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## Which side are you on? – Deducing the chamber of origin of atrial tachycardia

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### ABSTRACT

Atrial arrhythmias rising from the regions of the atria that are in close proximity to each other may pose a challenge in identifying the chamber to map and ablate in. In this report, we discuss a patient with left atrial tachycardia which initially mimicked right atrial tachycardia. We discuss the origins of the abnormal electrograms in the right atrium and how this provides a general understanding of the mechanism of double potentials.

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### 1. Case

A 64 year old male presented with exertional breathlessness progressing in severity over six months. He had been diagnosed to have dilated cardiomyopathy with non sustained atrial tachycardia and ventricular tachycardia elsewhere and advised implantation of a defibrillator. Review of electrocardiograms showed recurrent runs of atrial tachycardia at a rate of 200 beats per minute for 6–8 beats with 1–2 sinus beats intervening (Fig. 1). Variable AV block was seen during the runs of tachycardia and there was intermittent aberrancy at faster rates leading to the mistaken diagnosis of ventricular tachycardia. P waves were upright in the inferior leads, biphasic in V1 with a small initial negative component, negative in lead aVL, but positive in lead I. Diagnosis of focal atrial tachycardia was made with left ventricular dysfunction most likely secondary to the rapid ventricular rates during tachycardia. Although ECG features suggested that tachycardia origin was likely in the left atrium, they were not unequivocal, so it was decided to map both atria.

Mapping was done with CARTO 3 (Biosense Webster) system in the right atrium initially. Centrifugal activation was seen from the

earliest site in the high posteromedial right atrium (Fig. 2). Local activation was 25 ms ahead of the P wave onset. Double potentials were seen at multiple points in this region and a long, fractionated signal was seen inferiorly. Ablation at the earliest site was unsuccessful. Subsequently, the left atrium was accessed by a transseptal puncture and mapped. Earliest activation was seen from the anterior left atrium, just medial to the antrum of the right upper pulmonary vein, in close relation to the earliest activation noted in the right atrium (Fig. 2). Local activation was 30 ms ahead of the P wave and radiofrequency ablation resulted in immediate termination of the runs of tachycardia. At three months follow up, he remained free of tachycardia, showed normalization of the left ventricular ejection fraction and was asymptomatic. What is the explanation for the double potentials and fractionated signal in the right atrium?

### 2. Discussion

Abnormal electrograms including double potentials and fractionated electrograms indicate abnormal tissue and often are seen near the origin of focal atrial tachycardia [1]. But double potentials are only a marker of serial activation of two regions that are anatomically close but electrically separated. The posteromedial right atrium lies in proximity to the anterior left atrium and therefore activation in one of these chambers can produce a signal in a catheter in the other chamber. Although these are situated in close proximity, the atrial walls are

