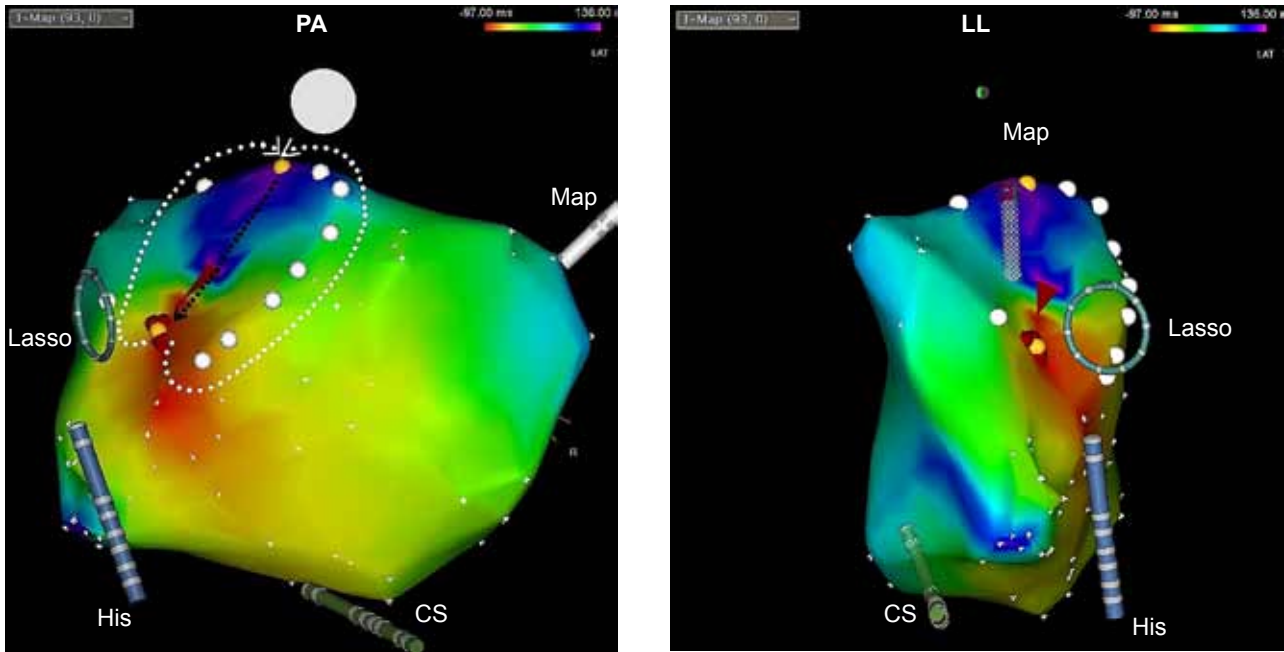
Atrial tachycardia with a cycle length (CL) of 360 ms and 2:1 atrioventricular conduction was induced by burst-pacing from the CS. During tachycardia, 1:1 left PV conduction across the previous circular lesions performed in the first ablation was detected by

the Lasso catheter. The right PVs were found to be isolated. After exclusion of right atrial tachycardia, entrainment mapping from the CS was performed, demonstrating fused intracardiac activation with a post-pacing interval (PPI) more than 30 ms longer than the tachycardia cycle length (TCL), ruling out perimitral macro-reentry. Following that, left atrial activation mapping was performed relatively to the timing reference derived from the CS catheter, using the Carto 3 system (Biosense-Webster, USA). Additionally, selective biplane PV angiographies in the right anterior oblique 30° and left anterior oblique 40° projections were performed, and the PV ostia were defined and tagged.

