



Antidromic tachycardia with RR interval variability: Take a closer look at the VH intervals

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Keywords:

Antidromic tachycardia
Wolff-Parkinson-White

Case presentation

A 25-year-old woman without structural heart disease was referred to our arrhythmia unit due to a history of well-tolerated recurrent

palpitations, which had never been graphically documented. An ECG showed manifest preexcitation (Fig. 1) suggestive of a posterior (left free wall) AP. During the EP study, there was baseline preexcitation, and the AH and HV intervals were 85 and 5 ms, respectively. A decapolar catheter was inserted into the coronary sinus (CS; interelectrode distance, 8–2 mm), and 3 quadripolar catheters were positioned in the high right atrium (HRA), His bundle region (H), and right ventricular apex (RV). Ventricular pacing showed concentric and decremental VA

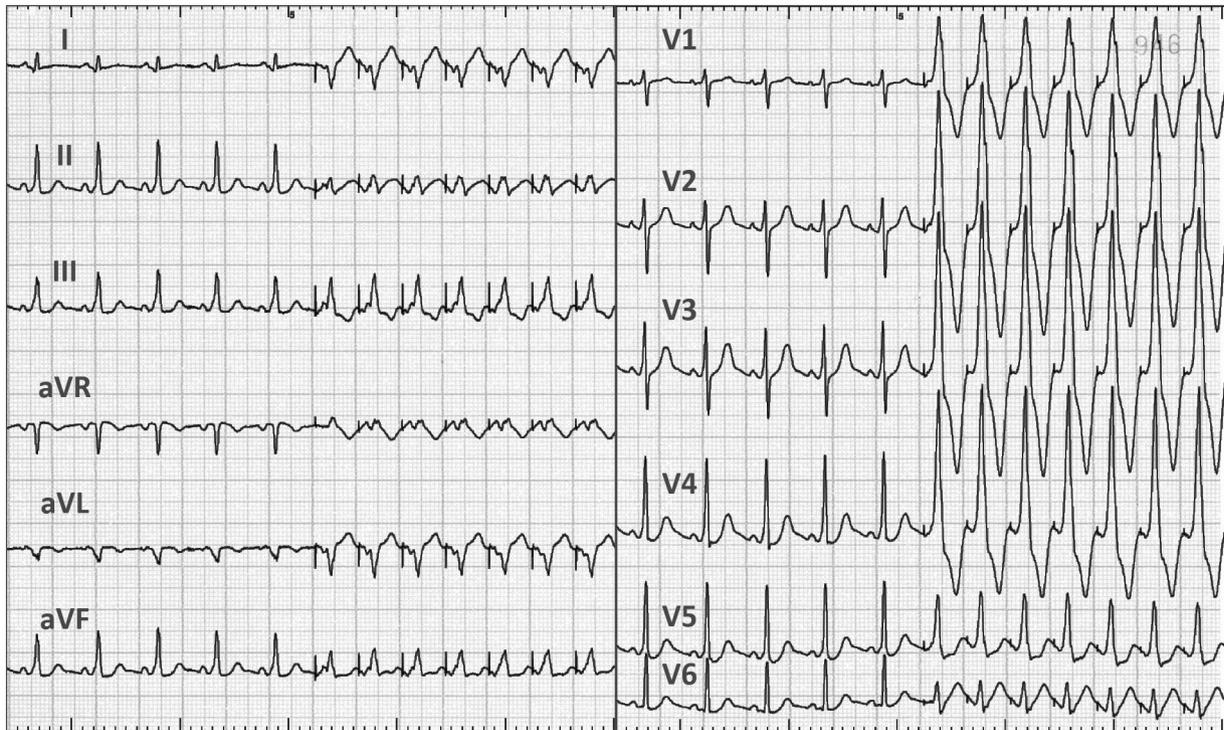


Fig. 1. 12-Lead ECG with manifest preexcitation suggestive of the presence of a posterior (left free wall) accessory pathway (5 first complexes). Pacing from distal coronary sinus at a cycle length of 400 ms showed marked preexcitation.

Abbreviations: ADT, Antidromic tachycardia; WPW, Wolff-Parkinson-White; AP, accessory pathway; HP, His-Purkinje; TCL, tachycardia cycle length.

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activation at all times, with no evidence of conduction over this or any other AP. Pacing from distal CS showed progressive preexcitation without decremental conduction over the AP (Fig. 1). There was no evidence of anterograde or retrograde dual AV nodal physiology.

