Responses to Ventricular Overdrive Pacing during Wide QRS Tachycardia: What is the Mechanism?

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A 35-year-old female presenting with recurrent palpitation was referred for electrophysiology study. Short episodes of narrow QRS tachycardia could be recorded on ECG (Figure 1). During electrophysiology study, the tachycardia could be readily induced with atrial pacing or programmed atrial extrastimuli, but usually terminated in seconds, making it difficult to perform diagnostic maneuvers. When it spontaneously changed to a wide QRS tachycardia, a burst of ventricular overdrive pacing (VOD) was delivered which turned it back to the narrow complex one (Figure 2). What can be learned from the response?

The first 4 beats in this tracing showed wide complex tachycardia (WCT) with right bundle branch block (RBBB) morphology and 1:1 ventriculo-atrial ratio. Candidates for diagnosis could be ventricular tachycardia, supraventricular tachycardia (SVT) with RBBB, bundle branch reentrant tachycardia and preexcited tachycardia. An H-V interval of 53ms approximating that during sinus rhythm excluded ventricular tachycardia from myocardium and preexcited tachycardia. The last 4 beats were clinical narrow complex tachycardia with the same cycle length, H-V interval and atrial activation sequence (earliest A at CS7-8) as the WCT, highly indicating that the WCT was SVT with functional RBBB caused by continuous concealed activation from left bundle branch before VOD peeled back the refractoriness of the right bundle branch in the following beats. In addition, A right-sided accessory pathway (AP) was unlikely given the same H-A interval (127ms, measured to CS7-8) with and without RBBB [1].

The middle 4 beats demonstrated progressive change in QRS, from fusion to probable fully-paced morphology during VOD. The 3rd beat advanced the subsequent A without atrial activation change, indicating the presence of a septal AP. However, post-pacing interval (PPI) was 125ms over tachycardia cycle length (TCL) with V-A-H-V response, which argued against AV reentrant tachycardia (AVRT) utilizing a septal AP. Note that His signal was found after the 3rd and the 4th stimulating artifact. Considering the extremely short interval between the 3rd artifact and the subsequent His (17ms), it could hardly be a retrograde His, but was activated in an orthodromic direction instead. A progressively increased A-H interval after the 2nd, 3rd

and 4thpacing suggested decremental conduction in AV node before orthodromic His capture. When atrium was entrained, anterograde conduction to the His bundle during VOD supported the diagnosis of AVRT and excluded AV nodal reentry ^[2]. The long PPI was associated with the delay of the first return His-V following VOD-induced decremental AV nodal conduction. The corrected PPI-TCL was 78ms after subtracting the difference between the first return A-H and tachycardia A-H from PPI ^[3]. The AP was successfully ablated at left posterior septum, which rendered the tachycardia non-inducible.

Conflicts of interest

The authors declare no conflicts of interest.

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Figure legends

Figure 1: 12-lead ECG showed a short episode of narrow QRS tachycardia.

Figure 2: VOD during WCT with a cycle length of 286ms and H-V interval of 53ms. Earliest atrial activation was at CS7-8 with H-A interval of 127ms. VOD caused orthodromic His activation with decremental conduction in AV node. The prolongation of A-H contributed to the relative long PPI following the cessation of VOD. After which it turned into a narrow QRS tachycardia with the cycle length, H-V, H-A and atrial activation sequence identical to the WCT. See text for discussion. CS=coronary sinus; VOD=ventricular overdrive pacing; WCT=wide complex tachycardia.



