Case Report

A Tachycardia with Varying QRS Morphology and RP Intervals: Differential Diagnosis and Therapy

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An interesting case of a tachycardia with varying QRS morphology and RP intervals is presented and the electrophysiological differential diagnosis is discussed.

Key words: **Long RP tachycardia, narrow-QRS tachycardia, wide-QRS tachycardia, entrainment.**

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In certain cases of supraventricular tachycardias with atypical characteristics, entrainment techniques are necessary to establish a diagnosis. This case demonstrates the inherent limitations of pacing manoeuvres in the differential diagnosis.

Case presentation

A 40-year-old lady was referred for evaluation of frequent episodes of paroxysmal tachycardia with both narrow- and wide-QRS morphology. The clinical examination was unremarkable, the 12-lead ECG normal, and stress echocardiography negative for the detection of ischemia or structural disease. Electrophysiological testing was undertaken and revealed normal atrioventricular (AV) conduction, without evidence of pre-excitation, and concentric retrograde atrial activation. Atrial pacing readily induced both narrow- and wide-QRS tachycardias with short or prolonged RP intervals, such as AH=204 ms, HA=144 ms, VA=98 ms for the narrow-QRS tachycardia with a cycle length (CL) of 348 ms, and AH=178 ms, HA=142 ms, VA=116 ms for the wide-QRS tachycardia with a CL of 320 ms (Figure 1). During atrial pacing at a cycle length of 340 ms the AH interval was 36 ms. Both tachycardias were interrupted by ventricular extrastimulation and an A-V response or by single ventricular extrastimuli that were not conducted to the atria. During tachycardia, significant spontaneous as well as ventricular extrastimulation-induced changes in ventriculo-atrial conduction times were noted, and at times there was transition from a short RP to prolonged RP tachycardia or *vice versa*. However, the retrograde atrial activation sequence remained the same (Figure 2). Ventricular pacing manoeuvres aimed at tachycardia entrainment were performed (Figure 3).

Discussion

The patterns of interruption of tachycardias by ventricular extrastimulation and the difference in AH between atrial pacing and tachycardia (>40 ms) ruled out an atrial tachycardia.[1] The short RP tachycardias (both narrow- and wide-complex) most probably represented slow-fast AV nodal re-entry tachycardia (AVNRT). They were always induced by atrial pacing following a conduction jump, VA was always <50 ms on the His bundle electrogram, the tachycardias were not reset by His-synchronous ventricular extrastimuli, and the difference between VA intervals during apical ventricular pacing and tachycardia was always >90 ms.[2] Considering

