Ventricular overdrive pacing during supraventricular tachycardia

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1. Case

An 18-year-old male with recurrent palpitations and documented regular, long RP, narrow QRS tachycardia was taken up for electrophysiology study. Four catheters were placed in the high right atrium, right ventricular apex, coronary sinus and His bundle location. Tachycardia was easily inducible with an atrial extra-stimulus. During tachycardia (cycle length = 356 ms), ventricular overdrive pacing (VOP) at cycle length of 320 ms was done (Fig. 1). What is the mechanism of tachycardia?

2. Discussion

Differential diagnosis for any regular, long RP, narrow QRS tachycardia includes atrial tachycardia (AT), orthodromic reentrant tachycardia (ORT) mediated by a slow conducting accessory pathway and atypical atrioventricular nodal reentrant tachycardia (AVNRT).

In Fig. 1, atrial cycle length (CL) is the same as the paced CL, confirming entrainment of the tachycardia. Earliest atrial activation is in the proximal CS and this is consistent with any of the diagnoses listed above. The surface QRS is relatively narrow and predominantly positive in lead aVF while the pacing catheter is placed in the RV apex. This suggests that some part of ventricle is activated through His-purkinje system and ventricular activation is a fusion between antegrade conduction over His-purkinje system and RV pacing. Stable fusion morphology of surface QRS during ventricular overdrive pacing (VOP) of supraventricular tachycardia (SVT) suggests the diagnosis of ORT. But this finding has interobserver variability and a narrow window for its observation [1].

Antegrade activation sequence in His bundle during VOP of SVT provides intracardiac evidence of fusion. This finding is diagnostic of ORT and can be seen even when there is minimal or concealed fusion in surface QRS [2,3]. It can be noted in Fig. 1 that the His activation occurs before the pacing spike and the sequence of activation in mid and distal His channels is also consistent with antegrade activation of the His bundle. Patients with AVNRT or AT will not show this finding because entrainment of AVNRT or overdrive pacing of AT requires retrograde activation of His bundle before the impulse reaches the atrium (Fig. 2). Although uncommon, it is possible to entrain ORT with retrograde activation of His bundle when collision of the wavefronts is occurring above the His bundle. Thus, while the antegrade His bundle activation during VOP confirms the diagnosis as ORT, retrograde His bundle activation makes the diagnosis of ORT unlikely but does not rule it out and needs further EP evaluation.

Rarely it is possible to get fusion in surface QRS morphology and antegrade His activation during VOP of AVNRT or AT with bystander
concealed posteroseptal accessory pathway if the retrograde His-purkinje conduction is too slow or absent. Thus, after ablation of accessory pathway, non-inducibility of SVT including AVNRT or AT should be confirmed.

This patient was diagnosed as a case of ORT mediated by a slow and decremental conducting concealed posteroseptal accessory pathway. Pathway was mapped during tachycardia and successfully ablated. No tachycardia was inducible after ablation.
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Declaration of competing interest

None for any of the authors.

References

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