Role of His Refractory Premature Ventricular Complexes in the Differential Diagnosis of a Left Bundle Branch Block Morphology Tachycardia

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A 65-year-old man, with no significant past medical history, presented with an episode of sudden onset palpitations and lightheadedness. He was hemodynamically stable and the ECG revealed a left bundle branch block (LBBB) morphology tachycardia at 214 beats per minute (Figure 1A). The tachycardia terminated spontaneously with conversion to normal sinus rhythm and LBBB (Figure 1B). Further evaluation included a 2-dimensional echocardiogram showing a left ventricular ejection fraction of 40% to 45%, left heart catheterization showing angiographically normal coronaries, and cardiac magnetic resonance imaging revealing atypical septal motion with left ventricular ejection fraction of 60% to 65% and no scar or ischemia. The patient underwent an electrophysiology study at baseline, the sinus cycle length was 890 ms, atrial-His interval was 64 ms, His-ventricular interval was 108 ms, and QRS duration was 120 ms. Spontaneous onset of the LBBB morphology tachycardia after a sinus beat is shown in Figure 2. The tachycardia cycle length is 306 ms, and the His-ventricular interval is 120 ms. The QRS while similar to the LBBB seen during sinus rhythm shows change in axis and the morphology in aVR is predominantly positive. There is no ventriculo-atrial conduction during the tachycardia. Attempts at entrainment from right ventricle (RV) led to termination of the tachycardia. His refractory premature ventricular complexes (PVCs) delivered from RV apex during tachycardia after the His depolarization advances the next H and V (Figure 3). What is the mechanism of the tachycardia?

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Discussion

A wide QRS complex tachycardia with LBBB morphology similar to that during sinus rhythm raises the possibility of supraventricular tachycardia (SVT) with LBBB and ventricular tachycardia (VT). Ventriculo-atrial dissociation and sinus atrial rhythm during the tachycardia rules out atrioventricular (AV) accessory pathway–mediated tachycardia and atrial tachycardia. SVT mechanisms that do not require atrial participation for the perpetuation of tachycardia remain in the differential: (1) AV nodal reentrant tachycardia (AVNRT) with a ventriculo-atrial block, (2) non-reentrant junctional tachycardia, and (3) orthodromic tachycardia using a nodofascicular or nodoventricular accessory pathway. Antidromic pathway–mediated tachycardia using nodofascicular or nodoventricular pathways is excluded from the prolonged His-ventricular interval during tachycardia. VT mechanisms that may present with LBBB morphology similar to that in sinus rhythm include bundle branch reentrant tachycardia (BBRT) and focal VT originating from the His-Purkinje system (HPS) or RV myocardium near the right bundle branch exit. Myocardial VT is excluded by H-H interval changes that preceded and predicted V-V interval changes and termination of tachycardia occurring repeatedly with V-H block (Figure 4). The remaining SVT mechanisms (1) to (3), as well as BBRT and focal HPS VT, can have a His bundle depolarization preceding QRS complex during tachycardia and changes in H-H interval driving changes in V-V interval. Entrainment from RV apex showing post-pacing interval equal to tachycardia cycle length could have helped to rule out AVNRT, but tachycardia terminated consistently with ventricular pacing.

Mode of Tachycardia Initiation

In Figure 2, the tachycardia initiates after a normal sinus beat with the usual LBBB and His-ventricular interval. All 3 remaining SVT possibilities may do this with a 2 for 1 response in the case of AVNRT, spontaneous initiation of the focal rhythm in case of junctional tachycardia, and reentry into nodoventricular or nodofascicular tracts. Among the 2 VT possibilities, His bundle depolarization preceding the first tachycardia beat suggests trans-septal reentry into the HPS (BBRT) after the sinus beat (Figure 5) or a focal VT from HPS. Although it would be unusual for BBRT to start this way, it cannot be excluded. A myocardial VT should not start with His activation and is again excluded. Incremental atrial pacing and ventricular pacing also induced the tachycardia but did not help the differential.

Interpreting His Refractory PVC

In general, the ability of a His refractory PVC to reset an SVT is diagnostic of the presence of an accessory pathway.
This is because the His bundle is already depolarized and cannot be reactivated retrogradely by the PVC. Furthermore, a His refractory PVC resetting an SVT without atrial activation excludes AV nodal rhythms unless there is a nodo-fascicular/nodoventricular pathway. But can a His refractory PVC reset an AV nodal rhythm in the absence of an accessory pathway?

**Exception to the Rule**

In the setting of LBBB, it is possible that a His refractory PVC from RV apex conducts trans-septally and lead to retrograde activation of the His bundle (Figure 6). This can result in subsequent resetting of BBRT (Figure 6A), nodoventricular reentry (Figure 6B), or AVNRT (Figure 6C). In the case of AVNRT, this is not a differential that physicians generally consider.
His Refractory PVCs during electrophysiology studies, and resetting of the rhythm by His refractory PVCs in the absence of an accessory pathway has not been described in the literature. Here, one would expect a V-H-H-V response with the His refractory PVC (unless there is simultaneous bundle branch reentry beat). As diagrammatically represented in Figure 6C, the retrograde activation of the His has to reach the AV node, reset the rhythm, and the next tachycardia beat would cause anterograde His and ventricular activation. The V-H-V resetting by PVC seen in this case (Figure 3) argues against AVNRT. Further, a late PVC results in termination of the tachycardia with retrograde block in the HPS (Figure 4). This rules out AVNRT and junctional tachycardia because retrograde conduction into His bundle is essential to reach the AV node and perturb any AV nodal rhythms.

The only possibilities remaining are BBRT and orthodromic reentry via nodoventricular or nodofascicular pathway. Differentiating these 2 tachycardia mechanisms is difficult because PVC and entrainment responses would be similar (Figure 6A and 6B). Analysis of His to right bundle branch interval (H-RBB) activation sequence may help in some cases. A shorter H-RBB time during tachycardia compared with sinus rhythm would indicate BBRT. This is because the

Figure 3. Premature ventricular complexes delivered after the His depolarization, advances the next H and V. The H-H and V-V intervals are marked. HBED indicates His bundle distal; HBEP, His bundle proximal; HRA, high right atrium; and RV, right ventricular apex.

Figure 4. Termination of tachycardia with a spontaneous premature ventricular complexes occurring after His activation. Note the termination occurring without terminal His activation. CS 9 to 10 through CS 1 to 2 indicates coronary sinus electrodes; HBED, His bundle distal; HBEP, His bundle proximal; HRA, high right atrium; and RV, right ventricular apex.
His is activated in a retrograde manner by the LBB during tachycardia, and by the time a His deflection is recorded, the reentry circuit has already turned around to start anterograde conduction of the RBB. However, if the H-RBB interval is unchanged or prolonged compared with sinus rhythm, it does not help the differential. The involvement of the RBB was confirmed during the transient catheter trauma-induced complete heart block and failure to induce the tachycardia during this period of time. A final presumptive diagnosis of BBRT was made, and a nodoventricular/nodofascicular reentry could not be definitively excluded. The patient underwent implantation of a dual chamber pacemaker and subsequent right bundle branch ablation. After ablation, the patient remained in complete heart block, and no arrhythmias were inducible.

This case illustrates the usefulness of PVC maneuver to differentiate AV nodal tachycardias from BBRT. The electrophysiology purist may consider the possibility of trans-septal conduction and retrograde His activation when interpreting His refractory PVCs in patients with LBBB.
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