

Always Worth to Keep in Mind

# Atrioventricular Nodal Reentrant Tachycardia with Upper Common Pathway Block

Jan Berg<sup>a,b</sup>, Daniel Hofer<sup>a</sup>, Feifan Ouyang<sup>c</sup>, Ardan M. Saguner<sup>a</sup><sup>a</sup> Department of Cardiology, University Heart Centre, University Hospital Zurich, Switzerland; <sup>b</sup> Department of Cardiology, Kantonsspital Aarau, Switzerland;<sup>c</sup> Department of Cardiology, University Hospital Hamburg-Eppendorf, Hamburg, Germany

## Summary

We present the case of a 34-year-old man with paroxysmal narrow complex tachycardias. An EP study revealed AVNRT with upper common pathway block. Our case shows a diagnostic approach and treatment for this rare scenario in the EP lab.

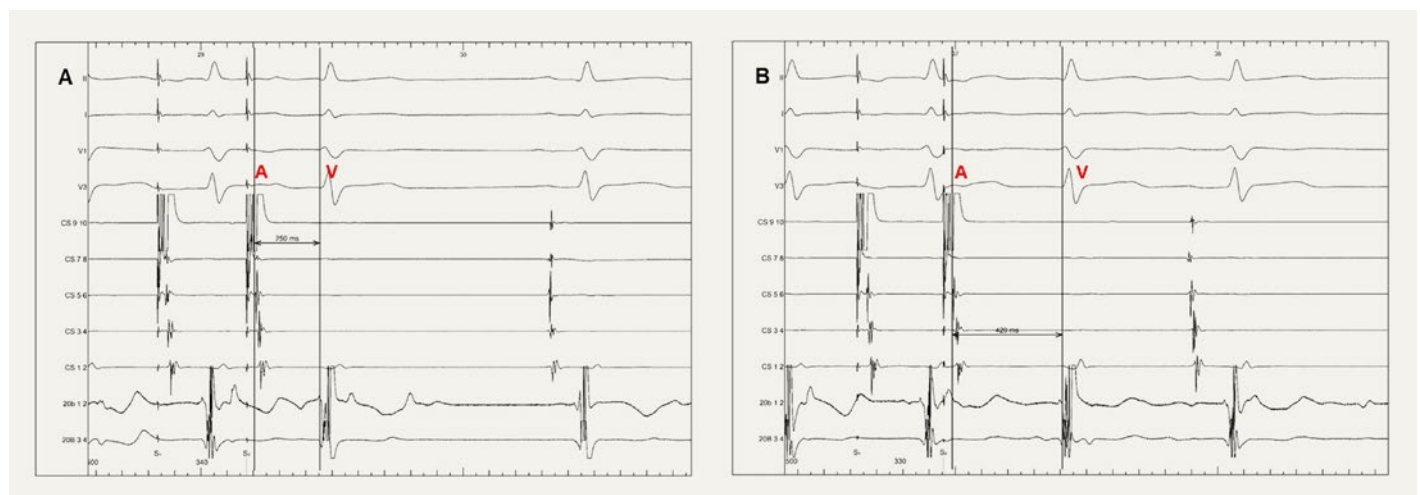
**Keywords:** electrophysiological study; catheter ablation; AV nodal reentry tachycardia

## Case

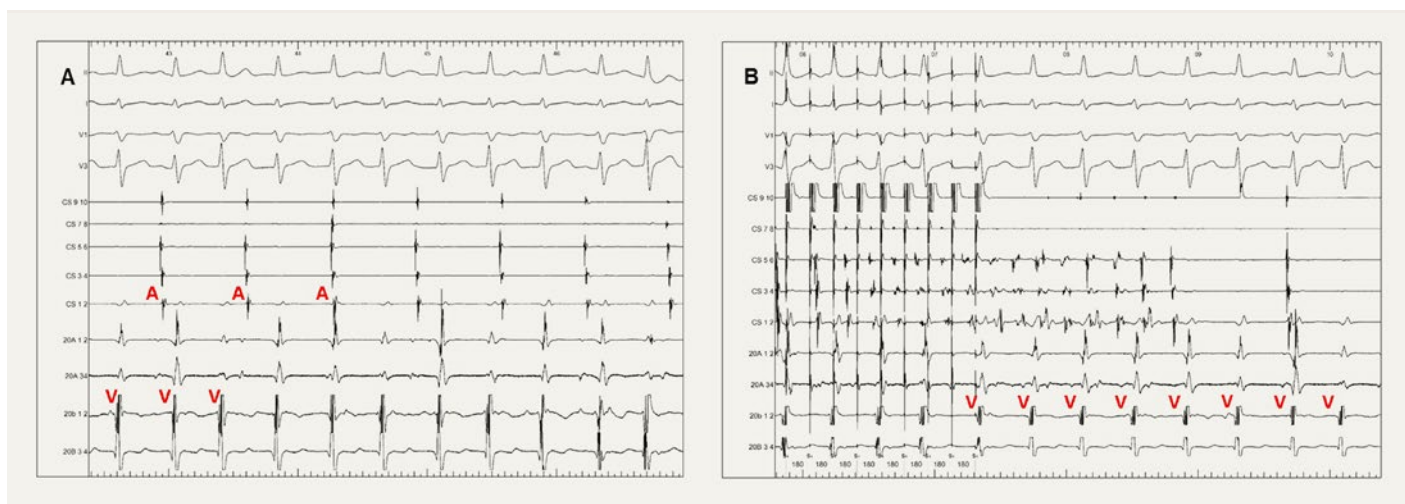
We present the case of a 34-year-old man who was referred to our hospital for an electrophysiologic study (EPS) due to recurrent palpitations. The 12-lead surface ECG had previously

