Multiple Shifts of the Earliest Retrograde Atrial Activation Site Along the Tricuspid Annulus During the Fast-Slow Form of Atrioventricular Nodal Reentrant Tachycardia by Radiofrequency Modification

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Summary
A 70-year-old woman was admitted for treatment of supraventricular tachycardia. Ventriculoatrial conduction was revealed through programmed ventricular stimulation; the coronary sinus ostium (CSos) was the earliest atrial activation site. The fast-slow forms of atrioventricular nodal reentrant tachycardia (AVNRT) were induced by ventricular extra-stimuli. During tachycardia, the earliest atrial activation site was located at the bottom of CSos. Radiofrequency (RF) energy application to this site resulted in the delay of local electrical potential, prolongation of tachycardia cycle length, and a shift of the earliest retrograde activation site to the roof of CSos. Subsequent ablation induced a similar shift to the inferior tricuspid annulus and to the right posterior septum. Finally, RF energy application to the right posterior septum resulted in the termination of tachycardia, which was not induced afterward. Multiple shifts in the earliest retrograde atrial activation site along the tricuspid annulus after each slow pathway ablation suggested that annular tissue plays a substantial role as a substrate for AVNRT.

Key words: Atrioventricular ring, Supraventricular tachycardia, Radiofrequency catheter ablation

Radiofrequency catheter ablation (RFCA) of the slow pathway (SP) is an established treatment for atrioventricular nodal reentrant tachycardia (AVNRT). In most cases with slow-fast and fast-slow forms of AVNRT, modification of SP conduction, through RF energy application, to the right posterior septum results in successful tachycardia elimination. However, ablation in the region was sometimes ineffective; hence, other approaches were considered due to SP’s spatial heterogeneity. Several previous reports demonstrated the potential existence of anatomically different multiple SPs, as well as successful ablation sites, such as para-septal regions around the triangle of Koch or the left atrial mitral annulus along the coronary sinus. However, retrograde SP bound by the tricuspid annulus has not yet been described adequately.

We report a case with the fast-slow form of AVNRT involving four retrograde SPs, one of which was located in the inferior tricuspid annulus. The earliest retrograde atrial activation site has shifted three times along the tricuspid annulus with prolongation of the tachycardia cycle length (TCL) after each SP ablation. This indicated that annular tissue plays an important role as a substrate for AVNRT.

Case Report
A 70-year-old woman without any significant past illnesses was referred for RFCA of tachycardia with regular wide QRS complex. She had been suffering from paroxysmal palpitations for the past three years, with an increased frequency of about two or three episodes per month. Results of the chest X-ray film, echocardiogram, and laboratory data were all normal. The 12-lead electrocardiogram (ECG) revealed normal sinus rhythm without any ventricular pre-excitation or bundle branch block at rest. However, there was left bundle branch block QRS morphology without discernible P waves during tachycardia (Figure 1A).

An electrophysiology study was performed after obtaining written informed consent. All antiarrhythmic drugs were discontinued a week before the procedure. Under local anesthesia, four electrode catheters were inserted into the femoral and brachial veins and positioned in the right
atrial appendage, His bundle region, coronary sinus (CS), and right ventricular apex. Atrial and ventricular extra-stimulus pacing showed decremental conduction. An abrupt increase of conduction time of ≥50 ms, referred to as jump-up, in response to a 10-ms decrement in the extra-stimulus coupling interval was not identified. During ventricular pacing, the CS ostium (CSos) was the earliest atrial activation site. Tachycardia was not induced by an atrial extra-stimulus, but it was reproducibly induced by ventricular extra-stimuli without evidence of jump-up (basic cycle length [BCL] 700 ms, S1S2 430 ms) with a V-A-V sequence (Figure 1B). During tachycardia, there is a change in QRS complex morphology with the same atrial activation sequence and TCL. Clinical wide QRS tachycardia with the left bundle branch block pattern was also observed. Continuation of tachycardia with 2:1 AV block posed atrial tachycardia (AT) or AVNRT as its differential diagnosis (Figure 1B).

The electrophysiology study also revealed an atrial-His (A-H) interval of 120 ms, His-atrial (H-A) interval of 197 ms, and TCL of 317 ms during tachycardia. Failure to reset tachycardia was observed by a ventricular extra-stimulus, delivered when the His bundle was refractory. Without TCL oscillation, VA intervals changed during tachycardia along with the development of bundle branch block. Aberrant ventricular conduction with a left bundle branch block configuration resulted in a shortened VA interval, whereas that with a right bundle branch block configuration did not (Figure 1C, Supplemental Figure). Attempts to entrain tachycardia through repeated ventricular pacing were also unsuccessful as it terminated reproducibly. Differential atrial overdrive pacing with CS elec-

