Electrophysiological Delineation of the Tachycardia Circuit in Atrioventricular Nodal Reentrant Tachycardia

Hiroshige Yamabe, MD; Ikuo Misumi, MD; Hironobu Fukushima, MD; Kazuhiro Ueno, MD; Yoshihiro Kimura, MD; Youichi Hokamura, MD

Background—The exact boundaries of the reentry circuit in atrioventricular nodal reentrant tachycardia (AVNRT) have not been convincingly defined.

Methods and Results—To define the tachycardia circuit, single extrastimuli were delivered during AVNRT to 8 sites of the right intra-atrial septum: 3 arbitrarily divided sites of the AV junction extending from the His bundle (HB) site to the coronary sinus ostium (CSOS) (sites S, M, and I) and the superior (S-CSOS), inferior (I-CSOS), posterior (P-CSOS), and posteroinferior (PI-CSOS) portions of the CSOS and the CSOS in 18 patients. The mean tachycardia cycle length (TCL) was 368±52 ms. Retrograde earliest atrial activation was observed at the HB site in all patients. The longest coupling intervals of single extrastimuli that reset AVNRT at sites S, M, I, I-CSOS, S-CSOS, P-CSOS, and PI-CSOS were 356±51, 356±51, 355±52, 357±51, 318±47, 305±53, 311±56, and 312±56 ms, respectively, and the following return cycles at these sites were 368±52, 368±53, 367±53, 367±53, 407±66, 431±73, 415±55, and 412±56 ms, respectively. The longest coupling intervals at sites S, M, I, and I-CSOS did not differ from each other and were longer than those at CSOS and S-, P-, and PI-CSOS (P<0.0001). The return cycles at sites S, M, I, and I-CSOS did not differ from the TCL, whereas those at CSOS and S-, P-, and PI-CSOS were longer than the TCL (P<0.0001). Conclusions—The perinodal atrium extending from the HB site to I-CSOS was involved in the tachycardia circuit. I-CSOS was thought to be the entrance of the slow pathway. (Circulation. 1999;100:621-627.)

Key Words: atrioventricular node ■ mapping ■ reentry

The prevailing clinical understanding of atrioventricular nodal reentrant tachycardia (AVNRT) is based on a model of dual AV node pathways that are functionally and anatomically distinct. However, the exact boundaries of the reentry circuit in AVNRT have not been convincingly defined. The purpose of the present study was to define the tachycardia circuit in AVNRT. Single extrastimulus testing is useful for determining the location of the pacing site relative to the reentry circuit. Using this diagnostic tool to identify the location of the reentry circuit, we examined whether the tachycardia involves the atrium in the circuit, in particular to determine the extent of the atrial myocardium involved in the reentry circuit in AVNRT.

Methods

Patients

Eighteen patients with typical AVNRT (7 men and 11 women; mean age 56 years, range 22 to 82 years) were included. All drugs were discontinued 3 days before the ablation procedure. All patients gave written informed consent. The protocol was approved by the hospital Human Research Committee. The inclusion criteria for patients were (1) induction of a sustained slow-fast form of AVNRT and (2) a stable tachycardia cycle length that varied by no more than 10 ms over 20 consecutive beats.

Electrophysiological Study

The study was performed with patients in a fasting, unsedated state. Three 6F quadripolar electrode catheters (USCI) were inserted percutaneously via the right femoral and subclavian veins and positioned in the His bundle (HB) region, right ventricular apex, and coronary sinus ostium (CSOS). One 7F large-tip (4-mm-long), deflectable quadripolar electrode catheter with a 2-mm interelectrode distance (Cordis Webster) was introduced into the right femoral vein and advanced to the right intra-atrial septum for atrial mapping, pacing, and ablation. Bipolar electrograms from the CSOS, HB region, right ventricular apex, and sequential right intra-atrial septum were filtered at 50 to 600 Hz and recorded along with the surface ECG (leads II and V1) with the use of a polygraph (RMC-2000, Nihon Kohden). The right atrium was paced at an output of 2 times the diastolic threshold and a pulse width of 2 ms with a cardiac stimulator (SEC-3102, Nihon Kohden). Dual AV nodal physiology was identified by an increment of ≥50 ms in the A1H2 interval in response to a decrement of 10 ms in the A2A3 interval during programmed atrial stimulation. AVNRT was defined by the previously published standard criteria.

