Case Report

Type A Wolff-Parkinson-White Syndrome Generating an Antidromic Atrioventricular (AV) Reentrant Tachycardia (AVRT) and an Orthodromic AVRT with a Long RP Interval Initiated only after Incomplete Impairment of an AV Accessory Pathway

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We report on a case of a 23-year-old male with Wolff-Parkinson-White syndrome. At baseline, constant right atrial pacing induced antidromic atrioventricular reentrant tachycardia (AVRT), whereas constant right ventricular (RV) pacing only revealed a normal His-Purkinje system. Mapping below the mitral annulus during sinus rhythm revealed fusion of atrial and ventricular potentials at multiple lateral sites. After unsuccessful ablation at these sites, constant RV pacing induced a long RP interval, orthodromic AVRT with the earliest atrial site being located at an anterior aspect, where successful ablation was later achieved. These phenomena may indicate an unexpected arrhythmogenic effect of initial ablations.
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Key words: Decremental accessory pathway conduction, De-novo arrhythmia, Catheter ablation

Introduction

We report on a rare case with Wolff-Parkinson-White syndrome requiring numerous radiofrequency (RF) current applications because of unique characteristics, thus causing a de-novo tachycardia.

Case report

A 23-year-old male with a structurally normal heart and recurrent episodes of wide QRS tachycardia was admitted to Nozaki Tokushukai Hospital for RF catheter ablation treatment of type A Wolff-Parkinson-White syndrome. Electrophysiologic study (EPS) and ablation were performed off antiarrhythmic agents after written informed consent was obtained. A 7-F, decapolar catheter (Daig Corp., Minnetonka, MN, USA) was inserted into the coronary sinus (CS) via the right internal jugular vein and its tip was positioned as distally as possible, being confirmed with the injection of contrast media. EPS was performed using three additional electrode catheters positioned at high right atrium (HRA), His

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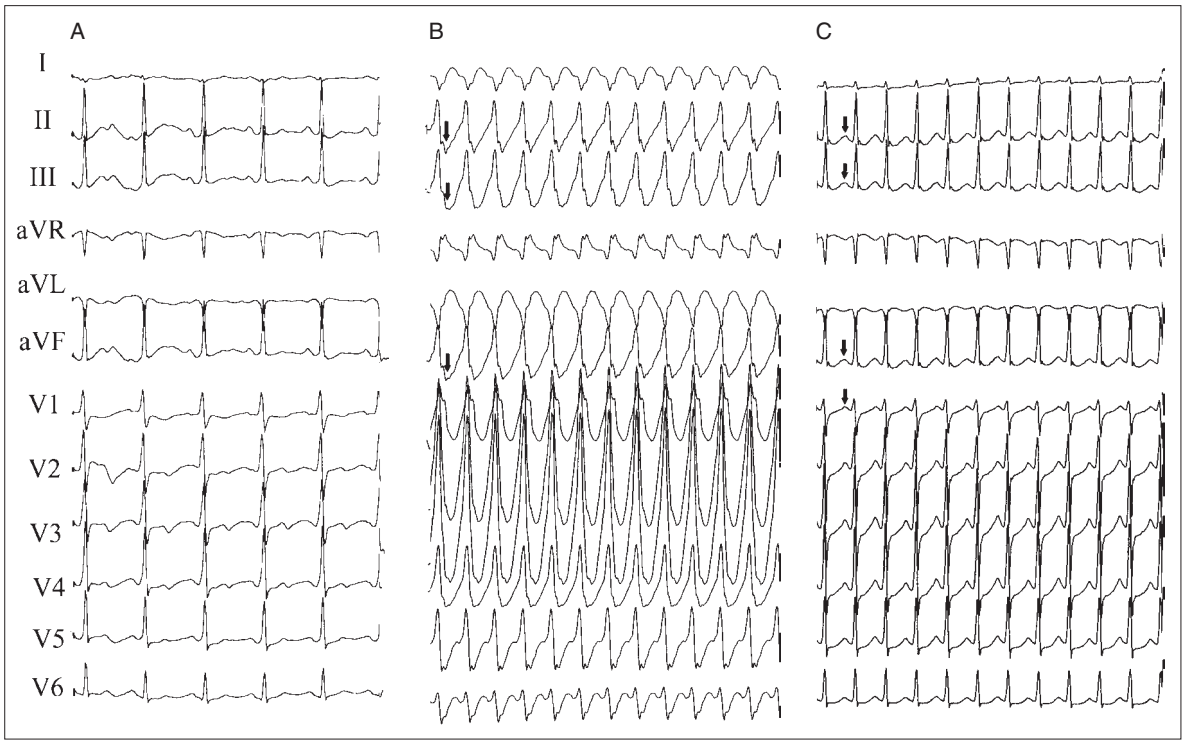

