The existence of upper common pathway: Evidence from concomitant atrioventricular nodal reentrant tachycardia and atrial fibrillation

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Introduction

Despite being the most common supraventricular tachycardia in clinical practice, atrioventricular nodal reentrant tachycardia (AVNRT) still often imposes challenges both conceptually but also from a procedural standpoint. The anatomic substrate for AVNRT is still not fully understood and controversy remains as to whether the reentrant circuit involves part of the atrium.[1]

The model of a reentrant circuit with a slow pathway in the right atrial inferoseptal region and a more anteriorly located fast pathway in the apex of the Koch triangle has served as a simplistic conceptual framework, which, however, has been challenged. The notion of an upper common pathway localized between the upper turnaround point within the atrioventricular (AV) node upper junction of fast and slow pathways and the atria as a separate anatomical entity has also been disputed.[2,3] This controversy stems predominantly from the lack of any supportive histological evidence. It has been long recognized, however, that unlike the AV reentrant tachycardia, the atrium is not an obligate part of the concept circuit, as evidenced by the fact that the atria can dissociate from the tachycardia during AVNRT and also during pacing maneuvers. Nonetheless, more recent evidence suggests that perinodal atrial tissue may be part of the tachycardia circuit.

We report a case of typical AVNRT that persisted with various degrees of ventriculoatrial (VA) block as well as during sustained atrial fibrillation (AF), suggestive of the presence of an upper common pathway.

Case report

A 35-year-old woman with previous history of documented regular narrow complex tachycardia (NCT) came for an

KEY TEACHING POINTS

- In some patients with atrioventricular nodal reentrant tachycardia (AVNRT), there is evidence of presence of an upper common pathway.
- The upper common pathway may be anatomical or functional.
- The presence of concomitant atrial fibrillation and AVNRT provides the evidence of an upper common pathway.
- The right atrium is not an essential component of the AVNRT circuit.

elective electrophysiological (EP) study. The last episode of tachycardia occurred 2 weeks before and did not respond to adenosine administered by the paramedics. It terminated spontaneously soon after the patient was transferred to the emergency department. The recorded tachycardia had a narrow QRS complex, with a cycle length (CL) of 330 ms and no evident retrograde P waves (Figure 1A).

