

CASES AND TRACES

Unusual Rhythm Behind a Narrow Complex Tachycardia

ECG CHALLENGE

A 18-year-old man presented to the emergency department with palpitations and fatigue. Twelve months earlier he had been submitted to an electrophysiological study and radiofrequency ablation for Wolff-Parkinson-White syndrome with recurrent narrow complex (orthodromic) paroxysmal supraventricular tachycardia. At admission, he presented with tachycardia at 205 beats/min and blood pressure was 105/71 mm Hg; there were no other relevant findings. The 12-lead ECG showed a regular narrow-complex tachycardia of 205 beats/min. Transthoracic echocardiography excluded structural heart disease. The tachycardia was transiently interrupted with intravenous adenosine and resumed after 8 seconds. An intravenous bolus of verapamil was administered that interrupted the tachycardia. Figure 1 shows the ECG after conversion.

What abnormalities are present? How should we analyze the ECG combined with his medical history? What is the best management of this situation?

Please turn the page to read the diagnosis.

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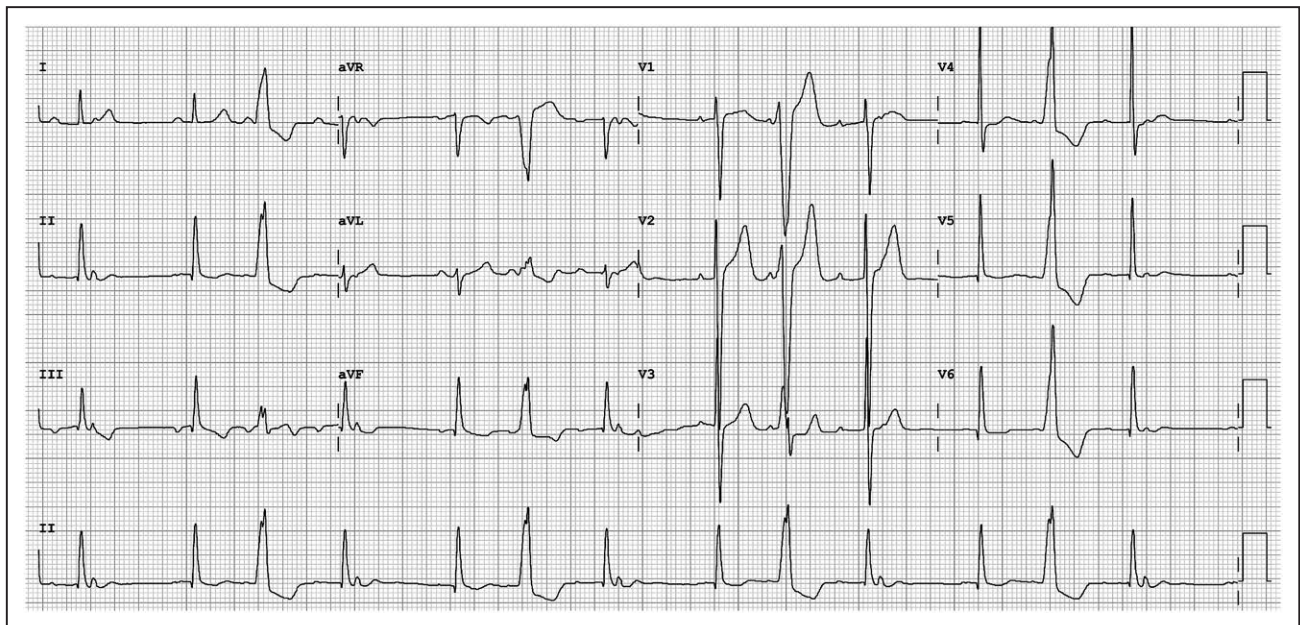


Figure 1. Twelve-lead ECG after conversion of narrow-complex tachycardia.