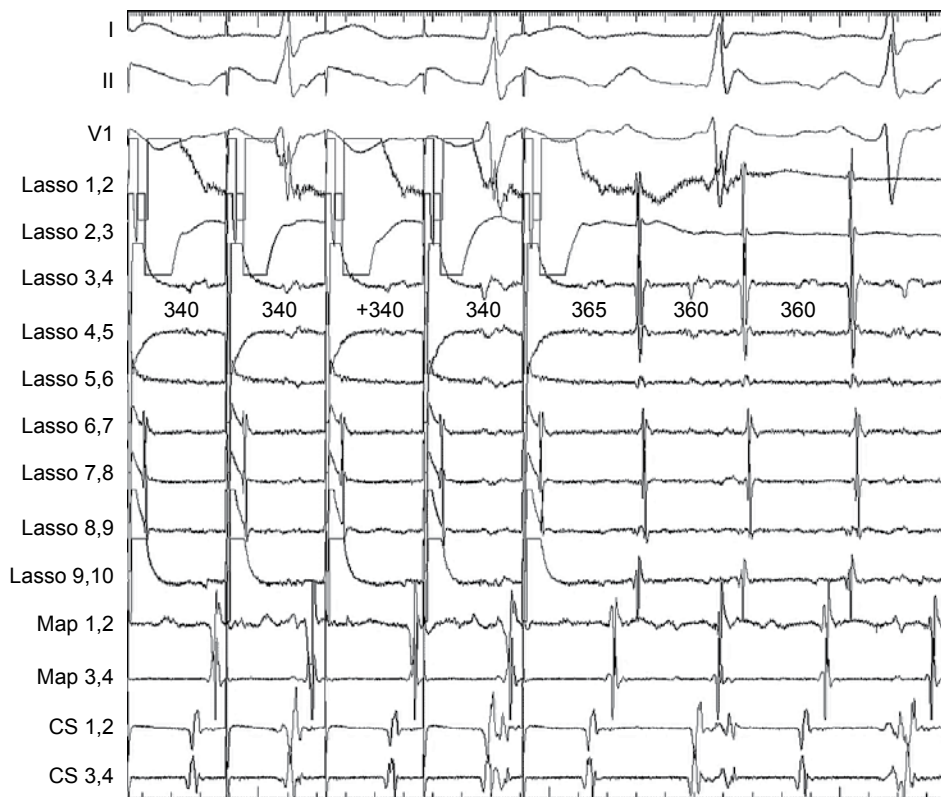
The earliest atrial activation was detected in the inferior part of the circular lesion-line deployed around the left PVs during the first ablation (Figure 1). The latest atrial activation was located in the superior part of the circular lesion (upper site of the left PV antrum). During tachycardia, less than 50% of the TCL was mapped, indicating a focal underlying mechanism. However, entrainment from within the left PV via the circular catheter (Figure 2) and also from the LA demonstrated a PPI almost identical to the TCL (PPI-TCL <30 ms). Furthermore, captured PV activation during entrainment demonstrated a P-wave morphology identical to that during tachycardia. These observations were consistent with the presence of a macro-reentrant circuit involving both left PV and LA, propagating within the PV (see also Figure 1). Initially, radiofrequency current was applied at the site of latest activation (entrance of the reentrant circuit), followed by tachycardia termination and restoration of sinus rhythm (Figure 3). Tachycardia termination occurred following left atrial activation not conducted to the PV. Furthermore, the PV activation sequence in sinus rhythm was changed compared to that during tachycardia. These observations confirmed that ablation was performed at the entrance site of the circuit and indicated the presence of another conducting gap along the PV ostial line. Mapping around the left PV ostia during SR revealed a second conduction gap (earliest PV activation) near the site of earliest activation (exit of the circuit). Ablation at that site resulted in complete left PV isolation (Figure 4). Following this, repetitive burst-pacing from the CS did not induce atrial tachycardia.

## Discussion

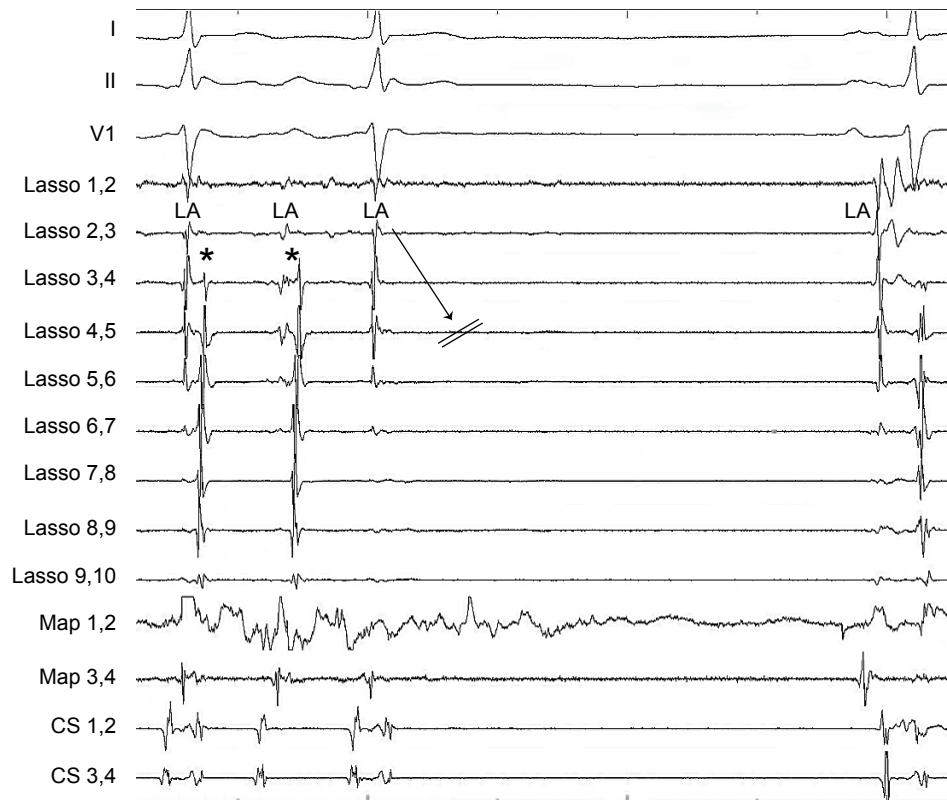
In this report, we present a case of a recently recog-