Study Protocol

To define the retrograde atrial activation sequence during AVNRT, atrial mapping was performed at 8 sites of the right intra-atrial...
septum: 3 arbitrarily divided sites of the AV junction extending from the HB site to CSOS (sites S [superior], M [middle], and I [inferior third of atrioventricular junction of septal leaflet between HB region and CSOS, respectively]) and the superior (S-CSOS), inferior (I-CSOS), posterior (P-CSOS), and posteroinferior (PI-CSOS) portions of the CSOS and the CSOS (Figure 1).

Single extrastimuli were delivered to these 8 sites during AVNRT beginning with the tachycardia cycle length and decreasing by 10 ms until the next His potential was advanced. The catheter positions during pacing were checked by biplane fluoroscopy. The pacing protocol was performed at least twice at each site. The longest coupling interval of the single extrastimulus that reset the tachycardia and the following return cycle at each site were measured. Extrastimuli that advanced the atrial potential in the HB region were excluded from the analysis. When resetting was confirmed, the coupling interval of the single extrastimulus was not shortened further to perform the single extrastimulation during the same tachycardia. If the tachycardia was terminated, it was reinitiated and the stimulation protocol was performed again from the start.

Catheter Ablation
Radiofrequency (RF) energy was delivered by the RF energy generator (CAT 500, Central Inc) as a continuous, unmodulated sine waveform at 500 kHz in a unipolar mode between the tip of the ablation catheter and a large skin electrode placed under the patient’s back. Slow pathway ablation was instituted in a stepwise fashion. RF energy (20 W for 30 seconds at each site) was delivered along the tricuspid annulus, starting at the level of the PI-CSOS with the ablation catheter positioned to record an atrial-to-ventricular electrogram ratio ≤0.5. If tachycardia was induced after the energy application, the catheter tip was then advanced more superiorly to the next adjacent site in a stepwise fashion.

Statistics
Values are expressed as mean ± SD. Differences between electrophysiological parameters were analyzed with Student’s t test. A value of P < 0.05 was considered statistically significant.

Results
The slow-fast form of AVNRT was induced in association with typical antegrade AV duality (jump >50 ms in the AH interval with a 10-ms decrement in A1A2) in all patients. The mean tachycardia cycle length was 368 ± 52 ms.

Retrograde Atrial Activation Sequence During AVNRT
The retrograde atrial exit from the fast pathway during AVNRT was located at the HB site in all patients. The intra-atrial conduction intervals between the earliest atrial electrogram at the HB site and that at each mapping site are shown in Figure 2.

Response to Single Extrastimuli
In all patients, resetting of the AVNRT was observed at all sites. The longest coupling intervals of a single extrastimulus that reset the tachycardia at sites S, M, I, I-CSOS, CSOS, S-CSOS, P-CSOS, and PI-CSOS were 356 ± 51, 356 ± 51, 355 ± 52, 357 ± 51, 318 ± 47, 305 ± 53, 311 ± 56, and 312 ± 56 ms, respectively (Table 1). The longest coupling intervals at sites S, M, I, and I-CSOS were significantly longer than those at CSOS, S-CSOS, P-CSOS, and PI-CSOS (P < 0.0001) (Table 1). The return cycles at sites S, M, I, and I-CSOS were significantly shorter than those at CSOS, S-CSOS, P-CSOS, and PI-CSOS (P < 0.0001) (Table 2). The return cycles at sites S, M, I, and I-CSOS were significantly shorter than those at CSOS, S-CSOS, P-CSOS, and PI-CSOS (P < 0.0001) (Table 2). The return cycles at sites S, M, I, and I-CSOS were not different from the tachycardia cycle length, whereas those at CSOS, S-CSOS, P-CSOS, and PI-CSOS were longer than the tachycardia cycle length (P < 0.0001) (Table 2). Thus, sites S, M, I, and I-CSOS were concluded to be located within the tachycardia triangle of Koch. Small shaded circles show 8 sites of right intra-atrial septum where mapping and single extrastimulation were performed during AVNRT. FO indicates fossa ovalis; IVC, inferior vena cava; TT, tendon of Todaro; and TV, tricuspid valve.
circuit, whereas CSOS, S-CSOS, P-CSOS, and PI-CSOS were not. Because the retrograde atrial exit site was located at the HB site in all patients, atrial tissue extending from the HB site to I-CSOS was concluded to be involved in the tachycardia circuit. I-CSOS was considered to be the entrance of the slow pathway, because it was located most distally to the retrograde atrial exit site.

