# A case of atrial fibrillation with preexcitation and duodromic tachycardia

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## Introduction

Orthodromic atrioventricular reentrant tachycardia (AVRT), where the atrioventricular (AV) node serves as the anterograde limb and the accessory pathway serves as the retrograde limb, is the most common type of arrhythmia in patients with Wolff-Parkinson-White (WPW) syndrome. Antidromic AVRT, where the accessory pathway serves as the anterograde limb and the AV node serves as the retrograde limb, is far less common—having been clinically documented in fewer than 5% of patients with WPW syndrome and found to be inducible in fewer than 10% of patients with WPW undergoing electrophysiology study.[1] Duodromic tachycardia (defined as 2 distinct accessory pathways, with 1 serving as an anterograde and the other as a retrograde limb) is an even rarer phenomenon. We present a case of a patient with WPW and numerous inducible supraventricular tachycardias (SVT), including duodromic tachycardia.

## Case report

A 32-year-old male patient presented to the emergency room with atrial fibrillation (AF) and evidence of preexcitation on 12-lead electrocardiogram (ECG) (Figure 1A). Initial efforts to restore normal sinus rhythm through multiple cardioversion attempts proved unsuccessful. Consequently, an intravenous infusion of procainamide was administered, resulting in the successful restoration of sinus rhythm (Figure 1B). The ECG in sinus rhythm revealed persistent preexcitation.

Initially, the observed preexcitation pattern was thought to align with a right-sided free wall accessory pathway with a precordial transition at lead $V_3$.[2] The patient was subsequently brought to the electrophysiology lab in a fasting state. A standard 4-catheter study was performed with catheters placed in the high right atrium, coronary sinus (CS), His bundle region, and right ventricular apex. The CARTO® electroanatomic mapping system (Biosense Webster, Diamond Bar, CA) was used. Pacing from the right atrium exhibited rapid 1:1 preexcitation consistent with a right-sided pathway and a pathway block cycle length of 230 ms. Pacing from the right ventricle resulted in concentric atrial activation in most instances. However, in some instances, eccentric atrial activation was observed where the earliest atrial electrogram was observed. The latter finding raised suspicion for the presence of a left lateral accessory pathway (LLAP).

Four distinct SVTs were induced over the course of the electrophysiology study (Figure 2), including:

(1) A narrow complex tachycardia most consistent with orthodromic reciprocating tachycardia (ORT), that used the His-Purkinje system/AV node as the anterograde limb and right-sided accessory pathway as the retrograde limb (Figure 3A). The 12-lead ECG from this arrhythmia is depicted in Figure 3B, demonstrating a narrow complex tachycardia with an rS complex in aVL. Pacing from the CS during this arrhythmia resulted in transient CS dissociation from the tachycardia (Figure 3C).
(2) A narrow complex tachycardia most consistent with ORT, that used the His-Purkinje system/AV node as the anterograde limb and LLAP as the retrograde limb (Figure 3D).
(3) A wide complex tachycardia most consistent with duodromic tachycardia, that used the right-sided accessory pathway as the anterograde limb and the LLAP pathway as the retrograde limb (Figure 3E).
(4) A wide complex tachycardia most consistent with antidromic AVRT that used the right-sided accessory pathway as the anterograde limb and the His-Purkinje system/AV node as the retrograde limb (Figure 3F).

We use the term "most consistent with" in describing the above arrhythmias because diagnostic pacing maneuvers were not performed. For example, what we presume to be arrhythmia "4" above could theoretically have been atrioventricular nodal reentrant tachycardia with bystander right lateral accessory pathway (RLAP) activation. A late (His-refractory) premature atrial contraction could have proven the diagnosis of antidromic AVRT.