**Figure 1.** Three-dimensional electroanatomic reconstruction of the left atrium is shown in the posterior-anterior (PA) and left lateral (LL) views. The left pulmonary vein (PV) ostium is tagged by white dots. Activation propagates from the inferior region of the left PV (area of earliest activation, red color) to the superior region of the left PV (area of latest activation, blue color), as indicated by the white dotted lines. Subsequently, activation continues within the vein, as the black dotted line indicates schematically. The yellow dot in the superior region of the PV ostium indicates the area of ablation that resulted in tachycardia termination (entrance site). The deep red dots in the inferior region of the ostium indicate the sites where radiofrequency current was applied and the yellow dot indicates the area of complete PV isolation (exit site). Also shown are the circular catheter located in the left inferior PV (Lasso), the mapping catheter (Map), the catheter positioned in the coronary sinus (CS), and the catheter for the His bundle recording (His).



**Figure 2.** Surface ECG leads I, II, and V<sub>1</sub>, along with intracardiac electrograms from the Lasso catheter (Lasso 1,2 up to Lasso 9,10), the mapping catheter (Map 1,2 and Map 3,4), and the coronary sinus catheter (CS 1,2 and CS 3,4). Entrainment pacing at a cycle length of 340 ms is performed from the Lasso catheter, which is located within the left pulmonary vein (PV). The post-pacing interval (365 ms) was almost identical to the tachycardia cycle length (360 ms), indicating the contribution of the PV to the reentrant circuit.



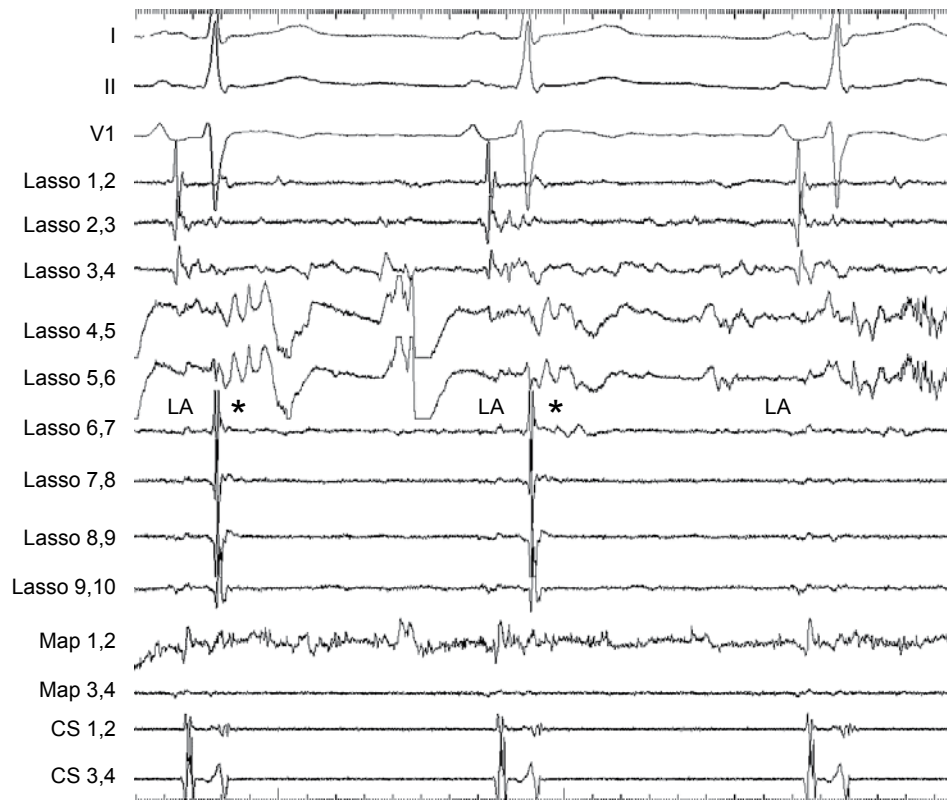
**Figure 3.** Surface electrocardiographic leads I, II, and  $V_1$ , along with intracardiac electrograms from the Lasso catheter (Lasso 1,2 up to Lasso 9,10), the mapping catheter (Map 1,2 and Map 3,4), and the coronary sinus catheter (CS 1,2 and CS 3,4). During ablation in the superior area of the pulmonary vein (PV) ostium (upper yellow dot in Figure 1), the tachycardia was terminated. The last three beats of the tachycardia and the first beat in sinus rhythm are presented. During tachycardia, a far-field left atrial (LA) electrogram, along with a local PV electrogram (asterisk) are recorded by the Lasso catheter. The earliest PV activation is recorded at Lasso 8,9 bipole. Tachycardia was terminated with a conduction block from LA to PV (arrow and double line). This is indicative of ablation at the entrance site of the reentrant circuit. During sinus rhythm (last beat), the activation sequence in the left PV was changed compared to that in tachycardia (earliest activation at Lasso 6,7), indicating a different entrance site (gap).

