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CASE SERIES

Impairment of the antegrade fast pathway in patients with atrioventricular nodal reentrant tachycardia can be functional and treated by slow pathway ablation: a case report study

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Background Slow pathway (SP) ablation is considered to be the standard treatment for symptomatic atrioventricular nodal reentrant tachycardia (AVNRT). This may be challenging in patients with documented PR interval prolongation due to the potential increased risk of atrioventricular (AV) block in some patients.

Case Summary We report two cases of symptomatic recurrent AVNRT refractory to medical treatment with significant baseline PR interval prolongation (304 ms and 336 ms). In both of these cases, the baseline electrophysiological study demonstrated dual AV nodal physiology with a functional antegrade fast pathway and evidence for transient normalization of the PR interval. Slow/fast AVNRT was confirmed in both cases. Slow pathway ablation was successfully performed resulting in normalization of the PR interval to 144 ms and 168 ms with no evidence of AV block. After a mean follow-up of 30 months, the patients remained asymptomatic with normal PR interval, no recurrence of AVNRT, and no documentation of high degree AV block.

Discussion Our cases illustrate a common dilemma when dealing with patients with AVNRT and prolonged baseline interval. We show that SP ablation is feasible and safe as long as a preserved antegrade FP is present.

Keywords Atrioventricular nodal reentrant tachycardia • Prolonged PR interval • Slow pathway ablation • Functional fast pathway impairment

Learning points

- Catheter ablation in patients with atrioventricular nodal reentrant tachycardia and prolonged baseline PR interval is challenging and carries a high risk of atrioventricular block.
- Defining fast pathway (FP) antegrade conduction properties is the main clue to predict its behaviour after slow pathway (SP) ablation.
- Fast pathway impairment can be partially functional due to electrotonic interactions and concealed conduction. In this case, reproducible PR shortening can be observed before ablation. Isoprenaline and exercise testing can provide additional information about the FP properties.
- Functional FP inhibition would resolve after SP ablation with subsequent PR shortening.
- When antegrade FP conduction is preserved, SP ablation is feasible and safe.

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Introduction

Catheter ablation is the treatment of choice for symptomatic recurrent atrioventricular nodal reentrant tachycardia (AVNRT). Atrioventricular (AV) block is a rare but potentially serious complication which has been reported to occur in 1% of the cases.[1] The presence of a prolonged PR interval prior to ablation may increase the risk of AV block up to 25–30%.[2,3] The mechanisms underlying PR prolongation in the presence of a preserved antegrade fast pathway (FP) conduction remain poorly understood. Predicting antegrade conduction after slow pathway (SP) ablation may be challenging. We report two cases of symptomatic recurrent AVNRT refractory to medical treatment with a prolonged PR interval which underwent successful SP ablation followed by immediate PR normalization. Both patients provided a written consent.

Timeline

Clinical presentation	• Palpitations refractory to medical interventions • Long PR at baseline • Recurrent surpra-ventricular tachycardias requiring medical intervention
Interventions	• Confirm the typical atrioventricular nodal reentrant tachycardia by pacing maneuvers • Demonstrate the presence of dual nodal physiology with a preserved conduction through the fast pathway • Ablation targets the slow pathway in sinus rhythm with continuous monitoring of the A-V conduction
Result and outcome	• No adverse events • Normalization of the PR interval • No arrhythmia recurrence during the follow-up