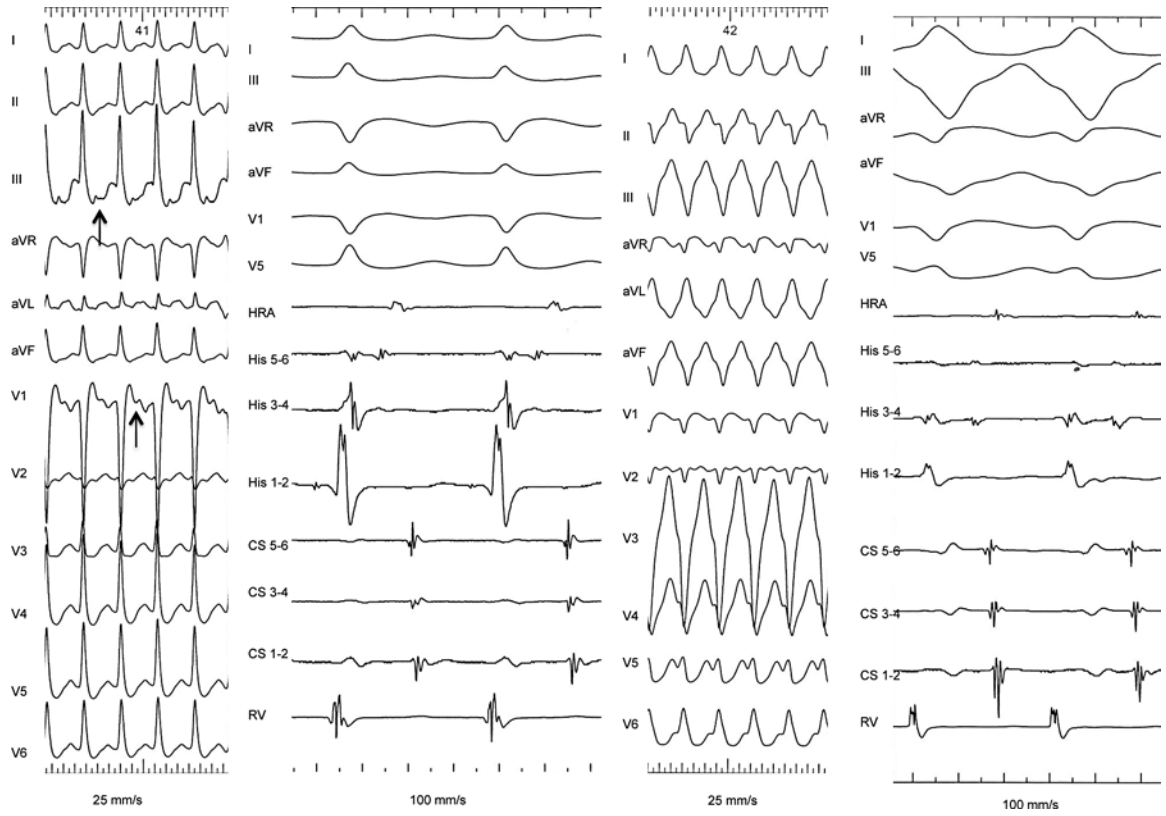

Figure 1. Surface ECGs and electrograms during narrow- and wide-QRS tachycardias. Arrows indicate presumed retrograde P waves. I-V6 – ECG leads; His – His bundle electrogram; CS – coronary sinus; RV – right ventricle.

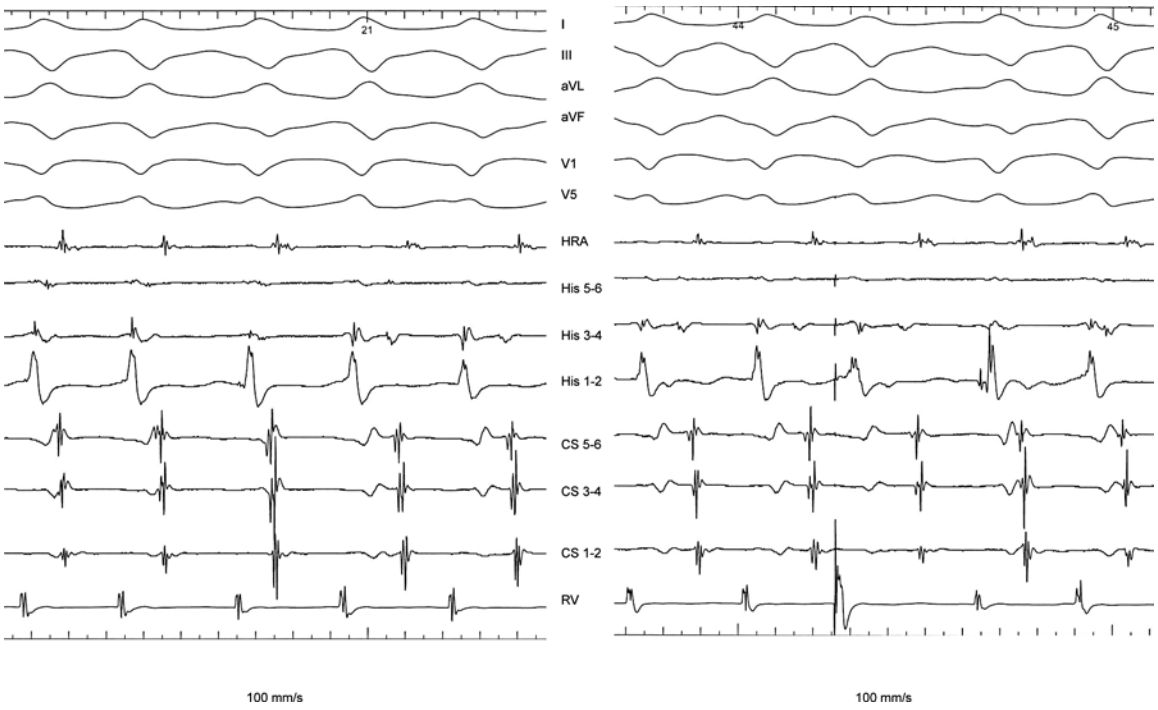

Figure 2. VA interval changes without retrograde activation sequence, spontaneously (left panel) or following ventricular extrastimulation (right panel). Abbreviations as in Figure 1.