**KEYWORDS** Wolff-Parkinson-White; Atrioventricular reentrant tachycardia; Supraventricular tachycardia; Preexcitation; Electrophysiology study; Catheter ablation

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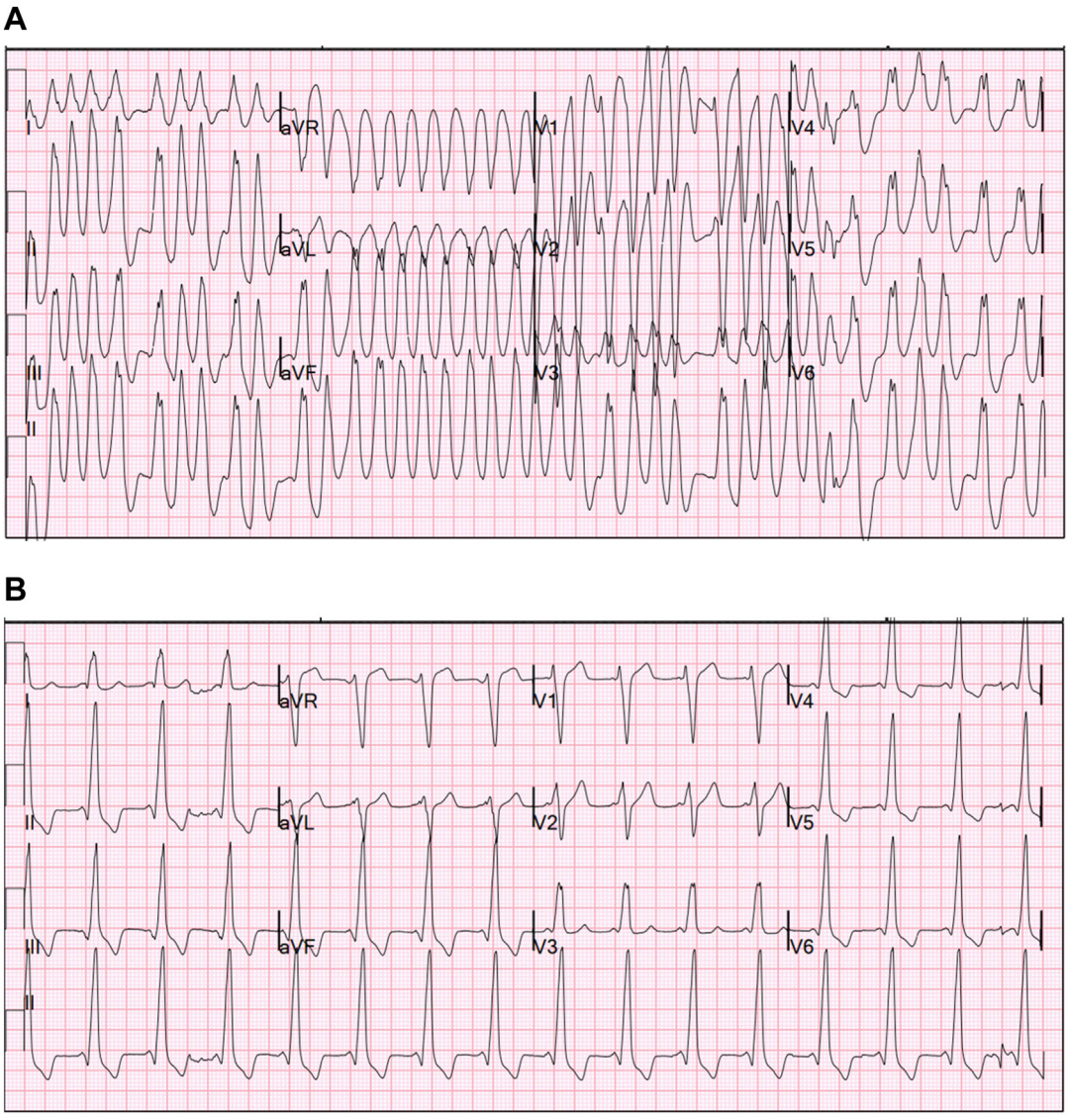


**Figure 1** **A:** Presenting electrocardiogram: atrial fibrillation with pre-excitation. **B:** Normal sinus rhythm with manifest preexcitation following administration of procainamide.

Ablation was initially performed on the LLAP during ventricular pacing. Subsequently, the right-sided AP was successfully ablated at approximately 11 o'clock on the tricuspid annulus. The postprocedural 12-lead ECG showed no evidence of preexcitation, and no SVT could be induced following ablation of the right-sided pathway.