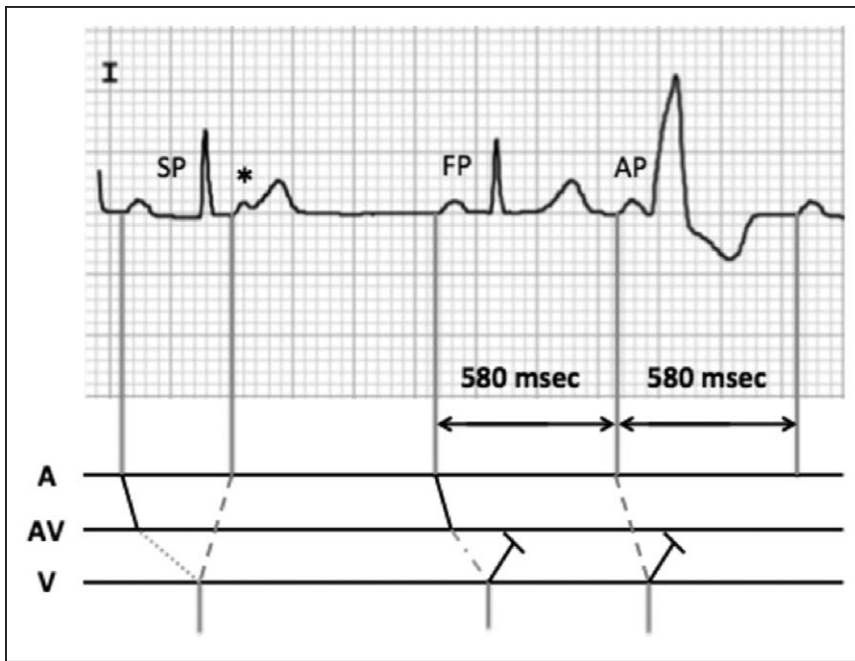


Figure 2. Dual atrioventricular nodal physiology and accessory pathway.

Regular sinus rhythm with an interval of 580 ms. Three different PR intervals are observed representing antegrade conduction over the SP (dotted line, 240 ms), the FP (dot-dashed line, 170 ms), and the AP (dashed line, 100 ms). A narrow P wave (*) seen 120 ms after nodal ventricular activation suggests retrograde conduction by the AP. The broad QRS complex corresponds to a preexcited ventricular beat conducted by the anteroseptal AP. A indicates atrium; AP, accessory pathway; AV, atrioventricular node; FP, fast pathway; SP, slow pathway; and V, ventricle.

RESPONSE TO ECG CHALLENGE

The ECG in Figure 1 is remarkable for showing sinus rhythm with 3 different PR intervals representing 3 distinct pathways of conduction to the ventricle. A spontaneous change in PR interval during regular sinus rhythm is noted; there are 3 different PR intervals (170, 100, and 240 ms). The shortest interval is related to a wide-complex QRS, suggesting antegrade conduction over the accessory pathway (AP). The delta wave is positive in inferior and anterolateral leads, with left bundle-branch block pattern and axis $>30^\circ$, suggesting

a right anteroseptal AP. The atrioventricular conduction is, therefore, performed alternately by the slow perinodal pathway (SP), the fast perinodal pathway, and an AP, forming a repeating pattern simulating ventricular trigeminy. An amplified view of the same ECG illustrating this mechanism is shown in Figure 2.

Review of 12-lead ECG during tachycardia (Figure 3) shows a narrow complex tachycardia of 205 beats/min, with an RP interval of 120 ms. It is noteworthy that the RP interval and morphology of the P wave during tachycardia are similar to the retrograde P wave observed after SP conduction during sinus rhythm, sug-