On presentation to the EP laboratory, she was in sinus rhythm (SR). Quadripolar catheters were advanced in the right ventricular apex (RVA), His position, and right atrium (RA). A deflectable decapolar catheter was inserted in the coronary sinus via the right femoral vein. The basic intervals at a sinus CL of 820 ms were AH: 83 ms; HV: 42 ms. With ventricular pacing, the retrograde conduction was concentric and decremental. During programmed atrial stimulation there was evidence of dual AV node physiology and possibly presence of more than 1 antegradely contacting slow pathway. A nonsustained 1:1 regular NCT with a CL varying between 280 and 350 ms was easily inducible with atrial pacing, without the use of isoprenaline. The atrial activation during the tachycardia was concentric and the septal VA time was 20 ms (Figure 1B). On 2 occasions, the tachycardia was induced after a 2-for-1 response (Figure 1C). The tachycardia terminated spontaneously on more than 1 occasion with the
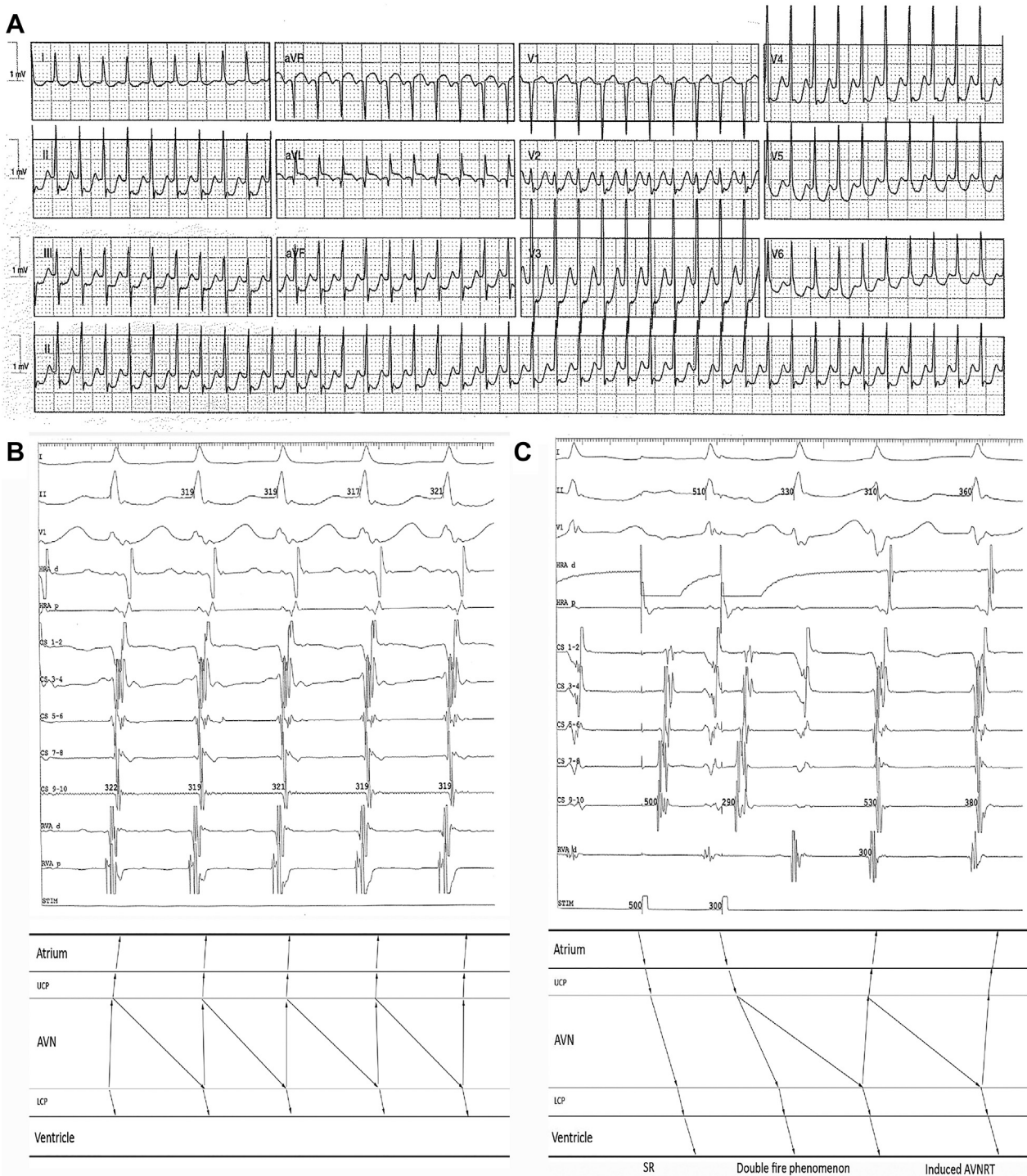
Figure 1   **A:** Electrocardiogram during narrow complex tachycardia. **B:** Intracardiac electrogram showing typical slow-fast atrioventricular nodal reentrant tachycardia (AVNRT). **C:** Intracardiac electrogram showing 2-for-1 response and induction of AVNRT.

