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Manuscript Title:

5 shades of grey in a Broad QRS tachycardia. What is the mechanism?

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Case Presentation

30-year-old gentleman with mild form of Ebstein's anomaly complained of recurrent palpitations but had no documented tachycardia. His baseline electrocardiogram (ECG) showed pre-excitation which was suggestive of a right posterior accessory pathway (Figure 1).

He was taken up for an electrophysiology study with the intent of radiofrequency ablation. Four catheters were placed – i) 2-5-2 spaced quadripolar catheter at the His region, ii) 2-5-2 spaced quadripolar catheter at the Right Ventricular apex, iii) 2-5-2 spaced decapolar catheter in the coronary sinus (CS) and iv) 3.5mm tip quadripolar ablation catheter in the right atrial appendage. Baseline intervals showed normal AH (Atrial-His) of 90ms and short HV (His-Ventricular) interval of 9ms. On atrial extra stimulus, there was loss of pre-excitation with QRS showing typical right bundle branch block aberrancy (RBBB) with tachycardia induction at a cycle length of 320ms (Figure 2). The initial few beats of tachycardia showed varying degrees of fusion with the QRS morphology finally settling for a typical left bundle branch

block (LBBB) morphology. What is the mechanism of this tachycardia where the trace shows 5 different QRS morphologies?

Discussion:

The basal train of programmed atrial pacing resulted in a QRS [labelled 1 in Figure 3] that was a fusion between antegrade AV nodal conduction and the right posterior accessory pathway. On atrial extra stimulus at 600ms – 300ms, there was blockade in the accessory pathway conduction and the impulse conducted antegradely through the Atrioventricular (AV) node and His Purkinje system with RBBB aberrancy with QRS duration of 130ms consistent with Ebstein's anomaly [labelled 2 in Figure 3]. The HV interval for this beat was 54ms. The next atrial activation [labelled A in Figure 3] was earliest in the proximal CS electrode which could have been due to retrograde conduction up the right posterior pathway and the VA interval was 176ms. It then conducted antegradely with a HV interval of 45ms resulting in a narrower QRS [labelled 3 in Figure 3]. This could suggest that it resulted from a fusion of AV nodal conduction and a premature ventricular beat arising from the right ventricle. It could also have resulted from a fusion of antegrade conduction down the AV node and another right sided accessory pathway which probably had decremental conduction. The next atrial impulse [labelled B in figure 3] had the same activation sequence with a VA interval of 130ms. The prior VA interval which followed QRS labelled 2 was longer because of the RBBB aberrancy. The atrial impulse [labelled B in figure 3] conducted antegradely with a wider QRS [labelled 4 in Figure 3] with a very short HV interval of 15ms. This suggests antegrade conduction predominantly down an accessory pathway with minimal contribution by the AV node. The VA interval following this QRS [labelled 4] was 130ms and remained so thereafter. From the next atrial impulse [labelled C in figure 3] onwards, the ventricles were depolarised only

through the proposed decremental right sided accessory pathway resulting in a stable LBBB QRS morphology [labelled 5 in Figure 3] with the His electrogram buried in the local ventricular electrogram.

On measuring the AA, HH and VV intervals during the first beat of the tachycardia it was clearly seen that AA drives the VV interval. Since the HH interval was more than the tachycardia cycle length (Figure 3) it can be inferred that His was not part of the circuit and was activated passively. Hence, AV nodal dependant tachycardia like Atrioventricular nodal reentrant tachycardia (AVNRT) and Atrioventricular reentrant tachycardia (AVRT) can be ruled out. Hence the possible differentials are,

1. Ventricular tachycardia
2. Atrial tachycardia with varying ventricular fusion resulting from antegrade nodal and pathway activation.
3. Duodromic tachycardia with antegrade conduction over a right sided decremental pathway (probably atriofascicular pathway) and retrograde conduction over the right sided posterior accessory pathway.

Ventricular tachycardia was ruled out as at the initiation of tachycardia the chamber activated was the atrium.

Atrial tachycardia with conduction over a right sided decremental pathway is a possibility with the first few beats demonstrating QRS fusion due to conduction through both the AV node and the pathway; the subsequent beats showing typical LBBB morphology due to conduction only through the pathway with His being activated retrogradely. However, an identical atrial activation sequence during both ventricular pacing [noted during the study] and tachycardia made it unlikely. The repeated induction of the tachycardia with an atrial extra made reentry the likely mechanism.

The His being a bystander made duodromic tachycardia (pathway to pathway tachycardia) with antegrade conduction over a right sided decremental pathway and retrograde conduction over the right posterior accessory pathway the most likely possibility. In the first few beats of the tachycardia, AV node was activated antegradely as a bystander causing varying "shades" of QRS fusion. As the antegrade conduction through the right sided decremental accessory pathway showed typical LBBB aberrancy with no delta waves; the His electrogram being buried in the local ventricular electrogram with earliest ventricular activation at the RV apex implied that the accessory pathway inserted into the right bundle – His purkinje system which was suggestive of an atriofascicular pathway. This case demonstrates that the presence of varying degrees of fusion in a wide QRS tachycardia does not always imply ventricular tachycardia.

The right posterior accessory pathway was mapped antegradely and at 6 o clock position on the tricuspid annulus there was a pathway potential, where it was successfully ablated. The atriofascicular pathway was mapped for its potential. It was present at 9 o clock position on the tricuspid annulus and successfully ablated. Postablation there was no evidence of any other pathway conduction on waiting for a period of 30minutes. Intravenous Adenosine given at 30minutes demonstrated AV and VA block. Postablation 12 lead ECG showed sinus rhythm with RBBB aberrancy like QRS labelled 2.

Duodromic tachycardia are generally very rare. The two accessory pathways involved in the reentry circuit are usually at considerable distance from each other for the maintenance of tachycardia[1]. The sustenance of duodromic tachycardia requires one of the limbs of the circuit to have slow conduction or decremental AV node like properties[2,3]. In this case even though both the limbs of the circuit were in proximity, the decremental properties of the atriofascicular pathway that is incapable of retrograde conduction provided the necessary delay to sustain the

tachycardia. This case highlights the importance of measuring individual intervals towards identifying the His as a bystander, which helped ascertain the mechanism.

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Figure legends:

Figure 1: 12 lead ECG showing sinus rhythm with pre-excitation

Figure 2: Intracardiac electrogram showing initiation of tachycardia with atrial extra stimulus. From top to bottom, II, aVF, V1 and V6 represent lead II, avF, V1 and V6 of the 12 lead ECG. HRA-D represents distal bipole of right atrial catheter in right atrial appendage. His 3-4 to 1-2 represent proximal to distal poles of His bundle catheter. CS 9-10 to 1-2 represent proximal to distal poles of a decapolar catheter in coronary sinus. RVD represent distal bipole of the catheter in right ventricular apex.

Figure 3: Intracardiac electrogram showing initiation of tachycardia with atrial extra stimulus. AA interval of 320ms is followed by VV interval of 320ms while HH interval is 350ms. Catheter position and abbreviation same as in Figure 2.