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**Figure 1.** Electrocardiogram (ECG). A: Surface 12-lead ECGs during the sinus rhythm (left) and tachycardia (right). B: Intracardiac ECG during the induction of the fast-slow form of AVNRT. Long RP tachycardia was induced by a single, right ventricular apical extra-stimulus (BCL, 700 ms; S1-S2, 430 ms). The first two paced stimuli (S1 and S2) captured the atria with a long VA interval (254 ms) and the earliest atrial activation site at the electrogram of coronary sinus (CS) ostium (CS 9-10), consistent with retrograde conduction over a slow pathway. The dashed Vertical lines mark the earliest atrial activation on the CS ostium. Despite the changes in QRS morphology and ventricular rate over time, the atrial cycle length and activation sequence remained the same, suggesting that the tachycardia circuit was confined to the supraventricular region. I, II, V1, and V5 represent surface ECG: HIS D and P represent distal to proximal His bundle electrogram, and CS 1-2 to 9-10 represent distal to proximal CS recordings. BCL indicates basic cycle length; CS, coronary sinus; HIS, His bundle; HRA, high right atrium; and RV, right ventricle. C: Comparison of intracardiac ECG during tachycardia with different QRS morphologies. The VA interval variability was associated with QRS morphology with constant atrial tachycardia cycle length (317 ms). The development of the left bundle branch block resulted in VA interval changes, suggesting that the main conduction pathway between His and ventricular myocardium is the left bundle (Supplemental Figure). These findings strongly indicated that the VA interval was more strongly associated with infra-Hisian aberrant conduction below the turnaround point than decremental conductivities of the retrograde slow pathways. RBBB indicates right bundle branch block; and LBBB, left bundle branch block.
trodites, located proximally, distally, and at the high right atrium during the tachycardia, revealed the maximal difference in the post-pacing AV intervals was 8 ms, suggesting the presence of VA linking, leading to the diagnosis of AVNRT.9,10 The atrial activation sequence during tachycardia was identical to that during the ventricular pacing, and activation mapping using an ablation catheter revealed the earliest atrial activation site, which is located at the bottom of CS ostium. Based on these findings, this tachycardia was diagnosed as a fast-slow form of AVNRT.

RF energy was first applied at the bottom of CS ostium (Figure 2, site 1), where the earliest atrial activation with local fragmented potential was recorded during tachycardia using an irrigated-tip catheter (Navistar Thermocoil, Biosense Webster) with a maximal temperature of 42°C and maximal power output of 20 W. Afterward, a delay of local electrical activation was observed along with a formation of double potentials and the TCL was prolonged from 317 to 321 ms. However, tachycardia persisted, and the earliest retrograde atrial activation site shifted to the roof of the CS ostium (Figure 2, site 2). RF energy application to this site further prolonged the TCL from 321 to 344 ms, accompanied by delayed activation of the atrial electrogram at the ablation site.

Subsequent activation mapping demonstrated a shift in the earliest retrograde activation site to the inferior tricuspid annulus (Figure 2B). Due to its distant location from the para-septal lesion, a differential ventricular overdrive pacing was performed to differentiate atrioventricular reciprocating tachycardia, or AT, from AVNRT (Figure 3). The V-A-V response, the PPI-TCL (TCL subtracted from PPI, which was over 115 ms), and cPPI-TCL (corrected for decrement in AV nodal conduction) were exhibited. Based on the measurement from the pacing stimulus to the consistent atrial electrocardiogram, the SA interval was longer in the basal region, which was nearer to the earliest activation site compared to the apex region (basal, 332 ms; apex, 306 ms). In addition, the difference in the shortest local AV interval of CS during ventricular entrainment and during tachycardia was more than 48 ms. The results were consistent with AVNRT.9,10,12 RF energy application to the site repeatedly prolonged the TCL from 344 to 355 ms with delayed activation of the atrial electrocardiogram at the ablation site (Figure 2, site 3).

Due to the presence of persisting tachycardia, a need arises to search for the earliest atrial activation site, which was located in the posterior septum region with continuous fragmented potential (Figure 2, site 4). Application of low RF energy to the site by 10 W led to the termination of tachycardia, and a gradual titration increase in the RF power level to 30 W was performed. An electrophysiology study subsequently revealed the presence of intermittent VA conduction with a long effective refractory period of 700 ms, but it demonstrated neither echo beat nor antegrade and retrograde dual pathways. The patient was then discharged without antiarrhythmic drugs and was free from palpitation thereafter.

Discussion

This case presented multiple forms of fast-slow AVNRT using four different retrograde SPs, all of which are located along the tricuspid annulus. The RF energy application for each SP resulted in the delay of local electrical potential and a shift in the earliest atrial activation site accompanied by prolongation of TCL. The prolongation was a result of increased VA interval, which reflects the prolongation of retrograde conduction time among different retrograde SPs, suggesting a shift in the retrograde limb of tachycardia and transition of the tachycardia circuit. Autonomic tone changes may be a cause of the prolongation of TCL and VA intervals. However, immediate changes and consistent prolongation of TCL and VA intervals after each RF energy application suggested that the modification of retrograde SPs was a primary underlying mechanism. Of interest, after RF energy application to site 2, the third earliest SP was located in the inferior tricuspid annulus, which was distal from the para-septal region over the triangle of Koch. Thus, there were four anatomically different retrograde SPs with distinct conduction velocity in this case.

