Demonstration of the Exact Anatomic Tachycardia Circuit in the Fast-Slow Form of Atrioventricular Nodal Reentrant Tachycardia

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Background—The tachycardia circuit in the fast-slow form of atrioventricular nodal reentrant tachycardia (FS-AVNRT) has not been convincingly defined.

Methods and Results—To define the tachycardia circuit, single extrastimuli were delivered during FS-AVNRT to 9 intra-atrial sites in 12 patients: the His bundle (HB) site; the superior portion of the HB site (S-HB); 3 arbitrarily divided sites on the AV junction extending from the HB site to the coronary sinus ostium (CSOS) (sites S, M, and I); the superior, posterior, and posteroinferior portions of the CSOS (S-CSOS, P-CSOS, and PI-CSOS, respectively); and the CSOS. The inferior portion of coronary sinus ostium (I-CSOS), at which the earliest retrograde activation was observed, was excluded. At each site, the longest coupling interval of the single extrastimulus that reset the tachycardia and the subsequent return cycle was measured. The mean tachycardia cycle length was 370±55 ms. The longest coupling intervals at sites S-HB, HB, S, M, I, CSOS, S-CSOS, P-CSOS, and PI-CSOS were 328±53, 360±55, 358±55, 358±54, 360±55, 338±56, 323±54, 331±56, and 321±58 ms, respectively, and the subsequent return cycles were 408±58, 371±55, 370±55, 372±56, 370±55, 396±56, 411±60, 405±58, and 412±59 ms, respectively. The longest coupling intervals at sites HB, S, M, and I were longer than those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS (P<0.0001). The return cycles at sites HB, S, M, and I did not differ 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).

Conclusions—The perinodal atrium extending from the HB site to the I-CSOS is an integral limb of the reentry circuit in FS-AVNRT. (Circulation. 2001;104:1268-1273.)

Key Words: atrioventricular node ■ mapping ■ reentry

The mechanism of atrioventricular nodal reentrant tachycardia (AVNRT) is based on a substrate with 2 functionally and anatomically distinct AV node pathways. Recent evidence suggests that the reentry circuit in AVNRT involves the perinodal atrium. Little is known, however, about the exact boundaries of the reentry circuit in the fast-slow (FS) form of AVNRT. The purpose of the present study was to define the tachycardia circuit in FS-AVNRT. Using the single extrastimulation method, we investigated whether or not the tachycardia involves the atrium in the circuit, and more specifically, the extent of atrial myocardium involved in the reentry circuit in FS-AVNRT.

Methods

Patients
A total of 12 patients with FS-AVNRT (6 men and 6 women; mean age 48 years, range 16 to 81 years) were included in the study. Written informed consent was obtained from each patient. The protocol was approved by the Hospital Human Research Committee.

The inclusion criterion for sustained FS-AVNRT was a stable tachycardia cycle length varying by no more than 10 ms over 20 consecutive beats.

Electrophysiological Study
Three 6F quadripolar electrode catheters (USCI) were percutaneously inserted and positioned in the His bundle (HB) region, right ventricular apex, and coronary sinus ostium (CSOS). Two 7F large-tip (4 mm in length), deflectable quadripolar electrode catheters with a 2-mm interelectrode distance (Cordis Webster) were percutaneously inserted 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 sites on the right intra-atrial septum were filtered between 50 and 600 Hz and recorded along with the surface ECG (leads II and V1) using 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 using a cardiac stimulator (SEC-3102, Nihon Kohden).

Dual AV nodal physiology was identified by a sudden jump (≥50 ms) in the AH or HA interval in response to the programmed atrial or ventricular extrastimulation. FS-AVNRT was diagnosed if the tachycardia was initiated without an antegrade jump in AH interval.
or if the VA interval during tachycardia was $>60$ ms and the HA interval was longer than the AH interval during tachycardia. Tachycardia incorporating a midseptal or paraseptal accessory pathway was excluded by showing (1) that tachycardia continued despite the occurrence of AV block (patients 1, 2, 4, and 11); (2) that the tachycardia interval was longer than the AH interval during tachycardia.

I-CSOS

Figure 1. Schematic drawing of the triangle of Koch. The small shaded circles show the 10 sites of the right intra-atrial septum where the mapping and single extrastimulation were performed. FO indicates fossa ovalis; IVC, inferior vena cava; TT, tendon of Todaro; and TV, tricuspid valve.

