Atrioventricular nodal reentrant tachycardia: Evidence of an upper common pathway in some patients

Claudio Hadid, MD,* Sergio Gonzalez, MD,† Jesús Almendral, MD, PhD‡

*From the *Hospital General de Agudos Cosme Argerich, Buenos Aires, Argentina, †Instituto de Cardiología, Tucumán, Argentina, and ‡Grupo HM Hospitales, University CEU–San Pablo, Madrid, Spain.*

Introduction

Atrioventricular nodal reentrant tachycardia (AVNRT) is the most common form of paroxysmal supraventricular tachycardia.[1] The concept of dual atrioventricular (AV) nodal physiology as the underlying substrate is well established. Although AVNRT was originally described as being confined within the compact AV node, whether some atrial tissue is part of the reentrant circuit remains unsettled. Josephson and Kastor[2] and Miller et al[3] proposed an entire intranodal circuit with an upper common pathway (UCP) localized between the upper turnaround point within the AV node (upper junction of fast and slow pathways) and both atria. In contrast, Otomo et al[4] and Heidbüchel and Jackman[5] described the AVNRT circuit as composed of the AV node, atrial myocardium, at least 2 atrionodal connections (ie, slow and fast pathways), and a lower common pathway.

KEY TEACHING POINTS

- The fact that the atria can dissociate from the tachycardia during atrioventricular nodal reentrant tachycardia (AVNRT) is indicative of the atria not being a component of the tachycardia circuit.
- Continuous atrial pacing at a cycle length equal to or longer than the tachycardia cycle length (TCL) resulted in Wenckebach AV conduction. The site of Wenckebach block likely is at an upper common pathway with different conduction properties during anterograde (atrial pacing) and retrograde (AVNRT) directions.
- An upper common pathway exists at or above the upper turnaround site in some patients with AVNRT.

Case reports

We describe 3 cases of AVNRT that are suggestive of the presence of a UCP.

Case 1

A 55-year-old woman had atypical AVNRT induced at electrophysiological study (EPS). The tachycardia cycle length (TCL) was 394 ms (Figure 1A), AH interval 183 ms, and HA interval 211 ms. Immediately after interruption of the tachycardia by atrial stimulation, continuous atrial pacing at the TCL was performed, and AV conduction with Wenckebach phenomena was seen (Figure 1B).

Case 2

A 62-year-old woman had slow–fast AVNRT induced at EPS. TCL was 411 ms. After 1 attempt at cryoablation of the slow pathway, AVNRT was still inducible but TCL increased to 470 ms (Figure 2A). The tachycardia showed some irregularities and oscillations in cycle length in the presence of constant VA intervals, suggesting variations in conduction time over the slow pathway or multiple slow pathways. After interruption of the tachycardia by ventricular stimulation, continuous atrial pacing was started. Wenckebach AV block was seen at a cycle length of 540 ms (Figure 2B).

Case 3

A 64-year-old man had documented supraventricular tachycardia. At EPS, AH "jump" and double AV response were seen (Figure 3A). Induced tachycardia showed retrograde HA Wenckebach block, followed by dissociation of the atria from the tachycardia (Figure 3B). AVNRT was diagnosed, and the tachycardia was rendered noninducible after slow pathway ablation.

In cases 1 and 3, slow pathway ablation was performed at the base of the triangle of Koch, near the coronary sinus ostium. In case 2, applications in the mid to high septum were required. In fact, the successful cryoenergy application had to be terminated prematurely because of PR prolongation.

KEYWORDS Ablation; Atrioventricular nodal reentrant tachycardia; Atrioventricular nodal reentrant tachycardia circuit; Fast pathway; Slow pathway; Supraventricular tachycardia; Upper common pathway
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Address reprint requests and correspondence: Dr Claudio Hadid, Hospital General de Agudos Cosme Argerich, Cardiologia, Pi y Margall 750, Buenos Aires C1155AHD, Argentina. E-mail address: claudio.hadid@gmail.com.