Figure 1 Surface ECG during sinus rhythm (**A**), an antidromic AVRT (**B**) and an orthodromic AVRT (**C**). Downward arrows in **panels B** and **C** indicate retrograde P-waves.

bundle area (HBE) and right ventricular apex (RV). Ablation was attempted either at a site showing fusion of atrial and ventricular potentials or a site recording an accessory pathway (AP) potential during sinus rhythm on the condition of a 50 Watt power or 60 °C temperature limit using a 500-kHz continuous-wave current generator (Japan Lifeline Inc., Tokyo, Japan). RF energy was applied below the mitral valve for 60 to 120 seconds if a delta wave was eliminated within 10 seconds but if not, discontinued after a maximum of 20 seconds. **Figure 1-A** shows a baseline 12-lead ECG during sinus rhythm, which suggested that the atrioventricular (AV) AP was localized in the lateral or anterolateral wall of the left ventricle (LV) according to Arruda's ECG algorithm.[1] **Figure 1-B** indicates surface ECG of an antidromic AVRT initiated by a constant HRA pacing at the rate of 220 beats/minute (BPM) at baseline, whereas **Figure 1-C** shows an orthodromic AVRT caused during straight RV pacing for the first time after an incomplete impairment of the AP by RF ablation. Note that in **Figures B** and **C**, negative P-waves immediately after QRS complex in inferior leads and positive P-waves before QRS in inferior leads and V1, marked by downward arrows, are observed, respectively.

The antidromic AVRT

Figures 2-A and **B** show responses to stimuli applied from the CS1-2 electrode pair during the tachycardia of **Figure 1-B**. Since the QRS morphology showed maximal preexcitation, this tachycardia was considered to utilize the AP as an anterograde limb. Furthermore, to clarify that the AP played an essential role in the tachycardia, atrial stimulation was applied with a coupling interval of 245 ms, or 15 ms shorter than the tachycardia cycle length (**Figure 2-A**). This caused a premature atrial excitation followed by ventricular capture and another atrial response (sequence of A-Vs-As). **Figure 2-B** shows the response to entrainment pacing with a cycle length of 250 ms during the tachycardia. Similarly, the Vs-As response was observed upon cessation of pacing. These findings imply that the tachycardia was not an AV nodal reentrant tachycardia with a bystander AP but an antidromic AVRT itself, a classic type according to Josephson.[2] Furthermore, since the difference between the post-pacing interval (PPI) and the tachycardia cycle length was just 10 ms, the atrial insertion site of the AP is considered to be very close to the atrial myocardium adjacent to the CS1-2, or slightly more anterior. Although stimulation from the RV up to a rate of 200 BPM did not reveal the presence of any

retrograde conduction other than via the normal His-Purkinje system (not shown), the presence of a retrograde conduction via the AP was unveiled by a pair of PVCs with a morphology of RBBB appearing during manipulation of the ablative catheter into the LV before ablation. The earliest atrial activation corresponded to a low-frequency potential marked by an asterisk at the CS3-4, possibly originating from the left atrium,[3] followed by the CS5-6, then the CS1-2, CS7-8, CS9-10 and finally the HRA (**Figure 2-C**). Since no fusion of ventricular and atrial potentials was observed on the CS 3-4, the retrograde conduction over the AP may be a relatively slow and/or oblique pathway. Mapping the LV wall below the mitral annulus using an ablative catheter during sinus rhythm was able to reveal fusion of atrial and ventricular potentials in the proximity of the CS3-4, at 4 o'clock phase along the mitral annulus in the left anterior oblique (LAO) projection (**Figure 2-D**), and CS1-2, at 3 o'clock (**Figure 2-E**). However, RF energy applied to these sites resulted in only a transient elimination of the delta wave (not shown).