Case 1

A 47-year-old woman was referred to our institution 1 year after an initial attempt of SP ablation complicated by PR prolongation. She was still complaining of recurrent episodes of palpitations. Baseline electrocardiogram (ECG) found an alternation between a prolonged PR interval of 304 ms and a normal PR interval of 168 ms (*Figure 1*). A narrow complex tachycardia was documented on 12-lead ECG despite treatment with Verapamil and Flecainide. The tachycardia cycle length was 328 ms. Transthoracic echocardiography (TTE) showed a structurally normal heart. Baseline electrophysiological study (EPS) demonstrated a normal HV interval (40 ms) and prolonged AH interval (208 ms). The tachycardia was easily inducible by rapid atrial pacing and atrial extrastimuli testing at a coupling interval of 600/400 ms. Of note, no AH jump (AH discontinuity of > 50 ms during atrial pacing with atrial extra-stimulus decrement of 10 ms interval) was documented. Low dose isoprenaline restored the PR interval to a normal range (130 ms) with an AH interval of 90 ms. Pacing manoeuvres helped to rule out the presence of concealed accessory pathway and confirmed the diagnosis of AVNRT. Mapping of the slow and FPs was performed during tachycardia, showing the earliest site of atrial activation at the superior septum consistent with retrograde FP activation. Ablation was performed posterior to the triangle of Koch based on an electrophysiological approach targeting SP potentials as described by Haissaguerre *et al.*[4] During ablation, a junctional rhythm was induced followed by PR normalization with a PR interval of 144 ms (*Figure 2*). Following ablation, the FP effective refractory period (ERP) was 600/570 ms, and the antegrade Wenckebach point was at 400 ms. Follow-up at 26 months showed no recurrence of tachycardia or significant AV block and maintenance of normal PR interval.

Case 2

An 80-year-old man, with a history of hypertension presented with a history of recurrent palpitations despite treatment with beta blockers. Baseline ECG showed a prolonged PR interval (336 ms) which was alternating with normal PR interval of 168 ms and intraventricular conduction abnormalities in the form of right bundle branch block and left anterior hemiblock (*Figure 1*). Spontaneous Wenckebach phenomenon was noticed at the 24-h Holter monitoring. ECG during palpitations ECG during palpitations showed a wide complex tachycardia with a heart rate of 120 b.p.m., the same ventricular morphology as during sinus rhythm and no evidence for retrograde P wave. Transthoracic echocardiography showed a structurally normal heart. The EPS showed a prolonged AH interval (236 ms) and a normal HV interval (46 ms). The tachycardia was easily inducible by atrial burst pacing at a cycle length of 400 ms. An interesting phenomenon of PR shortening after atrial premature complex (APC) was reproducible (*Figure 3*). Programmed atrial pacing with atrial extrastimuli reproducibly shortened the PR interval to a normal range. A similar approach as mentioned previously was used, allowing successful SP ablation with PR shortening (post-ablation PR: 168 ms). The EPS post-ablation found antegrade ERP of 600/540 ms and a Wenckebach point at 600 ms. The patient did well after the procedure with no recurrence of the tachycardia. Treadmill test performed 33 months later showed a normal adaptation of the PR interval with no high degree block; Wenckebach phenomenon without high degree block was still notable at rest and during the 24-h Holter monitoring.

Discussion

Dual nodal physiology is a main property of the atrioventricular node (AVN) that results from longitudinal dissociation of the slow and the FPs.[5] Nevertheless, certain physiological properties of the AVN continue to remain somewhat elusive resulting in this fundamental structure being described by Zipes.D as ‘a riddle wrapped in a mystery inside an enigma’.

Atrioventricular nodal reentrant tachycardia in patients with a significantly prolonged PR interval is uncommon occurring in less than 3% of the cases. The reasons for PR prolongation in patients with AVNRT and preserved antegrade FP conduction are not fully understood and predicting antegrade conduction in this group after SP

