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Narrow complex tachycardia with discordant 12-lead RP intervals

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ABSTRACT

We present a case of supraventricular tachycardia (SVT) with discordant RP intervals on 12-lead ECG, rendering the diagnosis initially challenging. However, clues present on the ECG during SVT along with an ECG in sinus rhythm helped to elucidate the mechanism and the underlying diagnosis.

Case presentation

A 78-year-old man presented to the emergency department with a 30-min history of sudden onset, regular palpitations. He denied syncope, pre-syncope, chest pain or dyspnea. He had experienced similar, shorter-lived episodes in the preceding months. His past medical history was remarkable only for hypertension and hypercholesterolemia for which he took Irbesartan and Atorvastatin. Clinical examination revealed a heart rate of 170 bpm with a blood pressure of 140/80 mmHg. A 12-lead ECG showed a narrow complex tachycardia (Figs. 1 and 2 left panel) which was cardioverted successfully with the Valsalva manoeuvre (Fig. 2, right panel). What is unusual about the RP interval during the tachycardia? What is the diagnosis?

Interpretation

Figs. 1 and 2 (left panel) show a narrow complex, regular tachycardia at 170 bpm. Small positive deflections are noted in V1 and V2, suggestive of P waves, buried in the initial phase of the T waves with a 1:1 AV ratio (Fig. 2, red arrowheads). These divide the RR interval creating RP and PR intervals of 140 ms and 220 ms, respectively. The differential diagnosis of a short RP (RP < PR) supraventricular tachycardia (SVT) should include AVNRT, orthodromic AVRT and atrial tachycardia (AT), particularly given the presence of first-degree AV block in sinus rhythm as shown in Fig. 2 (right panel). Typical AVNRT is unlikely given the RP interval far exceeding 90 ms, a finding more consistent with AVRT [1], although this seems implausible in a septuagenarian with recent onset symptoms. Successful cardioversion with Valsalva manoeuvres is not a typical feature of AT but may occur when the underlying mechanism is triggered activity.

However, closer inspection of the ECG reveals the presence of further small, negative deflections distorting the terminal phase of the QRS complex best seen in inferior leads (Fig. 2, black arrowheads). These deflections also resemble P waves, creating a second RP interval of 80 ms and a PR interval of 280 ms, features more consistent with typical AVNRT. Thus, what is the diagnosis and the explanation for the discordant RP intervals?

A subsequent electrophysiological study provided the following findings: (i) the presence of retrograde atrial activation occurring earliest at the proximal coronary sinus with a short septal VA interval (<70 ms) [3] (Fig. 3); (ii) tachycardia induction was dependent on a critical AH interval with manifestation of a sudden "jump" in AH interval, indicating the presence of dual nodal pathways; (iii) His refractory premature complexes did not reset the tachycardia, thus excluding the presence of an accessory pathway; (iv) a V-A-V response was observed after ventricular entrainment, thus excluding atrial tachycardia; (v) a first post-pacing interval after tachycardia entrainment with correction for the atrioventricular interval of 200 ms [2]. Taken together, these findings were consistent with a diagnosis of typical AVNRT – anterograde conduction via the slow pathway and retrograde conduction via the fast pathway.

The ECG in sinus rhythm (Fig. 2, right panel) provides the explanation for the discordant RP intervals; the key abnormality is the long duration of the P wave (170 ms) which exhibits a biphasic morphology (positive-negative) in II, III, and aVF. This is an example of advanced interatrial block (A-IAB) due to blocked interatrial conduction through Bachmann's bundle. Consequently, left atrial activation is delayed and occurs "retrogradely" (i.e. caudocranially), via muscular connections located in the vicinity of the coronary sinus ostium. This creates a prolonged biphasic P wave (>120 ms) with a negative terminal component in inferior leads [4]. Fig. 1 is the electrocardiographic manifestation of the combination of A-IAB and typical AVNRT. The tachycardia makes the two phases of the P wave impossible to discern in any one lead. However, closer inspection permits identification of the distinct P

