Heterogeneity of Anterograde Fast-Pathway and Retrograde Slow-Pathway Conduction Patterns in Patients With the Fast–Slow Form of Atrioventricular Nodal Reentrant Tachycardia: Electrophysiologic and Electrocardiographic Considerations

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Objectives. This study sought to define the electrophysiologic and electrocardiographic characteristics of fast–slow atrioventricular nodal reentrant tachycardia (AVNRT).

Background. In fast–slow AVNRT the retrograde slow pathway (SP) is located in the posterior septum, whereas the anterograde fast pathway (FP) is located in the anterior septum; however, exceptions may occur.

Methods. Twelve patients with fast–slow AVNRT were studied. To determine the location of the retrograde SP, atrial activation during AVNRT was examined while recording the electrograms from the low septal right atrium (LSRA) on the His bundle electrogram and the orifice of the coronary sinus (CS). Further, to investigate the location of the anterograde FP, single extrastimuli were delivered during AVNRT both from the high right atrium and the CS.

Results. The CS activation during AVNRT preceded the LSRA in six patients (posterior type); LSRA activation preceded the CS in three patients (anterior type), and in the remaining three both sites were activated simultaneously (middle type). In the anterior type, CS stimulation preexcited the His and the ventricle without capturing the LSRA electrogram (atrial dissociation between the CS and the LSRA), suggesting that the anterograde FP was located posterior to the retrograde SP. In the posterior and middle types, high right atrial stimulation demonstrated atrial dissociation, suggesting that the anterograde FP was located anterior to the SP. In the posterior and middle types, retrograde P waves in the inferior leads were deeply negative, whereas they were shallow in the anterior type.

Conclusions. Fast–slow AVNRT was able to be categorized into posterior, middle and anterior types according to the site of the retrograde SP. The anterior type AVNRT, where an anteriorly located SP is used in the retrograde direction and a posteriorly located FP in the anterograde direction, appears to represent an anatomical reversal of the posterior type which uses a posterior SP for retrograde and an anterior FP for anterograde conduction. Anterior type AVNRT should be considered in the differential diagnosis of long RP (RP > PR intervals) tachycardias with shallow negative P waves in the inferior leads.

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Two types of atrioventricular nodal reentrant tachycardia (AVNRT) have been defined on the basis of the surface electrocardiographic (ECG) patterns and electrophysiologic findings (1–3). During the common form of AV nodal reentry, a slow pathway (SP) is used in the anterograde direction and a fast pathway (FP) is operative in the retrograde direction (slow–fast AVNRT). In the uncommon form, the direction of impulse propagation within the reentrant circuit is reversed (fast–slow AVNRT). The underlying basis for both types of AVNRT is functional and anatomic duality of pathways in the confines of the triangle of Koch, with an FP located anterior to the compact AV node (anterior input) and a SP located in the posteroseptal right atrium (posterior input) (4). Thus, during fast–slow AVNRT, the impulse propagates down the FP in the anterior septum and up the SP in the posterior septum, with the earliest retrograde atrial activation occurring near the orifice of the coronary sinus (CS). Although slow–fast AVNRT has been studied extensively (1–3,5), relatively little is known of the fast–slow form. The purpose of this study was to define the electrophysiologic and ECG characteristics of fast–slow AVNRT. Specifically, our findings provide new electrophysiologic evidence that in some patients with fast–slow AVNRT, the anterogradely conducting FP is located posterior to the retrograde SP within the confines of the triangle of Koch,
which has previously been considered rare in fast-slow AV nodal reentry.

Methods

**Definition of terms.** Atrioventricular nodal reentrant tachycardia was defined using standard electrophysiologic criteria (2,5–7). Dual AV nodal pathway physiology was defined by a sudden increase in the atrium-His (AH) interval of $\geq 50$ ms with a 10-ms decrement in the extrastimulus coupling interval, or by a sudden jump in the AH interval during incremental atrial pacing. Fast-slow AVNRT was defined when the tachycardia was associated with a long RP and a short PR interval (8) (RP/PR >1), as well as with a long His-atrium (HA) and short AH interval (HA/AH >1).

