

# Atrial Tachycardia Arising from an Epicardial Site with Venous Connection Between the Left Superior Pulmonary Vein and Superior Vena Cava

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**Atrial Tachycardia.** We describe a case of atrial tachycardia originating from an epicardial site with a venous connection between the left superior pulmonary vein (LSPV) and superior vena cava (SVC). Initial endocardial mapping with multiple electrodes catheters demonstrated early endocardial activation at both the SVC and LSPV. However, radiofrequency applications at the SVC failed to terminate the atrial tachycardia. With three-dimensional electroanatomic mapping, the earliest endocardial activation was found to be in the left atrial appendage (LAA). However, radiofrequency energy applications at multiple sites in the LAA resulted in only transient termination of the tachycardia. A left atrial angiogram demonstrated a venous connection between the LSPV and SVC, overlying the LAA. An application of radiofrequency energy with a saline-irrigated ablation catheter delivered at the earliest activation site in the LAA terminated the tachycardia. The tachycardia did not recur during 18-month follow-up. (*J Cardiovasc Electrophysiol*, Vol. 14, pp. 540-543, May 2003)

*atrial tachycardia, endocardial mapping*

## Introduction

Recent clinical studies have demonstrated that the thoracic veins are common sources of atrial arrhythmias.<sup>1-3</sup> Both clinical and anatomic studies have demonstrated the presence of

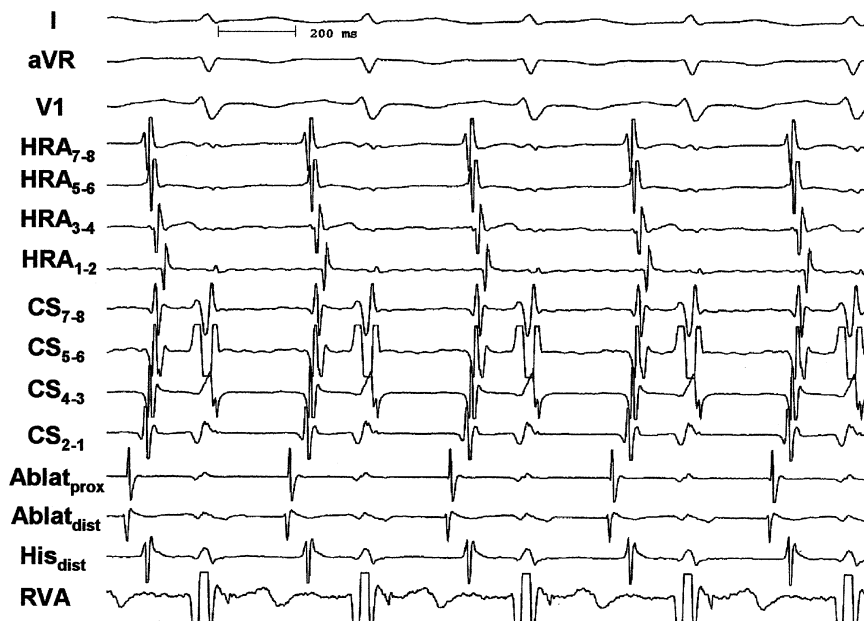
electrical connections between pulmonary veins or other venous structures such as the ligament of Marshall.<sup>4,5</sup> This case report describes a patient with incessant atrial tachycardia that was found to be arising at a site near or within a venous connection between the left superior pulmonary vein and the superior vena cava.