## Discussion

Multiple accessory pathways in WPW syndrome is defined as the presence of 2 or more pathways separated by at least 1–3 cm.[3] The incidence ranges from 3% to 20% in surgical studies and from 5% to 18% in catheter radiofrequency ablation procedures.[4] Although the concept of duodromic tachycardia is widely accepted, it is a rare finding and its incidence is unknown. Vali and colleagues[5] recently reported a case of duodromic tachycardia with the anterograde limb utilizing an atriofascicular pathway. Our case differs in that the observed RLAP was conventional and capable of retrograde conduction. As a result, we were able to observe 4 distinct tachycardias, including ORT, utilizing the RLAP.

Localization of pathway site by 12-lead ECG is complicated in the presence of multiple accessory pathways. In this case, the accessory pathway was initially presumed to be right free wall accessory pathway in accordance with the observed precordial transition at $V_3$. However, close examination of both initial ECGs (Figure 1A and 1B) demonstrated a QS pattern in aVL, suggesting some degree of left-to-right activation. This raises the possibility that the left-sided pathway may have been capable of anterograde conduction. Lead malposition is unlikely to explain this finding, as lead I displayed a dominant R wave on presenting ECGs. For similar reasons, it is unlikely that these findings can be explained on the basis of left posterior fascicular block.

We did, however, observe changes in lead aVL that would support the hypothesis that the LLAP was capable of

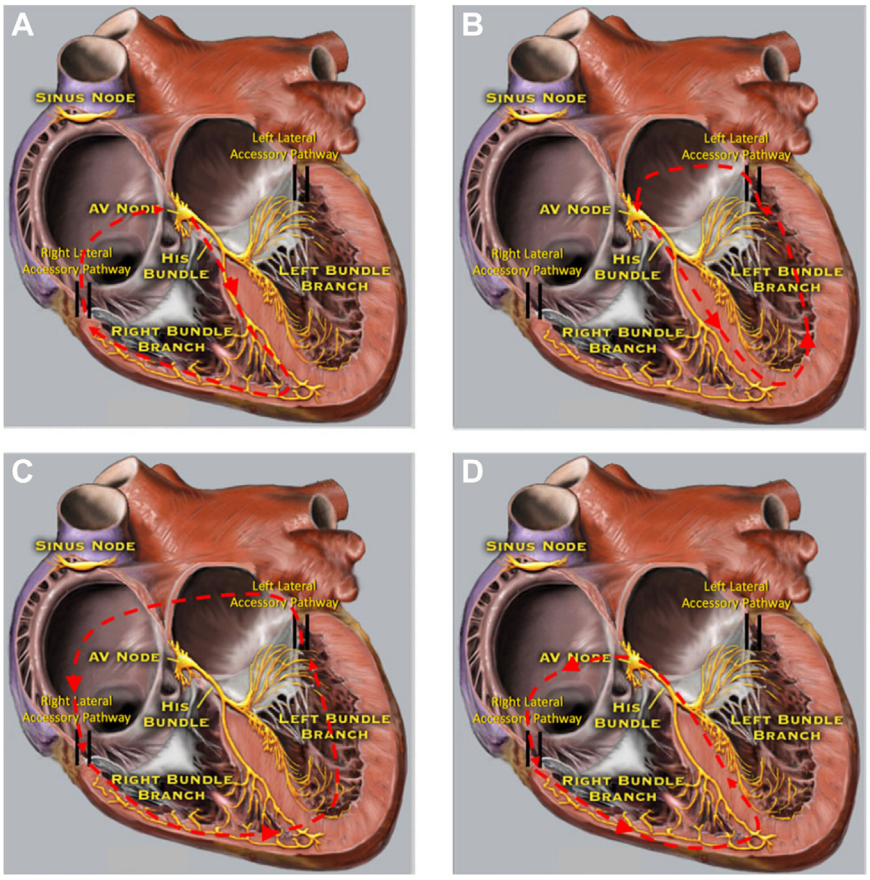


**Figure 2** **A:** Orthodromic reciprocating tachycardia (ORT), retrograde conduction via right lateral accessory pathway. **B:** ORT, retrograde conduction via left lateral accessory pathway. **C:** Duodromic tachycardia, anterograde conduction via right lateral accessory pathway, retrograde conduction via left lateral accessory pathway. **D:** Antidromic atrioventricular reentrant tachycardia, anterograde conduction via right lateral accessory pathway, retrograde conduction via His-Purkinje system/atrioventricular node.

anterograde conduction. During antidromic AVRT utilizing RLAP, lead aVL had a dominant R wave, making it unlikely that there was simultaneous bystander activation down the LLAP. In contrast, during what we presumed to be ORT utilizing the RLAP as the retrograde limb, aVL displays an rS complex (Figure 3B). When this is compared to the postablation ECG, aVL was found to have an Rs complex. This would suggest that the tachycardia depicted in Figure 3A and 3B could have been associated with some degree of left-to-right activation. One possibility for this observation would be ORT with retrograde activation via the RLAP and bystander anterograde activation down the LLAP.