Study Protocol
To define the retrograde atrial activation sequence during FS-AVNRT, atrial mapping was performed at 10 sites on the right intra-atrial septum: the HB site; the superior portion of the HB site (S-HB); 3 equidistantly divided sites of the AV junction extending from the HB site to the CSOS (sites S, M, and I); the superior, inferior, posterior, and posteroinferior portions of the CSOS (S-CSOS, I-CSOS, P-CSOS, and PI-CSOS, respectively); and the CIROS (Figure 1). The position of this mapping catheter was determined by its relation to the HB and coronary sinus catheters in the right (30°) and left (60°) anterior oblique fluoroscopic views. After right atriography in a biplane view, the S-HB and PI-CSOS were formed during the same tachycardia in all patients. The catheter positions during pacing were checked by biplane fluoroscopy. The pacing protocol was performed at least twice at each site.

Catheter Ablation
Radiofrequency energy was delivered 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 using a radiofrequency energy generator (CABL-IT, Central Inc). Radiofrequency energy was delivered to the earliest site of retrograde atrial activation during retrograde conduction over the slow pathway. A current of 20 W for 30 seconds was delivered with the temperature limit set at 60°C.

Results
Retrograde slow pathway conduction with the earliest atrial activation at the I-CSOS was observed in all patients. In 7 patients, retrograde AV nodal duality accompanying the shift in the earliest retrograde atrial activation from the HB site to I-CSOS was demonstrated (patients 1 to 3, 6, 10, 11, and 12). Sustained FS-AVNRT was inducible by ventricular incremental or extrastimulus pacing in all patients. The mean AH and HA intervals during tachycardia were 109±31 and 261±69 ms, respectively, and the mean tachycardia cycle length was 370±55 ms.

Response to Single Extrastimulation
Single extrastimuli were delivered to 9 atrial sites excluding the I-CSOS. The longest coupling intervals of the single extrastimuli that reset the AVNRT at sites S-HB, HB, S, M, I, CSOS, was measured as the interval from the pacing artifact to the subsequent local electrogram. The single extrastimulation was performed during the same tachycardia in all patients. The catheter positions during pacing were checked by biplane fluoroscopy. The pacing protocol was performed at least twice at each site.

Statistics
Values are expressed as mean±SD. Differences between electrophysiological parameters were analyzed using the Student’s t test. P<0.05 was considered statistically significant.
S-CSOS, P-CSOS, and PI-CSOS were 328±53, 360±55, 358±55, 360±55, 338±56, 323±54, and 321±58 ms, respectively (Table). The longest coupling intervals at sites HB, S, M, and I did not differ from each other and were significantly longer than those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS \((P<0.0001)\) (Table). The difference between the tachycardia cycle length and the longest coupling interval at sites HB, S, M, and I did not differ from each other and were shorter than those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS \((P<0.0001)\) (Figure 3A).

The return cycles at sites S-HB, HB, S, M, I, CSOS, S-CSOS, P-CSOS, and PI-CSOS were 408±58, 371±55, 370±55, 372±56, 370±55, 396±56, 411±60, 405±58, and 412±59 ms, respectively (Table). The return cycles at sites HB, S, M, and I were significantly shorter than those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS \((P<0.0001)\) (Table). The return cycles at sites HB, S, M, and I did not differ from their respective tachycardia cycle lengths, whereas those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS were longer than the tachycardia cycle length \((P<0.0001)\) (Table). The difference between the return cycle and the tachycardia cycle length at sites HB, S, M, and I did not differ from each other and were shorter than those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS \((P<0.0001)\) (Figure 3B). Thus, sites HB, S, M, and I were concluded to be located within the tachycardia circuit, whereas S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS were not. Since the retrograde atrial exit site was located at the I-CSOS in all patients, atrial tissue extending from the HB site to I-CSOS was concluded to be involved in the tachycardia circuit.

The return cycles at sites S-HB, HB, S, M, I, CSOS, S-CSOS, P-CSOS, and PI-CSOS were 408±58, 371±55* 370±55* 372±56* 396±56† 411±60† 405±58† 412±59† ms, respectively (Table). The return cycles at sites HB, S, M, and I were significantly shorter than those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS \((P<0.0001)\) (Table). The return cycles at sites HB, S, M, and I did not differ from each other and were shorter than those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS \((P<0.0001)\) (Figure 3B).