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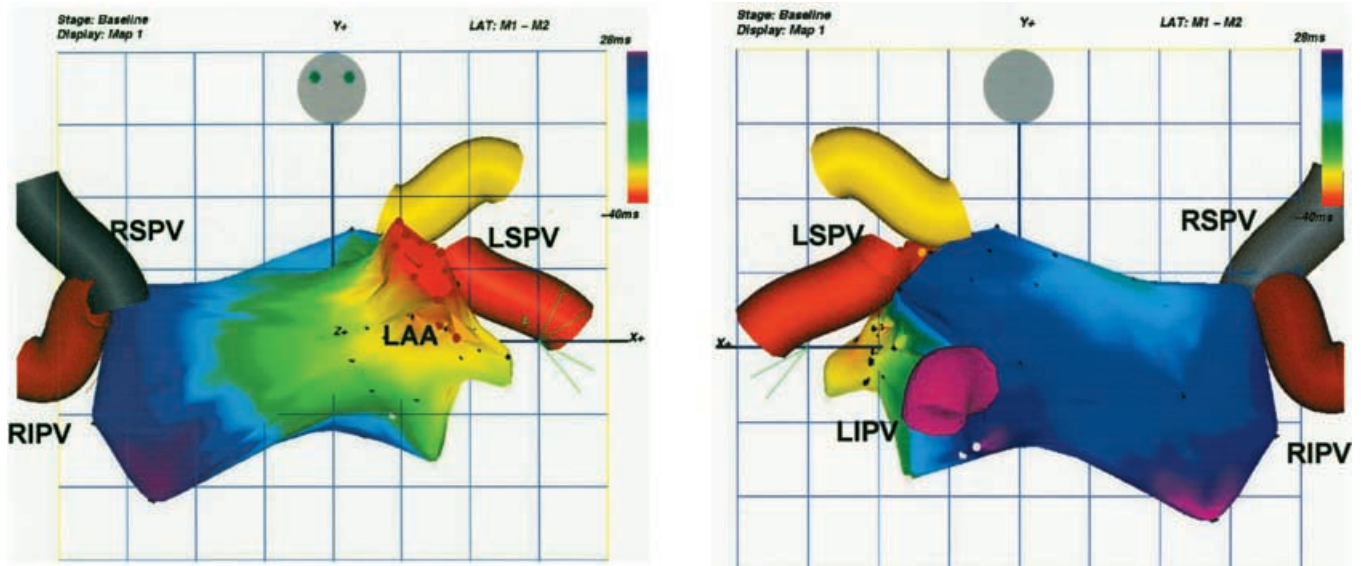
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## Case Report

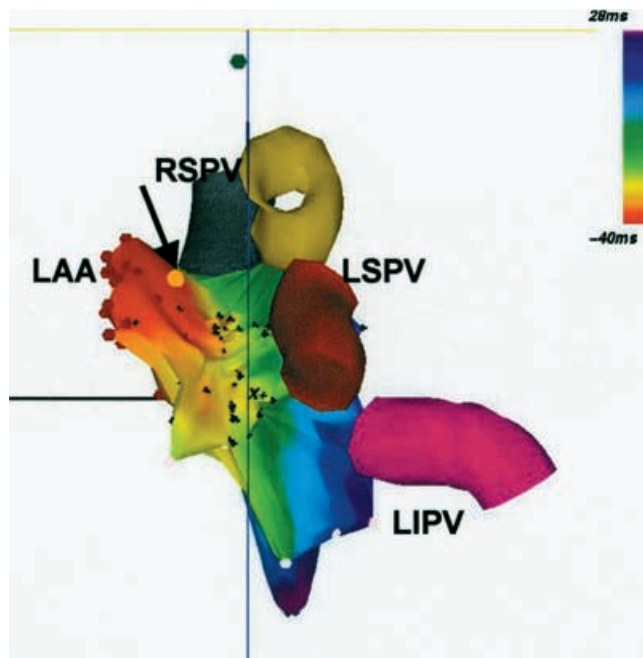
**A 14-year-old boy with almost incessant, drug-refractory atrial tachycardia was referred for radiofrequency catheter ablation. He had a 2-year history of palpitations, and ECG showed**



**Figure 1.** Surface ECG leads I, aVR, and V<sub>1</sub> and intracardiac electrogram recordings during atrial tachycardia. Early endocardial activation (asterisk) was recorded at the junction of the superior vena cava and high right atrium (HRA<sub>7-8</sub>) and at the ostium of the left superior pulmonary vein (Ablat<sub>dist</sub>). Ablat = ablation catheter; CS = coronary sinus; His = His bundle; HRA = high right atrium; RVA = right ventricular apex.