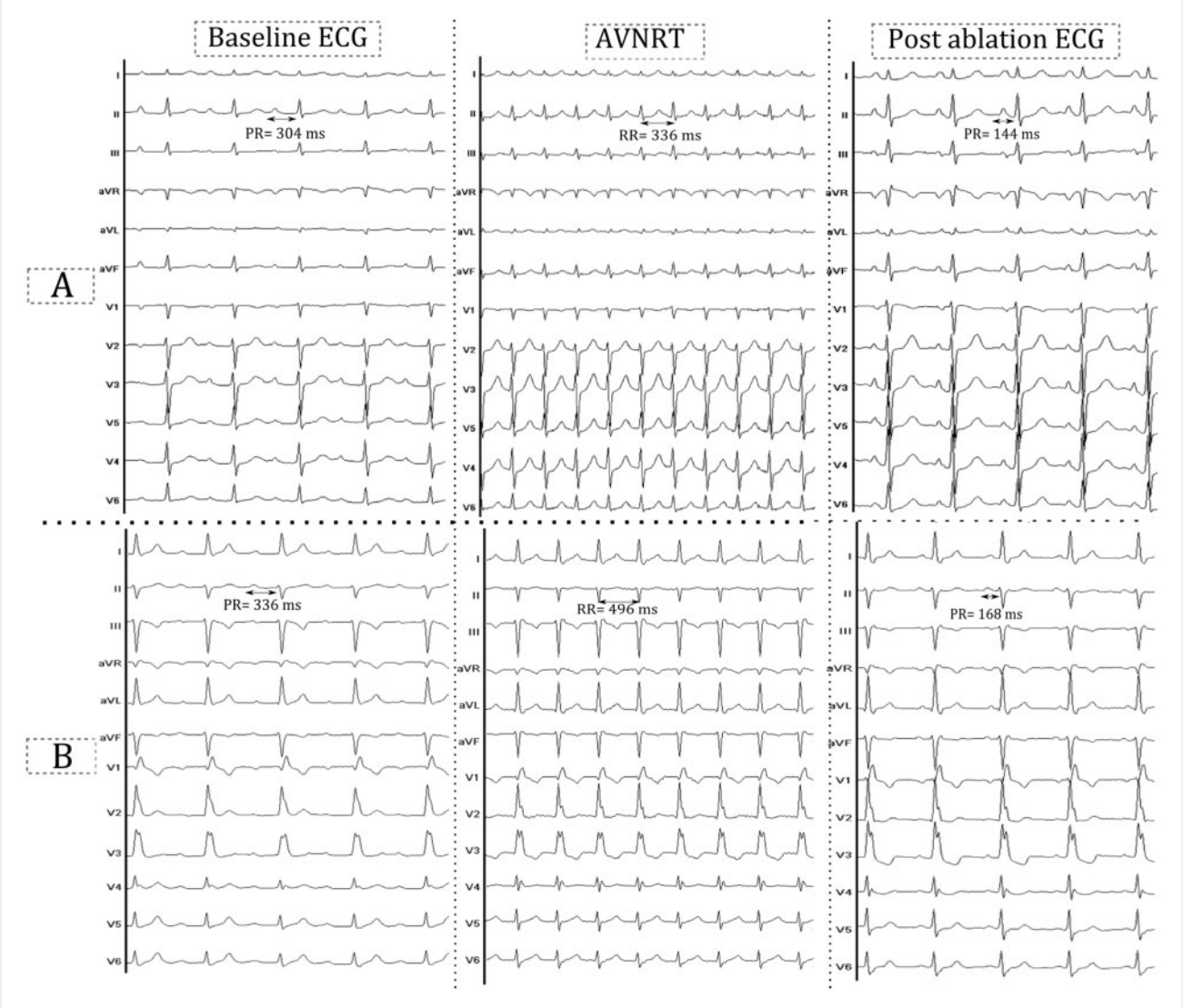

Figure 1 (*A* and *B*) refer respectively to Cases I and II. Baseline ECG shows a prolonged PR interval in both cases with intraventricular conduction abnormalities in *B* (right bundle branch block and anterior hemiblock). ECG during tachycardia is highly suggestive of slow/fast atrioventricular nodal reentrant tachycardia (AVNRT) in both cases. Slow pathway ablation resulted in normal PR interval with no longer inducible tachycardia. (Paper speed is 25 mm/s.)

ablation is challenging. We considered that FP conduction is preserved in the presence of dual nodal physiology with long episodes of normal PR interval. PR normalization after isoprenaline infusion or during exercise testing provide additional clue in favour of preserved antegrade conduction properties of the FP. These tests may be helpful to stratify the risk of AVB in the presence of prolonged baseline PR interval.