The return cycles at sites S-HB, HB, S, M, I, CSOS, S-CSOS, P-CSOS, and PI-CSOS were 408±58, 371±55, 370±55, 372±56, 370±55, 396±56, 411±60, 405±58, and 412±59 ms, respectively (Table). The return cycles at sites HB, S, M, and I were significantly shorter than those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS \((P<0.0001)\) (Table). The return cycles at sites HB, S, M, and I did not differ from each other and were shorter than those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS \((P<0.0001)\) (Figure 3B). The return cycles at sites S-HB, HB, S, M, I, CSOS, S-CSOS, P-CSOS, and PI-CSOS were 408±58, 371±55, 370±55, 372±56, 370±55, 396±56, 411±60, 405±58, and 412±59 ms, respectively (Table). The return cycles at sites HB, S, M, and I were significantly shorter than those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS \((P<0.0001)\) (Table). The return cycles at sites HB, S, M, and I did not differ from each other and were shorter than those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS \((P<0.0001)\) (Figure 3B). The return cycles at sites S-HB, HB, S, M, I, CSOS, S-CSOS, P-CSOS, and PI-CSOS were 408±58, 371±55, 370±55, 372±56, 370±55, 396±56, 411±60, 405±58, and 412±59 ms, respectively (Table). The return cycles at sites HB, S, M, and I were significantly shorter than those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS \((P<0.0001)\) (Table). The return cycles at sites HB, S, M, and I did not differ from each other and were shorter than those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS \((P<0.0001)\) (Figure 3B). The return cycles at sites S-HB, HB, S, M, I, CSOS, S-CSOS, P-CSOS, and PI-CSOS were 408±58, 371±55, 370±55, 372±56, 370±55, 396±56, 411±60, 405±58, and 412±59 ms, respectively (Table). The return cycles at sites HB, S, M, and I were significantly shorter than those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS \((P<0.0001)\) (Table). The return cycles at sites HB, S, M, and I did not differ from each other and were shorter than those at S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS \((P<0.0001)\) (Figure 3B). Thus, sites HB, S, M, and I were concluded to be located within the tachycardia circuit, whereas S-HB, CSOS, S-CSOS, P-CSOS, and PI-CSOS were not. Since the retrograde atrial exit site was located at the I-CSOS in all patients, atrial tissue extending from the HB site to I-CSOS was concluded to be involved in the tachycardia circuit.

The tracings during resetting of the FS-AVNRT in patient 10 are shown in Figures 4 and 5. The extrastimulus delivered from site M with a coupling interval of 310 ms shortened the interval between His potentials preceding and following the extrastimulus to 310 ms, and the subsequent return cycle was identical to the tachycardia cycle length (Figure 4A). A single extrastimulus delivered from the HB site with a coupling interval of 310 ms also reset the tachycardia, and the subsequent return cycle was identical to the tachycardia cycle length (Figure 4B).
A single extrastimulus delivered from the S-HB site with a coupling interval of 310 ms was unable to reset the tachycardia (Figure 5A). Single extrastimuli with coupling intervals of 300 and 290 ms were also unable to reset the tachycardia. The tachycardia was reset initially when the coupling interval was shortened to 280 ms; however, the return cycle after the extrastimulus was longer than the tachycardia cycle length (Figure 5B).

Catheter Ablation
Radiofrequency energy application to the I-CSOS successfully eliminated the slow pathway conduction in all patients. The mean number of radiofrequency applications required for successful ablation was 2.61. There were no complications associated with the ablation.

Discussion
The precise tachycardia circuit in AV nodal reentry remains controversial. Mendez and Moe,12 and more recently, Iinuma et al13 demonstrated that the perinodal tissue was a necessary link in the AV node reentrant circuit in their models. Alternatively, observations made by other investigators support an intranodal location of the reentrant circuit.14,15 The underlying basis for the FS-AVNRT is a functional and anatomic duality of pathways in the confines of the triangle of Koch.6 A clinical study has confirmed that surgical procedures could cure the FS-AVNRT without significantly impairing AV nodal conduction.16 The participation of atrial tissue in the reentry circuit in the FS-AVNRT has also been suggested electrophysiologically.17 In addition, it has been shown that the slow pathway can be selectively eliminated by applying radiofrequency energy near the CSOS inferior to the AV node in the FS-AVNRT.18,19 These findings strongly suggest that the atrial tissue inferior to the compact AV node is used in the reentry circuit of the FS-AVNRT.