confirmed a narrow complex tachycardia with a single QRS morphology at a heart rate of around 180 bpm with slightly varying R-R intervals. The QRS morphology during tachycardia was the same as in sinus rhythm and

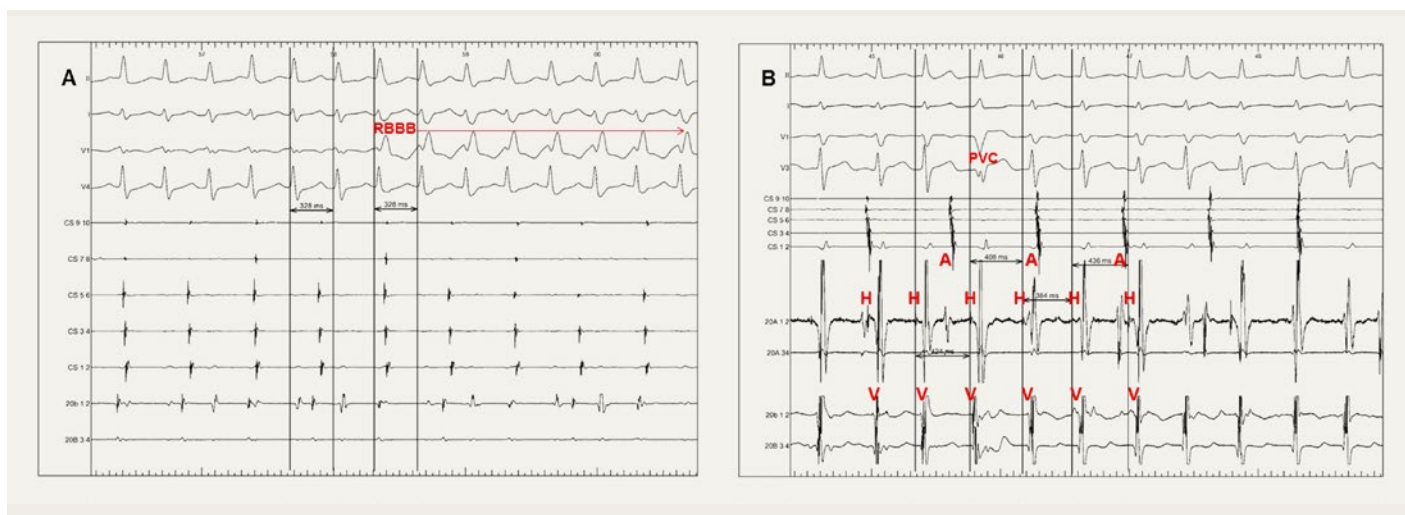
was therefore deemed to be of supraventricular origin. The supraventricular tachycardia terminated after administration of 18 mg adenosine IV. Transthoracic echocardiography excluded a structural heart disease; lab values were unremarkable. EPS showed normal baseline intracardiac intervals (AH interval 112 ms, HV interval 42 ms, AV block cycle length baseline 360 ms). Ventricular stimulation at a drive train cycle length of 600 ms showed ventriculoatrial (VA) dissociation. Physiology of both AV nodal was present (fig. 1, AV jump). A narrow complex tachycardia with slightly varying tachycardia cycle length (TCL) of



**Figure 1:** Programmed atrial stimulation with a drive train S1 of 500 ms and S2 of 340 ms (panel A) and S2 of 330 ms (panel B) showing an AV (AH) jump of 170 ms (420-250 ms). Atrial signals are indicated with a red A, ventricular signals with a red V. I, II, V1 and V3 indicate surface ECG. CS1-2 (distal) to CS9-10 (proximal): bipolar endocardial signals from a decapolar catheter placed in the coronary sinus. 20B 1-2 and 3-4: bipolar endocardial signals from a quadripolar catheter placed in the apex of the right ventricle.