There is another intriguing possibility regarding the tachycardia depicted in Figure 3A and 3B. There appears to be a small potential just preceding the local ventricular electrogram on the His channels. This potential occurs after the onset of the QRS. If it is indeed a His or right bundle potential, it raises the possibility of simultaneous antidromic activation of the LLAP and RLAP with retrograde activation to the atrium via that HIS and AV node. Depending on the timing of wavefronts down both accessory pathways, this could yield a narrow QRS. Unfortunately, it was difficult to ascertain whether this potential was in fact a His or right bundle electrogram. A clear-cut retrograde His would have certainly supported this diagnosis but was not observed. In addition, we would have expected a QS complex in aVL in the case of simultaneous antidromic pathway activation. In this case an rS complex was observed. In fact, a QS complex in aVL was never observed during the case and was only seen on the patient's presenting ECGs.

One explanation for these observation is that brisk AV nodal and RLAP conduction might have masked anterograde LLAP conduction. It is possible that, had we ablated the right lateral pathway first, LLAP conduction may have become manifest. Another strategy that might have been considered at the beginning of the case is the administration of adenosine. This may have helped to unmask any anterograde conduction across the LLAP through suppression of AV nodal conduction.

During ORT utilizing the RLAP we also observed transient dissociation of the CS with pacing of the proximal CS electrode (Figure 3C). This would suggest that the proximal CS is not an obligate portion of the circuit. This is consistent with earlier observations by Morady and colleagues[6] where they demonstrated that various regions of the atria or CS could be dissociated from ORT depending on the location of the pathway. Figure 3C also underscores the importance of careful examination of electrograms. CS pacing ultimately