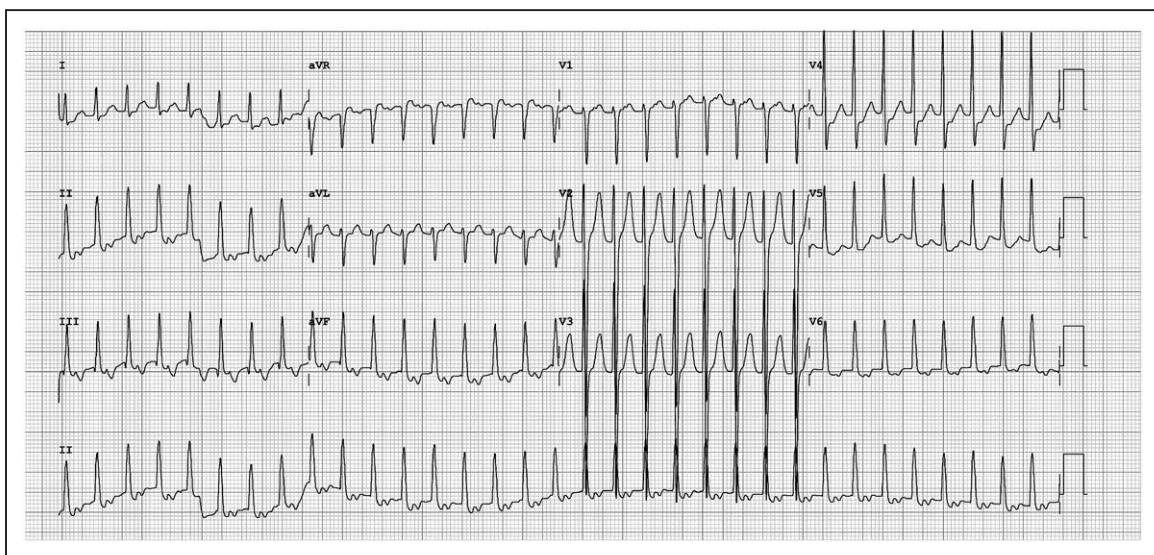


Figure 3. Twelve-lead ECG showed regular narrow complex tachycardia of 205 beats per minute with an RP interval of 120 ms, suggesting orthodromic AVRT.

AVRT indicates atrioventricular reentrant tachycardia.

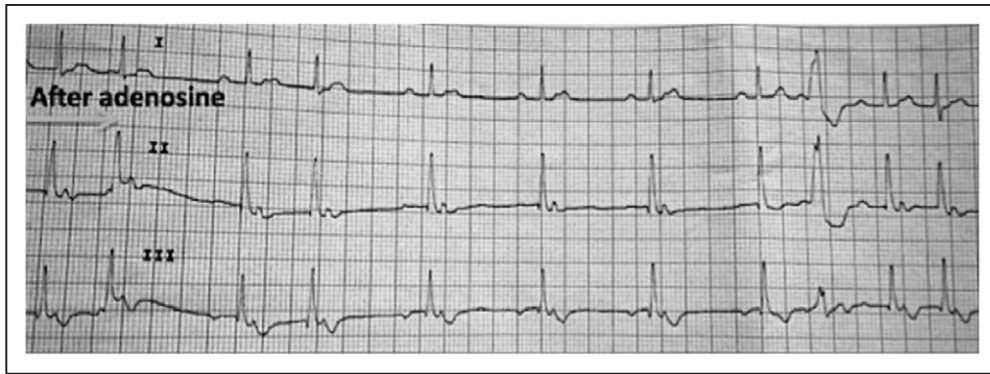


Figure 4. Rhythm strip immediately after adenosine bolus.

Interruption of the tachycardia is illustrated with resumption of sinus rhythm. Sinus P waves are followed by narrow QRS with a long RP (240 ms), suggesting slow pathway conduction. Immediately after the QRS complex, a retrograde P wave is consistently observed, representing retrograde conduction over the accessory pathway. There is an isolated broad QRS, with a delta wave preceded by a short PR (100 ms) demonstrating antegrade conduction over the accessory pathway; after this preexcited QRS, the tachycardia recurred.

gesting involvement of the SP as the antegrade limb of the tachycardia and the AP as the retrograde limb. In addition, retrograde P waves are narrow, positive in the

inferior leads and negative in V1, suggesting antero-septal breakout of atrial activation compatible with a bidirectionally conducting AP.