With programmed atrial stimulation (basic CL 500 ms) and a single premature beat at a coupling interval of 270 ms, a non-sustained, wide QRS-complex tachycardia was repeatedly induced with a QRS morphology compatible with a maximal preexcitation pattern (Fig. 2A). Tachycardia CL was 299 ms, with an A-V-H activation sequence compatible with an ADT incorporating the AP as the anterograde limb and the contralateral bundle-branch/AV node as the retrograde limb. During this non-sustained tachycardia, the H deflection was clearly visible after the ventricular component (V) of the His bundle catheter (Fig. 2A), with a constant VH interval (onset of QRS to onset of the H) of 144 ms, an HA

interval of 67 ms and without changes in the TCL. The tachycardia always ended in the AV node.

With administration of isoprenaline, the same tachycardia was easily induced again (Fig. 2B) and sustained, with a CL of 233 ms. Now the H deflection was located before the V and the VH and AH intervals were 81 and 82 ms respectively (Fig. 2B). Late atrial premature beats introduced near the atrial insertion (distal CS) when the H was refractory resulted in preexcitation of the subsequent ventricular complex, advancement of the next H with the same VH interval, and retrograde preexcitation of the next atrial depolarization. Atrial entrainment of the tachycardia resulted in a similar QRS configuration.

Interestingly, during the first beats of this sustained tachycardia, the VH interval was irregular, with the H deflection sometimes occurring after the ventricular component, sometimes obscured within it and