Potential explanations of PR prolongation in this population include:

(1) Preferential conduction over the SP in the presence of impaired FP antegrade conduction. These patients tend to exhibit two relatively distinct PR intervals representing alternating antegrade conduction over the FP and SP following atrial or ventricular premature complexes. These patients uncommonly present with AVNRT due to the presence of more extensive FP disease. In these cases, SP ablation had an associated high risk of AV block and retrograde FP ablation can be considered as alternative.[6,7]

(2) Preferential SP conduction in the presence of preserved FP antegrade conduction. Although the exact mechanism of these are not completely understood, electrotonic interactions are proposed as an important contributing factor.[8–10] In fact, the SP conduction may inhibit conduction via the FP by affecting the local recovery properties. This may result in concealed conduction through the FP that will further prolong its refractory period. This phenomenon was unmasked in Case 2 by concealed APC which prolongs the post-depolarization refractoriness and subsequently the SP refractory period.[11] Resetting of the AV nodal conduction properties is then associated with a normal PR due to the conduction via the FP. Autonomic tone may also modulate

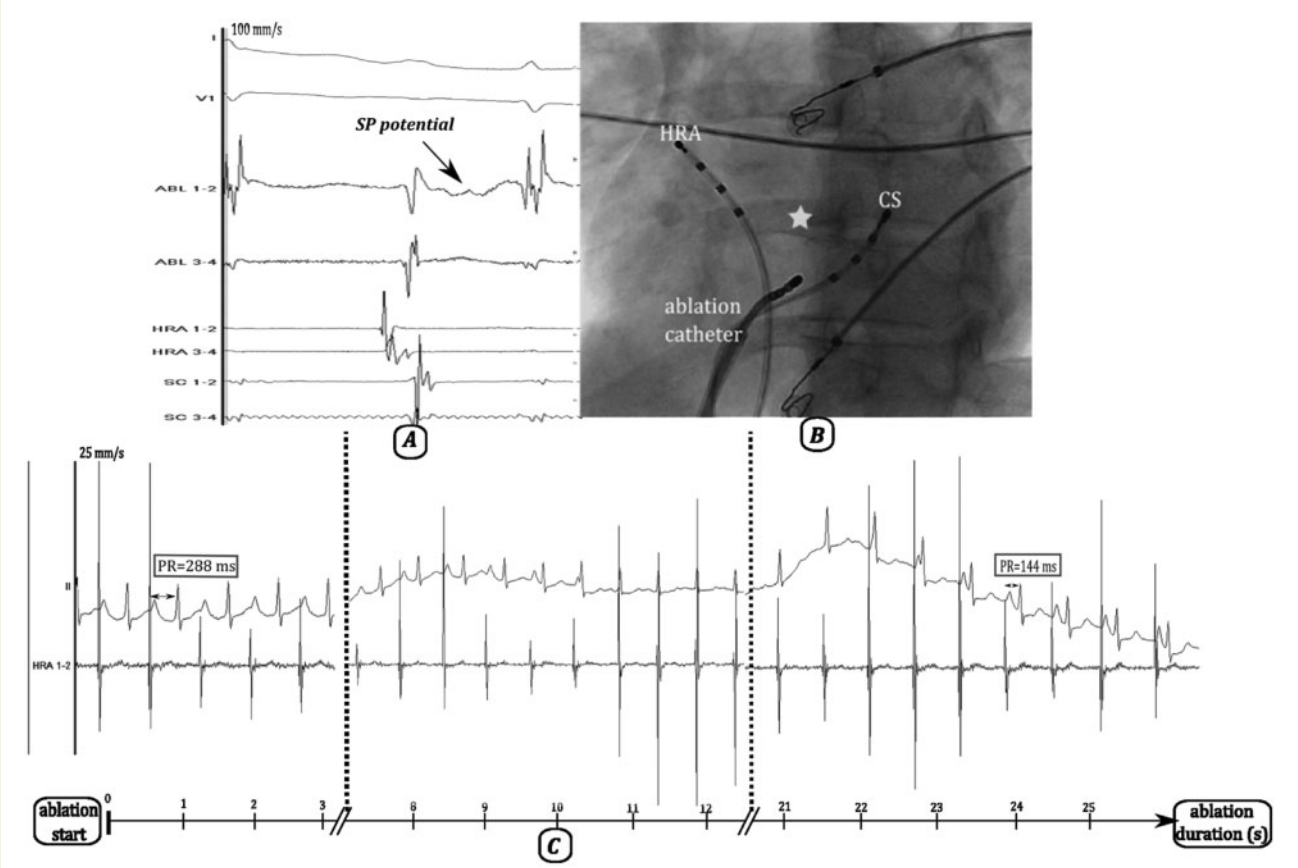

Figure 2 (*A*) Slow pathway potential recorded at the distal electrodes (Abl 1–2) of the ablation catheter; (*B*) Left anterior oblique (LAO) 30° fluoroscopic view showing the site of successful ablation corresponding to the site of the slow pathway potential; (*C*) Ablation at the site shown in (*B*) shows a prolonged PR interval at start (288 ms), a junctional rhythm was induced followed by a PR shortening to 144 ms when the ablation was stopped. The star refers to the His bundle location. Abl, ablation catheter; CS, coronary sinus; HRA, high right atrium; SP, slow pathway.

conduction via the AVN and contributes to an alternation in the PR interval. Electrotonic interactions would resolve after SP ablation with subsequent PR shortening.