The tracings during resetting of the AVNRT in patient 5 are shown in Figures 3, 4, and 5. The extrastimulus delivered from site S with a coupling interval of 340 ms shortened the interval between His potentials before and after the extrastimulus to 340 ms, and the following return cycle was identical to the tachycardia cycle length (Figure 3A). A single extrastimulus delivered from site M with a coupling interval of 340 ms also reset the tachycardia, and the following return cycle was identical to the tachycardia cycle length (Figure 3B). Figure 4 shows the recordings during delivery of a single extrastimulus from I-CSOS. A single extrastimulus with a coupling interval of 340 ms reset the tachycardia, and the following return cycle was identical to the tachycardia cycle length (Figure 4A). When the coupling interval of the single extrastimulus was shortened to 320 ms, the interval between the His potentials before and after the extrastimulus shortened further to 330 ms (Figure 4B).

Figure 5 shows the recordings during delivery of a single extrastimulus from PI-CSOS. A single extrastimulus with a coupling interval of 340 ms was unable to reset the tachycardia (Figure 5A). Single extrastimuli with coupling intervals of 330, 320, and 310 ms were also unable to reset the tachycardia. The tachycardia was reset initially when the coupling interval was shortened to 300 ms; however, the return cycle after the extrastimulus was longer than the tachycardia cycle length (Figure 5B).

**Catheter Ablation**

RF ablation was successful in eliminating inducible AVNRT in all patients. The mean number of RF applications required for successful ablation was 3±3. The atrial-to-ventricular electrogram amplitude ratio at the successful ablation site was 0.2±0.1. RF energy application along the tricuspid annulus at the level of PI-CSOS was not efficacious in any of the patients. The successful ablation site was located at the level of I-CSOS in 14 patients (78%) and at the level of site I in 4 patients (22%). There were no complications associated with ablation.

**Discussion**

The exact anatomic tachycardia circuit in typical AVNRT has not been convincingly defined. Mendez and Moe,6 and more recently, Iinuma et al7 demonstrated that the perinodal tissue was a necessary link in the AV node reentrant circuit in their model. Alternatively, another experimental study provided evidence in favor of an intra-AV nodal reentry circuit.8 Also, some recent descriptions of the AV node have suggested that the slow and fast pathways exist within the AV node.9,10 Clinically, Josephson and Miller11 proposed the concept of intra-AV nodal reentry. However, several studies have confirmed that surgical procedures could cure AVNRT without significantly impairing AV nodal conduction.12,13 This suggests that cure is achieved by damaging the putative atrial part of the reentrant circuit.13 The involvement of the atrial tissue in the reentry circuit of AVNRT was also demonstrated.
electrophysiologically. Also, it was shown that RF energy application near the CSOS inferior to the AV node can selectively eliminate the slow pathway conduction. In addition, Keim et al demonstrated the location of the site of the antegrade slow pathway using ice mapping techniques. Histological findings in patients also have provided evidence that the AV node itself does not participate in the slow pathway conduction. These findings strongly suggest that the tissue inferior to the compact AV node is used in the reentry circuit of the AVNRT.

Atrial Myocardium Participated in the Reentry Circuit

Resetting of AVNRT by the atrial extrastimulus without advancing the atrial potential at the HB site indicates that the atrial myocardium participates in the tachycardia circuit in AVNRT. Lai et al reported that the single extrastimulus delivered from the posteroinferior interatrial septum during late diastole began to reset the AVNRT once the extrastimulus started to depolarize the intervening tissue. In the present study, the extrastimulus began to reset the AVNRT as soon as it was delivered during late diastole at sites S, M, I, and I-CSOS. Furthermore, the return cycles at these sites were almost uniformly identical to the tachycardia cycle length. These results strongly suggest that sites S, M, I, and I-CSOS are involved in the tachycardia circuit of the AVNRT.

Anatomy of the AV Nodal Region

Tawara first described a superior dense network of nodal tissue and an inferior, more open part of the AV node into which atrial bands gradually merged. The anatomic area in which the compact AV node is situated is quite complex. The compact AV node is surrounded by transitional cells whose structure and function are intermediate between those of atrial and compact nodal cells. The connections between atrial and transitional cells are so gradual that no clear anatomic demarcations can be detected. Thus, sites S, M, I, and I-CSOS, which were considered to be within the tachycardia circuit, may contain the transitional cells. If one considers the compact AV node and the surrounding transitional cells as a functional AV node unit, then the AV nodal reentry is confined to the AV node. Therefore, the disagreement about the presence or absence of an upper common pathway may be related to this definition of the anatomy.