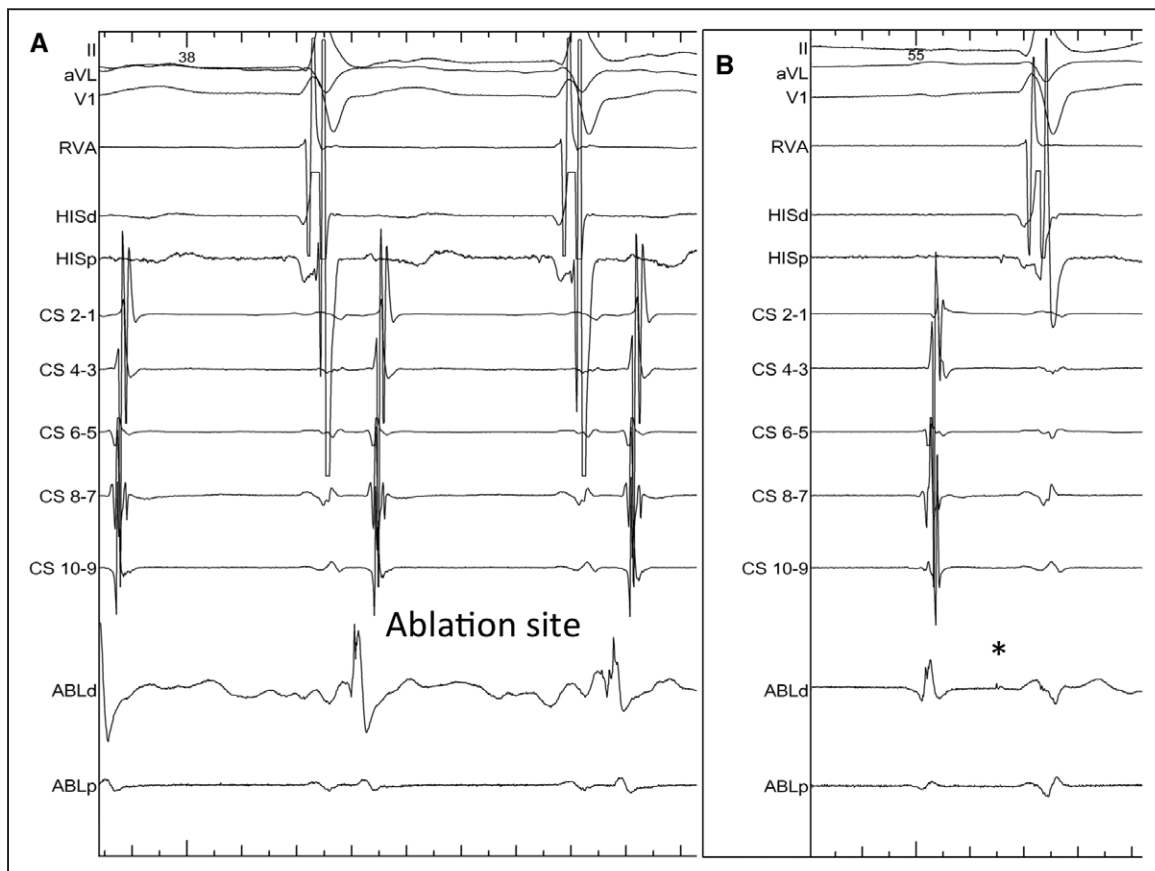


Figure 5. Intracardiac electrograms during the first EP study.

A, Orthodromic AV reentrant tachycardia with the ablation catheter showing continuous AV activation. Ablation at this site interrupted the tachycardia and led to PR interval prolongation, leading to immediate cessation of radiofrequency delivery. **B**, After ablation there is evidence of a near-field His potential (asterisk) at the ablation site and first-degree AV block because of AH interval prolongation, suggesting injury to the fast AV nodal pathway. Sweep speed=100 mm/s. II, aVL, and V1 indicate surface ECG leads; ABLd, distal ablation catheter; ABLp, proximal ablation catheter; AV, atrioventricular; CS, coronary sinus; EP, electrophysiological; HISd, distal His bundle; HISp, proximal His bundle; and RVA, right ventricular apex.