In the cases which we present, PR prolongation may be the result of all these interactions in patients with potentially impaired FP (either by previous ablation or by conduction tissue degeneration).

The results of catheter ablation in patients with AVNRT and prolonged baseline PR interval were reported in few studies. Natale *et al.*[12] included seven patients with a mean baseline PR interval of 280 ms. Slow pathway ablation successfully eliminated the arrhythmia and shortened the PR to 230 ms in five patients. Lee *et al.*[7] reported 14 cases with prolonged AH interval of (172 ± 18 ms). Slow pathway was ablated in eight cases, and FP was ablated in the remaining cases. No PR shortening was noticed in both groups. After a mean follow-up of 25 months, one patient receiving SP ablation developed 2/1 AV block requiring a pacemaker. Pasquié *et al.*[13] studied 10 patients with slow fast AVNRT and moderately prolonged PR (222 ms ± 15 ms). Slow pathway ablation was associated with a transient complete AVB in two patients and a PR prolongation from 220 ms to 320 ms in one patient. The PR interval was unchanged in the remaining cases.

After a mean follow-up of 39 months, no patient presented with an AV block necessitating the implantation of a permanent pacemaker. In some reports, no PR interval shortening occurred attesting a previous conduction via the FP,[2] and in other reports, SP ablation was complicated by immediate[14] or delayed AV block.[3,15] Of note, dual nodal physiology could not be demonstrated in all cases described in the literature.

In our cases, SP ablation was safe with PR shortening and long-term freedom from high degree AV block. We preferred an electrophysiological approach targeting SP potential due to the higher risk of AV block in this population. Other authors adopted an anatomical approach with similar results.[12] The presence of preserved antegrade FP conduction is a key point. We believe that SP ablation may be safe and feasible in patients with recurrent AVNRT and baseline prolonged PR interval as long as the FP is partially functionally impaired and maintains a preserved antegrade conduction.

Conclusion

The association of AVNRT with prolonged PR interval is rare. The pathophysiological mechanisms remain scarcely enlightened. Slow