Atrial Myocardium Participated in the Reentry Circuit
Resetting of the FS-AVNRT by the atrial extrastimulus without capturing the earliest retrograde atrial potential indicates that the atrial myocardium participates in the tachycardia circuit. Nawata et al20 also reported that the FS-AVNRT was reset by atrial single extrastimuli. In the present study, the extrastimulus delivered during late diastole began to reset the FS-AVNRT, and the subsequent return cycles were almost uniformly identical to the tachycardia cycle length at 4 atrial sites (sites HB, S, M, and I). These results strongly suggest that the atrial myocardium extending from the HB site to the I-CSOS is involved in the tachycardia circuit. Furthermore, the HB site was considered to
be the entrance of the fast pathway because of these atrial sites, it was the site located most distally to I-CSOS.

Tachycardia Circuit in the FS-AVNRT

Sung et al\textsuperscript{21} demonstrated a different retrograde atrial activation sequence between the fast and slow pathway conduction. More recently, McGuire et al\textsuperscript{22} showed that 2 distinct septal activation sequences were present in typical and atypical AVNRT. The present study demonstrates that the reentrant impulse during the FS-AVNRT initially exits into the atrium at the I-CSOS, then propagates through the atrial component, and finally enters the fast pathway at the HB site. This circuit is consistent with the findings reported previously.

Recently, Yamabe et al\textsuperscript{4} demonstrated that the perinodal atrium extending from the HB region to I-CSOS was an integral limb of the reentry circuit in the slow-fast form of AVNRT. In the present study, it was shown that the same extent of atrial myocardium was also involved in the reentry circuit in the FS-AVNRT. This circuit is consistent with the findings reported previously.

Recently, Sorbera et al\textsuperscript{28} reported successful ablation of the left-sided slow pathway in the slow-fast form of AVNRT. Also, Hwang et al\textsuperscript{29} demonstrated atypical AV nodal reentry with eccentric retrograde left-sided activation. It is possible that the left-sided perinodal transitional cells could form an essential component of FS-AVNRT. Delivery of single extrastimulation to the left endocardial surface might provide more information about the tachycardia circuit.

The catheter positions were determined only fluoroscopically. Therefore, these positions might not be exactly the same during mapping and extrastimulation. To minimize the catheter movement, we recorded frequent fluoroscopic images and reviewed the electrogram characteristics during the acquisition of the data.

We concluded that the I-CSOS was within the tachycardia circuit on the basis of the fact that ablation at this site was successful. However, we did not perform single extrastimulation at the I-CSOS. Thus, it is possible that the I-CSOS was in close proximity to the circuit but outside the circuit.

Conclusions

Mapping of the right intra-atrial septum using a single extrastimulation method revealed that the perinodal atrial tissue extending from the HB site to the I-CSOS is an integral limb of the reentry circuit in the FS-AVNRT.

References


With regard to the substrates of the slow pathway, Meijler et al\textsuperscript{23} demonstrated discrete anteroseptal and posteroseptal atrionodal inputs. McGuire et al\textsuperscript{24} suggested that transitional tissues are the major constituents of the slow pathway. More recently, Inoue et al\textsuperscript{25} demonstrated the inferior extensions of the AV node in human hearts. In addition, it has been shown that the destruction of the inferior extensions of the AV node underlies successful slow pathway ablation.\textsuperscript{26} The findings of the present report, as well as previously reported results,\textsuperscript{21,22} are consistent with the anatomic findings of Inoue et al.\textsuperscript{25} In the present study, it was also shown that the exit site of the slow pathway was separated by the intervening atrial tissue from the entrance of the fast pathway during tachycardia. These findings suggest the presence of a discrete anatomic structure for the slow pathway, as suggested by Inoue et al.\textsuperscript{25}

Recently, heterogeneity of the fast and slow pathway conduction patterns in the FS-AVNRT was reported.\textsuperscript{20} Similarly, multiple forms and variant forms of AVNRT have been reported.\textsuperscript{10,11,18} Wu et al\textsuperscript{10} proposed that a double loop figure-8 reentry was the mechanism of multiple forms of AVNRT. McGuire et al\textsuperscript{11} suggested that multiple atrial exits of the AV nodal tissue formed the substrate for multiple forms of AVNRT. These observations suggest that dual AV nodal pathways may not exist as a discrete entity, but rather that the perinodal AV node may include several pathways with varying electrophysiological properties.\textsuperscript{27}


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