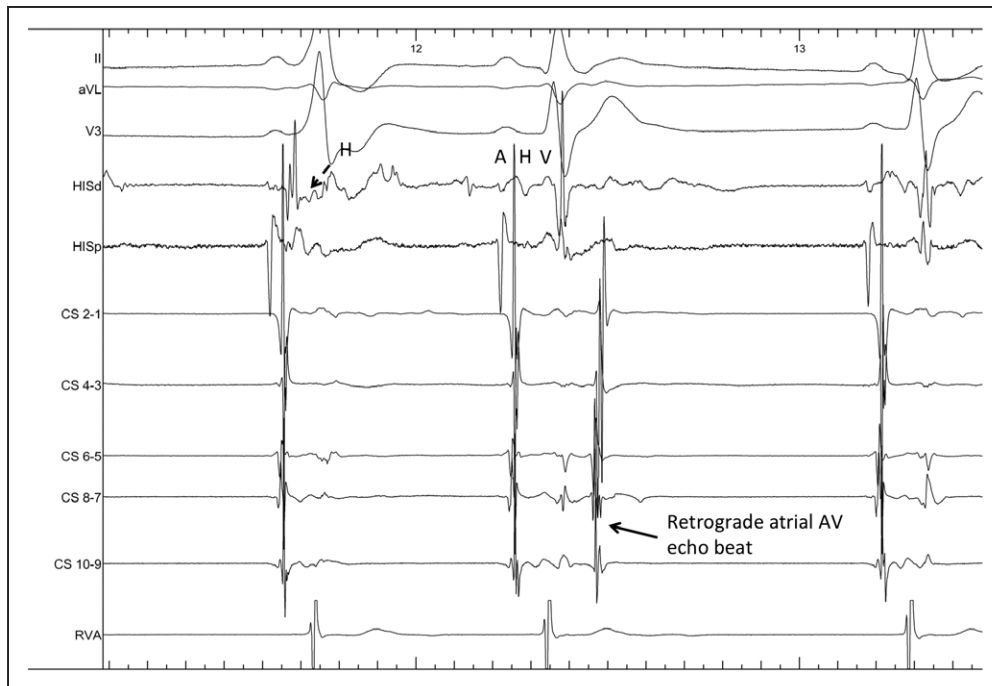


Figure 6. Intracardiac electrograms during sinus rhythm show a preexcited beat with negative HV interval (dashed line) followed by a sinus beat with AV nodal conduction and retrograde AV echo beat similar to the pattern observed in Figure 1.

II, aVL, and V3 indicate leads on surface ECG; AV, atrioventricular; CS, coronary sinus; HISd, distal His bundle; HISp, proximal His bundle; and RVA, right ventricle apex.

Response to intravenous adenosine bolus during tachycardia is shown in Figure 4. There is ECG evidence of interruption of tachycardia in the antegrade (atrioventricular [AV] nodal) limb. In addition, sinus beats have consistent retrograde conduction by the AP resulting in a 2:1 AV relationship. There is evidence of incomplete inhibition of the SP with persistent antegrade conduction. The fast perinodal pathway tissue has been described to be more responsive to adenosine in comparison with the SP.¹

At a previous electrophysiological study, the patient presented with persistent preexcitation and AP was mapped to the anteroseptal region of the tricuspid annulus. Radiofrequency ablation of the peri-Hisian AP during orthodromic AV reentrant tachycardia (Figure 5A) was prematurely interrupted because of the appearance of a high-frequency His potential at the ablation site immediately after abolition of the AP (Figure 5B). We therefore hypothesize this may have led to incomplete ablation of the AP and inadvertent damage to the fast perinodal pathway, resulting in the current pattern of alternating AV conduction.

The literature reports the occurrence of dual AV nodal physiology in 8% to 40% of patients with AP,² leading to a variety of possible reentrant circuits. Although this phenomenon is commonly observed in the electrophysiological laboratory, it is rarely evidenced in the surface ECG. A repeat EP study was performed confirming dual AV nodal physiology and documenting intermittent antegrade AV pathway conduction (Figure 6) with consistent AV echo beats during antegrade nodal conduc-

tion. An orthodromic AV reentrant tachycardia was reinduced, confirming the role of the AP in the mechanism of the clinical tachycardia. After 3-dimensional mapping with the EnSite Precision system with annotation of the His potential, successful reablation of the para-Hisian AP was performed. At the 6-month follow-up visit, the patient did not have recurrent preexcitation or tachycardia.

ARTICLE INFORMATION

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Disclosures

None.

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