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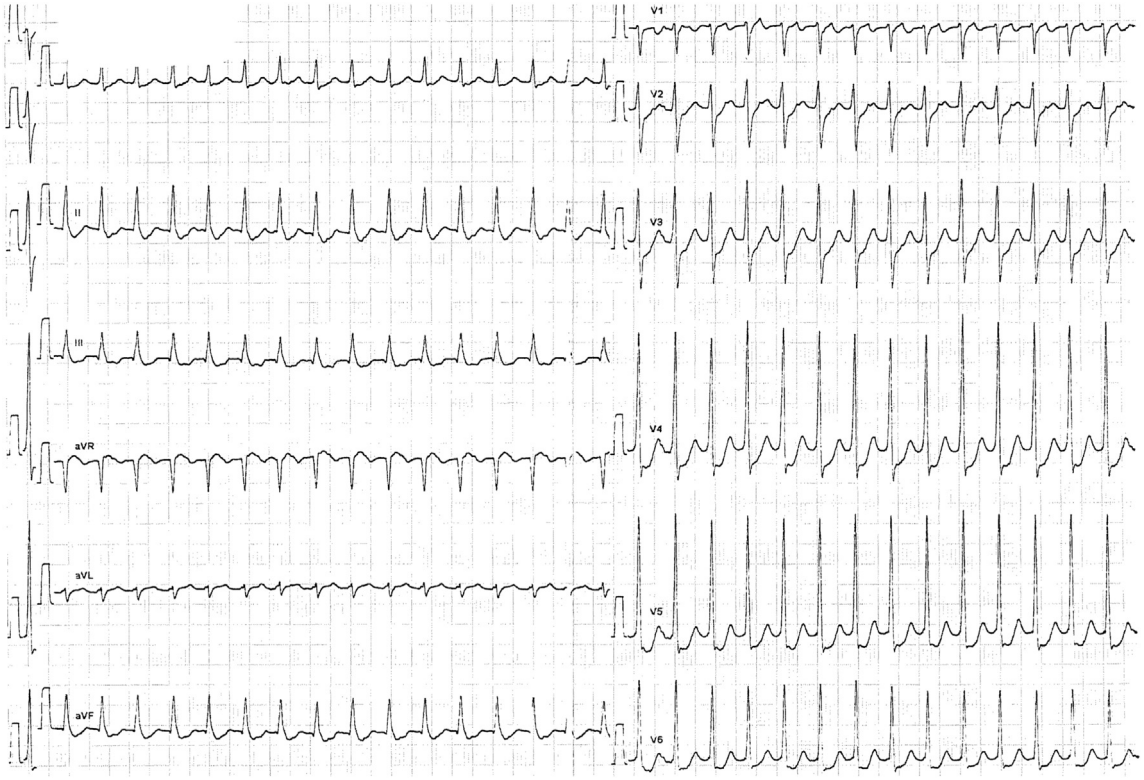

Fig. 1. 12-lead ECG of narrow complex tachycardia.

wave phases separately. During AVNRT, phase 1 of the P wave is visible as a negative deflection in inferior leads reflecting mainly the retrograde, caudocranial atrial activation which is relatively unaffected by the A-AIB. Conversely, phase 2 of the P wave represents the delayed atrial component which is only visible in the transverse plane (V1 and V2).

The patient underwent successful catheter ablation and remained symptom-free during follow-up. As recommended by current guidelines

