Typical atrioventricular nodal reentrant tachycardia with 2:1 conduction block: What is the mechanism?

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Keywords: atrioventricular nodal reentrant tachycardia, conduction block

CASE

A 66-year-old woman with a history of frequent premature ventricular contractions (PVCs), and wide QRS complex tachycardia, underwent electrophysiological testing. During the electrophysiological study, multipolar catheters were positioned in the coronary sinus (CS), His bundle (HB) region, and right ventricular apex. She underwent successful catheter ablation of PVCs at the mitral annulus. Following this, an induction of wide QRS complex tachycardia was attempted. Programmed atrial stimulation demonstrated no evidence of dual AV nodal physiology. Any pacing maneuvers from the right side could not induce any tachycardias. Programmed ventricular stimulation from the left ventricle induced a nonsustained supraventricular tachycardia (SVT) with 2:1 atrioventricular (AV) conduction block (Figure 1). An SVT with 2:1 AV conduction block was induced again, but the His-atrial (HA) interval was shorter than that during the first SVT (Figure 2). This SVT immediately converted to a wide QRS complex tachycardia with 1:1 AV conduction (Figure 2). During the tachycardia, there was a slight oscillation in the tachycardia cycle length (Figures 1 and 2), and the preceding His to His (HH) intervals equaled the following intervals between the atrial activations (AA intervals). Rapid ventricular pacing was performed, and the tachycardia was terminated without any retrograde atrial capture. What is the mechanism?

DISCUSSION

In this case, the wide QRS complex tachycardia was considered to be an SVT with an aberrancy. Furthermore, the SVT was suggested to be a slow-fast type AV nodal reentrant tachycardia (AVNRT) for several reasons. First, the SVT occurred with 2:1 AV conduction, and an AV reciprocating tachycardia was ruled out. Second, the SVT was induced with a V-A-H-V-A pattern. Third, the SVT was terminated by ventricular pacing without any retrograde atrial capture. Fourth, during the SVT with a slight oscillation in the tachycardia cycle length, the preceding HH intervals equaled the following AA intervals. The last three findings were consistent with typical AVNRT. In fact, in this case, the slow pathway was mapped, and successfully ablated. Thereafter, no tachycardias were induced by any pacing maneuvers on or off an isoproterenol infusion.

It has been reported that 2:1 AV conduction block sometimes occurs during an AVNRT. In this case, different electrophysiological findings were recorded when 2:1 AV conduction block occurred during the typical AVNRT in Figures 1 and 2. First, when the AV conduction block occurred, an HB potential was present in Figure 1 whereas it was absent in Figure 2. Therefore, the AV conduction block occurred at the infra-Hisian region in Figure 1 and at the supra-Hisian region in Figure 2. Second, in Figure 1, the AA interval and atrio-His (AH) interval before the AV block were shorter than those after the AV block. However, in Figure 2, the AA interval before the AV block was longer than that after the AV block. Third, when 2:1 AV conduction block occurred, the HA intervals were longer in Figure 1 than those in Figure 2. The HA intervals with 2:1 AV conduction block in Figure 1 were similar to those with 1:1 AV conduction in Figure 2. In Figure 1, the alternating shortening of the AH intervals could be explained by an involvement of the upper common pathway. When the retrograde activation conducted from the fast pathway to the slow pathway through the upper common pathway, the activation would conduct over the...
slow pathway antegrade while the activation conducted to the atrium over the fast pathway retrogradely. The shortening of the AH intervals led to the shortening of the following AA (=AH+HA) and HH (=HA+AH) intervals. After the short HH intervals, the infra-Hisian region did not recover from the refractoriness, resulting in an occurrence of the infra-Hisian conduction block. The HH intervals with 2:1 AV conduction block in Figure 1 were as short as those with 1:1 AV conduction and an aberrancy in Figure 2. These findings suggested that in Figure 1, the AV conduction was completely blocked at the infra-Hisian region, but in Figure 2, that was partially blocked with the aberrancy. In Figure 2, the shortening of the HA intervals could be explained by an involvement of the lower
common pathway. When the antegrade activation conducted from the slow pathway to the fast pathway through the lower common pathway, the activation would conduct over the fast pathway retrogradely while the activation conducted to the HB over the slow pathway antegradely. This mechanism was supported by the evidence that the AVNRT was sustained without recording the HB electrograms. In Figure 2, the shortening of the HA intervals would have led to the shortening of the following HH (=HA+AH) intervals although HB electrograms were not recorded. After the short HH intervals, the supr-Hisian region below the lower common pathway and above the HB did not recover from the refractoriness, resulting in the occurrence of the supr-Hisian conduction block.

In summary, in this case, the involvement of the upper and lower common pathways during the typical AVNRT induced 2:1 AV conduction block.

CONFLICT OF INTEREST
The authors declare no conflict of interests for this article.

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