The orthodromic AVRT with a long RP interval

Figure 3-A shows that the tachycardia of **Figure 1-C** was initiated by constant RV pacing with a cycle length of 460 ms during sinus rhythm for the first time after an incomplete impairment of the AP by above ablations (26 applications in total). Its atrial activation sequence was quite different from that of the retrograde atrial activation over the AP as shown in **Figure 2-C**. Namely, the earliest activation was registered at the distal pair of the ablative catheter (asterisks on ABL1-2) positioned at 2 o'clock phase of the LAO view, followed by the CS1-2 electrode pair, then the CS3-4 coinciding with the HRA, finally almost simultaneously the CS5-6 and the CS7-8. Conduction intervals from the earliest site to individual atrial potentials were measured, of which values were shown by figures with arrows. **Figures 3-B** and **C** reveal the initiation and cessation of RV pacing with a cycle length of 260 ms during the tachycardia with a cycle length of 290 ms, respectively. In **Figure 3-B**, the 1st RV stimulus delivered at a coupling interval of 240 ms from the previous His potential (shown as H-St; 240), 50 ms earlier than the estimated timing of an anterograde His potential, could capture atrial myocardia by 15 ms prematurely. In **Figure 3-C**, the cessation of the pacing was followed by the As-H-V response with the same atrial activation sequence as that of the tachycardia, implying the entrainment phenomenon. Furthermore, the stimulation at a still shorter cycle length of 250 ms could provoke the same one and at 240 ms terminate the tachycardia by ventriculo-atrial (VA) conduction block at last (not shown). Also, VA conduction time occupied a significant portion of the tachycardia cycle length, for example VA/AV >1, and the conduction time gradually prolonged in a rate-dependent fashion (not shown). Therefore, the mechanism of this narrow QRS tachycardia was considered to be a long RP interval, orthodromic AVRT using a left sided AP with a decremental conduction property for a retrograde limb, but not an atrial tachycardia.[4] Simultaneous registration of atrial depolarizations on the CS3-4 and the HRA electrogram possibly indicates that the HRA region was activated via the Bachmann's bundle. Alternatively, the synchronization may imply that the tachycardia was uncommon AVNRT with the CS ostium as the earliest retrograde atrial site. However, this probability is not likely at all, because in addition to the above findings, positive P-waves in leads II, III, aVF and V1 during the tachycardia, shown by arrows in **Figure 1-C**, strongly suggest that the earliest site should differ from the posteroseptal area of the right atrium.

The successful ablation site

Figures 4-A, **-B** and **-C** show the intracardiac recordings, ECG and fluoroscopic images at the successful ablation, respectively. Namely, an RF application below the mitral annulus at 2:30 o'clock phase in the LAO view during sinus rhythm, the 28th trial from the beginning, resulted in the disappearance of delta wave. Importantly, **Figure 4-A** indicates that the electrogram from the successful site did not show fusion of atrial and ventricular potentials, which may imply that the AP crossed the mitral annulus at an angle[5] and/or earlier RF applications resulted in anterograde conduction delay over the AP.[6] In total, the number of RF applications was 32 and the duration of delivery 1031 seconds. After a successful RF ablation, programmed stimuli from multiple sites could not provoke any conduction over the AP or any variety of tachycardia and stimulation from RV could reveal only a retrograde decremental conduction via AVN. Finally, it was fluoroscopically confirmed throughout the procedure that no electrode catheter had been displaced.

Discussion

Unique characteristics of the present case are as follows. First, multiple sites demonstrating fusion of atrial and ventricular potentials during sinus rhythm