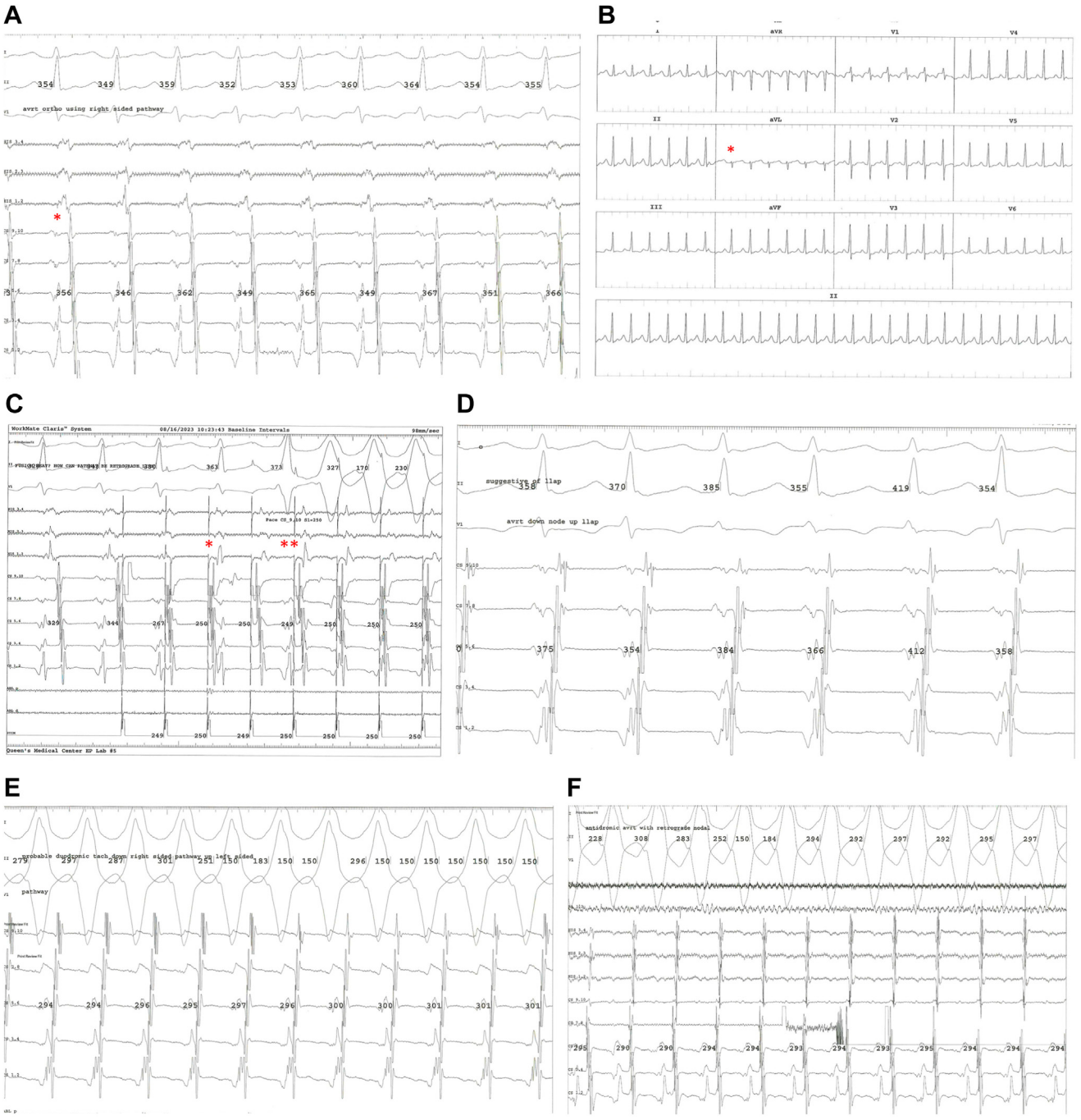


**Figure 3** **A:** Orthodromic atrioventricular reentrant tachycardia (AVRT), retrograde conduction via right-sided accessory pathway: note the potential (*asterisk*) on the His electrograms that occurs after the onset of the QRS. **B:** Twelve-lead electrocardiogram corresponding to panel A; note the rS complex in aVL (*asterisk*). **C:** Coronary sinus (CS) pacing results in transient CS dissociation from the orthodromic reciprocating tachycardia utilizing right lateral accessory pathway (RLAP). Following this the tachycardia terminates owing to premature engagement of the RLAP (*asterisk*). The following beat (*double asterisk*) is a fusion beat between conduction via the RLAP and the His-Purkinje system. The subsequent beats appear to be fully preexcited. **D:** Orthodromic AVRT, retrograde conduction via left lateral accessory pathway. **E:** Duodromic tachycardia, anterograde conduction via right-sided accessory pathway and retrograde conduction via left-sided accessory pathway. **F:** Antidromic AVRT, retrograde conduction via His-Purkinje/atrioventricular node

terminates the tachycardia by prematurely engaging the RLAP. The subsequent beat following termination appears to be fusion between anterograde conduction down the RLAP and the His-Purkinje system. In contrast, the subsequent pace beats appear to be fully preexcited. This observation could be explained by the phenomena of repetitive concealed conduction via retrograde invasion of the His-Purkinje system.

Finally, our case also draws attention to risk factors associated with sudden cardiac death in patients with WPW.

Increased risk of sudden cardiac death secondary to rapid conduction and degradation into ventricular fibrillation is associated with male sex, history of symptomatic tachycardia, multiple accessory pathways, and a short preexcited R-R interval of <250 ms during AF—all characteristics that our patient demonstrated.[7] Recognition of these risk factors is important in identifying high-risk patients for whom catheter ablation should be promptly pursued.

## Conclusion

We present a rare case of duodromic tachycardia in a patient presenting with preexcited AF. The patient was found to have multiple SVTs involving both accessory pathways, including duodromic tachycardia with the right accessory pathway as the anterograde limb and left lateral accessory pathway as the retrograde limb. To our knowledge, this has not been widely reported in the literature and its prevalence is unknown. One important observation was the presence of a QS in lead aVL on the presenting ECG. Although other leads suggested the presence of a right-sided pathway, the presence of the QS in aVL may have been a clue alerting us to the presence of an additional left-sided pathway. In this case, successful radiofrequency catheter ablation of both pathways was achieved and no other SVTs were inducible following ablation.

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