nized, iatrogenic, post-PVI reentrant tachycardia, mapped and ablated using the Carto 3 electroanatomic system, following a double transeptal, single Lasso technique. Electrophysiologists should be aware of this novel arrhythmia; otherwise, they may be led to the wrong diagnosis due to some misleading characteristics observed during tachycardia mapping.

The presence of a double electrical gap along a previously deployed circular lesion around the PV antrum is a prerequisite for the development of LA-PV tachycardia. One of these gaps is the exit point (site of earliest left atrial activation) and the other is the entrance point (site of latest left atrial activation). Due to this circuit, LA-PV tachycardia is characterized by a regular CL and has to be differentiated from other post-AFib ablation regular tachycardias, such as perimitral atrial flutter, which is the most commonly observed macro-reentrant arrhythmia.<sup>7</sup> Entrain-

ment mapping from the CS is useful for this purpose, as described in the present case. Additionally, 1:1 LA-PV and PV-LA conduction relapse, as well as concealed entrainment from sites inside the PV, and also from the LA near the gap area, are consistent with the reentrant mechanism. Furthermore, pacing within the vein results in a P-wave with a morphology similar to that of the tachycardia.

However, since part of the circuit is located within the PV, activation mapping of the entire cycle length of the tachycardia is not possible. This observation might be misleading and the tachycardia could be interpreted as a focal one. Exposure of the tachycardia mechanism is important for the subsequent mapping technique and ablation sites, since both gap areas must be detected and complete PVI must be achieved. Radiofrequency current deployment at the entrance site of the circuit results in tachycardia ter-



**Figure 4.** Surface ECG leads I, II, and V<sub>1</sub>, along with intracardiac electrograms from the Lasso catheter (Lasso 1,2 up to Lasso 9,10), the mapping catheter (Map 1,2 and Map 3,4), and the coronary sinus catheter (CS 1,2 and CS 3,4). In sinus rhythm, radiofrequency current was applied at the exit site of the reentrant circuit (red dots in Figure 1), and the pulmonary vein (PV) was isolated (inferior yellow dot in Figure 1). Note the artifacts on Lasso 4,5 and 5,6 near the ablation spot. During the first two beats, a far-field left atrial (LA) electrogram, along with a local PV electrogram (asterisk) are recorded by the Lasso catheter. During the last beat, no PV activity is recorded, indicating complete isolation of the vein.

mination, as shown in this case. The PV activation sequence in sinus rhythm following ablation of the entrance site (latest LA activation) is different compared to that during tachycardia, because LA activation is conducted to the PV through another gap.

The hypothesis that an LA-PV reentrant tachycardia could possibly be developed following AFib ablation was initially put forward by Merino, four years ago.<sup>13</sup> Recently, Satomi and his colleagues reported eight cases of post-PVI LA-PV reentrant tachycardia, studied by the double Lasso technique.<sup>14</sup> These cases were diagnosed over an almost two-year period, among 45 patients with left atrial macro-reentrant tachycardia after an initial PVI procedure. In the majority of those patients, tachycardia exhibited a focal pattern, as in the present case, while in two of them more than 80% of the tachycardia CL could be mapped, which was consistent with a typical macro-

reentrant pattern. Satomi observed an identical PV activation sequence between tachycardia and sinus rhythm when radiofrequency energy was applied at the exit site of the reentrant circuit. This was attributed to the fact that the LA activation in sinus rhythm is conducted to the PV myocardial sleeves through the same gap at the entrance site. The sequence was altered when radiofrequency was applied at the exit site, in accordance with our observations.

The patients included in Satomi's report developed LA-PV atrial tachycardia within 50 days following PVI.<sup>14</sup> Our report may suggest that LA-PV atrial tachycardia could occur even later after a PVI procedure. Additionally, according to Satomi's report, LA-PV atrial tachycardia seems to be an infrequent entity. Interestingly, in the same department, four cases of documented and treated LA-PV tachycardias have been ablated over the last five months. As PVI be-

comes a more widely applied treatment for AFib, it may be shown in the future that the incidence of LA-PV tachycardia is higher. It is possible that a more systematic diagnostic approach in every case of regular atrial tachycardia after PVI could demonstrate that this novel tachycardia is not as infrequent as was initially thought.

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