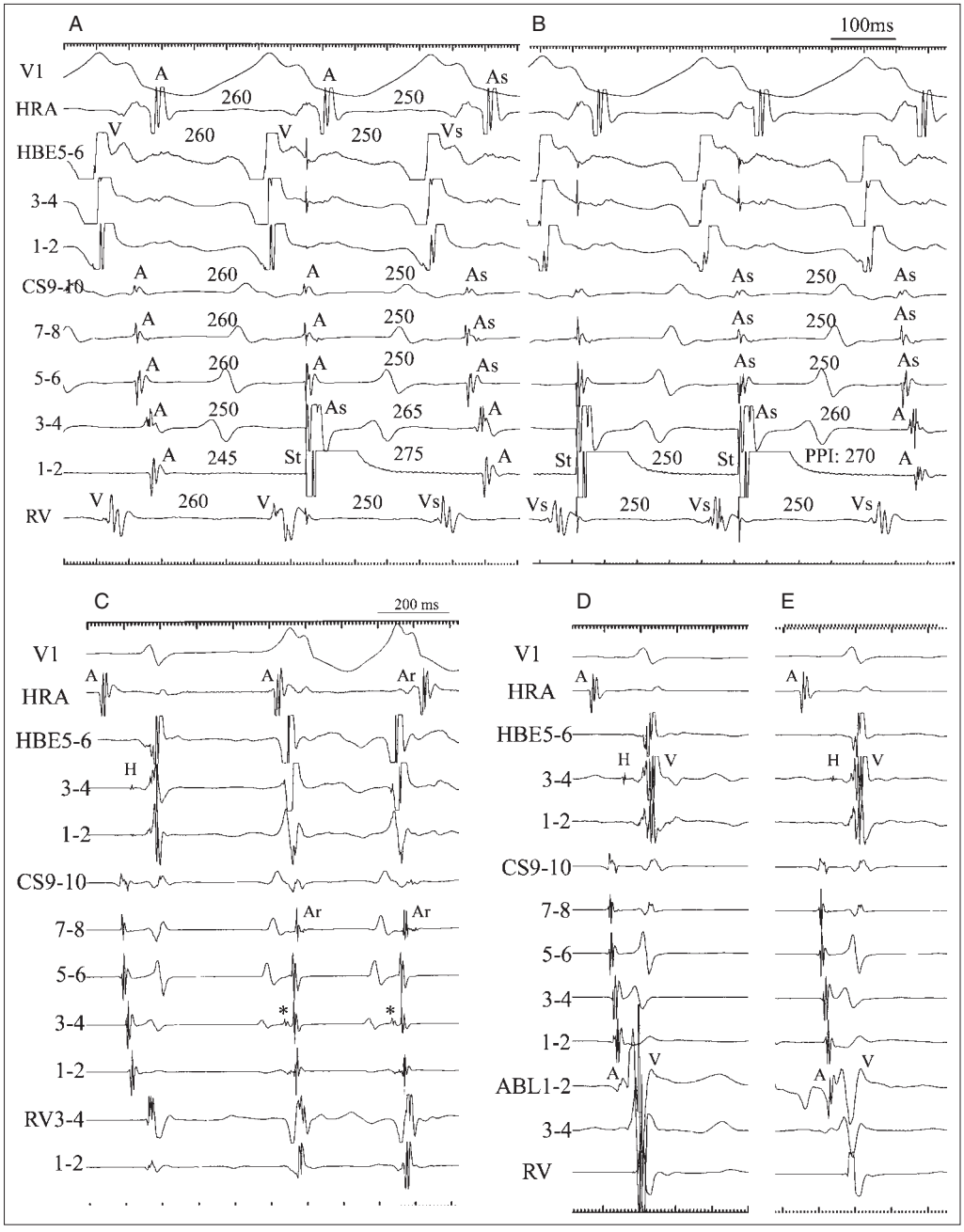

Figure 2 EPS records associated with the antidromic AVRT and unsuccessful ablation.
Surface ECG lead V1 and intracardiac electrograms from the high right atrium (HRA), His bundle region (HBE, 1-2: distal, 5-6: proximal), coronary sinus (CS, 1-2: distal, 9-10: proximal) and right ventricular apex (RV) are shown from the top to the bottom. A and V: atrial and ventricular potentials, St: stimulus artifact, As and Vs: atrial and ventricular potentials preexcited by the stimulus, PPI: postpacing interval. Figures above individual electrograms indicate A-A or V-V interval. In **panel A**, atrial stimulation from the CS1-2 electrode with a coupling interval of 245 ms prematurely captures the ventricular myocardium (Vs), followed by atrial depolarizations (As) except that at the CS3-4, which is directly captured by the stimulation. Similarly in **panel B**, pacing with a cycle length of 250 ms entrained the tachycardia with the PPI being just 10 ms longer than the tachycardia cycle length. Ar: retrograde atrial potential, H: His potential, ∗: possible left atrial potential. **Panel C** shows that the retrograde conduction over the AP was unveiled by paired PVCs with RBBB morphology before ablation. Although main deflections of the last atrial depolarization, possible CS musculature, were registered earliest on the CS5-6, a small potential marked with an asterisk on the CS3-4 was even earlier. **Panels D** and **E** show intracardiac recordings at an energy application near the CS3-4 and CS1-2 (ABL1-2 and 3-4: distal and proximal electrograms on the ablative catheter, respectively), respectively. Both panels reveal fusion of A and V potentials on the ABL1-2.