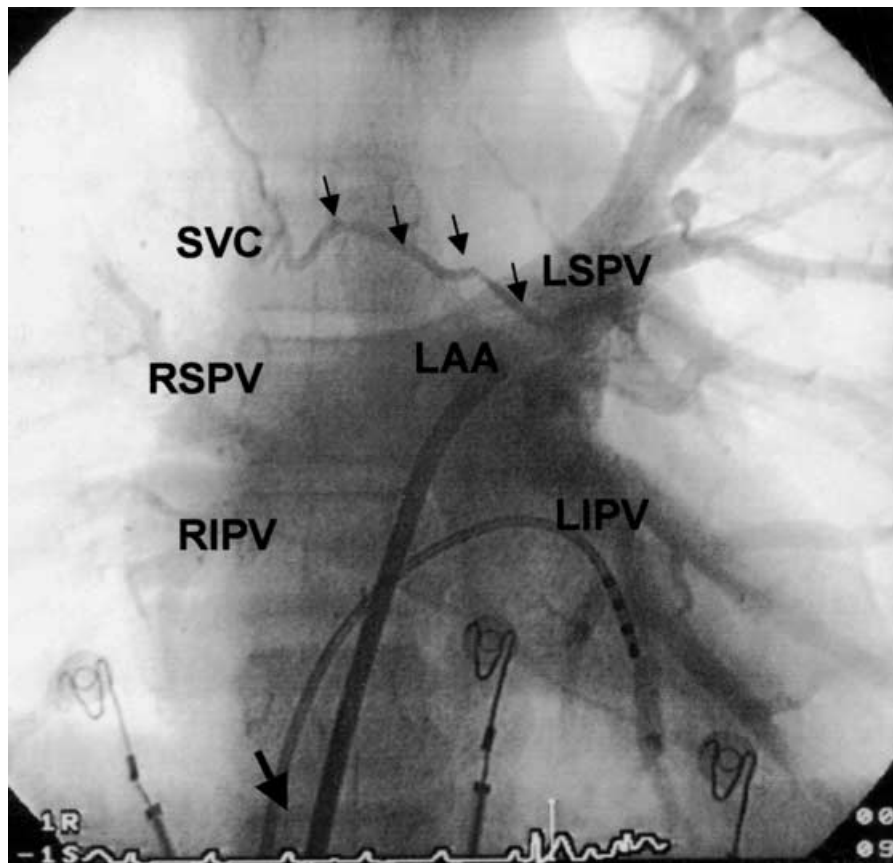
**Figure 2.** A: Anteroposterior view of isochronal activation map of the left atrium during atrial tachycardia shows the earliest activation (red) is located inside the left atrial appendage. The red tags represent sites of radiofrequency energy applications. B: Posteroanterior view of isochronal activation map of the left atrium during atrial tachycardia demonstrates more rapid conduction (light blue) at the superior left atrium where Bachmann's bundle is located. LAA = left atrial appendage; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein.



**Figure 3.** Left lateral view of an isochronal activation map of the left atrium during atrial tachycardia. Earliest activation (red) is located inside the left atrial appendage (LAA). The orange tag at the posterior aspect of the LAA represents the successful ablation site with a saline-irrigated ablation catheter. Although the posterior wall of the LAA is about 1 cm away from the left superior pulmonary vein (LSPV), it is in close proximity to the superior vena cava, which should be located anterior to the right superior pulmonary vein. This suggests that the venous connection between the LSPV and the superior vena cava runs an oblique course and lies at the posterior wall of the LAA. LIPV = left inferior pulmonary vein; RSPV = right superior pulmonary vein.

frequent episodes of narrow-QRS complex tachycardia at a rate of 150 beats/min. Echocardiography showed no structural abnormalities. An electrophysiologic procedure performed at another hospital demonstrated the tachycardia to be an atrial tachycardia. Radiofrequency catheter ablation was attempted at multiple sites of early endocardial activation at the junction between the superior vena cava and right atrium, but the tachycardia could not be terminated.