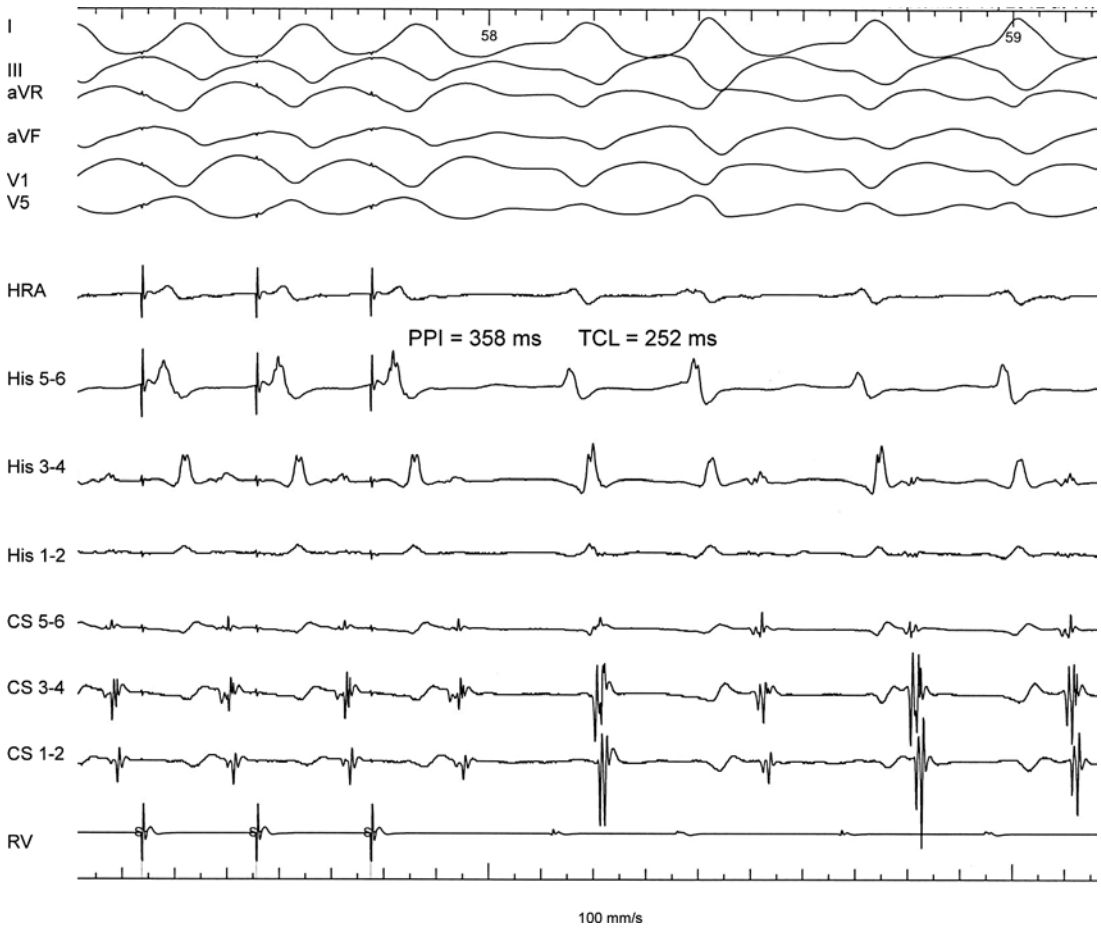

Figure 3. Entrainment of the tachycardia with a pre-stimulation cycle length (TCL) of 252 ms. The post-pacing interval (PPI) is 108 ms and, after correction according to pacing-induced atrioventricular incremental conduction (post-pacing AH - basic AH), becomes 97 ms. Abbreviations as in Figure 1.