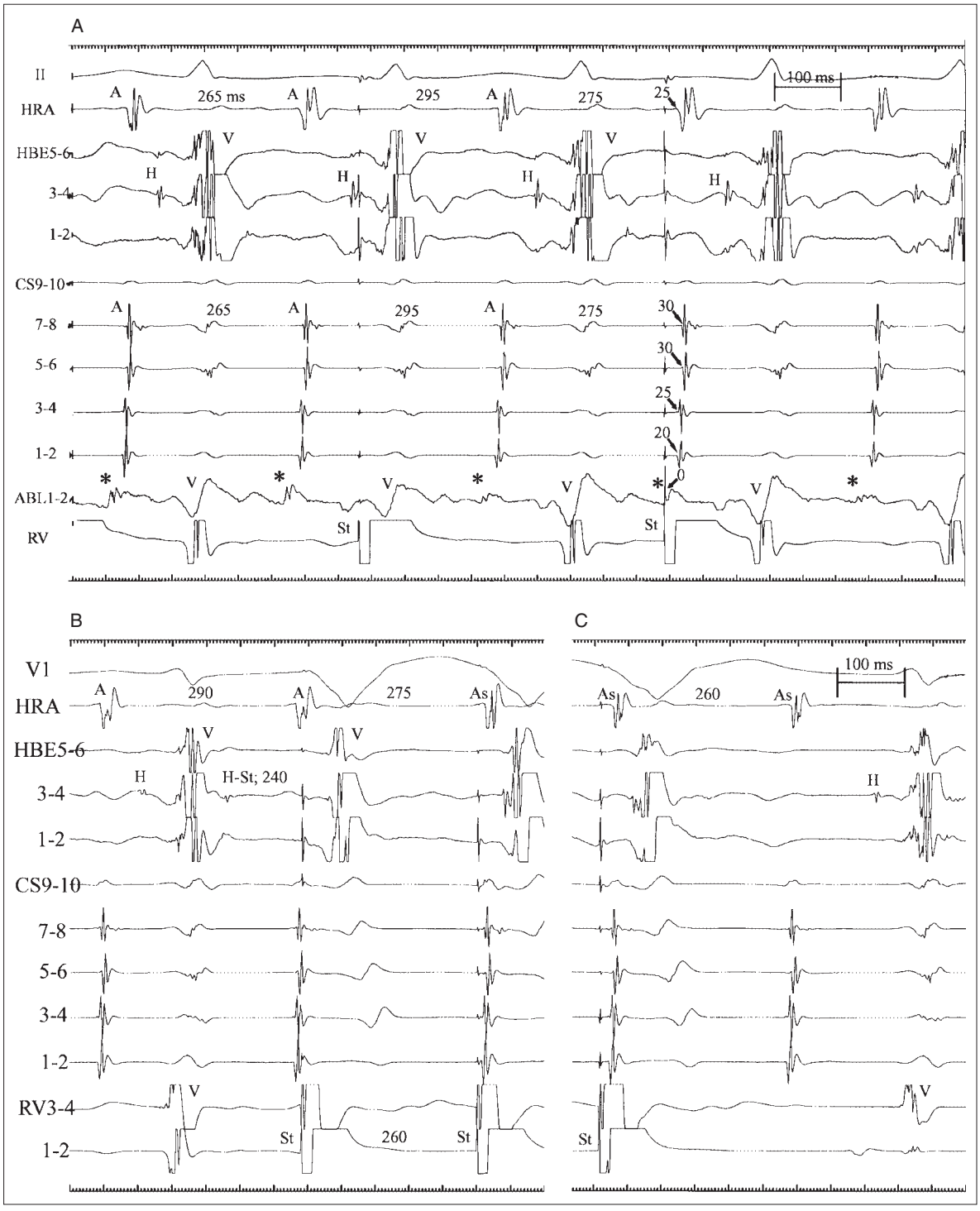

Figure 3 EPS records during the orthodromic AVRT.
Panel A shows the tachycardia induced by constant RV pacing with a cycle length of 460 ms during sinus rhythm. The earliest atrial activation was marked by an asterisk on the ABL1-2 and its activation sequence was quite different from that over the AP as shown in **Figure 2-C**. The ABL1-2 was positioned at 2 o'clock in the LAO view; furthermore, judging from the amplitude ratio of atrial and ventricular potentials, the tip of the ABL catheter was considered to be positioned above the mitral valve. Figures with arrows indicate time intervals from the 2nd stimulus artifact to the onset of each atrial potential. **Panels B** and **C** reveal the initiation and termination of RV pacing with a cycle length of 260 ms during the tachycardia, respectively. The former shows that the 1st stimulus could reset atrial depolarization (As) and the latter shows that after the cessation of stimulation the tachycardia resumed with the "As-H-V" response. See text for details.