**Figure 2:** Panel A shows ventriculoatrial dissociation during narrow complex tachycardia. Panel B shows a short episode of an atrial run with A-A intervals <200 ms (precursor of atrial fibrillation) during narrow complex tachycardia (dual tachycardia). Atrial signals are indicated with a red A, ventricular signals with a red V. I, II, V1 and V3 indicate surface ECG. CS1-2 (distal) to CS9-10 (proximal): bipolar endocardial signals from a decapolar catheter placed in the coronary sinus. 20A 1-2 and 3-4: bipolar endocardial signals from a quadripolar catheter placed near the His. 20B 1-2 and 3-4: bipolar endocardial signals from a quadripolar catheter placed in the apex of the right ventricle.



**Figure 3:** Panel A shows no change in varying tachycardia cycle length (TCL) during tachycardia with and without right bundle branch block aberration. Panel B shows narrow complex tachycardia with VA dissociation. Documentation of a premature ventricular complex during refractory His, but the response on the next His cannot be judged due to varying TCL (424 ms, 408 ms, 384 ms, 436 ms). Atrial signals are indicated with a red A, ventricular signals with a red V, His signals with a red H. I, II, V1, V3 and V4 indicate surface ECG. CS1-2 (distal) to CS9-10 (proximal): bipolar endocardial signals from a decapolar catheter placed in the coronary sinus. 20A 1-2 and 3-4: bipolar endocardial signals from a quadripolar catheter placed near the region of His. 20B 1-2 and 3-4: bipolar endocardial signals from a quadripolar catheter placed in the apex of the right ventricle.

around 380 ms, which corresponded to the clinical tachycardia, was induced during atrial burst stimulation without isoproterenol infusion. The tachycardia could be terminated by atrial pacing and immediate VA dissociation without retrograde conduction through the AV node (fig. 2A) was noted during tachycardia. The atrial rhythm during the tachycardia with VA dissociation was a sinus rhythm at a cycle length of around 600 ms. Atrial activation sequence and cycle length did not significantly vary during VA dissociation.

A short sequence of an atrial run with A-A intervals <200 ms was induced by atrial burst

stimulation and did not influence the ongoing narrow complex tachycardia (dual tachycardia, fig. 2B). During the clinical tachycardia, the QRS complex was the same as in sinus rhythm and intermittently showed right bundle branch block aberration. The HV interval during sinus rhythm was normal and no structural heart disease was detected. These observations made bundle branch reentrant ventricular tachycardia and fascicular ventricular tachycardia unlikely. The observations above and the fact that the His always preceded the V, made a ventricular tachycardia (VT) from the ventricular myocardium unlikely.

HV was normal during tachycardia and TCL during right bundle branch block aberration did not change (fig. 3A). Left bundle branch block aberration during tachycardia was never observed, therefore, atrioventricular reentrant tachycardia (AVRT) with VA dissociation using a nodofascicular/nodoventricular fibre as the antegrade limb or a nodoventricular fibre as the retrograde limb of the tachycardia circuit were highly unlikely. As shown in figure 3B, the response of a premature ventricular beat induced by the right ventricle catheter, which fell into His refractoriness, could not be reliably assessed due to varying TCL. Based

## Case Report

on our observations, the most likely diagnosis was an atrioventricular nodal reentrant tachycardia (AVNRT) with upper common pathway block, although prior to ablation we were not able to exclude a tachycardia originating from the His bundle and orthodromic AVRT using a nodofascicular fibre as the retrograde limb. Therefore, we performed ablation of the slow pathway in loco typico using a 7F 4 mm D-curve catheter in the CARTO3 system (both Biosense Webster, Diamond Bar, California, USA) with up to 35 Watts, reaching a temperature of 50°C. Junctional beats were noted during radiofrequency ablation. After ablation and a waiting period of 30 minutes, no more AH (AV) jumps were present and no tachycardia was inducible. This supported our working diagnosis of AVNRT with upper common pathway block. At three months follow-up, no more palpitations and tachycardia were reported by the patient, indicating a successful ablation.

## Discussion

Previous case reports have demonstrated VA block during AVNRT, supporting the independence of the tachycardia circuit from atrial tissue and thus indicating the existence of an upper common pathway [1–5]. Similarly, there are rare reports of the simultaneous presence of atrial fibrillation/atrial runs and AVNRT on the surface electrogram [6] and during EPS, which we also noted in our case [7, 8].