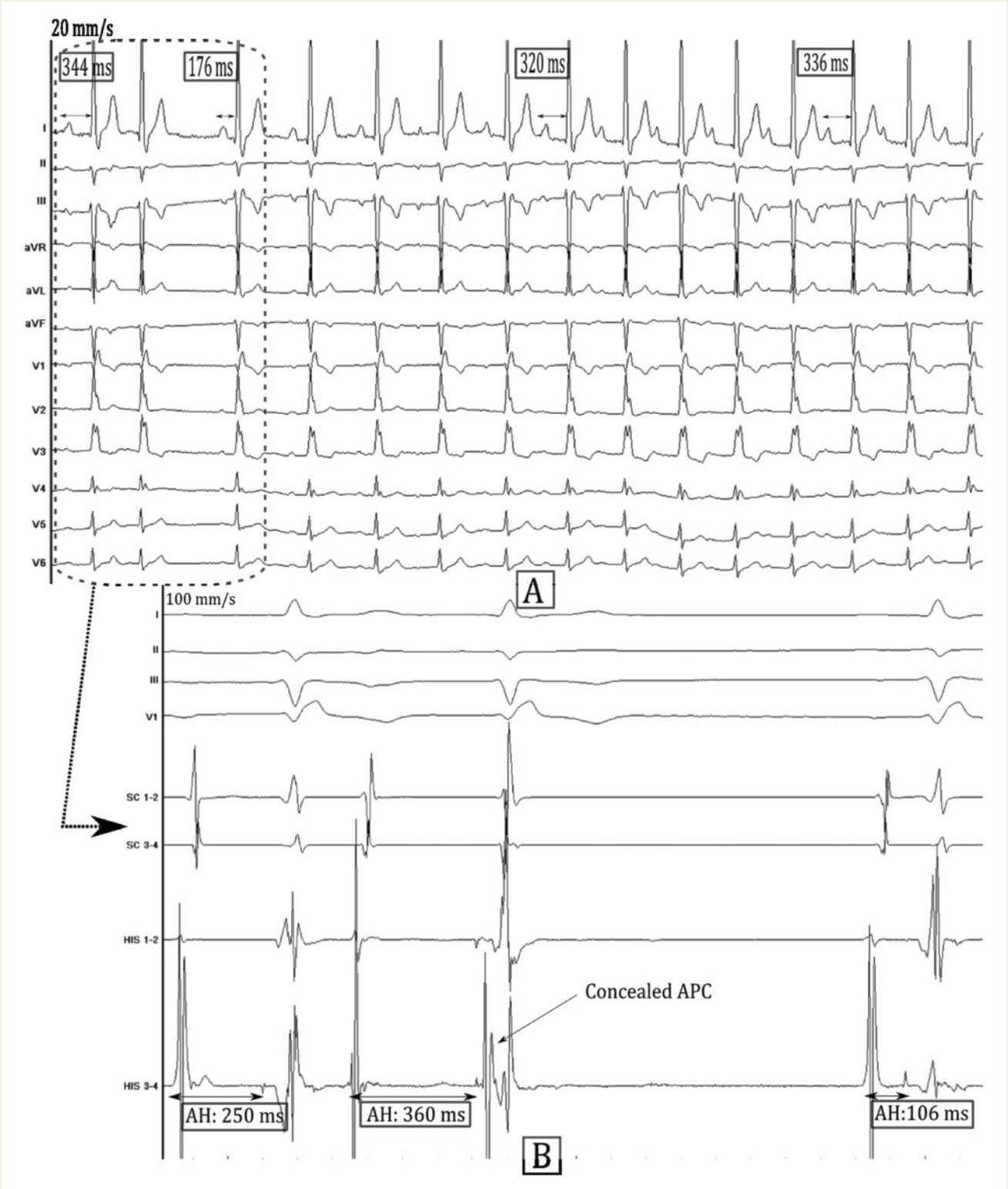

Figure 3 (*A*) 12-lead ECG with amplification of lead DI shows a PR shortening after an atrial premature complex (APC), PR is then prolonged progressively until 336 ms; (*B*) The corresponding endocavitary signals of the three first beats demonstrate that PR interval actually follows a concealed atrial premature complex that resetted the atrioventricular node and allowed conduction via the fast pathway with subsequent AH shortening.

pathway ablation may be feasible and safe as long as a preserved antegrade FP with evidence for normal PR interval before ablation are present.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patients in line with COPE guidance.

Conflict of interest: none declared.

