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## Orthodromic atrioventricular reentrant tachycardia using a concealed isoproterenol-sensitive accessory pathway

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### ABSTRACT

Accessory pathways (APs) represent the substrate for atrioventricular reentrant tachycardia. Catecholamine-sensitivity is an uncommon feature of APs and has been almost exclusively been described in APs with antegrade conduction. We present the rare case of a catecholamine-dependent concealed AP that was only unmasked upon isoproterenol stimulation and successfully ablated. This case highlights the importance of systematic isoproterenol stimulation in patients referred for ablation of supraventricular tachycardia - in particular if the baseline electrophysiology study is negative. Otherwise, ablation targets may be missed.

**Learning objective:** The absence of retrograde ventriculo-atrial conduction does not automatically exclude the presence of a concealed accessory pathway. Systematic isoproterenol stimulation should be part of any electrophysiology study for supraventricular tachycardia, to search for catecholamine-sensitive accessory pathways that may be otherwise missed.

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### 1. Introduction

Accessory pathways (APs) represent the substrate for atrioventricular reentrant tachycardia (AVRT) which is the second most common form of paroxysmal supraventricular tachycardia. While most APs present bidirectional (antegrade and retrograde) conduction properties, approximately 30–40% of orthodromic AVRTs are related to concealed APs exhibiting a unidirectional, typically exclusive retrograde, conduction pattern [1,2]. Most APs preserve a stable, non-decremental conduction pattern that is not influenced by catecholamines or alterations of the autonomic nervous system [1,3]. Catecholamine-sensitivity is an uncommon feature of APs and has been almost exclusively been described in APs with antegrade conduction [4–6]. We present the rare case of a catecholamine-dependent concealed AP that was only unmasked upon isoproterenol stimulation.

### 2. Case report

A 61-year-old female presented with recurrent paroxysmal

supraventricular tachycardia. A narrow QRS complex tachycardia with ventricular response at 180–185 beats per minute was repeatedly documented (Fig. 1A) and sensitive to Adenosine. Her resting ECG was unremarkable and did not show signs of ventricular pre-excitation (Fig. 1B). A transthoracic echocardiogram was normal. The patient was referred for an electrophysiology study and catheter ablation.

### 3. Procedure

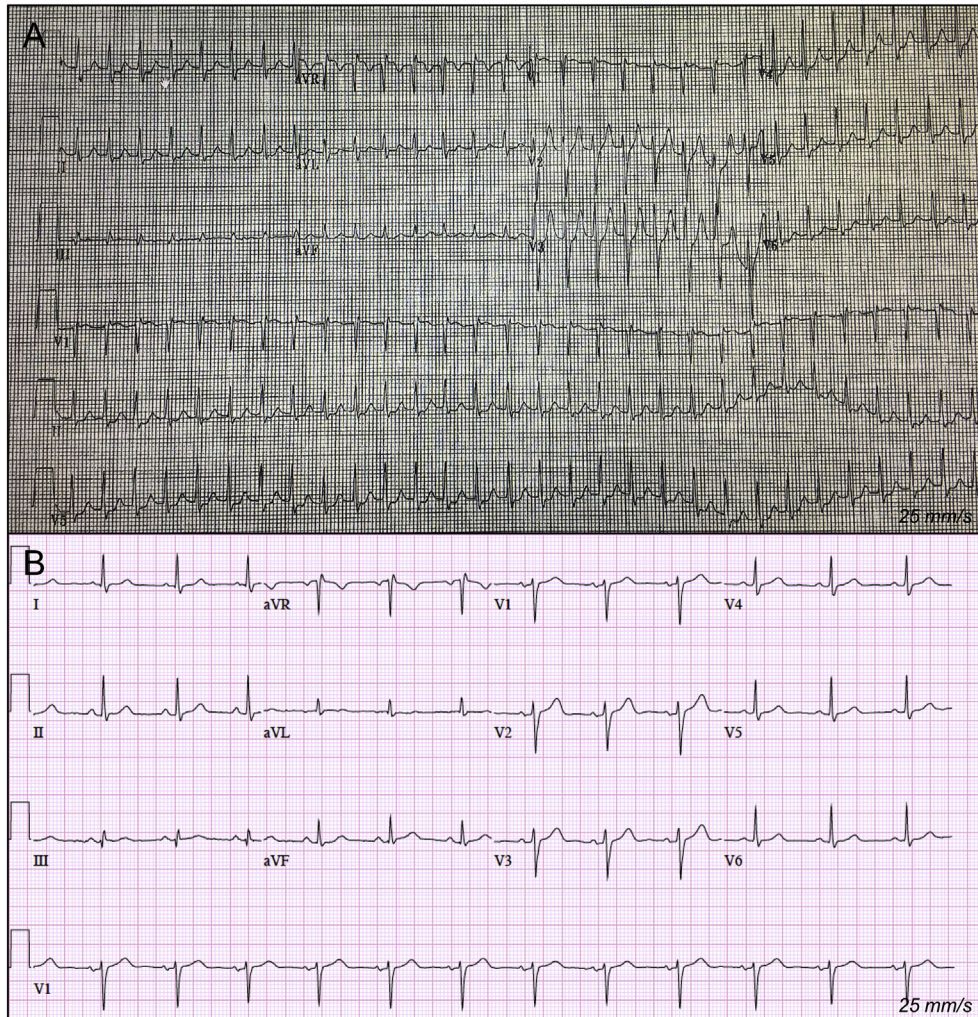
The electrophysiology study demonstrated normal AH (64 ms) and HV (36 ms) intervals and absence of spontaneous preexcitation. At baseline, there was no ventriculo-atrial (VA) conduction during decremental stimulation at various cycle lengths (CLs) (400, 500, 600 and 700 ms) (Fig. 2A). There was no evidence of dual AV node physiology (right atrial refractory period at 500/210 ms) and aggressive atrial burst pacing did not induce any tachycardia.

