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Short Communication

Wide and narrow QRS tachycardias: What is the mechanism?

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ABSTRACT

A 50-year-old lady was referred for radiofrequency catheter ablation of narrow QRS tachycardia that was terminated with intravenous adenosine. Twelve-lead Electrocardiogram (ECG) was normal during sinus rhythm. The electrophysiological study showed an Atrio-Hisian (AH) interval of 104 ms and Hisio-Ventricular (HV) interval of 45 ms during sinus rhythm. Atrial pacing reproducibly induced regular broad (left bundle branch block morphology) and narrow QRS tachycardias. A spontaneous premature ventricular ectopic from right ventricular apex has resulted in transition of the tachycardia from wide to narrow. What are the likely mechanisms?

1. Commentary

Fig. 1 shows a regular narrow QRS tachycardia with a stable cycle length (CL) of 310 ms and fixed septal Ventriculo-atrial (VA) interval of 150 ms. The earliest atrial activation was recorded at coronary sinus (CS) 9,10 dipoles placed at CS ostium. The differentials entertained for this narrow QRS tachycardia with long VA interval are atypical atrioventricular (AV) nodal reentrant tachycardia and orthodromic AV reentrant tachycardia (AVRT) with slowly conducting accessory pathway and atrial tachycardia. Fig. 2 shows a regular broad QRS tachycardia of LBBB morphology, with stable Tachycardia Cycle Length (TCL) of 310 ms and septal VA interval of 150 ms. The earliest atrial activation during the tachycardia was also recorded at CS 9,10 dipoles. The tachycardia with LBBB morphology showed a H–V-A activation pattern with His bundle electrogram (EGM) preceding the local ventricular EGM suggesting that the broad QRS tachycardia is likely to be supraventricular tachycardia with LBBB aberrancy. Identical atrial activation pattern, identical TCL and identical VA interval suggest similar mechanism for both tachycardias. Since development of LBBB does not alter the TCL and VA interval, it could be due to AV nodal reentry or AV reentry involving a right-sided accessory pathway or atrial tachycardia. Fig. 3 shows transition of wide QRS tachycardia to narrow QRS tachycardia in response to a spontaneous (PVE from the right ventricular apex. It is observed that PVE has resulted in advancement of the atrial EGM or atrial preexcitation followed by prolongation of AV interval and subsequent narrowing of QRS. Resetting of the tachycardia by the PVE suggests the mechanism as AV reentry.

Subsequent to the atrial preexcitation, in response to the PVE, the AV interval has prolonged followed by perpetuation of tachycardia with normalization of QRS, identical atrial activation pattern, identical TCL interval and identical VA interval. The AV delay after the atrial preexcitation represents anterograde conduction delay at the node, and it has given adequate time for the Left Bundle Branch (LBB) to come out of refractoriness, that is, gap phenomenon and the tachycardia continues with narrow QRS. It is also likely that the PVE has also made retrograde concealment of the LBB and peeled off its refractoriness.

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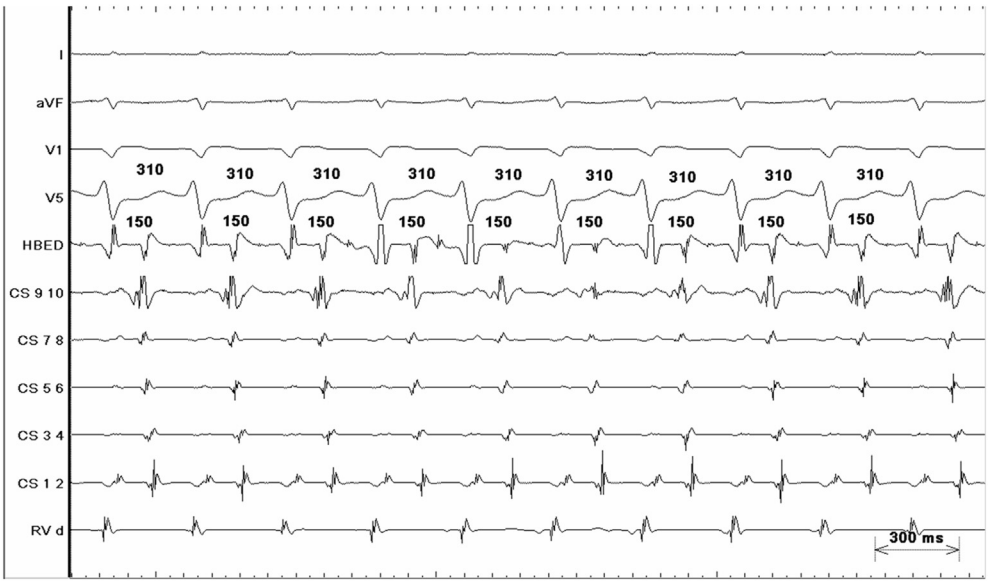

Fig. 1. Represents surface electrocardiogram (I, aVF, V1, V5) and intracardiac electrograms His distal (HBED) coronary sinus (CS) 9,10 to CS 1,2 and right ventricular apex (RVA) showing narrow QRS tachycardia.