References

1. Blomström-Lundqvist C, Scheinman MM, Aliot EM, Alpert JS, Calkins H, Camm AJ, Campbell WB, Haines DE, Kuck KH, Lerman BB, Miller DD, Shaeffer CW, Stevenson WG, Tomaselli GF, Antman EM, Smith SC, Alpert JS, Faxon DP, Fuster V, Gibbons RJ, Gregoratos G, Hiratzka LF, Hunt SA, Jacobs AK, Russell RO, Priori SG, Blanc J-J, Budaj A, Burgos EF, Cowie M, Deckers JW, Garcia MAA, Klein WW, Lekakis J, Lindahl B, Mazzotta G, Morais JCA, Oto A, Smiseth O, Trappe H-J. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias—executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (writing committee to develop guidelines for the management of patients with supraventricular arrhythmias) developed in collaboration with NASPE-Heart Rhythm Society. *J Am Coll Cardiol* 2003;**42**:1493–1531.
2. Sra JS, Jazayeri MR, Blanck Z, Deshpande S, Dhala AA, Akhtar M. Slow pathway ablation in patients with atrioventricular node reentrant tachycardia and a prolonged PR interval. *J Am Coll Cardiol* 1994;**24**:1064–1068.
3. Reithmann C, Remp T, Oversohl N, Steinbeck G. Ablation for atrioventricular nodal reentrant tachycardia with a prolonged PR interval during sinus rhythm: the risk of delayed higher-degree atrioventricular block. *J Cardiovasc Electrophysiol* 2006;**17**:973–979.
4. Haissaguerre M, Gaita F, Fischer B, Commenges D, Montserrat P, d'Ivernois C, Lemetayer P, Warin JF. Elimination of atrioventricular nodal reentrant tachycardia using discrete slow potentials to guide application of radiofrequency energy. *Circulation* 1992;**85**:2162–2175.
5. Kurian T, Ambrosi C, Hucker W, Fedorov VV, Efimov IR. Anatomy and electrophysiology of the human AV node. *Pacing Clin Electrophysiol* 2010;**33**:754–762.
6. Reithmann C, Hoffmann E, Grünewald A, Nimmermann P, Remp T, Dorwarth U, Steinbeck G. Fast pathway ablation in patients with common atrioventricular nodal reentrant tachycardia and prolonged PR interval during sinus rhythm. *Eur Heart J* 1998;**19**:929–935.
7. Lee SH, Chen SA, Tai CT, Chiang CE, Wen ZC, Chen YJ, Yu WC, Fong AN, Huang JL, Cheng JJ, Chang MS. Atrioventricular node reentrant tachycardia in patients with a prolonged AH interval during sinus rhythm: clinical features, electrophysiologic characteristics and results of radiofrequency ablation. *J Interv Card Electrophysiol* 1997;**1**:305–310.
8. Liu Y, Zeng W, Delmar M, Jalife J. Ionic mechanisms of electronic inhibition and concealed conduction in rabbit atrioventricular nodal myocytes. *Circulation* 1993;**88**:1634–1646.
9. Van Hare GF. Developmental aspects of atrioventricular node reentry tachycardia. *J Electrocardiol* 2008;**41**:480–482.
10. Spitzer KW, Sato N, Tanaka H, Firek L, Zaniboni M, Giles WR. Electrotonic modulation of electrical activity in rabbit atrioventricular node myocytes. *Am J Physiol* 1997;**273**:H767–H776.
11. Liu S, Olsson SB, Yang Y, Hertervig E, Kongstad O, Yuan S. Concealed conduction and dual pathway physiology of the atrioventricular node. *J Cardiovasc Electrophysiol* 2004;**15**:144–149.
12. Natale A, Greenfield RA, Geiger MJ, Newby KH, Kent V, Wharton JM, Kearney M. Safety of slow pathway ablation in patients with long PR interval: further evidence of fast and slow pathway interaction. *Pacing Clin Electrophysiol* 1997;**20**:1698–1703.
13. Pasquié JL, Scalzi J, Macia JC, Leclercq F, Grolleau-Raoux R. Long-term safety and efficacy of slow pathway ablation in patients with atrioventricular nodal re-entrant tachycardia and pre-existing prolonged PR interval. *Europace* 2006;**8**:129–133.
14. Rigden LB, Klein LS, MIttani RD, Zipes DP, Miles WM. Poster Session IV. *Pacing Clin Electrophysiol* 1995;**18**:914–932.
15. Li YG, Grönefeld G, Bender B, Machura C, Hohnloser SH. Risk of development of delayed atrioventricular block after slow pathway modification in patients with atrioventricular nodal reentrant tachycardia and a pre-existing prolonged PR interval. *Eur Heart J* 2001;**22**:89–95.