Conflicting responses to two ventricular entrainments in a narrow QRS tachycardia: What is the mechanism?

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Case

A 37 year-old woman who had a history of a paroxysmal supraventricular tachycardia (SVT) with a short RP interval was referred for an electrophysiological study. Her clinical tachycardia was induced by programmed atrial stimulation with an AH jump. A single His refractory premature atrial contraction (PAC) delivered via the proximal coronary sinus electrodes advanced the next His timing (Figure 1A). The response of tachycardia to right ventricular (RV) entrainment at pacing cycle lengths (PCL) of 340 ms are shown in Figure 1B, but sequential RV entrainment at PCL of 350 ms yielded a conflicting response compared to the previous RV entrainment (Figure 2A). Figure 2B, shows the moment of the tachycardia perturbation by the second attempt of RV entrainment. Based on these electrophysiological findings, what is the mechanism of the conflicting responses of the two ventricular entrainments?

Discussion

The differential diagnosis of a short RP SVT with an HAV activation sequence with a VA interval of < 70 ms mainly includes junctional tachycardia (JT), typical slow-fast atrioventricular nodal reentrant tachycardia (AVNRT), and orthodromic reciprocating tachycardia (ORT) using a nodofascicular/nodoventricular (NF/NV) bypass tract (BT) (ONF/VRT) usually inserting into a slow pathway (SP) of the AV node. Rarely atrial tachycardia (AT) with a long AV interval is also possible, however, the identical atrial activation sequence during both tachycardia and ventricular entrainment reduces this possibility (Figure 1 B). In Figure 1A, a PAC delivered during His refractoriness advanced the next His electrogram by 12 ms. This response is highly suggestive of AVNRT and makes ONF/VRT less likely, because in the case of ONF/VRT, before the His becomes refractory, the tachycardia wavefront would have already penetrated the SP over the NF/NV BT, and then engaged in the remaining fast pathway (FP) or another SP retrogradely, rendering a normal atrioventricular conduction system refractoriness at the timing of the PAC. This finding also excludes JT, because any perturbation of the subsequent His activation by a His refractory PAC indicates antegrade SP conduction during AVNRT.[1] The response of the tachycardia to RV entrainment at a PCL of 340 ms demonstrated an AHAV response with a long post pacing interval (PPI) (Figure 1 B), and those findings are also consistent with AVNRT. However, conflicting responses were observed during a consecutive second attempt of RV entrainment at a PCL of 350 ms. After the cessation of the RV entrainment, unlike the previous entrainment, the first and second atrial electrograms were driven by the penultimate and last pacing stimulus, respectively (Figure 2A). This response is suggestive of slow conduction of the tachycardia's retrograde limb, which is not expected in typical slow-fast AVNRT. We also focused that the first returning His electrogram preceded the last entrained atrial electrogram. During a ventricular entrainment of AVNRT, the collision between the orthodromic and antidromic wavefronts generally occurs within the SP and the His bundle is antidromically activated. When the last ventricular stimulus is delivered, the first returning (orthodromic) His activation is made by the last entrained atrial activation, therefore, it cannot precede the last atrial activation with a PCL. Furthermore, repetitive retrograde penetration into the SP by ventricular overdrive pacing (VOP) often causes a post-pacing AH decrement, so the first returning His activation commonly occurs in excess of the TCL. Hence, these phenomena are not expected in pure AVNRT. Figure 2B exhibited the moment of the tachycardia perturbation during the second attempt of ventricular entrainment, which provides conclusive findings to infer the tachycardia mechanism. During the early phase of VOP, an SVT perturbation occurred within the transition zone (TZ), indicating the presence of BT conduction.[2] The HAV activation sequence during the tachycardia excludes an atrioventricular reentry, but an orthodromic reentry using an NV/NF BT inserting into SP of the AV node could elicit this phenomena. In this instance, after retrograde conduction from the NV/NF BT to the SP, the FP and His bundle are activated in parallel from the distal SP and the His bundle electrogram can precede the atrial electrogram when an antegrade His bundle conduction is faster than a retrograde FP conduction. Additionally, tachycardia reset within the TZ suggests NV BT more than NF BT.[3] Taken together, at this point AVNRT with bystander NV BT and ONVRT can be considered. Although it was not shown in the case description, His-refractory premature ventricular stimulus delivered during the tachycardia advanced the next His and subsequent atrial electrogram, which confirms a presence of slowly-conducting BT. Theoretically, in case of AVNRT with bystander NV BT, ventricular entrainment could be achieved over a BT conduction. In that case, the paced stimuli that activates the His bundle retrogradely should not enter the tachycardia circuit, thus the His electrogram is expected to be captured orthodromically during ventricular entrainment. In our case, we can surmise that the His was activated antidromically during the second RV entrainment because the orthodromic His activation was seen only after the cessation of VOP, hence ventricular entrainment of the AVNRT over the NV BT less likely. In contrast, ventricular entrainment of orthodromic NVRT can be achieved with the retrograde His capture, because significant conduction delay produced in NV BT would move the wavefront collision site above the lower common pathway. In summary, the initial mechanism of the tachycardia was AVNRT with a bystander NV BT, but it is thought to be transformed into an ONVRT during the second attempt of ventricular entrainment, and thereby conflicting post ventricular entrainment responses were demonstrated. Although the large difference between the PPI and TCL in the second ventricular entrainment is more consistent with AVNRT, rate related retrograde decrement NV BT conduction can generate

artificially large PPI for ventricular entrainment. As well as, in our case, significant shortening of the PPI at the second ventricular entrainment compared with the first ventricular entrainment supports that the tachycardia circuit has changed to be closer to the pacing site.

In the case of AVNRT with a bystander NV BT, which is attached to SP, a ventricular pacing stimulus can penetrate the excitable gap over the BT, and when the pacing stimulus cannot conduct orthodromically due to the refractoriness of SP, the AVNRT is terminated. However, if the tachycardia wavefront that has already passed the distal SP conducts to FP and His bundle, initiation of an ONVRT with subsequent ventricular entrainment can be achieved by continued VOP. When considering the circuit of AVNRT and ONVRT as a figure of eight reentry that shares the SP into which the NV BT is inserted, this transformation is feasible. To eliminate both the AVNRT and ONVRT, radiofrequency energy was delivered to the SP, after which no further SVT was inducible.

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Figure legends

Figure 1. A. A premature atrial contraction introduced during His refractoriness advanced the next His electrogram. B. The tachycardia response to the first attempt of ventricular entrainment exhibited an AHAV response with a long post pacing interval (PPI).

Figure 2. A. Response to the second attempt of ventricular entrainment also exhibited an AHAV response but the PPI was shorter than the first attempt of entrainment. During ventricular overdrive pacing, the atrial cycle length became shorter than the tachycardia cycle length, which confirmed the achievement of entrainment. B. The moment the tachycardia perturbation occurred during the second attempt of ventricular entrainment. The next atrial interval was advanced by 10 ms by the fourth pacing stimulus (asterisk) with a fused QRS morphology.