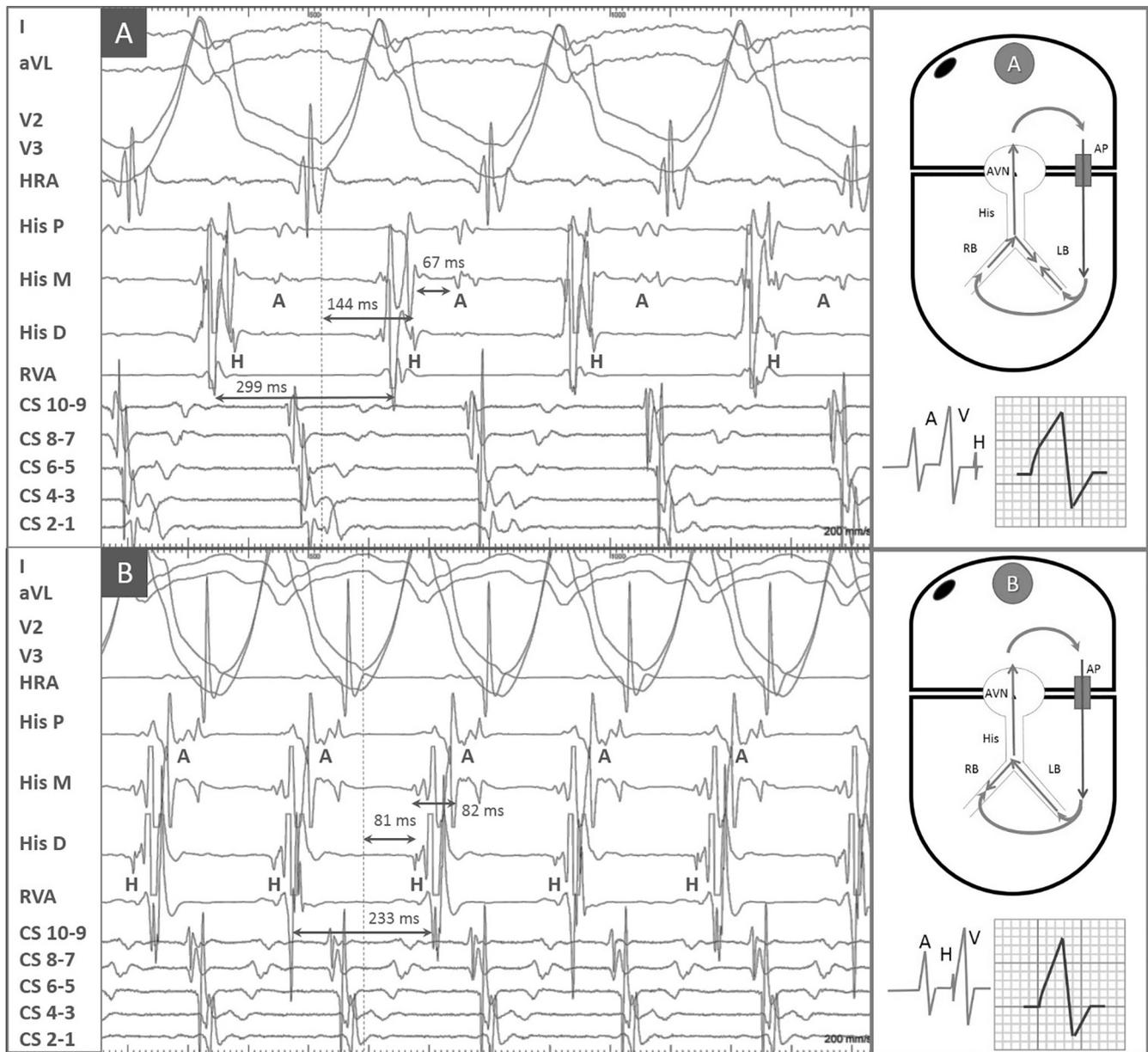


Fig. 2. A: Intracardiac recordings during the non-sustained antidromic atrioventricular reciprocating tachycardia (cycle length 299 ms). The His electrogram is clearly visible after the ventricular component of the His bundle catheter, with a constant long ventricular–His interval of 144 ms (dotted line indicates onset of delta wave) and a His–atrial interval of 67 ms, suggesting retrograde activation of the His–Purkinje system via the right bundle branch, serving as the retrograde limb of the circuit (see schema A). B: During the sustained antidromic atrioventricular reciprocating tachycardia (cycle length 233 ms), the His electrogram is now visible before the ventricular component of the His bundle catheter, with a short ventricular–His interval of 81 ms and a His–atrial interval of 82 ms, suggesting the use of the left bundle branch as the retrograde limb of the circuit (schema B). Shown are surface leads I, aVL, V2 and V3. HRA: high right atrium; His: His bundle electrogram (proximal, medium, distal); RVA: right ventricular apex; CS: coronary sinus bipolar recordings (10 proximal, 1 distal); AVN: AV node; AP: accessory pathway; RB: right bundle; LB: left bundle.