atrial electrogram being consistently the last, essentially ruling out atrial tachycardia as the mechanism. With isoprenaline the inducible tachycardia became sustained. His synchronous premature ventricular contractions did not advance or delay the atrial electrogram and/or the tachycardia. Entrainment from the RVA at a CL 10–20 ms shorter than the tachycardia cycle length (TCL) consistently elicited

a V-A-V response with a postpacing interval (PPI) – TCL difference of 170 ms and stimulus atrial – VA difference of 115 ms. Attempts to entrain the tachycardia with atrial pacing at 260 ms (TCL of 280 ms) dissociated the atrium without influencing TCL, which continued with the same activation sequence and the same CL after the end of the atrial pacing (Figure 2A). These findings excluded AV reentrant
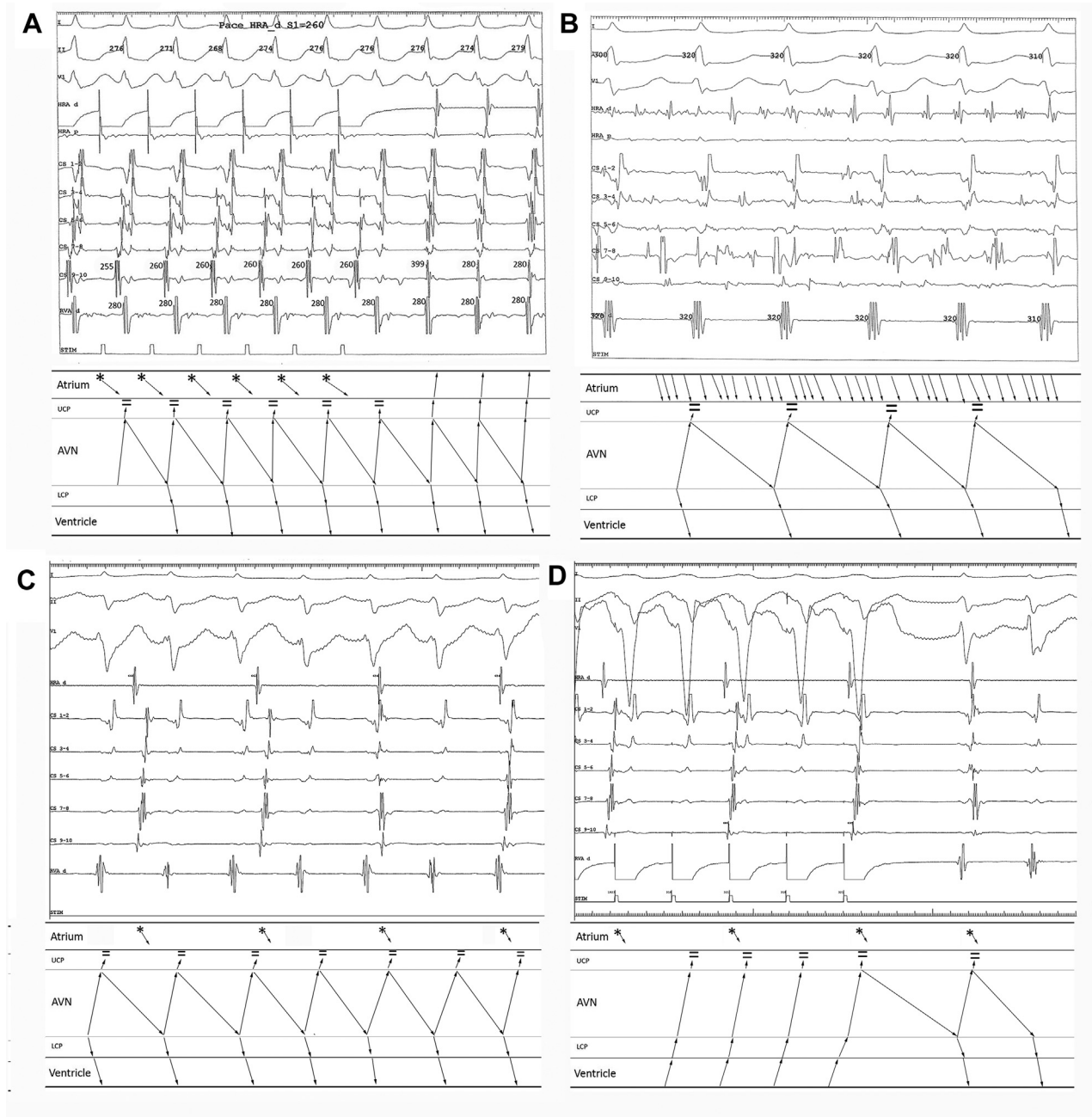
Figure 2 Evidence of existence of upper common pathway in atrioventricular nodal reentrant tachycardia (AVNRT). **A:** Atrial pacing during AVNRT with A-V dissociation. **B:** Atrial fibrillation and AVNRT, showing dissociation of atrium from tachycardia. **C:** AVNRT with retrograde complete vemtriculoatrial (VA) block and VA dissociation. **D:** Ventricular pacing during AVNRT with VA dissociation.

