Published in final edited form as:

Heart Rhythm. 2009 May ; 6(5): 716–717. doi:10.1016/j.hrthm.2008.06.006.

A DIAGNOSTIC MANEUVER DURING NARROW-COMPLEX TACHYCARDIA: WHAT IS THE ARRHYTHMIA MECHANISM?

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A 14-year-old male with a history of Wolff-Parkinson-White syndrome and recurrent palpitations despite medical therapy was referred for electrophysiology (EP) study and possible ablation. He had undergone two previous unsuccessful ablation attempts. Ambulatory monitoring revealed self-terminated episodes of narrow-complex tachycardia at 160–180 beats per minute (BPM). Echocardiography showed normal ventricular function and no evidence of structural heart disease.

At EP study, the patient was found to be in sinus rhythm at a cycle length of 813 milliseconds (ms) with atrial-His (AH) and His-ventricular (HV) intervals of 80 ms and zero, respectively. A duodecapolar catheter was placed in the right atrium and mapping/ablation catheters were placed both in the epicardial space and endocardially in the right atrium. Ventricular pacing showed retrograde conduction with eccentric atrial activation. A narrow-complex tachycardia with cycle length 380 ms (158 BPM) was induced by ventricular pacing. Overdrive pacing from the ventricle during tachycardia repeatedly resulted in a VAV response on cessation of pacing. During tachycardia, a single His-refractory ventricular extrastimulus was delivered through the right ventricular catheter (Figure 1). Catheter positions are shown in Figure 2. What is the arrhythmia mechanism?

Commentary

The 12-lead ECG (in Figure 1) shows delta waves that are negative in lead II and positive in the precordial leads with an early R/S transition, suggesting a posteroseptal accessory pathway (AP)¹. Intracardiac recordings during tachycardia show advancement of atrial activation by a His-refractory ventricular extrastimulus, ruling out atrioventricular nodal reentrant tachycardia. Atrial activation sequence is consistent with an orthodromic atrioventricular reentrant tachycardia (AVRT) using the AV node as the antegrade limb and a right-sided atrioventricular accessory pathway as the retrograde limb. However, close examination of the atrial activation sequence (Figure 1) before the extrastimulus reveals earliest retrograde activation in D 9,10, which is on the lateral tricuspid annulus, near 9:00 in the left anterior oblique (LAO) projection (Figure 2). The coronary sinus ostium is activated much later than the right atrial annular sites. This would not be expected from a posteroseptal pathway, as was suggested by the pattern of antegrade preexcitation. A mismatch between the origin of ventricular preexcitation and the site of atrial activation during ventricular pacing or

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orthodromic AVRT raises the possibility of multiple APs² or an oblique course of the AP³.⁴ In this case, atrial activation during tachycardia 1 was probably the result of concealed conduction resulting in a block of the non-participating accessory pathway or even fusion of retrograde activation (via two accessory pathways). The premature extrastimulus was delivered with a coupling interval shorter than the refractory period of the right lateral AP. This resulted in AP conduction block and a switch to retrograde atrial activation via a second AP with continued orthodromic AVRT, as illustrated in Figure 3. After the extrastimulus, earliest atrial activation was seen in D 5,6, near 7:00 on the tricuspid annulus. Conduction block in the first accessory pathway during tachycardia 2 was likely maintained by concealed antegrade penetration. Both pathways were targeted and ablated endocardially utilizing epicardial mapping to guide endocardial catheter location⁵.

After 3 months of follow-up, the patient remains free of tachycardia and has no preexcitation on 12-lead electrocardiogram.

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Figure 1.

A: Twelve-lead electrocardiogram prior to ablation, showing preexcitation pattern consistent with a manifest posteroseptal atrioventricular accessory pathway.

B: Intracardiac tracings during supraventricular tachycardia induced by atrial pacing. Note change in atrial activation sequence induced by ventricular extrastimulus, with earliest activation shifting from D9,10 to D5,6. (ABL, endocardial ablation catheter; ABL Epi, epicardial ablation catheter; HRA, high right atrial catheter; HIS, His bundle catheter; D1-D20, duodecapolar right atrial catheter; CS1-CS10, coronary sinus catheter)

Figure 2.

Left anterior oblique and right anterior oblique fluoroscopic views of catheters. Abbreviations are as in Figure 1. (ABL, endocardial ablation catheter; ABL Epi, epicardial ablation catheter; HRA, high right atrial catheter; HIS, His bundle catheter; D1-D20, duodecapolar right atrial catheter; CS1-CS10, coronary sinus catheter)

Figure 3.

Schematic representation of proposed mechanism of change in atrial activation sequence by His-refractory premature ventricular complex (PVC). During tachycardia #1, the pattern of retrograde atrial activation is likely due to concealed conduction causing functional block of the non-participating accessory pathway. The PVC coupling interval is shorter than the refractory period of the participating accessory pathway, causing pathway block. This results in relief of block in pathway #2, allowing atrial activation by this pathway and switch to tachycardia #2. Continued block in accessory pathway #1 is likely mediated by concealed conduction causing functional block.