**Classification of fast-slow AVNRT.** We categorized the fast-slow AVNRT as posterior, middle and anterior types depending on the site of the retrograde SP. The sites of the SP were determined on the basis of the temporal relationship between the electrogram of the low septal right atrium (LSRA) at the His bundle recording site and the CS orifice electrogram. Posterior type fast-slow AVNRT was defined when retrograde atrial activation in the CS orifice preceded the LSRA activation by $\geq 10$ ms, anterior type when the LSRA activation preceded the atrial activation in the CS orifice by $\geq 10$ ms and middle type when retrograde atrial activation was recorded simultaneously (time difference $\leq 5$ ms) in the region of the CS orifice and the His bundle recording site.

**Patients.** Between February 1977 and December 1993, 138 patients with AVNRT (52 men and 86 women) were studied in our electrophysiology laboratory. Twenty-three patients (17%), 11 men and 12 women with a mean age of 52 ± 14 years (range, 26 to 64 years), were found to have an inducible fast-slow AVNRT. Among those 23 patients, 12 patients were enrolled in this study, having satisfied the following inclusion criteria. First, both AV reentry using a slowly conducting posteroseptal accessory pathway and intraatrial reentry in the low atrial septum were excluded with certainty. It is well appreciated that definitive distinction of AV nodal reentry from these two reentry mechanisms is often difficult (see later). Second, during the induced tachycardia, intracardiac electrograms both from the LSRA site and the orifice of the CS (or proximal CS) were simultaneously recorded. Third, during a sustained episode of tachycardia single programmed atrial extrastimuli were delivered both from the high right atrium (HRA) near the right atrial appendage and from the proximal CS, approximately 1 to 2 cm from the orifice, to investigate the effects of extrastimulation on the tachycardia. The entire cycle of atrial diastole was scanned by programmed extrastimulation until atrial refractoriness was encountered. Seven more recent cases with fast-slow AVNRT (i.e., after December 1993) were excluded because the above stimulation protocol was not entirely performed in each of these patients. This was because, after the introduction of the catheter ablation technique into our laboratory, electrophysiologic procedures were mainly performed only for radiofrequency catheter ablation rather than for detailed analysis of the electrophysiologic phenomena. Eleven of the 12 patients had no evidence of structural heart disease and one patient (case 5) had dilated cardiomyopathy. The patient profiles and tachycardia characteristics are shown in Table 1.

**Electrophysiologic study.** Electrophysiologic studies were performed using standard catheterization techniques (9), after informed written consent was obtained. Antiarrhythmic medications were discontinued for at least five half-lives of the longest-acting drug. Three conventional, quadripolar or bipolar electrode catheters with an interelectrode distance of 5 to 10 mm were introduced percutaneously via the femoral vein and positioned at the HRA site, across the tricuspid valve for His bundle recording (bipolar electrode catheter) and at the right ventricular apex. The His bundle electrode catheter with an interelectrode distance of 5 to 10 mm presumably covers a relatively large area near the AV node. This appears to allow the His bundle catheter to record the earliest atrial activation site at the anterior septum during retrograde impulse conduction through the AV node. An additional quadripolar electrode catheter with an interelectrode distance of 5 to 10 mm was introduced via the right jugular vein and placed in the CS, approximately 1 to 2 cm from the orifice, with the proximal pair located near the CS orifice. The distal pair of electrodes on each quadripolar catheter was used for stimulation, while the proximal pair of electrodes was used for recording the local bipolar electrogram. The tracings from the surface ECG leads (usually I, II and V_{1}) and intracardiac electrograms, filtered at a band-pass of 50 to 500 Hz, were displayed on an oscilloscope and recorded on a Mingograf (Siemens-Elema, Solna, Sweden) or thermal recorder at a paper speed of 100 mm/s. Anterograde and retrograde AV nodal functions were assessed using both extrastimulus testing and the incremental pacing technique. Extrastimulus testing was generally performed at a basic drive cycle length of 600 ms. Incremental atrial or ventricular pacing was initiated at a cycle length (multiple of 100 ms) slightly shorter than the spontaneous sinus cycle length, and the pacing cycle length was progressively shortened until second-degree AV or ventriculoatrial block occurred. During the induced tachycardia, retrograde atrial activation was carefully determined while simultaneously recording the electrograms from the LSRA and the CS orifice (or proximal

**Abbreviations and Acronyms**

<table>
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<tr>
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<tr>
<td>AH</td>
<td>atrium-His</td>
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<tr>
<td>AV</td>
<td>atrioventricular</td>
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<td>AVNRT</td>
<td>atrioventricular nodal reentrant tachycardia</td>
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<td>CS</td>
<td>coronary sinus</td>
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<td>ECG</td>
<td>electrocardiographic</td>
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<td>FP</td>
<td>fast AV nodal pathway</td>
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<tr>
<td>HA</td>
<td>His-atrium</td>
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<tr>
<td>HRA</td>
<td>high right atrium</td>
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<td>LSRA</td>
<td>low septal right atrium</td>
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<td>SP</td>
<td>slow AV nodal pathway</td>
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CS). Single programmed atrial extrastimuli were delivered during a sustained episode of the tachycardia both from the HRA and the CS orifice, to determine the FP location (see following). Entrainment pacing study was not systematically performed.