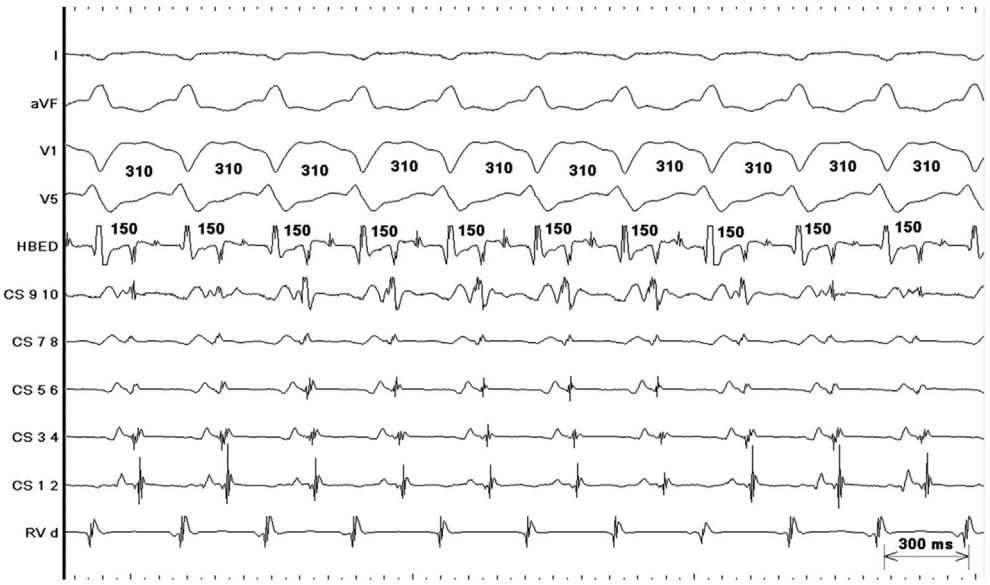

Fig. 2. Represents surface electrocardiogram (I, aVF, V1, V5) and intracardiac electrograms His distal (HBED) coronary sinus (CS) 9,10 to CS 1,2 and right ventricular apex (RVA) showing broad QRS tachycardia of LBBB morphology. LBBB, left bundle branch block.

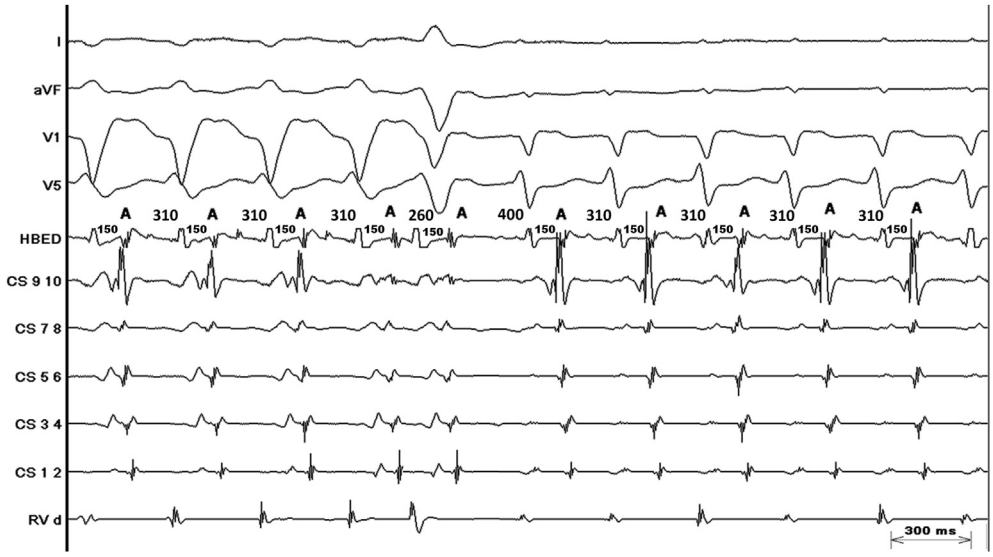

Fig. 3. Represents surface electrocardiogram (I, aVF, V1, V5) and intracardiac electrograms His distal (HBED) coronary sinus (CS) 9,10 to CS 1,2 and right ventricular apex (RVA) showing transition of broad QRS tachycardia of LBBB morphology to narrow QRS tachycardia in response to a spontaneous PVE. LBBB, left bundle branch block; PVE, premature ventricular ectopic.

During AVRT development of ipsilateral Bundle Branch Block (BBB) enlarges the tachycardia circuit due to transseptal conduction. This typically results in increase in tachycardia CL and VA interval. Free wall accessory pathways (APs) are associated with a VA increase >35 ms, and for septal APs, the increase in the VA interval is usually <25 ms. As the contra lateral bundle branch is a bystander, conduction delay or block in it will not affect the tachycardia CL or VA time in orthodromic AVRT. Hence, the most likely mechanism in this case is AV reentry using a right free wall AP.

The pathway was mapped to 8 O'clock of tricuspid annulus and ablated.

Conflicts of interest

All authors have none to declare.