

Supraventricular tachycardia with cycle length variation and apparent VA dissociation: what is the mechanism?

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We report on a 33-year-old woman who underwent an electrophysiologic study because of a documented supraventricular tachycardia. The induced tachycardia presented with cycle length variations and an apparent ventriculo atrial (VA) dissociation on the surface ECG. Careful analysis of the activation sequence revealed the correct diagnosis of an atrioventricular nodal re-entrant tachycardia using two different slow pathways in an anterograde and retrograde directions.

A 33-year-old woman was referred for an electrophysiologic study because of a documented supraventricular tachycardia. The patient suffered from paroxysmal tachycardias with a typical on/off-phenomenon for 2 years. Structural heart disease had been ruled out.

Stimulation from the RV-apex showed retrograde conduction over the bundle of His without any evidence of an accessory pathway. Programmed atrial stimulation with one extra stimulus revealed a dual atrioventricular (AV) nodal physiology. Under isoprenaline, a supraventricular tachycardia was inducible. On the first glance, this tachycardia presented with VA dissociation on the surface ECG (*Figure 1*). Is that true? What is the correct diagnosis?



Figure 1 Shown are the corresponding intracardiac electrograms. The white arrows demonstrate the activation sequence (continuous arrows: fast pathway, dashed arrows: slow pathways). White, ECG; yellow, His catheter; green, CS catheter; purple, RV catheter; FP, fast pathway; SP 1, slow pathway 1; SP 2, slow pathway 2.

The correct diagnosis is an AV nodal re-entrant tachycardia (AVNRT) which uses two different slow pathways either in an anterograde or in a retrograde direction. This results in cycle length variations and an apparent VA dissociation. The arrows in *Figure 1* demonstrate the activation sequence (FP: fast pathway, SP: slow pathway). Starting with a retrograde FP conduction, the anterograde conduction passes through a slow pathway (SP 1) as in common AVNRT. The following retrograde conduction uses the second slow pathway (SP 2). This is followed by a dual response in the anterograde direction via the fast pathway and one of the slow pathways. With retrograde conduction through the fast pathway the sequence starts again.

The differential diagnosis of an AVNRT with AV Wenckebach block in an upper common pathway had been ruled out by atrial stimulation at the tachycardia cycle length.

After successful slow pathway modulation (4 mm tip, non-irrigated, 30 W, 43°C), the AVNRT was no longer inducible. The patient was discharged without any complaints the day after.

Conflict of interest: none declared.