Several pacing manoeuvres can be conducted to rule out possible differential diagnoses for AVNRT with upper common pathway block, among which are junctional tachycardia, intrahisian reentry and nodofascicular/-ventricular tachycardia. Atrial pacing manoeuvres may help differentiate AVNRT from (focal) junctional tachycardia: 1) a premature atrial complex delivered during His refractoriness may influence AVNRT through early engagement of the slow pathway, but

should be unable to influence junctional tachycardia. 2) an earlier premature atrial complex (delivered before His activation) that advances the immediate His activation without terminating the tachycardia favours junctional tachycardia.

It is very difficult to differentiate AVNRT with upper common pathway block from orthodromic AVRT using a nodofascicular/-ventricular fibre as the retrograde limb, which can both be associated with VA dissociation. In this regard, a premature ventricular stimulus from the apex of the right ventricle delivered during His-bundle refractoriness can advance the next His or terminate the tachycardia, if a nodofascicular/-ventricular fibre is present and constitutes the retrograde limb of the tachycardia circuit [4, 5, 8]. This manoeuvre was not performed in our case since the TCL was constantly varying, hence the response of the next His was difficult to judge. Therefore, we cannot exclude the possibility of the presence of a nodofascicular fibre as the retrograde limb of the tachycardia circuit or as an innocent bystander pathway, of which the treatment of choice is also ablation of the slow pathway in most cases [9, 10]. Other rare differential diagnoses for AVNRT with upper common pathway block are orthodromic AVRT using a nodofascicular accessory pathway, junctional tachycardia and intrahisian reentry. However, ablation of the slow pathway led to non-inducibility of the tachycardia in our case.

Consequently, our case suggests AVNRT with VA dissociation due to upper common pathway block as the most likely diagnosis.

## Correspondence

PD Dr. med. Ardan M. Saguner  
Department of Cardiology  
University Heart Centre, University Hospital Zurich  
Rämistrasse 100  
CH-8091 Zürich  
Ardan.Saguner[at]jusz.ch

## Disclosure Statement

No financial support and no other potential conflict of interest relevant to this article was reported.

## References

- Wellens HJ, Wessdorp JC, Duren DR, Lie KI. Second degree block during reciprocal atrioventricular nodal tachycardia. *Circulation*. 1976;53(4):595–9.
- Bauernfeind RA, Wu D, Denes PO, Rosen KM. Retrograde block during dual pathway atrioventricular nodal reentrant paroxysmal tachycardia. *Am J Cardiol*. 1978;42(3):499–505.
- Portillo B, Mejias J, Leon-Portillo N, Zaman L, Myerburg RJ, Castellanos A. Entrainment of atrioventricular nodal reentrant tachycardias during overdrive pacing from high right atrium and coronary sinus. With special reference to atrioventricular dissociation and 2:1 retrograde block during tachycardias. *Am J Cardiol*. 1984;53(11):1570–6.
- Calo L, Lamberti F, Ciolli A, Santini M. Atrioventricular nodal reentrant tachycardia with ventriculoatrial block and unsuccessful ablation of the slow pathway. *Journal of cardiovascular electrophysiology*. 2002;13(7):705–8.
- Kose S, Amasyali B, Aytemir K, Kilic A, Iyisoy A, Celik T, et al. Ventriculoatrial block during atrioventricular nodal reentrant tachycardia suggesting existence of an upper common pathway. *International heart journal*. 2005;46(2):333–8.
- Brugada P, Wellens HJ. Electrophysiology, mechanisms, diagnosis, and treatment of paroxysmal recurrent atrioventricular nodal reentrant tachycardia. In: *Tachycardias*. Springer; 1984:131–57.
- Chen J, Josephson ME. Atrioventricular nodal tachycardia occurring during atrial fibrillation. *Journal of cardiovascular electrophysiology*. 2000;11(7):812–5.
- Saluja D, Beauregard L-A, Patel A, Coromilas J. The simultaneous presence of sustained atrial fibrillation and atrioventricular nodal reentrant tachycardia. *Heart Rhythm*. 2015;12(1):229–33.
- Han FT, Riles EM, Badhwar N, Scheinman MM. Clinical Features and Sites of Ablation for Patients With Incessant Supraventricular Tachycardia From Concealed Nodofascicular and Nodoventricular Tachycardias. *JACC: Clinical Electrophysiology*. 2017;3(13):1547–56.
- Nazer B, Walters TE, Dewland TA, Naniwadekar A, Koruth JS, Najeeb Osman M, et al. Variable Presentations and Ablation Sites for Manifest Nodoventricular/Nodofascicular Fibers. *Circulation Arrhythmia and electrophysiology*. 2019;12(9):e007337.

Anzeige



**BUCH-SHOP**  
EMH Schweizerischer Ärzteverlag

Wir freuen uns  
auf Ihren Besuch!

Lesen, was lesenswert ist!

- Umfangreiche Auswahl an Fachbüchern
- Spannende Kriminalromane
- Kinderbücher, Comics ... und mehr!

shop.emh.ch