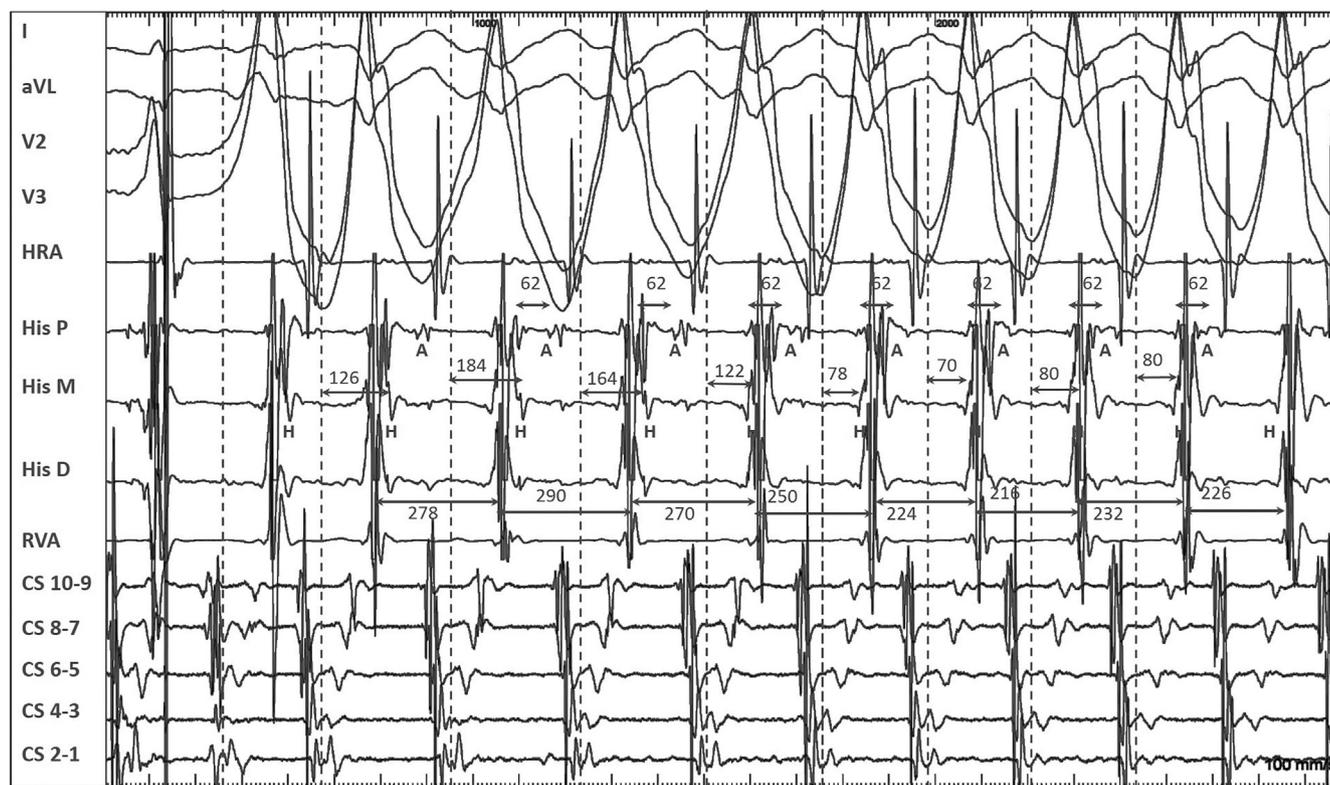


Fig. 3. Intracardiac recordings during the sustained antidromic atrioventricular reciprocating tachycardia that used a single left lateral accessory pathway. The cycle length of the tachycardia changed as a result of different ventricular-His intervals, without modification in the His-atrial interval (dotted lines indicates the onset of delta wave). This modification in the RR intervals after variable ventricular-His intervals indicates the requirement of the His-Purkinje system in the tachycardia circuit and rules out an AV reentrant tachycardia with bystander accessory pathway conduction. Shown are surface leads I, aVL, V2 and V3. HRA: high right atrium; His: His bundle electrogram (proximal, medium, distal); RVA: right ventricular apex; CS: coronary sinus bipolar recordings (10 proximal, 1 distal).

sometimes before it, without any changes in the HA interval (Fig. 3). This phenomenon was accompanied by subsequent changes in the tachycardia cycle length (faster with shorter VH intervals). A final diagnosis of an ADT with a single, non-decremental conduction AP located in the left free wall was made and with a single radiofrequency application the AP was abolished and no more tachycardias were inducible, with or without isoprenaline.

Discussion

Antidromic tachycardias (ADT) in the setting of Wolff-Parkinson-White (WPW) syndrome are rare. This finding has been clinically documented in less than 3% of patients, and incidence detected by electrophysiological (EP) study varies from 5% to 10% [1]. Most patients with an ADT are young males without structural heart disease, and in a majority of cases, a single accessory pathway (AP) serves as the anterograde limb, with the His-Purkinje (HP) system-AV node forming the retrograde limb. The AP is usually located in the right or left free wall, although some rare cases involving septal APs have also been described [2].

Assessment of an ADT is often challenging due to the different electrophysiological maneuvers that must be performed to confirm that it is a true ADT. Additional challenges include efforts to establish the type of AP according to its anterograde conduction properties (conventional or decremental) and the exact tachycardia circuit. Differential diagnosis must include the following: 1) orthodromic reentry tachycardia with aberration, 2) atrial tachycardia or flutter with preexcitation, 3) ADT due to AP conduction (duodromic tachycardia), 4) AV nodal reentry tachycardia with bystander AP conduction, 5) orthodromic tachycardia with bystander AP conduction, 6) ventricular tachycardia, and 7) nodofascicular or nodoventricular tachycardias.

In our case, during the process of diagnoses, we observed two important phenomena. First, the initial non-sustained and the second tachycardia had different VH intervals: long (144 ms) and short (81 ms), respectively, with the His potential after the ventricular component in the first one and before it in the second one, most probably reflecting that the first tachycardia used the contralateral bundle branch as the retrograde limb (via transseptal activation) and the faster one the ipsilateral bundle branch (see schema on Fig. 2). The HA interval was also slight different within the first (67 ms) and the second tachycardia (82 ms), most probably reflecting a delay in the retrograde AV node conduction. The presence of isoprenaline was important during the induction and maintaining of the second tachycardia, perhaps facilitating anterograde conduction over the AP and modifying the conduction properties of the intraventricular and HP system.

As a second important point, during the first beats of the sustained tachycardia, the RR interval changes were always preceded by changes in the VH interval, maintaining the same HA interval. This alternation in the tachycardia CL depending on changes in VH intervals clearly indicates the requirement of the HP system in the tachycardia circuit, being the best evidence for a macroreentrant tachycardia and against an AV nodal reentry tachycardia with bystander AP conduction.