Tachycardia Circuit in AVNRT

The present study demonstrated that the reentrant impulse during AVNRT initially exits into the atrium at the HB site, then propagates the atrial component, and finally enters the slow pathway at the I-CSOS. This circuit is consistent with the findings previously reported. In the study by Iinuma et al, the reentrant circuit included the so-called sinus septum, which would be equivalent to sites P- and S-CSOS where they were not part of the circuit in the present study. This may be a result of the difference in the anatomic structure between humans and rabbits. Also, it is possible that the reentrant circuit for single echo beats in the study by Iinuma et al is not the same as for sustained AVNRT.

Recently, Inoue and Becker reported the presence of a rightward inferior extension of the AV node and suggested that this extension may represent the anatomic substrate for the slow pathway. Their results strongly suggest that the

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Mean ± SD 368±52 368±52* 368±53* 367±53* 407±66‡ 431±73†‡ 415±55‡ 412±56‡

TCL indicates tachycardia cycle length. Values are ms.

*P<0.0001 vs CSOS, S-CSOS, P-CSOS, and PI-CSOS, respectively; †P<0.01 vs CSOS; ‡P<0.0001 vs TCL.
entrance of the slow pathway is anatomically defined and is usually located at the inferior margin of the CSOS. The findings of the present report are consistent with their anatomic findings. However, the ablation lesions of the slow pathway were found to be in the atrial myocardium in the cases in which histology was done. Thus, it remains debatable whether or not the destruction of the inferior extension of the AV node is required for the cure of AVNRT. Meanwhile, other substrates for the slow pathway have been suggested. An experimental study has demonstrated that AV nodal conduction is determined by discrete anteroseptal and posteroseptal atrionodal inputs that are associated with differential functional properties. Clinically, Markowitz et al suggested that successful slow pathway ablation is achieved by selective ablation of the posterior atrionodal input. McGuire et al, however, suggested that transitional, rather than atrial, tissues are the major constituents of the slow pathway. Also, several investigators proposed an anatomic expansion of the AV node, which included the transitional zone tissue, thus maintaining the intra-AV nodal concept of AVNRT.

The results of single extrastimulation revealed that I-CSOS was the entrance of the slow pathway. Indeed, RF energy application along the tricuspid annulus near the I-CSOS was successful in most patients. This result is consistent with those previously reported. However, a more superior energy application was required in 4 patients. This may depend on a difference in the width of the slow pathway along its course. Supposing that the tachycardia circuit is partially formed functionally, the width of the slow pathway at the entrance may be wider than that at the other more superior sites. Furthermore, the site of application of energy was not the atrium, where the extrastimulation was performed, but rather the site along the tricuspid annulus, where the atrial electrogram was smaller than the ventricular electrogram to avoid the risk of AV block. Thus, an application of energy to the entrance might not be sufficient.

Recently, a variety of retrograde atrial exits from the AV node has been reported. Although such a variant retrograde atrial activation was not observed in the present study, it is likely that in some patients the atrionodal inputs to the fast pathway may be anatomically displaced more inferiorly. Similarly, the presence of multiple antegrade slow AV nodal pathways has been reported. These observations suggest that dual AV nodal pathways may not exist as a discrete entity, but rather that the proximal AV node may include several pathways with varying electrophysiological properties.

**Conclusions**

This study shows that perinodal atrium extending from the HB region to I-CSOS is an integral limb of the reentry circuit in AVNRT and that the entrance of the slow pathway is situated at the I-CSOS.
AVNRT is physiological rather than anatomic.32 Because we without AVNRT, which suggests that the defect causing was no structural difference between patients with and the rest of the atrium. Second, a previous study showed that there entirely intranodal. It is possible that perinodal atrial tissue common pathway does not necessarily imply that the circuit the electrophysiological evidence suggestive of an upper possible that in some patients, the circuit is entirely intranodal of confinement of the reentry circuit to the AV node. It is First, our findings do not completely rule out the possibility Study Limitations

First, our findings do not completely rule out the possibility of confinement of the reentry circuit to the AV node. It is possible that in some patients, the circuit is entirely intranodal and that in others, it involves the perinodal atrium. Moreover, the electrophysiological evidence suggestive of an upper common pathway does not necessarily imply that the circuit is entirely intranodal. It is possible that perinodal atrial tissue is necessary to the circuit but becomes uncoupled from the rest of the atrium. Second, a previous study showed that there was no structural difference between patients with and without AVNRT, which suggests that the defect causing AVNRT is physiological rather than anatomic.32 Because we studied only AVNRT patients, we cannot determine the difference between patients with and without AVNRT. The physiological substrate that causes reentry in AVNRT, especially in relation to the anatomic structure, needs to be elucidated.

References

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