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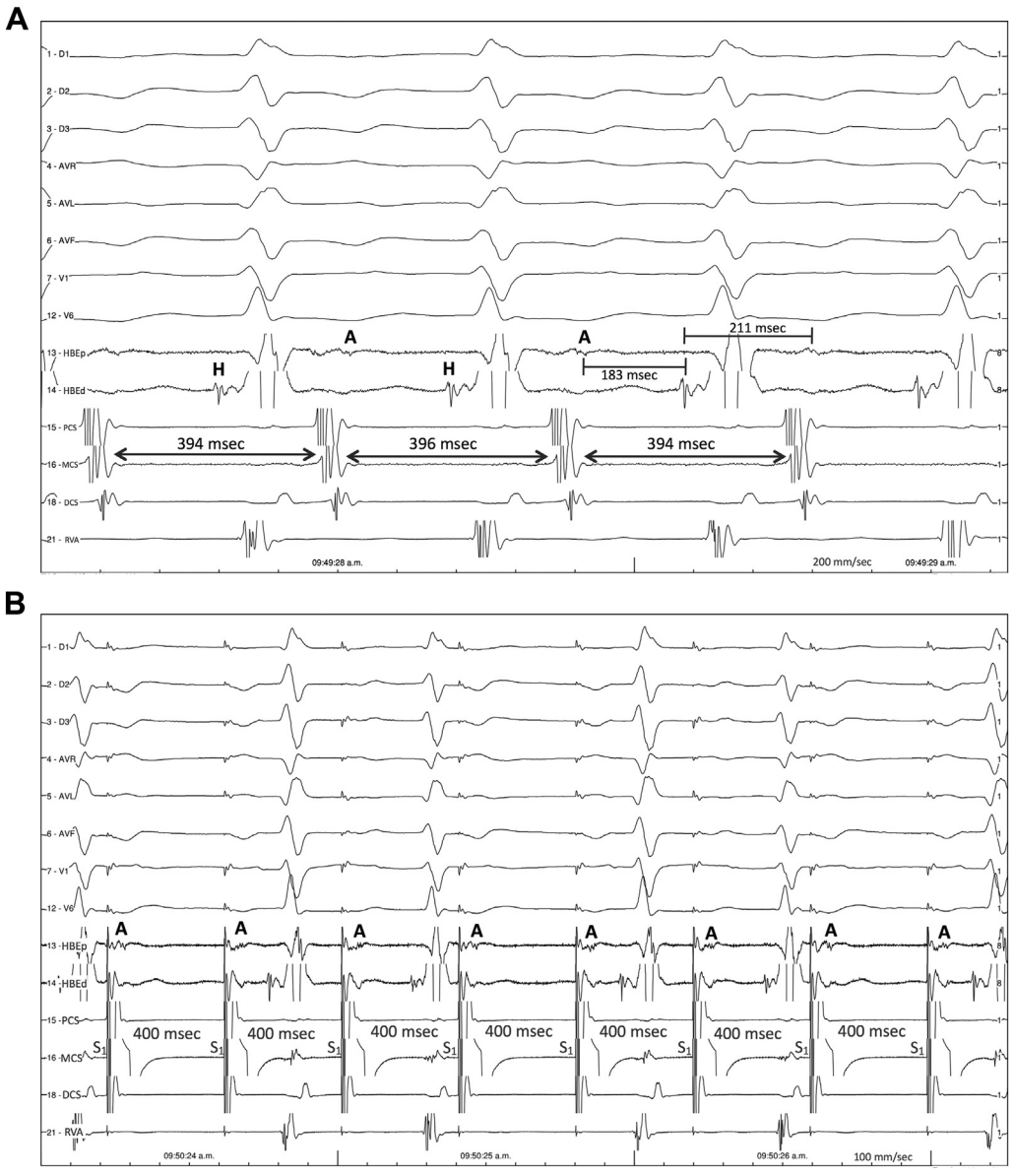

Figure 1 **A:** Fast–slow atrioventricular nodal reentrant tachycardia induced in case 1. **B:** Wenckebach AV conduction during continuous pacing at the tachycardia cycle length in the same patient. DCS = distal coronary sinus; HBE = His-bundle electrogram; MCS = middle coronary sinus; PCS = proximal coronary sinus; RVA = right ventricular apex.