Changes in VH intervals during an ADT have been previously described; these, however, are a rare phenomenon, and the incidence in previous series of ADT has not been described. Atie et al. [3] reported that 28% of ADTs in a series of 36 patients had sudden changes in the VA interval (17% because of changes in the VH interval and 11% in the HA interval), and in the classic paper by Packer et al. [4], 12 and 6 out of 30 ADTs had short and long VH intervals, respectively, and the His component was not seen in the other 12 cases.

The main question is why these changes in VH interval occur. Kuck et al. [5] speculated that changes in retrograde conduction time were

caused by different retrograde entries of the impulse into the bundle branch system and modifications in the refractoriness after the onset and during the tachycardia, mainly due to the fatigue phenomenon in one branch. Takahashi et al. [6] described one case of an ADT with a gradual shortening of the VH interval (and in the subsequent RR intervals) and speculated that the most plausible explanation was the resolution of the functional retrograde right bundle branch. Tchou et al. [7], in an elegant study, demonstrated that resolution of functional retrograde right bundle branch block can occur gradually as a result of migration of the site of the block from the distal to the proximal right bundle branch, with shortening of the TCL. Finally, Gandhavadi et al. [8] studied thirteen cases of ADT due to atriofascicular APs with anterograde decremental conduction properties, and all of them showed different and sudden changes in VH intervals (long and short) with subsequent changes in the TCL, suggesting the shift of conduction over the right bundle branch to the anterior or posterior left bundle fascicles.

In our case, the explanation might be in the beat beat-to-beat modification of the functional conduction properties of the HP system, changing the site of entry gradually from the contralateral to the ipsilateral bundle branch, although no definite proof could be provided because we did not make a detailed mapping of the right or left bundle branch potentials or myocardial activation.

But for practical purposes, our most important conclusion is that in the differential diagnosis of a wide QRS complex tachycardia in the setting of WPW syndrome, the finding of different VH intervals preceding changes in RR intervals without changes in the HA interval and in

the atrial activation sequence immediately suggests the antidromic type of circus movement tachycardia as the underlying mechanism and rules out an AV nodal reentry tachycardia with the AP as innocent bystander.

References

- [1] Brembilla-Perrot B, Pauriah M, Sellal JM, Zinzus PY, Schwartz J, de Chillou C, et al. Incidence and prognostic significance of spontaneous and inducible antidromic tachycardia. *Europace* 2013;15:871–6.
- [2] Man DC, Sarter BH, Coyne RF, Callans DJ, Schwartzman D, Gottlieb CD, et al. Antidromic reciprocating tachycardia in patients with paraseptal accessory pathways: importance of critical delay in the reentry circuit. *Pacing Clin Electrophysiol* 1999;22:386–9.
- [3] Atie J, Brugada P, Brugada J, Smeets JL, Cruz FS, Peres A, et al. Clinical and electrophysiologic characteristics of patients with antidromic circus movement tachycardia in the Wolff-Parkinson-White syndrome. *Am J Cardiol* 1990;66:1082–91.
- [4] Packer DL, Gallagher JJ, Prystowsky EN. Physiological substrate for antidromic reciprocating tachycardia. Prerequisite characteristics of the accessory pathway and atrioventricular conduction system. *Circulation* 1992;85:574–88.
- [5] Kuck KH, Brugada P, Wellens HJ. Observations on the antidromic type of circus movement tachycardia in the Wolff-Parkinson-White syndrome. *J Am Coll Cardiol* 1983;2:1003–10.
- [6] Takahashi K, Nagashima K, Okumura Y, Watanabe I, Iso K, Hirayama A. Resolution of the functional retrograde right bundle branch block during antidromic atrioventricular reciprocating tachycardia. *HeartRhythm Case Rep* 2017;3:519–22.
- [7] Tchou PJ, Trohman R, Kidwell G, Mehdirad AA. Retrograde migration of the site of functional block: a mechanism underlying resolution of functional retrograde bundle branch block during AV reentrant tachycardia. *J Cardiovasc Electrophysiol* 1996;7:335–40.
- [8] Gandhavadi M, Sternick EB, Jackman WM, Wellens HJ, Josephson ME. Characterization of the distal insertion of atriofascicular accessory pathways and mechanisms of QRS patterns in atriofascicular antidromic tachycardia. *Heart Rhythm* 2013;10:1385–92.