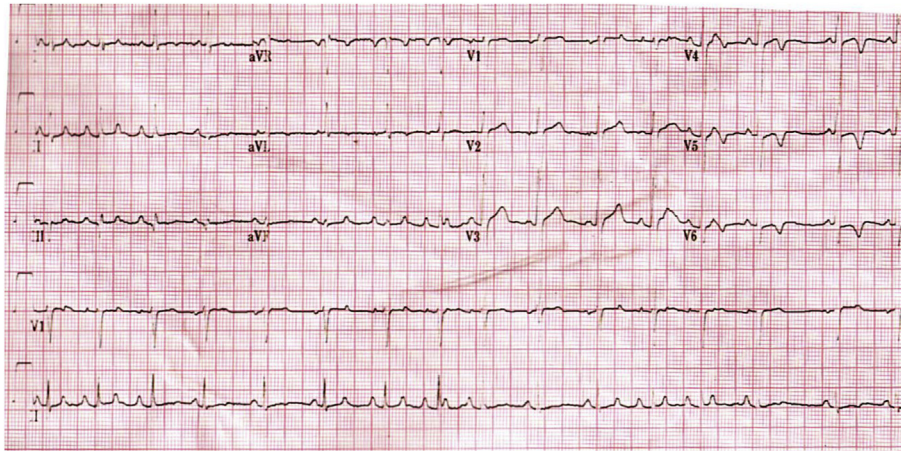
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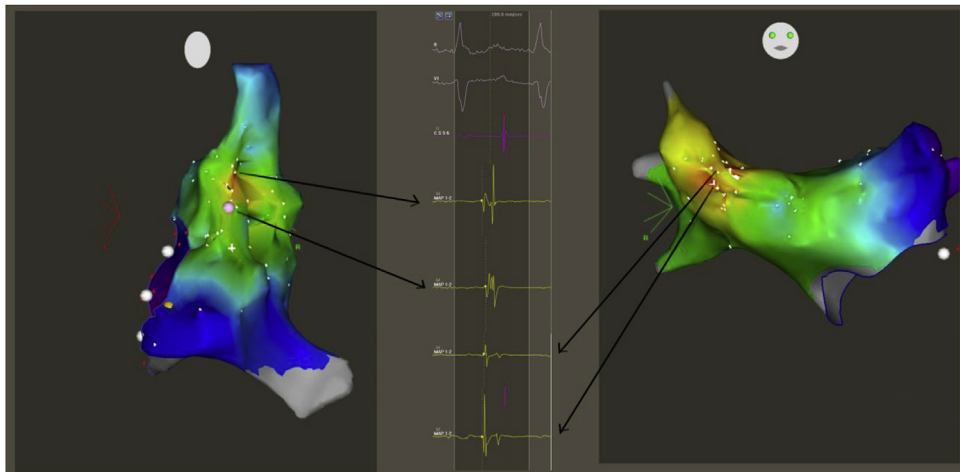
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**Fig. 1. Electrocardiogram at presentation.** Twelve lead electrocardiogram with rhythm strips of leads V1 and lead II. Runs of atrial tachycardia are seen, with few sinus beats in between.



**Fig. 2. Activation maps of right and left atria.** Panel on left shows a posterior view of the activation map of the right atrium with centrifugal activation spreading from the posteromedial aspect. Panel on right shows earliest activation in the corresponding anterior left atrium. Panel in the middle is a composite image showing the bipolar electrograms recorded from the distal bipole of the mapping catheter at two locations each in the right and left atria.

separated by connective tissue which electrically insulates them (Fig. 3A).

Fig. 3B is a schematic illustration of the mechanism of the double potentials seen in our case. Activation beginning in the anterior left atrium has to traverse a certain distance before it activates the posterior right atrium. This initial activation of the left atrium is recorded as a low frequency far-field potential in the right atrium with the local activation occurring after some interval as the second component because of the delay in the impulse reaching the local site. The local and far-field activation are also recorded as a double potential in the left atrium. We hypothesise that fractionated, long duration electrogram recorded inferiorly in the right atrium may indicate the region of electrical connection between the two atria, with activation of both atrial walls in a direction perpendicular to the orientation of the muscle fibers resulting in slow, anisotropic conduction.

Double potentials in the posteromedial right atrium in left atrial tachycardia has been described by Soejima et al. [2]. As described by the authors, reversal of the components during sinus

rhythm can help in elucidating the origin of the components. In our case, the potentials in both the right and left atria showed reversal of the components during sinus rhythm as compared to that during tachycardia (Fig. 4). This confirms that during tachycardia the double potentials reflect left followed by right atrial activation.

These same concepts can also be applied to other situations where signals from two adjacent structures need to be differentiated. These include, for example, superior vena cava and right upper pulmonary vein, left atrial appendage and left upper pulmonary vein or the coronary sinus and left atrium. Common principles to identify the electrograms originating from different structures in all these situations include frequency characteristics to differentiate far-field from near-field and change in timing of signals with altered activation or by pacing in proximity to one structure. Most important is an understanding of anatomy and recognition of situations where such potential confusion may occur.

Understanding the mechanism of the abnormal potentials and

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