Recurrent Supraventricular Tachycardia with a Different Retrograde Atrial Activation Sequence: What is the Mechanism?

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A young man underwent 2 catheter ablation procedures because of frequent episodes of paroxysmal supraventricular tachycardia (SVT). A left accessory pathway was ablated in the first procedure. The patient underwent the second ablation procedure due to a recurrent SVT demonstrated by 12-lead electrocardiogram two weeks after the first procedure. During the second procedure, a SVT with a different retrograde atrial activation sequence was present. Programmed stimulation at the right ventricular apex showed multiple retrograde atrial activation sequences. Thereafter, careful analysis of the intracardiac electrograms helped us to successfully ablate the SVT.

Key Words: Ablation • Electrophysiology • Supraventricular tachycardia

CASE REPORT

A 26-year-old male underwent a catheter ablation procedure because of frequent episodes of supraventricular tachycardia (SVT). During the electrophysiological study, an atrioventricular reentry tachycardia (AVRT) using the atrioventricular nodal (AVN) pathway for antegrade conduction and a left accessory pathway (AP) for retrograde conduction was present. After multiple attempts, the AP seemed to be blocked by radiofrequency delivered at the anterolateral mitral annulus area (Figure 1). However, the patient underwent the second procedure due to a recurrent SVT demonstrated by 12-lead electrocardiogram two weeks after the first ablation procedure. During the second procedure, a narrow QRS tachycardia with an interesting retrograde atrial activation sequence was present (Figure 1C). S1S2 stimulation at the right ventricular apex (RVA) showed multiple retrograde atrial activation sequences (Figure 2). The purpose of this article was to determine the underlying nature of the mechanism for the SVT and the different atrial activation sequences during RVA pacing.

DISCUSSION

An interesting phenomenon of this tachycardia (Figure 1C) in the second ablation procedure was the discontinuous activation between the proximal coronary sinus (CS, CS ostium to CS5-6) and distal CS (CS5-6 to CS12). The double atrial potentials at CS5-6 may indicate a conduction block between CS3-4 and CS7-8. After careful mapping, the earliest retrograde atrial activation was found in close proximity to CS5-6. These observations suggested that the mechanism of the tachycardia was AVRT using the remaining left AP as the retrograde limb. The previous ablation in the first procedure did not totally eliminate the AP; however, a conduction block between the atrial end of the AP and the proximal CS was created (Figure 1B). The relatively long ventriculoatrial interval in the proximal CS (Figure 1C) during retrograde AP conduction was caused by the long travel-
ing distance from the atrial end of AP clockwise (in the left anterior oblique view) to the proximal CS due to the conduction block at CS5-6. The multiple retrograde atrial activation sequences during S1S2 stimulation at the RVA (Figure 2) were caused by different conduction pathways (AVN and/or AP) at proximal and distal CS. In response to the S1S2 stimulation in Figure 2A, the retrograde atrial activation route in the proximal CS changed from the AVN (the 1st and 2nd beats) to the AP (the 3rd beat). S1S2 stimulation in Figure 2B did not show a decremental conduction in the proximal CS, suggesting that this activation sequence was via retrograde AP. The atrial activation sequence in Figure 2B was identical to that during the SVT (Figure 1C), further supporting a retrograde AP conduction. S1S2 stimulation in Figure 2C induced a decremental conduction in the proximal CS, suggesting that this activation sequence, identical to the first and second beats in Figure 2A, was via retrograde AVN. In Figures 2A, 2B and 2C, the atrial activation in the distal CS was consistently conducted via the retrograde AP. In Figure 2D, the AP conduction (the first and second beats) was interrupted in distal CS and an abrupt increase in ventriculoatrial interval was noted (the third beat), suggesting that the CS distal was activated via the retrograde AVN for the third beat. Double atrial potentials at the CS5-6 were clearly observed at the third beat.

The AP was finally eliminated by ablation delivered in close proximity to the CS5-6. Rapid intravenous injection of 20 mg of atropine showed ventriculoatrial dissociation, proving the elimination of the AP. The patient did not report any palpitation during his subsequent 8-month follow-up.

A similar phenomenon of intra-atrial conduction block along the mitral valve annulus caused by extensive radiofrequency applications was also reported in pre-
vious studies. Together, these studies and our case suggest that careful and detailed mapping is the necessary prerequisite to kill the AP with one burn.

REFERENCES