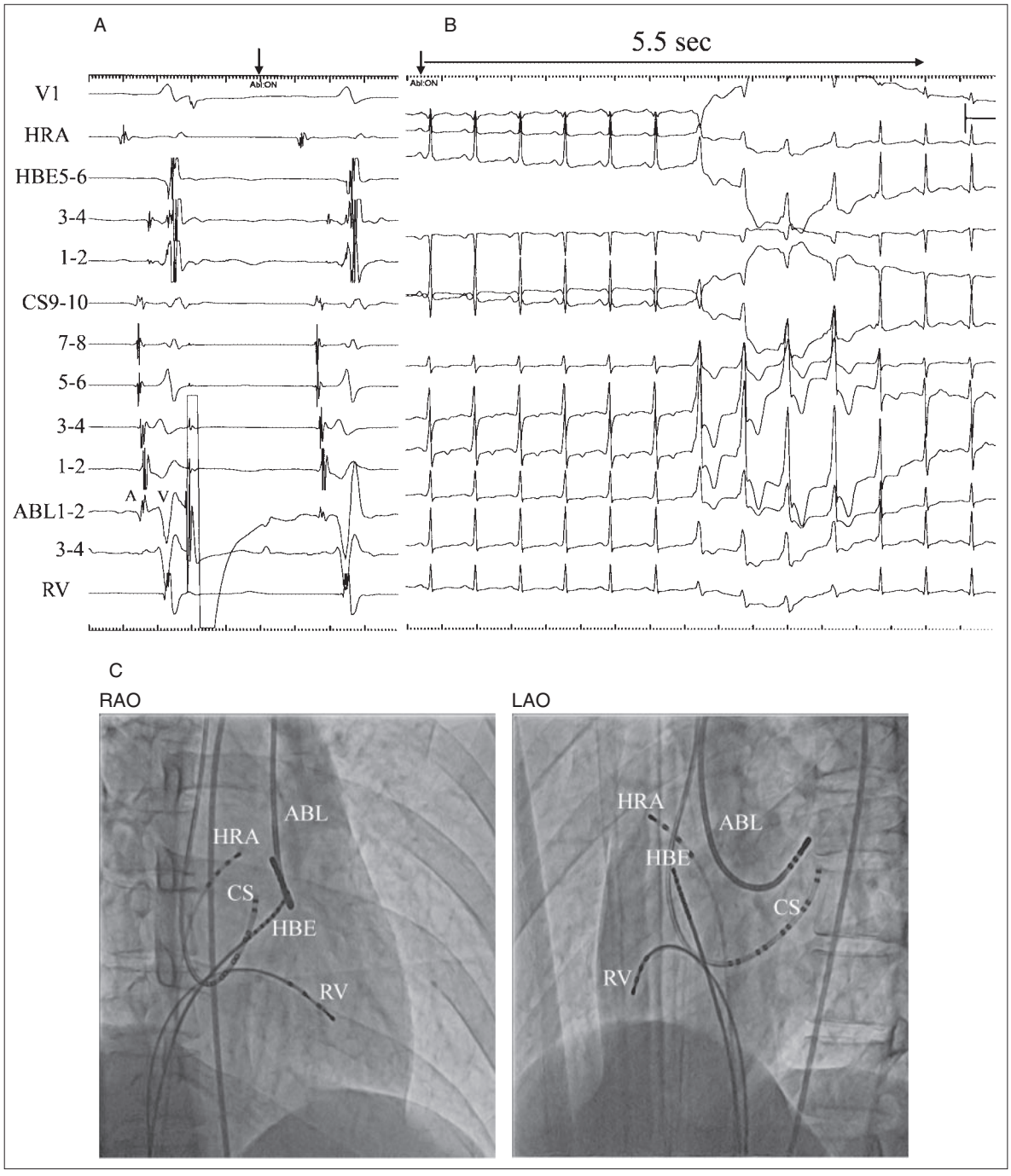

Figure 4 Successful ablation.
The downward arrow indicates the beginning of energy application. **Panel A** shows EPS records at the time of initiation of the successful ablation. Note that the atrial and ventricular potentials on the ABL1-2 electrogram are not fully fused as compared with the **Figures 2-D** and **E**. **Panel B** reveals a surface 12-lead ECG showing the time course from the initiation of the ablation to the disappearance of the delta wave, requiring 5.5 sec. **Panel C** shows fluoroscopic images of the successful ablation site, of which the left and right columns indicate 30 degrees right anterior oblique (RAO) and 60 degrees LAO views, respectively. Note that the tip of the ablative catheter is positioned at about 2:30 o'clock.