tachycardia. During the tachycardia, intermittent loss of atrial depolarizations were noted, as well as periods of Wenckebach and 2:1 – 3:1 VA periodicity with fixed VA interval in the ventricular electrograms that were linked to an atrial electrogram. Further attempt to entrain the tachycardia from the RVA at this point led at the end of the pacing train to complete VA dissociation, with the ventricular rate continuing unchanged (CL of 280–300 ms) and the atrial activity now having a CL of 640–700 ms (likely at the sinus rate). The QRS morphology and the HV interval remained the same as at baseline (45 ms), during both the 1:1

tachycardia and the dissociated tachycardia with V>A. Ventricular pacing at a CL faster than the tachycardia did not have any effect on the tachycardia and the complete retrograde VA block persisted. Adenosine was administered intravenously (6 mg followed by 12 mg) and also had no effect. While the dissociated tachycardia persisted, the patient felt very unwell and the systolic blood pressure dropped to 90 mm Hg. A 50 J synchronized DC shock restored SR with a rate of 90 beats per minute and 1:1 AV conduction and the same basic intervals as at the beginning of the study. With burst atrial pacing the same 1:1 tachycardia with similar

CL (280–310 ms) was easily reinduced. Shortly after its reinitiation, the same phenomenon with the complete VA dissociation and continuation of the tachycardia reoccurred. Following this, however, and without any interruption of the tachycardia or any change in its CL, the onset of AF was noted in the coronary sinus catheter (Figure 2B). The AF terminated spontaneously after approximately 3 minutes, and without any interruption of the tachycardia the previous complete AV dissociation (Figure 2C) continued for a few more minutes before the tachycardia also terminated spontaneously with restoration of SR. In addition, RV over-drive pacing during tachycardia showed V-A dissociation (Figure 2D).

A slow pathway ablation was performed in the postero-septal region between the inferior aspect of the coronary sinus ostium and the tricuspid valve annulus with a Biosense Webster 3.5 mm bidirectional catheter (upper temperature limit 60°C, maximal power output 40 W). Junctional tachycardia was induced after each application. Postablation, no inducible tachycardia and no evidence of presence of dual AV node physiology were noted.

The patient had no further episodes of tachycardia 3 months after the procedure. (Prior to this she had several episodes every month.)

Discussion

Our case supports the long-debated concept of the presence of an upper common pathway in some patients with AVNRT. To our knowledge, there is only 1 other similar case in the literature where a dual tachycardia (AVNRT and AF) occurred.[4]

Previously reported different VA block patterns during AVNRT,[2,5] intermittent loss of atrial depolarizations, and variable HA conduction times during a fixed His-to-His inter-val with 1:1 VA relationship were thought to suggest the presence of an upper common pathway between the reentrant circuit and the atrium.[2] The differential diagnosis of an NCT with a VA block pattern includes junctional tachycardia, intrahisian reentry, and nodofascicular tachycardia using the His-Purkinje system for antegrade conduction and a nodofascicular pathway for retrograde conduction. In our case there was no evidence of pre-excitation at any point at baseline or during the study, nor did we observe the initiation of the tachycardia with ventricular extrastimuli without retro-grade His deflection. The rate of the tachycardia was faster than one would expect in junctional tachycardia owing to increased automaticity; however, no pacing maneuvers were performed to specifically rule out this possibility. After the slow pathway ablation there has been no recurrence of the tachycardia.