Discussion

Cases 1 and 2 show similar findings in different types of AVNRT. Continuous atrial pacing at a cycle length equal to or longer than the TCL resulted in Wenckebach AV conduction. It is unlikely that such block occurs in a structure that conducted in the same direction 1:1 during tachycardia. In contrast, a UCP would conduct in the retrograde direction during tachycardia and in the anterograde direction during atrial pacing, and thus could have different conduction properties in both directions. This finding has been considered diagnostic of a UCP.[3] The same phenomenon, but occurring during ventricular pacing (absence of retrograde 1:1 conduction), was assumed to be indicative of the presence of a lower common pathway.[5] In case 3, AVNRT with retrograde Wenckebach conduction is followed by transient VA dissociation. The occurrence of block without tachycardia termination is indicative of the site of block being outside the tachycardia circuit. The fact that the atria can dissociate from the tachycardia is indicative of the atria not being a component of the tachycardia circuit.

Decades ago, Josephson and Kastor[2] addressed this issue by introducing atrial premature depolarizations during tachycardia. The ability to depolarize the atrial tissue surrounding the AV node (ie, the atrium recorded at the His-bundle electrogram, at the ostium, and within the coronary sinus) with an extrastimulus, without affecting the tachycardia, was suggestive of the atria not being part of the tachycardia circuit. The authors suggested that a subatrial or intranodal UCP may be