existed. It has been suggested that, in such a case, the atrial or ventricular insertion of the AP might have consisted of multiple components or had a broad-bundle shape.[7] Those cases frequently required repeated energy applications to eliminate the AP conduction. Second, judging from the successful

ablation site showing no fusion of atrial and ventricular potentials, the AP may have had a strong angulation with the AV borders. Third, only an antidromic AVRT was induced by programmed atrial pacing at baseline, whereas an orthodromic AVRT with a long RP interval was initiated by RV stimulation for the first time after multiple RF applications not resulting in complete elimination of anterograde conduction over the AP. Although it is difficult to explain why the latter tachycardia developed only after multiple ablations, one possible explanation is that the retrograde conduction property over the AP was more susceptible to heat injury than the anterograde one, and hence, such a de-novo tachycardia using the modified AP as a retrograde limb was provoked. Few reports have described an AP acquiring a decremental conduction property following RF ablation.[6,8] Shih-Ann Chen et al. reported that, among 166 APs in 142 patients, 5 pathways recurred with the appearance of decremental conduction properties in a mean follow-up period of 14 months.[8] These authors speculated the mechanism as follows. RF energy might have destroyed the major components of the fast-conducting AP fibers, or changed the geometry and conduction characteristics of viable fibers away from the lesion center, thus making conduction properties of the recurrent pathway like depressed fast conducting fibers or a slowly conducting AV-node-like tissue. Alternatively, RF energy may have destroyed most fibers in the AP network but spared a minor component or a few fibers with poorer, decremental conduction properties. From the cellular electrophysiological standpoint, it has been known that RF applications decrease the action potential amplitude, peak dV/dT and resting membrane potential along the border zone of the lesion, thus making conduction decremental.[9] As compared with their cases, a unique finding in our present case was that repeated RF energy applications had a significant effect on the retrograde conduction property of the AP with little effect on the anterograde one. Although the cause of such a discrepancy remains unknown, one possibility is that the AP in our case had already a different electrophysiological property between the anterograde and retrograde conduction at baseline and/or a complex geometry especially at the junctions with either the atrial or ventricular muscle, and nonspecific injury at these junctions caused by RF applications may have generated different degrees of impedance mismatch depending on three-dimensional interactions between the AP and the adjacent myocardium[10] or simply, numerous energy applications may have destroyed multiple components of the AP one after another. Whatever happened, such changes might have yielded the renovated atrial activation sequence different from the intrinsic VA conduction over the AP before ablation, resulting in an unexpected development of orthodromic AVRT only after repeated applications of RF energy. An alternative explanation is that the AP had longitudinal dissociation similar to dual AV node pathway physiology, or there was another AP having a ventricular junction adjacent to that of the main AP and a retrograde decremental conduction property before ablation. The former possibility could be ruled out since the atrial end of one retrograde pathway and that of the other were quite different. Although the latter possibility cannot be excluded completely, it is less likely since entrainment pacing applied at the RV during the antidromic AVRT did not unveil the presence of any retrograde conduction pathway other than the normal AVN-His Purkinje system (not shown) and no anterograde conduction over the main AP played a role as a bystander during the long RP tachycardia at all. Possible limitations of the present study include the following. (1) Unipolar electrograms were not recorded by the ablative catheter to detect subtle differences in the atrial and ventricular activation process. (2) Changes in the delta wave morphology following each RF application were evaluated only during sinus rhythm, and not during burst atrial pacing to provoke maximal preexcitation. Hence, subtle changes in the delta wave after ablation might have been missed. (3) Recording a clear atrial potential on the HBE electrogram throughout the procedure was absent. However, this would have little effect on the main focus of this paper, because the AP existed in the anterolateral wall of the LV far from the HBE. (4) No assessment associated with a VA conduction was achieved by para-His pacing before and after ablation. However, in such an AP localized at a LV free wall or with a decremental conduction property as in this case, the pacing method might have had little effect.[11]

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