Transition of a narrow to a wide QRS tachycardia: What is the mechanism?

A 48-year-old man underwent an electrophysiological catheter study following a history of worsening palpitations associated with pre-syncope. He had a structurally normal heart on echocardiography and no other significant past medical history. Figure 1A depicts his 12 lead surface ECG at baseline. A standard 4 wire catheter study was performed with a decapolar catheter situated more proximally than convention in the coronary sinus (electrodes 7 and 8 at the ostium). Quadrupolar catheters were situated at the high right atrium, right ventricular apex and bundle of His. A narrow complex tachycardia (VA time 115 ms) was easily induced which transitioned to a broad complex tachycardia (Figure 1B). Subsequent atrial overdrive pacing resulted in entrainment of the ventricle and an A-V-A response. Ventricular overdrive pacing resulted in constant manifest fusion and then tachycardia termination.
Figure 1A. 12-Lead surface ECG at baseline.
Figure 1B. Intracardiac electrograms. CS 7,8 is located at the CS ostium. There is narrow complex tachycardia (α), which transitions to a broad complex tachycardia (γ) following a His refractory ventricular premature activation (β). Following this, atrial activation is advanced and maintains the same atrial activation sequence. Note that His activation occurs at the onset of the QRS. There is a prolongation in AV interval (182ms to 210ms), with a LBBB morphology in lead V1 (asterisk) and a shortened HV interval.
What is the likely mechanism for the findings?

**Answer options**

A. Orthodromic atrioventricular tachycardia with development of left bundle branch block
B. Atrial tachycardia with aberrance or conduction via bystander pathway
C. Duodromic tachycardia
D. Atypical atrioventricular nodal re-entrant tachycardia with aberrance or conduction via bystander pathway
E. Orthodromic tachycardia transitioning to ventricular tachycardia
ANSWER TO MARCH 18th QUESTION

C. Duodromic tachycardia

Explanation
The patient’s 12 lead surface ECG shows manifest pre-excitation suggestive of a posteroseptal tricuspid annulus / coronary sinus os pathway.¹ The differential diagnosis for a narrow complex tachycardia with a VA interval >60 ms (α in Figure 1) includes orthodromic atrioventricular re-entrant tachycardia (AVRT), atrial tachycardia (AT) and atypical atrioventricular nodal re-entry tachycardia (AVNRT). There is a His refractory ventricular premature beat (β in Figure 1) which represents either a ventricular ectopic or spontaneous conduction down an accessory pathway. This results in advancement of the next atrial beat, confirming the presence of an accessory pathway (but not obligatory participation).

Now considering the broad complex tachycardia (ϒ in Figure 1), the differential diagnosis is: AT/AVNRT with aberrance or with conduction via a bystander accessory pathway; AVRT with ventricular activation via a pathway; or ventricular tachycardia (VT). The aforementioned response to atrial entrainment rules out VT and the constant manifest fusion during ventricular pacing implies ventricular participation in a re-entry circuit making an AT or AVNRT an unlikely mechanism. Accordingly, we are left with an orthodromic AVRT (seen initially in α), which transitions to a broad complex tachycardia (ϒ) following a His refractory ventricular premature beat (β). Following this premature ventricular beat, atrial activation is advanced and maintains the same atrial activation sequence indicating a reset of the tachycardia from the ventricle, and since the His must be refractory during this period, retrograde conduction must be via an accessory pathway. On the subsequent ventricular beat (ϒ), the His signal on the distal pole occurs at the onset of the QRS (Figure 1, Figure 2) implying that His bundle conduction alone is responsible neither for AV conduction nor for VA conduction during tachycardia.

Therefore, the broad complex tachycardia is duodromic AVRT utilizing accessory pathways for both anterograde (AV) and retrograde (VA) conduction (Figure 2).
Explanation continued
The premature activation (β) results in a shortening of the AA interval and this is associated with prolongation of the AH time. We hypothesize that this caused peeling of refractoriness to facilitate conduction via a second accessory pathway, responsible for QRS morphology seen from γ onwards.2 If one examines the AV intervals at β and γ (Figure 1), there appears to be decremental conduction antegradely. The more prominent LBBB morphology pattern in lead V1 (asterisk in Figure 1) and shortened HV interval would suggest the presence of an atrio-fascicular or Mahaim pathway in addition to an atrio-ventricular right sided accessory pathway.

This patient had two right sided accessory pathways as well as a Mahaim pathway. During sinus rhythm, a D-curve mapping catheter was employed to map the tricuspid annulus and identify the site of earliest ventricular activation at the right septal wall. RF ablation at this point (site 1), resulted in a change of pre-excitation morphology after 4 seconds of RF energy (Figure 3). However, a degree of pre-excitation was still present which became more evident on pacing in the right atrium. Further mapping identified the second accessory pathway located posteriorly on the tricuspid annulus (site 2). Ablation at this site resulted in further loss of pre-excitation after 7 seconds of applied RF energy (Figure 3). An antegrade curve following ablation was performed which revealed pre-excitation with a LBBB morphology (similar to γ), decremental conduction and a shortened HV interval, consistent with a Mahaim pathway.3 Ablation was performed at the site of a Mahaim potential (site 3) and abolished pre-excitation. Figure 3 shows the changes in the surface ECG following each ablation at the different sites. On repeat testing, tachycardia was no longer inducible and an absence of dual AV-nodal physiology was demonstrated. The patient remained symptom free at 3 month follow up.

References
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Figure 2. Hypothesized conduction pathways of beats $\alpha$, $\beta$ and $\gamma$. 

Leong KMW, Wright I, Linton NWF. Challenge of the Week: March 18th Answer
Figure 3. Change in preexcitation on the 12-lead surface following ablation at each site. PAC, premature atrial complex.