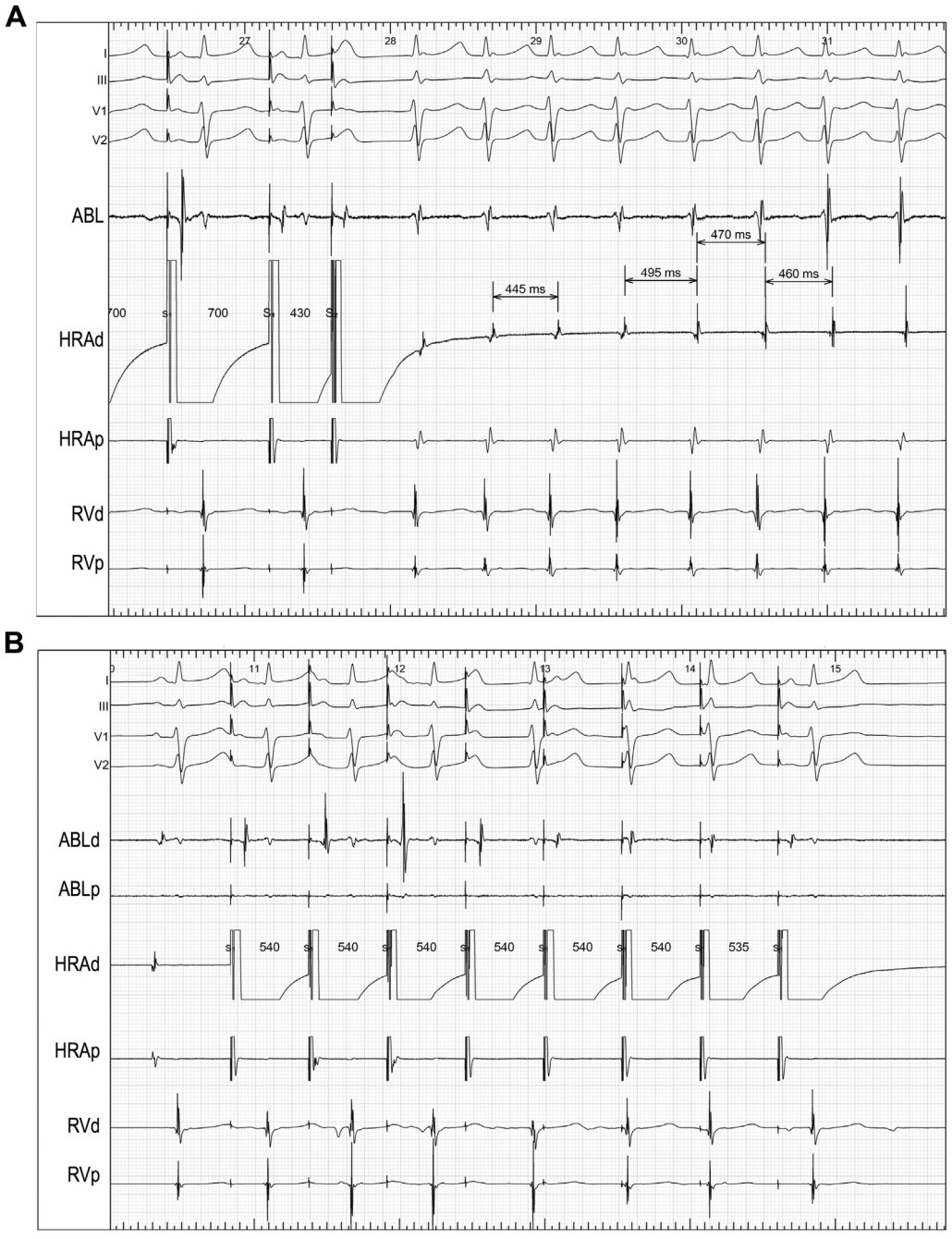

Figure 2 **A:** Slow–fast atrioventricular nodal reentrant tachycardia induced in case 2. **B:** Wenckebach AV conduction during continuous pacing slower than the tachycardia cycle length in the same patient. ABL = ablation catheter placed on the triangle of Koch; HRA = high right atrium; RV = right ventricle.

the necessary link between the slow and fast pathways without involving atrial tissue in the tachycardia circuit. In contrast, other authors have proposed that at least some atrial tissue may be necessary to link the fast with the slow pathway. The finding that most patients with slow–fast AVNRT have multiple, heterogeneous sites of early atrial activation during the arrhythmia rather than a focal breakthrough site argues against the concept of an anatomically discrete pathway.[6] McGuire et al[7] examined the electrical activation of the triangle of Koch and the proximal coronary sinus in 13 patients using a 60-point surgical plaque electrode. They described 2 groups of AVNRT, in which the earliest atrial activation was recorded at the apex of the triangle of Koch or near the coronary sinus ostium. In the 2 patients in whom rapid atrial pacing was performed during tachycardia, atrial activity could be advanced in all electrodes overlying the whole triangle of Koch without affecting subsequent ventricular activity, suggesting that atrial tissue did not participate in the reentrant circuit. In these 2 patients, dual sites of earliest atrial activation had been observed.