the prolonged RP tachycardias, the pattern of AH/HA>1 and VA>60 ms suggests either a slow-slow AVNRT or a concealed mid-septal accessory pathway. Examination of the narrow-QRS tachycardia reveals prominent retrograde P waves in leads III and V_1 (Figure 1, arrows) with a difference between the RP intervals in lead III and lead V_1 of 64 ms (208 ms - 144 ms). This is a rather crude index, but is suggestive of an atypical AVNRT.[3] The difference in AH during atrial pacing and tachycardia at similar cycle lengths also indicates AVNRT, which requires a ΔAH >40 ms, rather than a septal pathway, in which case the ΔAH is <20 ms.[1] Entrainment of the tachycardia with atrial pacing was attempted, but a clear transition zone could not be reliably established.[4] Entrainment with ventricular pacing was most probably achieved,[5] and always resulted in alternating VA intervals (Figure 3). The measured post-pacing interval tachycardia cycle length (PPI-TCL) was 108 ms, and when corrected for the pacing-induced incremental AV nodal conduction[6] it became 97 ms. In other entrainment attempts these numbers were even smaller. These values are in favour of AVRT due to a septal accessory pathway, rather than atypical AVNRT, and this manoeuvre has a higher diagnostic accuracy if the intervals suggest AV re-entry, rather than AV node reentry.[7] It should also be remembered that AVNRT (at least the slow-fast type) becomes entrained with much more difficulty than AVRT, and in the past this used to be a differentiation criterion. Differential ventricular pacing during sinus rhythm from the RV apex and the posterobasal area at varying cycles was also performed, to derive the so-called ventriculo-atrial index as described by Martinez-Alday et al.[8] Results were inconclusive, with values ranging between 6 and 10 ms. Differential ventricular pacing during tachycardia from the RV apex and base with termination of tachycardia was also accomplished.

The difference in VA times was 18 ms, again a rather inconclusive number, since >20 ms is required for diagnosis of AVNRT.[8] However, this index, although better reflecting the tachycardia circuit, may not be applicable in true septal pathways, since in the study of Segal and colleagues[9] it was mainly derived by comparing AVNRT with lateral pathways. Attempts to derive the atrial pre-excitation index[10] with ventricular extrastimuli did not yield reproducible results. A retrograde His could not be recorded during tachycardia or pacing manoeuvres in order to allow assessment of HA times or parahisian entrainment.[11,12]

The absence of any evidence of pre-excitation with atrial pacing makes the diagnosis of antidromic AVRT unlikely. Thus, the left bundle branch block (LBBB) tachycardia most probably represents either AVNRT or orthodromic AVRT with aberration. Antidromic AVRT or AVNRT with a bystanding atriofascicular Mahaim pathway is also possible, but no evidence was produced during atrial pacing in sinus rhythm or atrial extrastimulation during tachycardia.[13] Unfortunately no spontaneous transition from narrow- to wide-complex tachycardia was noted; that would have allowed the examination of HH intervals under the same conditions, considering the possibility of a macro–re-entrant loop. In addition, the lack of spontaneous transition to LBBB pattern does not allow definitive conclusions regarding changes in VA time. Should this be the case, the occurrence of a longer VA time with the development of LBBB (18 ms in our study) would be indicative of an operating concealed septal pathway.[14] Thus, it is obvious that pacing manoeuvres would not allow an unequivocal diagnosis in this case. A clue for diagnosis may be provided by Figure 2. Spontaneous as well as ventricular extrastimulation-induced changes in the retrograde VA time, without disturbing the atrial activation sequence and relative retrograde conduction times, are in favour of AVNRT that changes from typical to atypical patterns. The coexistence of various types of AVNRT in the same patient has been previously described by several groups, including ours.[15]

An ablation electrode was advanced to the anatomical site of the slow pathway and two RF pulses were delivered with induction of a transient, slow nodal rhythm. Following that, no tachycardia could be induced despite persistent isoprenaline challenges; antegrade and retrograde conduction were also entirely normal without evidence of atriofascicular conduction. One month later, the lady remains free of arrhythmia without any medication. Thus, AVNRT alternating between the slow-fast and slow-slow circuits, with and without aberrant conduction to the ventricles, is the most plausible diagnosis—although, theoretically at least, the possibility of inadvertent damage to a septal pathway that courses close to the vicinity of the slow pathway cannot be ruled out.

This case demonstrates the inherent limitations of pacing manoeuvres in the differential diagnosis of supraventricular tachycardias. In certain cases of tachycardias with atypical characteristics, multiple criteria and pacing techniques have to be employed in order to reach a diagnosis.

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