An electrophysiologic procedure was performed after informed consent was obtained. Decapolar electrode catheters were positioned in the coronary sinus and against the high anterolateral right atrial wall. A 7-French mapping and ablation catheter with a deflectable 4-mm distal electrode (Biosense Webster, Diamond Bar, CA, USA) was positioned at the ostium of the left superior pulmonary vein through a transseptal puncture. Endocardial mapping demonstrated endocardial activation times of  $-30$  msec and  $-70$  msec (relative to the onset of the P wave) in the high right atrium and left superior pulmonary vein, respectively (Fig. 1). Three-dimensional, nonfluoroscopic, electroanatomic mapping (CARTO, Biosense Webster) was performed and demonstrated that the earliest endocardial activation ( $-85$  msec) was in the left atrial appendage, with relatively late endocardial activation at the superior left atrium (endocardial activation time  $-30$  msec, Figs. 2 and 3). Radiofrequency energy applications at multiple sites with early endocardial activation in the left atrial appendage terminated the tachycardia (Figs. 2 and 3); however, the atrial tachycardia consistently recurred within 5 minutes after each radiofrequency energy application. Left atrial angiography performed during bradycardia induced by 20 mg of intravenous adenosine triphosphate<sup>6</sup> demonstrated a venous connection between the left superior pulmonary vein and superior vena cava running across the left atrial appendage (Fig. 4). A saline-irrigated ablation catheter was used to deliver an application of radiofrequency energy at a site in the posterior wall of the left atrial appendage where the activation time was  $-80$  msec, and this terminated the tachycardia (Fig. 3). The patient has remained free of symptomatic arrhythmias during 18-month follow-up.



**Figure 4.** Anteroposterior view of left atrial angiogram during AV block (large arrow) induced by adenosine triphosphate. There is a venous connection (small arrows) between the left superior pulmonary vein (LSPV) and the superior vena cava (SVC). Note that this venous connection courses over the ablation site in the left atrial appendage (LAA). LIPV = left inferior pulmonary vein; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein.

### Discussion

This case demonstrates that a venous connection between two thoracic veins may be a potential source of atrial tachycardia. Although earliest endocardial activation was in the left atrial appendage, the fact that relatively early endocardial activation was also present at the left superior pulmonary vein and superior vena cava makes it unlikely that the left atrial appendage was the site of origin of the tachycardia. Furthermore, endocardial activation at the superior left atrium occurred simultaneous with the superior vena cava, suggesting that the superior vena cava was not activated by the wavefront from the left atrium. These findings suggest the presence of an electrical connection between these anatomic sites. Origin of the tachycardia at the posterior aspect of the left atrial appendage, with transmission of wavefronts along the venous connection overlying the left atrial appendage to the left superior pulmonary vein and superior vena cava, is the most likely explanation for the findings in this case. A site of origin in the venous connection overlying the left atrial appendage also would explain why successful ablation was possible only with the use of an irrigated-tip ablation catheter, because a transmural lesion would be necessary to ablate a venous structure on the epicardial aspect of the left atrium. Whether the origin of the tachycardia was in an epicardial portion of the left atrial appendage or actually within the venous connection between the left superior pulmonary vein and the superior vena cava remains unclear.

The venous connection that was potentially the site of origin of the atrial tachycardia was demonstrated angiographically when contrast was injected during a period of transient asystole induced by adenosine triphosphate. A venous connection between the left superior pulmonary vein and superior vena cava has not been previously reported. However, had adenosine triphosphate not been used to induce asystole, it is unlikely that the venous connection observed in this case would have been visualized. Although visualization of this venous connection was not critical for curing this patient, it provided important insight into the underlying mechanism of the tachycardia. The true prevalence of this type of connection remains to be determined.

In conclusion, this case demonstrates that atrial arrhythmias may arise not only from the major thoracic veins but also from an epicardial site or a venous connection between a pulmonary vein and the superior vena cava.

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