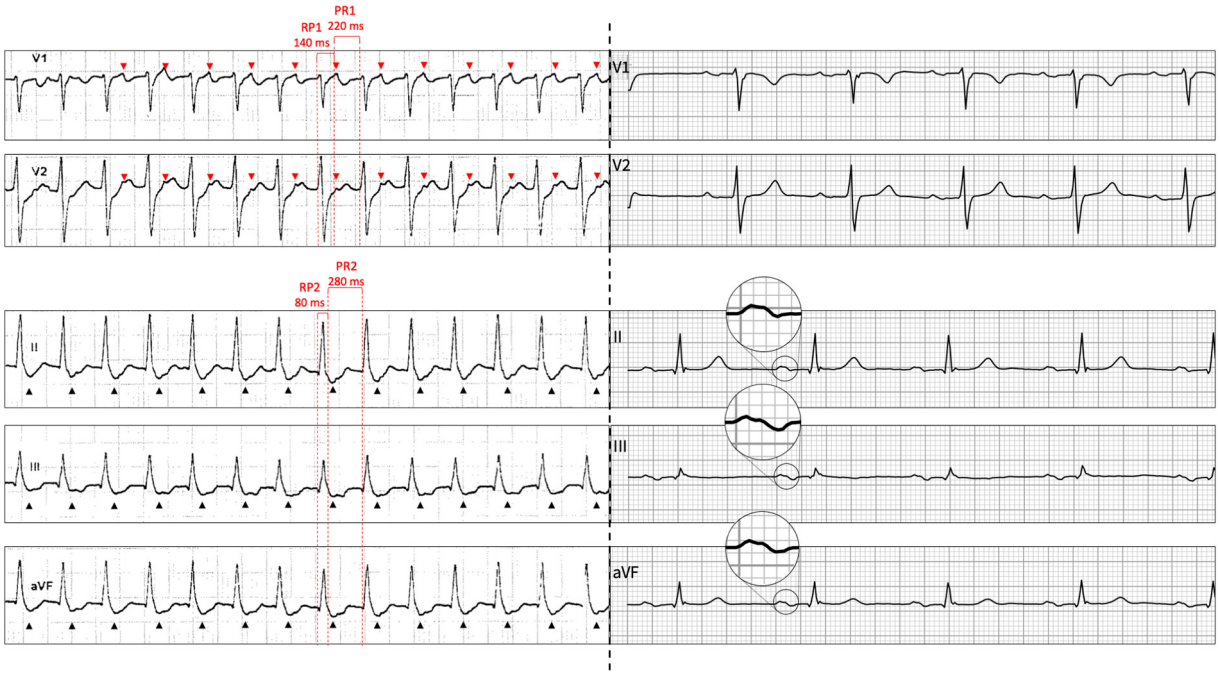

Fig. 2. *Left panel*: close-up of narrow complex tachycardia: in V1 and V2, P waves (red arrowheads) buried in the initial phase of the T waves divide the RR interval creating an RP interval of 140 ms and a PR interval 220 ms. In inferior leads, P waves (black arrowheads) create a second RP interval of 80 ms and a second PR interval of 280 ms; *Right panel*: sinus rhythm following cardioversion by the Valsalva manoeuvre. Advanced interatrial block (A-IAB) due to blocked interatrial conduction through Bachmann's bundle manifests as a prolonged P wave (170 ms) with a biphasic morphology (positive-negative) in II, III, and aVF.

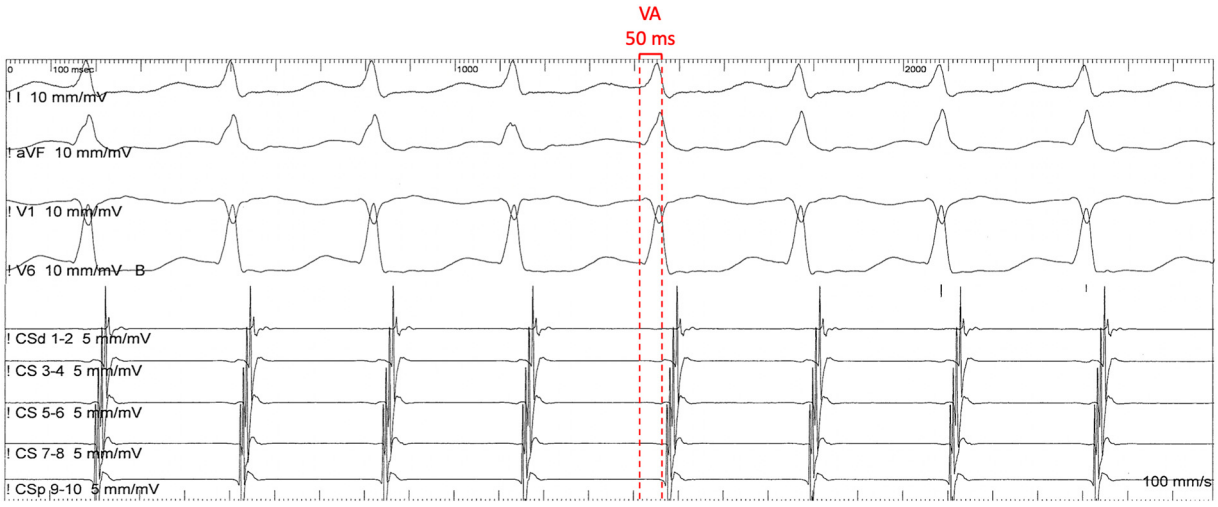

Fig. 3. Electrophysiological study.

[5], catheter ablation can be proposed as a first line treatment of symptomatic, recurrent AVNRT as it is associated with improved rhythm control and a lower rate of arrhythmia-related hospitalisation [6].

Discussion

The differential diagnosis of an SVT can typically be narrowed through measurement of the RP interval. However, in this unusual case, the presence of underlying A-IAB resulted in two distinct RP intervals during AVNRT, thus creating diagnostic uncertainty. Although the diagnosis was finally established with an electrophysiological study, close inspection of the ECG in sinus rhythm provided evidence of the underlying mechanism.

IAB is un underdiagnosed clinical entity caused by atrial structural remodelling and fibrosis. It exists in both partial (delayed conduction via Bachmann's bundle) and advanced forms (complete block of Bachmann's bundle). The prevalence of A-IAB increases with age with rates of 8% reported in patients between 70 and 80 years old, compared with 20% of centenarians [4].

The clinical relevance of A-IAB is related to its strong correlation with the occurrence of atrial arrhythmias, namely atrial fibrillation and atrial flutter, an association known as Bayés syndrome [7]. Our patient was not known for either arrhythmia. Although not currently recommended in current guidelines, given the strength of this association, additional screening by Holter monitoring may be justified in this case.

In summary, this case demonstrates the unusual combination of typical AVNRT and A-IAB. It highlights that, as a general principle, one should always consider the *earliest* timing of activation (e.g. RP intervals), and the *longest* activation duration (e.g. P waves) to avoid diagnostic wavering.

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Date availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author statement

TM and PP: data curation, data interpretation, drafting and review of the manuscript.

Declaration of Competing Interest

None.

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