The AVNRT circuit appeared in our case to be heteroge-neously coupled antegradely and retrogradely to the atrial tissue above it. It was isolated from the atrial fibrillatory activity as well as during the attempts to overdrive the tachy-cardia with atrial pacing, while such isolation did not appear to be present in SR during atrial pacing; it also demonstrated various degrees of retrograde VA dissociation during the

AVNRT but not during ventricular pacing (at baseline the retrograde Wenckebach CL was 360 ms and the tachycardia could be entrained with ventricular pacing at a CL 20 ms faster than the TCL when the atria were not fibrillating). These phenomena are in support of functional block, presumably due to nonuniform anisotropy.

Whether the upper common pathway is a discrete histological structure or simply functional has been a matter of debate for a long time.[6,7] There has been evidence suggesting that AVNRT most likely results from reentry in various locations in the AV nodal area but also involving the atrial perinodal area. The model of a reentrant circuit that consists of 2 anatomically distinct limbs that are confined to the AV node, although conceptually useful, is likely over-simplistic.[8–10] The concept of an upper common pathway, however, remains speculative despite previously observed various degrees of VA dissociation during AVNRT.[2,5,11] In our case the presence of an upper common pathway is speculated not only because of the various types of VA block noted but also, importantly, by the coexistence of AVNRT and sustained AF for part of our study. The disagreement as to whether an "upper com-mon pathway" exists as a discrete anatomic entity appears to stem predominantly from differences in the definitions of what comprises the "AV node." Previous morphologic studies of the AV node have shown a superior dense network of nodal tissue ("compact AV node"), an inferior portion of the AV node into which atrial bands gradually merged (transitional cell zone), and superficial transitional cells along the anterior limbus of the fossa ovalis. Therefore much of the disagreement on the role of the atrium in the genesis of the tachycardia circuit seems to stem from the exact definition of the extent of the AV node and specif-ically from the failure to recognize the transitional cell zone as part of the AV node.[5] It is also possible that the upper common pathway, rather than being a discrete histological entity, is rather functional, which explains the failure of previous histologic studies to demonstrate its presence.[3,7] This likely also explains the observations in our case. Another possibility is that previous histologic studies were simply unable to differentiate perinodal atrial myocardium from AV nodal transitional tissue with electro-physiologic characteristics resembling those of AV nodal cells that form part(s) of the tachycardia circuit and/or the upper common pathway.[7]

Intravenous adenosine has a class I indication for AVNRT termination, with success rates ranging from 78% to 96%.[12] However, our case did not demonstrate tachycardia termina-tion after adenosine administration. Although it has been previously postulated that adenosine administration induces AV conduction block in general, a previous animal study showed that 3 distinct groups of cells in the AV node (atrio-nodal, nodal, and nodal–His bundle cells) demonstrated different responses to adenosine administration, with the specific target for adenosine being the nodal cells.[13] Another study in normal human hearts showed a transient prolonga-tion of AH interval without affecting HV interval.[14] These

observations suggest that adenosine acts on the proximal portion of the AV junction, which is possibly proximal to the site of anatomical/functional block in our case. In addition, previous studies also demonstrated clear differences in the ability of adenosine to suppress conduction in fast and slow pathway and 38% of patients with typical AVNRT did not demonstrate retrograde fast pathway block after adenosine.[15] The differential response of antegrade and retrograde conduction to adenosine may also have a role in the failure response to adenosine.

Conclusion

The case we presented supports the debated notion of the presence of an upper common pathway between the reentrant circuit and the atrial myocardium in at least some of the patients with AVNRT. The presence of an upper common pathway explains not only the various degrees of VA block observed in our study but also the occurrence of 2 simultaneous tachycardias (AVNRT and AF).

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