An intravenous perfusion of isoproterenol was started at a rate of 1 µg/min and titrated up to 3 µg/min with marked acceleration of the underlying sinus rhythm. Repeat testing demonstrated 1:1 VA conduction (Fig. 2B). Retrograde atrial activation was initially concentric. At a CL of 300 ms, the atrial activation pattern became somewhat fused with non-decremental 1:1 VA conduction (fixed VA interval of 190 ms) and late atrial deflection on the His catheter (Fig. 3A). At a CL of 280 ms, an eccentric atrial activation pattern with earliest atrial activation at CS1-2 was observed thus

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**Fig. 1.** Baseline ECG and ECG during SVT.

(A) Shown is a typical example of the patient's recurrent SVT: a narrow QRS complex tachycardia at a rate of 185 beats per minute. There is evidence of retrograde P-waves (arrow) with an RP interval of approximately 80 ms. The patient's SVT was terminated by adenosine. (B) Resting ECG shows normal sinus rhythm with normal PR, QRS and QTc intervals. There was no evidence of spontaneous preexcitation.

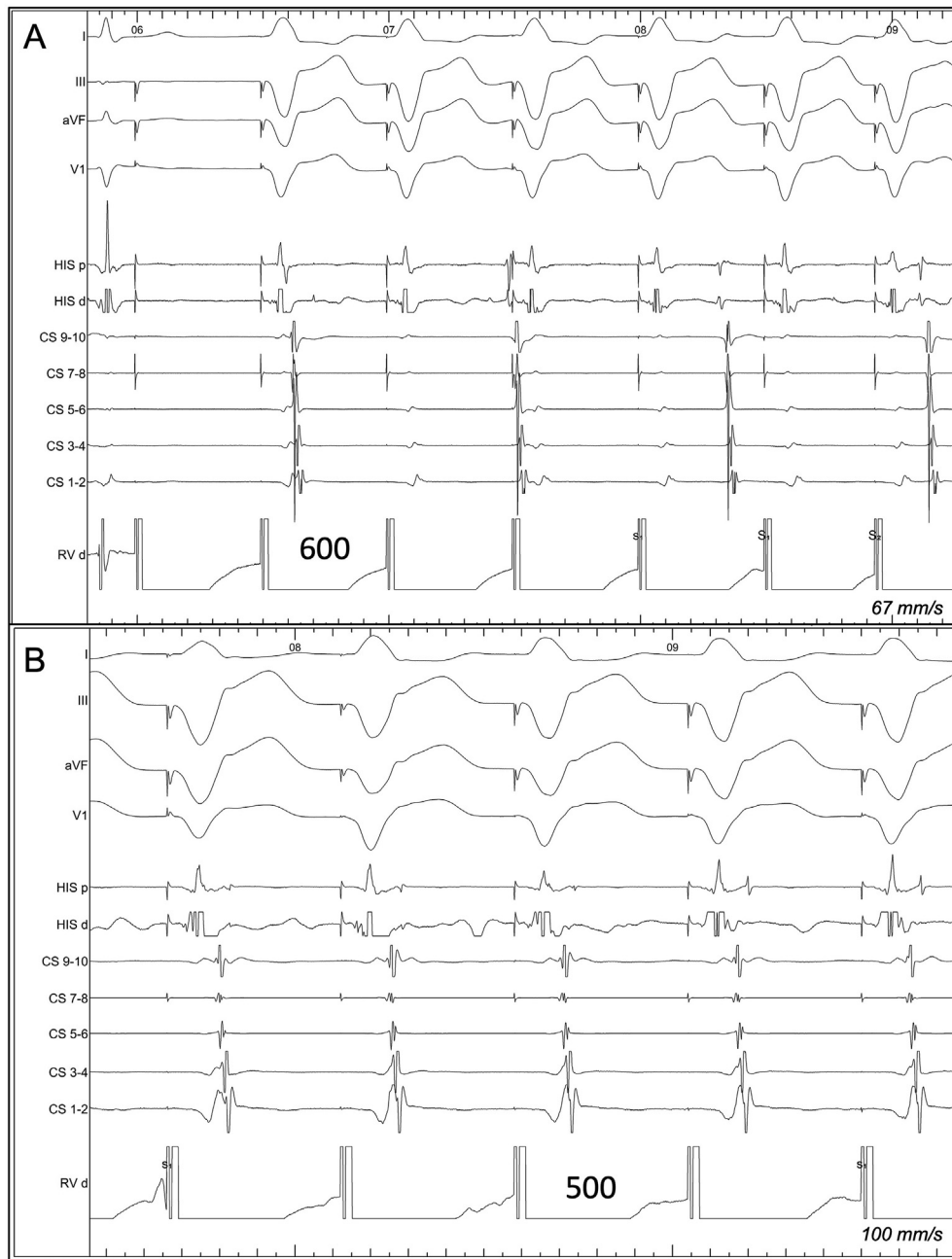
unmasking the presence of a left-sided concealed AP (Fig. 3B). The functional refractory period of the AP was at CL 240 ms (Supplementary Fig. 1). No antegrade AP conduction could be unmasked with isoproterenol and there was no evidence of ante- or retrograde dual AV node physiology. A stable supraventricular tachycardia at a CL of 290 ms was repeatedly inducible by atrial or ventricular stimulation (Supplementary Fig. 2A and B). The shortest VA interval was 84 ms with earliest atrial activation at CS1-2 and VA timing of 105 ms at the His region. Ventricular entrainment demonstrated a V-A-H-V response (Supplementary Fig. 3A) and atrial entrainment demonstrated a  $\Delta$ AH of only 15 ms. The final diagnosis was an orthodromic AVRT using an isoproterenol-sensitive concealed left AP as retrograde limb. The concealed AP was successfully ablated at 04.00 o'clock on the mitral annulus via a transseptal approach. There was no recurrence of AP conduction after a 30-min waiting period and subsequent challenge with adenosine and isoproterenol didn't induce any tachycardia.

#### 4. Discussion

The majority of so-called typical atrioventricular accessory pathways exhibit a fixed effective refractory period (ERP) and non-decremental conduction properties. However, a small proportion of

