EP CASE REPORT

# Recognition of short RP atrial tachycardia due to intra-atrial conduction delay: utility of a septal AH/HA ratio <1

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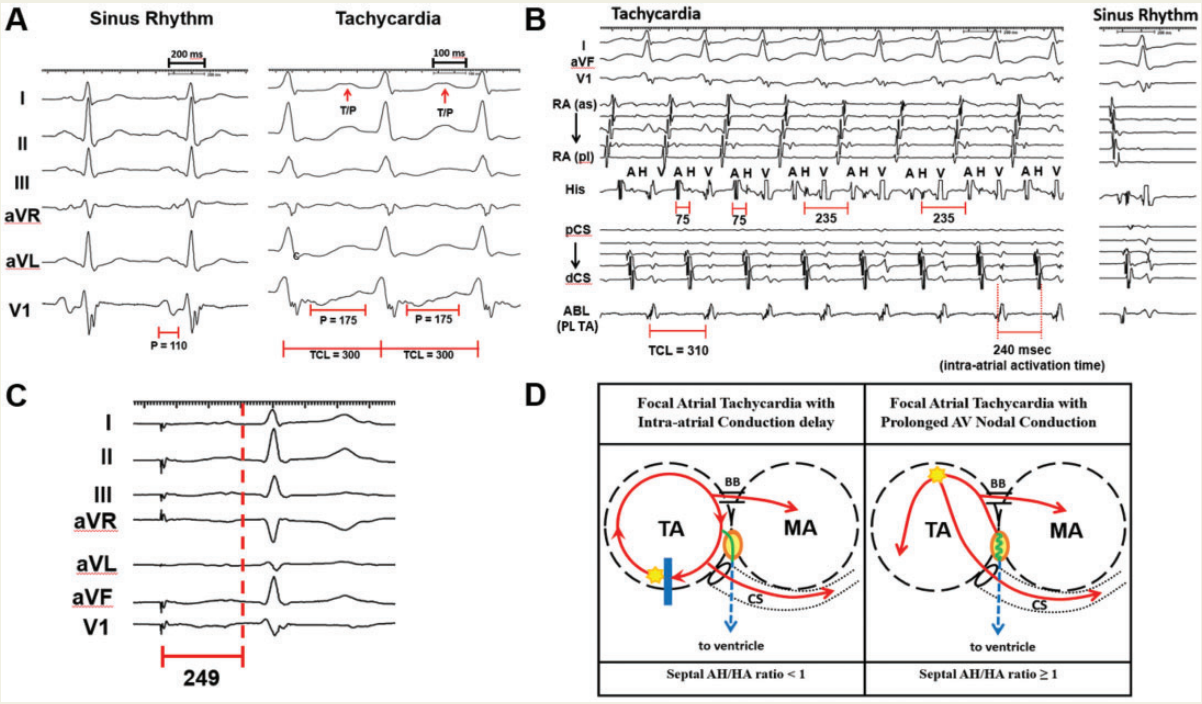
## Introduction

Short RP tachycardia is caused by typical atrioventricular nodal reentrant tachycardia, atrioventricular reentrant tachycardia or atrial tachycardia (AT) with first degree AV block. In the last mechanism, delayed AV nodal conduction during tachycardia causes the P wave to be cast close to the preceding R wave. However, first degree AV block can also be caused by prolonged intra-atrial conduction.

## Case

A 59-year-old man underwent ablation for cavotricuspid (CTI)-dependent right atrial flutter. He was not taking any anti-arrhythmic medications. Post-ablation programmed stimulation reproducibly initiated supraventricular tachycardia with a cycle length 300–310 ms. The rhythm strip showed short RP tachycardia with a prolonged P wave duration (175 ms), which was much longer compared with during sinus rhythm (110 ms) (*Panel A*) and comprised ~60% of the tachycardia cycle length.

The tachycardia was determined to be atrial tachycardia (AT) with first-degree atrioventricular (AV) block. Typically, this mechanism involves decremental AV nodal conduction during tachycardia that can be compounded upon pre-existing AV nodal disease. However, in our case, first-degree AV block was caused by prolonged intra-atrial conduction as a result of the focal AT arising lateral to the line of conduction block from CTI ablation. Therefore, the shortest potential conduction path of AT to the left atrium, through the CS, was interrupted by CTI conduction block. The intra-atrial activation time from the earliest activation of the focal AT to the latest activation along the distal CS was 240 ms. Had the AT originated medial to the ablation line or from a disparate site such as the left atrium, evidence for intra-atrial delay would not have been apparent during tachycardia and the arrhythmia would have had a long RP pattern. Such prolonged conduction is often manifested by a prolonged P wave duration and may have an iatrogenic aetiology, resulting from focal AT originating lateral to a previous line of conduction block within the atrium, such as from CTI or posterior mitral isthmus ablation. It is likely that a similar phenomenon could result from other conditions that cause intra-atrial conduction delay, such as surgical lesion lines or atrial myopathies.



The AH interval during AT was 75 ms and the HA interval was 235 ms, resulting in a short septal AH/HA ratio (<1) (*Panel B*). Diagnosis of this entity is differentiated from short RP AT due to pre-existing first-degree AV nodal block based on analysis of the septal AH and HA intervals. Findings also consistent with prolonged intra-atrial conduction was a P wave duration of 249 ms when pacing lateral to the CTI during sinus rhythm (*Panel C*). In the case of intra-atrial delay, the septal AH/HA ratio should be <1 since intra-atrial delay will be reflected by a relatively long HA interval, whereas in AT with prolonged AV nodal conduction, the AH/HA ratio will be ≥1 since delay is confined primarily to the AV node (*Panel D*).

This case highlights the often under-appreciated contribution of prolonged intra-atrial conduction, manifested by a prolonged P wave duration, to the spectrum short RP tachycardia. This particular form of AT manifests when its site of origin is adjacent to an area of intra-atrial conduction block. A septal AH/HA ratio <1 might be helpful for discriminating between focal AT with intra-atrial conduction delay and focal AT with prolonged AV nodal conduction.

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