# Sudden QRS widening while ablating a concealed left accessory pathway during left ventricular pacing

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**Case Presentation**

A 26-year-old man presented to emergency department with acute onset palpitations with a history of such episodes since two months. The electrocardiogram revealed a regular short RP narrow QRS tachycardia at the rate of 145 beats per minute. The echocardiogram showed a left ventricular ejection fraction of 0.25 during tachycardia but was normal during sinus rhythm. He underwent an electrophysiology study which revealed eccentric activation in the coronary sinus during right ventricular pacing and during tachycardia **(Figure 1)** , suggestive of a retrograde conducting concealed left free wall accessory pathway (AP). The

mitral annulus was mapped via the transseptal route and the AP was localized to the left anterolateral mitral annulus. Radiofrequency energy was delivered during entrainment of the orthodromic tachycardia by left ventricular (LV) lateral wall pacing**(Figure 2 left panel)** . A sudden change in QRS morphology was seen during energy **(Figure 2 right panel)** , coincident with elimination of accessory pathway conduction **(Figure 3 left panel)** . What is the mechanism?

**Commentary**

The intracardiac electrograms (EGMs) during delivery of radiofrequency energy reveal initially a RBBB-like morphology in lead V1, suggesting ventricular pacing from the left ventricle. The QRS duration is 130 ms, which is unusually short for left ventricular lateral wall pacing. Fused ventricular and atrial EGMs can be appreciated in the bipolar signal from the tip of the ablation catheter (RFD, **Figure 3 left panel** ). Retrograde AP conduction is seen until the fifth QRS complex, followed by ventriculo-atrial (VA) block; later there is variable VA conduction via the AV node. Immediately after the VA block, there is an abrupt vivid change in the QRS morphology with an increase in the QRS width to 187ms.

In trying to understand the rationale behind the change in QRS morphology, it is important to consider yet another important observation in the intracardiac electrograms. The clue lies in the EGMs on the His channel (HISD, **Figure 3 left panel** ). Clear sharp His signals (H) are seen to precede the ventricular EGMs during LV pacing until the retrograde VA block and change in QRS morphology. This suggests antegrade activation of the His bundle, which can happen only if LV pacing was being performed concomitant with the orthodromic atrioventricular reentrant tachycardia. The relatively narrow QRS complex is the result of fusion of ventricular activation from LV pacing and the tachycardia wavefronts. This is further suggested by the short HV interval of 20 ms **(Figure 3 right panel)** . This was actually the case here, as the LV was being overdrive paced at 30 ms shorter than the tachycardia cycle length, so as to maintain stability of the ablation catheter during ablation, in case of termination of tachycardia.

For a fusion to perpetuate between the orthodromic AVRT and ventricular pacing, the following conditions are necessary: pacing close to the insertion of the AP, pacing just shorter than the TCL, rapid AP conduction, brisk antegrade AV nodal conduction and sluggish retrograde AV nodal conduction. Basal pacing site near the pathway insertion leads to more ventricular muscle activation by the orthodromic tachycardia wavefront (1). Fusion can be identified by the presence of an orthodromically captured His or right bundle potential.

In conclusion, a relatively narrow QRS during LV lateral wall pacing along with evidence of antegrade activation of His bundle suggests fusion between ventricular pacing and the ongoing orthodromic tachycardia; here one would expect the QRS to change in morphology as soon as the tachycardia terminates during the energy delivery. While one concentrates on the atrial activation while ablating concealed accessory pathways during ventricular pacing, the QRS complex can display valuable evidence too!

**References:**

Veenhuyzen GD, Coverett K, Quinn FR, Sapp JL, Gillis AM, Sheldon R, Exner DV, Mitchell LB. Single diagnostic pacing maneuver for supraventricular tachycardia. Heart Rhythm. 2008 Aug;5(8):1152-8.

**Figure Legends:**

**Figure 1.** Surface electrocardiogram (I, aVF, V1, V6) and intracardiac electrograms - His bundle electrogram distal and proximal (HISD, HISP), coronary sinus (CS) 9,10 dipoles at CS ostium, & CS 1,2 dipoles at distal CS. Pacing is being done from HBD and is followed by tachycardia; both show identical eccentric atrial activation patterns (marked by arrows) with earliest activation at CS 3,4 channel.

**Figure 2. Left panel.** Fluoroscopic image showing location of left ventricle pacing site and ablation catheter positioned at the mitral annulus transeptally in RAO 30 (left upper panel) and LAO 40 (left lower panel). **Right panel.** 12-lead surface electrocardiogram at the initiation of radiofrequency energy delivery depicting sudden change in QRS morphology after the $6^{th}$ beat.

**Figure 3. Left panel.** Surface electrocardiogram (I, aVF, V1, V6) and intracardiac electrograms recorded from catheters as depicted in figure 2 left panel. The arrow after the 2$^{nd}$ beat denotes the artefact at the initiation of radiofrequency energy delivery. The orthodromically captured His bundle potentials on the HISD channel and the ventricular/atrial potentials on the RFD channel are marked with H, V and A respectively. Note the sudden change in QRS morphology and width with loss of ventriculo-atrial conduction after the 6$^{th}$ beat. **Right panel.** The shaded beat on the left panel at a higher speed of 300 mm/sec to demonstrate a short HV interval of 20 ms suggesting fusion between wavefronts originating from the ongoing othrodromic re-entrant tachycardia and the LV pacing.