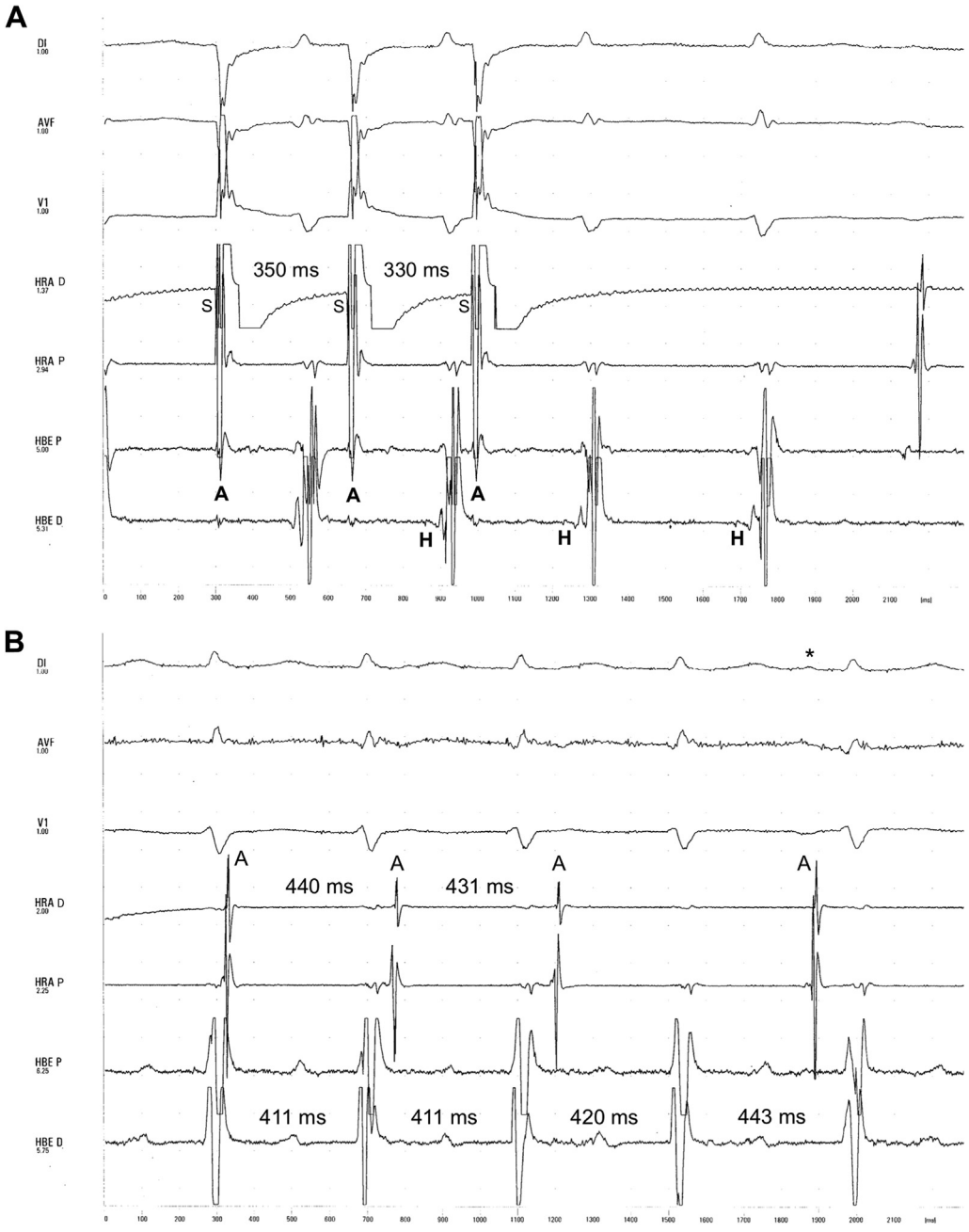

Figure 3 **A:** Double AV response during right atrial pacing in case 3. **B:** Induced tachycardia showed retrograde HA Wenckebach block, followed by dissociation of the right atrium from the tachycardia. HBE = His-bundle electrogram; HRA = high right atrium. *Sinus p wave.

Anatomically, the AV junction contains the conditions for the existence of functional pathways. A deep central portion is surrounded by successive layers of myocardium that gradually merge with atrial myocardium. In the region located anterior to the coronary sinus ostium, the terminal atrial tissue overlaps the AV node, showing a smooth rather than an abrupt transition.[8] This area has the lowest conduction velocity among cardiac tissues, including the node itself, and it has characteristics of functional longitudinal dissociation.[8] More recently, Katritsis and Becker[9] considered that atrionodal inputs, especially the inferior nodal extensions, seem to constitute a necessary limb of the tachycardia circuit in all forms of AVNRT.

Conclusion

The cases we presented support the concept that, at least in some patients with AVNRT, regardless of the type of AVNRT, a UCP is present between the tachycardia circuit and the atrial myocardium.

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