In fast-slow forms of AVNRT, the spatial heterogeneity of retrograde SP has been identified and investigated over the past few decades. Nawata, et al. categorized the fast-slow forms of AVNRT into posterior, middle, and anterior types according to the site of retrograde SP occurring within the triangle of Koch.13 Recent studies on a series of patients with anterior subtypes were subsequently confirmed to be the superior type of fast-slow AVNRT incorporating a superior SP located near the His bundle.14,15 Furthermore, atypical AVNRT with eccentric retrograde left-sided activation was identified following mapping over the triangle of Koch.16 In addition, several reports indicate multiple fast-slow forms of AVNRT may be associated with anatomically different SPs.17 Importantly, almost all of the atrial breakthrough sites in these fast-slow AVNRT cases were located along the septum and the annulus of mitral valves. In the present case, multiple shifts of the earliest atrial activation site were uniquely observed along the tricuspid valve.

Reportedly, inferior nodal extensions at the posteroseptal area are possible substrates for an SP in the tachycardial circuit of AVNRT.18 However, these extensions cannot solely explain the spatial heterogeneity of the atrial breakthrough site of the fast-slow AVNRT. Recent histological studies hypothesized the presence of specialized conduction tissues along the atrioventricular (AV) rings, which anatomically comprise the AV septum and annulus of the tricuspid and mitral valves. Rings of conduction tissue arise from inferior extensions of the AV node and pass both rightward and leftward to encircle the orifices of the tricuspid and mitral valves, finally reuniting to form an extensive retroaortic node.19 AV ring tissues are known to be embryologically derived from the AV canal, possessing gene expression profiles, and connexin isoforms similar to those possessed by the AV node.20 Calcium channel-dependent conduction properties along the AV ring produce electrical heterogeneity, such as slow and discontinuous conduction velocities, the dispersion of refractory period, and anisotropic distributions of intercellular resistance.21-23 Such functional tissue properties along the AV ring can cause waveform distortion, resulting in

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Figure 2. RF energy application sites and change in intracardiac ECG recordings. A: Fluoroscopic images of the ablation sites from right anterior oblique views. B: CARTO map and ablation points. A three-dimensional electroanatomic CARTO system was used for activation mapping and identification of ablation site. Atrial activation mapping using the CARTO system after ablation to site 2 is described. The depolarized wavefront revealed a centrifugal activation pattern from the inferior tricuspid annulus, located distally from the paraseptal region, which differentiates this type of tachycardia from atrial tachycardia or atrioventricular reciprocating tachycardia. The first ablation site (site 1) was illustrated in pink point, the second ablation site (site 2) in blue point, the third ablation site (site 3) was in red point, and the fourth ablation site (site 4) in yellow point. The white point showed the site where the His bundle potential was recorded. C: Change in intracardiac ECG recordings by each ablation. The change in intracardiac ECG recordings before and after each ablation is illustrated. The perpendicular red arrow marks local electrocardiogram recorded on the mapping catheter. Ablation for the earliest activation site resulted in the delay of local electrocardiographic potential and prolongation of tachycardia cycle length with a gradual increase in VA conduction time.
the generation of various conduction pathways between inferior extensions and atrial surface, which can act as a spatial heterogeneity mechanism of SPs in fast-slow AVNRT. In previous fast-slow AVNRT cases, the left side of the AV ring has been reported as the primary successful ablation site. The present case with multiple retrograde SPs bound by the tricuspid annulus provides an insight into the potential arrhythmogenicity of the right AV ring. Tissues lying along the entire AV ring could potentially function as an SP and a substrate of AVNRT.

Disclosure

Conflicts of interest: Masato Okada, Koji Tanaka, Yasuharu Matsunaga-Lee, Yuichi Ninomiya, Yuko Hirao, Taka-fumi Oka, Nobuaki Tanaka, Hiroyuki Inoue, Katsuomi Iwakura, Kenshi Fujii, and Koichi Inoue. None.

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Figure 3. Demonstration of AVNRT by differential ventricular overdrive pacing. The upper fluoroscopic image illustrates the catheter position during the ventricular overdrive pacing. The mapping catheter was located on the RV base near the earliest atrial activation site identified by CARTO mapping. The other electrographic catheter was located on RV apex away from the catheter located on RV base. RAO indicates right anterior oblique view; and RV, right ventricular.
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Supplemental Files
Supplementary Figure
Please see supplemental files; https://doi.org/10.1536/ihj.18-406