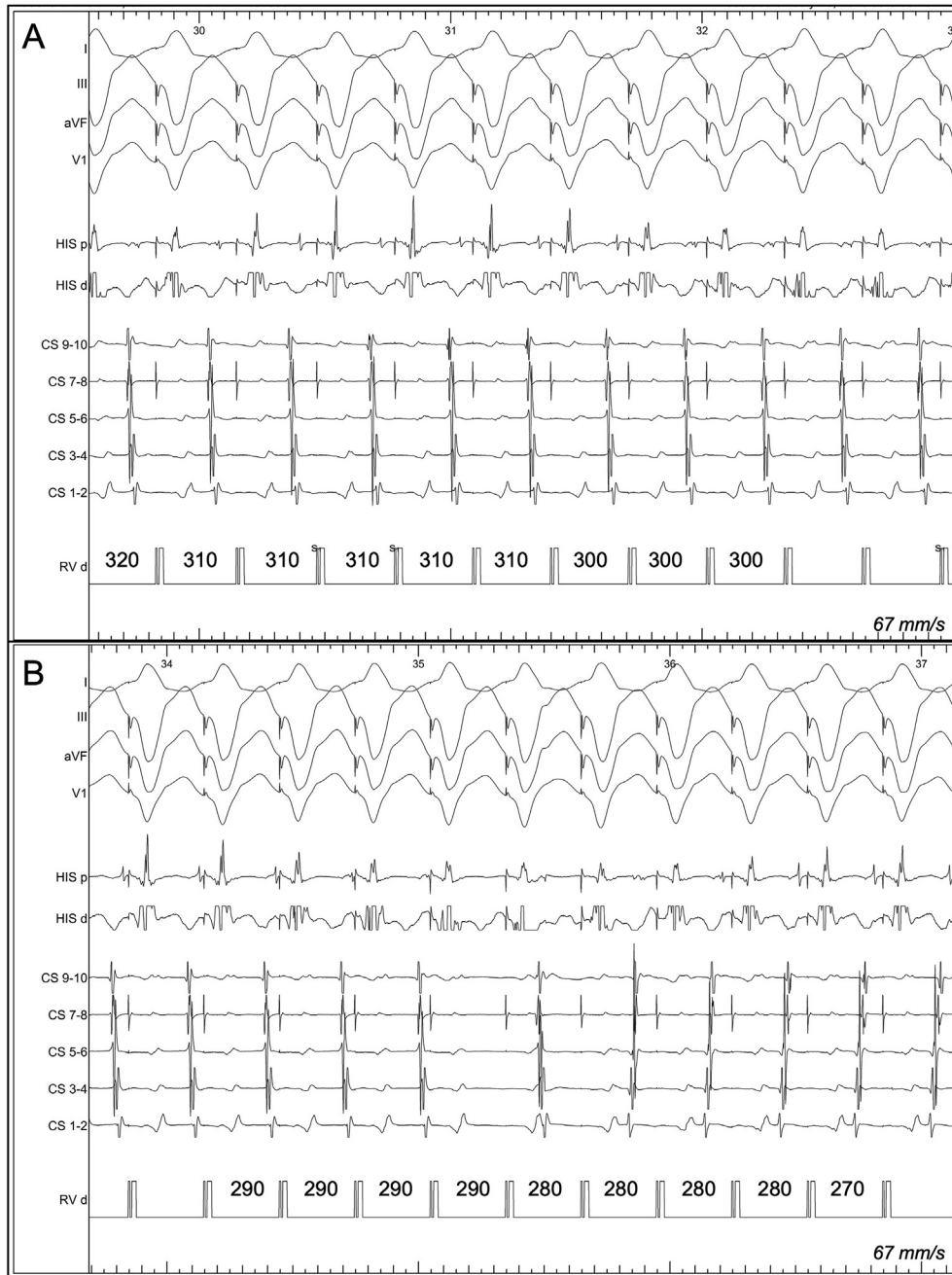
APs are characterized by atypical electrophysiological features including decremental antegrade/retrograde conduction or adenosine- or catecholamine-sensitivity [5–12].

Isoproterenol is an intravenous  $\beta_1/\beta_2$ -agonist and has been since many years the standard provocative agent during electrophysiology studies to facilitate intrinsic conduction properties and to induce supraventricular tachycardias [13,14]. The effect of isoproterenol on AP properties has been predominantly described in AP with antegrade conduction resulting in enhanced AP conduction and shortening of the antegrade and retrograde ERP [5,6]. Catecholamine-sensitive concealed APs has been described in some case reports and small series [4,9–12]. We report another case of a patient with a true concealed AP that was only apparent upon isoproterenol stimulation. Our patient had no spontaneous ventriculo-atrial conduction at baseline despite minimal sedation and extensive atrial and ventricular stimulation at various cycle lengths. Isoproterenol stimulation unmasked the presence of a concealed AP and facilitated the initiation of orthodromic AVRT using the AP as retrograde limb. The absence of antegrade pre-excitation during isoproterenol stimulation also confirmed the true concealed nature this AP. Potential mechanisms by which isoproterenol reversed the functional conduction block of the concealed AP may include direct enhancement of AP conduction and/or



**Fig. 2. Isoproterenol-dependent VA conduction.**

(A) At baseline decremental ventricular pacing at various cycle lengths showed absence of VA conduction. (B) Demonstration of constant 1:1 VA conduction under isoproterenol perfusion, retrograde atrial activation is initially concentric.



**Fig. 3. Unmasking of isoproterenol-dependent concealed accessory pathway.**

(A) Absence of decremental VA conduction starting from CL 300 ms (fixed VA interval of 190 ms). The retrograde atrial activation pattern appears somewhat fused and is not entirely concentric. (B) At CL 280 ms the retrograde atrial activation shows an eccentric pattern with earliest activation in CS1-2 (non decremental) demonstrating the presence of a concealed left accessory pathway.

shortening of the AP's ERP. Previous reports have suggested that improved conduction velocity seems to be the most likely effect of isoproterenol [5,12]. We cannot completely exclude the presence of a very slow retrograde AP conduction prior to isoproterenol stimulation, however there was no ventriculo-atrial conduction at ventricular stimulation with cycle lengths as slow as 700–800 ms.

The true incidence of isoproterenol-sensitive APs is unknown and this phenomenon is most likely underreported. Our observations also highlight the importance of routine isoproterenol stimulation in individuals referred for supraventricular tachycardia (SVT) ablation and negative findings during the baseline study. Treatable SVT causes and potential targets for ablation may be otherwise missed.

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### Declaration of competing interest

The authors have no conflict of interest to declare.

### Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ipej.2019.12.009>.

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