Successful tricuspid annulus ablation for adenosine sensitive atrial tachycardia originating from the mitral annulus.

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Abstract

A 57-year-old man with repetitive long R–P supraventricular tachycardia (SVT) was referred for radiofrequency catheter ablation. SVT was sensitive to adenosine triphosphate, was not linked between atrial and ventricular activation, and did not terminate with the ventricular response; thus, we speculated reentrant atrial tachycardia (AT). Although the mitral annulus (MA) was the earliest atrial activation site (EAAS; 5 o'clock), AT was ablated not at the EAAS, but at the tricuspid annulus (TA; 4 o'clock), 24 ms later than the EAAS. We suggest that preferential conduction from the TA to the EAAS of the MA may be involved in AT.

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Abstract : A 57-year-old man with repetitive long R–P supraventricular tachycardia (SVT) was referred for radiofrequency catheter ablation. SVT was sensitive to adenosine triphosphate, was not linked between atrial and ventricular activation, and did not terminate with the ventricular response; thus, we speculated reentrant atrial tachycardia (AT). Although the mitral annulus (MA) was the earliest atrial activation site (EAAS; 5 o'clock), AT was ablated not at the EAAS, but at the tricuspid annulus (TA; 4 o'clock), 24ms later than the EAAS. We suggest that preferential conduction from the TA to the EAAS of the MA may be involved in AT.

Keywords : atrioventricular annulus atrial tachycardia, adenosine triphosphate sensitive, radiofrequency ablation, coronary sinus musculature, preferential conduction

Introduction : Atrial tachycardia (AT) at the atrioventricular annulus (AVA) is reentrant and sensitive to adenosine triphosphate (ATP).[1] AT is usually ablated at the earliest atrial activation site (EAAS); however, AVA-AT can be ablated at the ipsilateral annulus far from the EAAS.[2] We report AT originating from the mitral annulus (MA) ablated at the tricuspid annulus (TA).

Case : A 57-year-old man with repetitive supraventricular tachycardia (SVT) was referred to our institution. P-wave morphology was negative in leads II, III, aVF, and V6, and positive in lead V1 (Figure 1A). After obtaining written informed consent, catheter ablation was performed.

Two decapolar and two quadripolar electrode catheters were positioned at the coronary sinus (CS), His bundle, high right atrium (HRA), and right ventricular apex, respectively. Programmed stimulation induced SVT (Figure 1B), which was sustained during isoproterenol infusion. Tachycardia cycle length (TCL) fluctuated from 370 to 390ms. The EAAS was CS7/8 (MA 5 o'clock). The ventricular response was sometimes lost without tachycardia termination. Dual atrioventricular (AV) nodal physiology and ventriculoatrial (VA) conduction were not observed. VA linking was not demonstrated in the HRA (288ms), distal CS (254ms), or proximal CS (CSp; 218ms) by 360ms pacing; thus, AT originating from the MA was suspected. Moreover, each return cycle after a single extra-stimulus from the HRA at induction was longer than AV decremental conduction delay. SVT was terminated by ATP (5mg bolus) with a prolonged TCL; however, the atrio-his interval was unchanged, suggesting ATP-sensitive reentrant AT.

Left atrium (LA) was mapped using the CARTO® system (Biosense Webster, Diamond Bar, CA, USA) with PentaRay® catheter (Biosense Webster). The EAAS was the MA (5 o'clock). Local activation of the ablation catheter was 8ms delay at the earliest MA site and was just simultaneous at the inside CS (Figure 2A and 2B), but SVT was not terminated for Radiofrequency (RF) ablation at these sites. Then, the RA around CS ostium was mapped. The earliest TA activation site (4 o'clock) was 24ms later than the EAAS (Figure 2C); however, AT terminated 1.9s after RF delivery (Figure 2D). After ablation, AT did not recur over 1 year. CARTO and fluoroscopic views at the successful site are shown in Figure 2E. This site was far from the CS ostium. The distance from the EAAS of MA to the successful site was 27.9mm.

Discussion : This reentrant AT had slow conduction with a decremental property resembling the AV node, and was ablated at the TA. We speculated the mechanism of the AT.

Horie et al. reported 3 of 7 cases of ATP-sensitive AT from CSp were ablated not the EAAS but the slow pathway area.[3] These cases may have had reentrant circuit involved perinodal tissue around rightward/leftward posterior AV nodal extensions, and after the EAAS ablation, CL was prolonged and the earliest site was changed to the CS ostium. However, our case was not observed.

The mechanism of the AT was revealed by the post-pacing interval (PPI). The PPI from CSp was similar to TCL and atrial activation sequence during pacing resembled AT, but the PPI from EAAS was not (Figures 3A,3B). The CSp was close to the exit of reentrant circuit. Furthermore, the success site had a 24ms delay from the EAAS. PPI was not evaluated at the success site; however, this site may be the entrance of slow conduction in the circuit and the circuit extended along posterior TA to the CSp. Moreover, Chauvin et al. described the anatomical/histological features of inferior RA to LA myocardial striated muscle connections around the CS.[4] We speculate that the reentrant circuit was connected between the reentrant circuit and the EAAS by CS musculature, leading to preferential conduction (Figure 3C).

In conclusion, ATP-sensitive AT originating from MA was successfully ablated at the TA. TA ablation around the CSp should be considered when ablation at MA and inside CS is ineffective.

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Figure legends

Figure 1 . A: Twelve-lead ECG. B: Intracardiac electrogram during SVT without isoproterenol. The earliest atrial activation site (EAAS) was CS 7/8 (black arrow). HRA: high right atrium, His: His-recording site, CS: coronary sinus, RVA: right ventricular apex, EAAS: earliest atrial activation site.

Figure 2. A, B: Intracardiac electrograms and fluoroscopic views at unsuccessful site. A: Mitral annulus. Focal activation was 8ms delay from earliest atrial activation site (EAAS). B: Inside CS. Focal activation was simultaneous from EAAS. C, D: Intracardiac electrogram at successful tricuspid annulus site; C: Before ablation. Focal activation was 24ms delay from EAAS. D: Ablation. Atrial tachycardia terminated 1.9s. E: Activation map and fluoroscopic view at the successful site. The success site was far from the EAAS. HRA: high right atrium, His: His-recording site, Nav: ablation catheter electrogram, CS: coronary sinus, RVA: right ventricular apex, EAAS: earliest atrial activation site, ABL: ablation catheter, AP: anteroposterior.

Figure 3 . A, B: Post-pacing interval (PPI) at the CS proximal (CSp) and the earliest atrial activation site (EAAS). A: PPI at CSp was similar to tachycardia cycle length (TCL), and intra-atrial activation sequence resembled atrial tachycardia. The last pace was orthodromic conduction for CS7/8 (red dashed arrow). B: PPI at EAAS. PPI was not similar to TCL and the last pace was antidromic (blue dashed arrows). C: Presumed reentrant circuit and preferential conduction. HRA: high right atrium, His: His-recording site, CS(M): coronary sinus (musculature), RVA: right ventricular apex, PENT: PentaRay® catheter, LAO: left anterior oblique, EAAS: earliest atrial activation site, CSp/d: proximal/distal CS, SCA: slow conduction area, PPI: post-pacing interval, TA: tricuspid annulus.

Figure (Figure 1: top, Figure 2: middle, Figure 3: bottom)