**Determination of the location of the anterogradely conducting fast pathway.** The location of the retrograde FP or SP can be determined electrophysiologically by identifying the site of earliest atrial activation during retrograde impulse conduction (10). However, it generally is not feasible to determine the location of the anterograde FP or SP by electrophysiologic study. This is because the earliest “ventricular” activation consistently occurs at the His bundle recording site, regardless of whether the anterograde AV nodal conduction is via the anterior or posterior AV nodal pathway. Nonetheless, we hypothesized, in this study, that the location of anterogradely conducting FP during fast–slow AV nodal reentry can be determined electrophysiologically by the use of programmed atrial stimulation from the CS orifice and the HRA on the basis of the assumptions illustrated schematically in Figure 1. In the hypothetical model (Fig. 1-I), programmed extrastimulus from the HRA is delivered during the posterior type of fast–slow AV nodal reentry in which the retrograde SP is located in the anterior septum (Fig. 1-II). The posterior wavefront from the CS orifice enters the AV node via a posterior input and preexcites the His bundle electrogram without advancing the timing of the LSRA electrogram (atrial dissociation between the CS orifice and the LSRA site). This atrial dissociation during the anterior type AV nodal reentry again provides evidence that the anterograde FP is in fact located posterior—rather than more anterior—relative to the SP site in the anterior septum.

A typical example of determination of the location of an anterograde FP is shown in Figure 2 (case 8). In Figure 2 the LSRA electrogram precedes the CS orifice electrogram by 5 ms (i.e., middle type fast–slow AVNRT), indicating that the retrograde SP is located in the mid-septum. However, the location of the anterograde FP (either anterior or posterior to the SP) is unknown. To determine the FP location, single programmed extrastimuli were delivered anteriorly from the HRA (A), and posteriorly from the CS orifice (B) during the tachycardia. In (A), a single extrastimulus at a coupling interval of 225 ms is delivered from the HRA. The anterior wavefront from the extrastimulus penetrates the tachycardia circuit and advances the LSRA, His bundle and the ventricular electrograms, thereby resetting the tachycardia. However, as shown by the interrupted arrow, the CS orifice electrogram (A1) on the CS3-4 recording is not advanced by the extrastimulus, indicating dissociation of atrial activation between the CS orifice and the LSRA site; the CS orifice electrogram is being activated retrogradely by the previous tachycardia beat conducting up the SP. In (B), a single extrastimulus is delivered posteriorly from the CS orifice. The extrastimulus advances both the His bundle and the ventricle, again resetting the tachycardia, however, the electrograms from the CS orifice and the LSRA site are both advanced by the CS extrastimulus, denoting absence of atrial dissociation between those two recording sites (atrial dissociation during resetting was not

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<th>Nature of Tachycardia</th>
<th>Cycle Length* (ms)</th>
<th>AH (ms)</th>
<th>HA (ms)</th>
<th>(CSOS–LSRA)† Interval (ms)</th>
<th>Type of AVNRT‡</th>
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AH = atrium-His; AVNRT = atrioventricular nodal reentrant tachycardia; CSOS = orifice of coronary sinus; HA = His-atrium; LSRA = low septal right atrium. *Cycle length indicates values observed during programmed atrial extrastimulation of AVNRT. †Minus value of (CSOS–LSRA) indicates that the retrograde activation of LSRA is earlier than that of CSOS. ‡See the Definition of terms section.
demonstrable from the CS orifice although the whole atrial diastole was scanned). The atrial dissociation demonstrated by the HRA extrastimulation (A) suggests that an anterograde FP is located anterior to the retrograde SP. The hypothesis that the anterograde FP is located anterior to the retrograde SP may also be supported by the findings during extrastimulation from the CS orifice (B). If the anterograde FP were to be located more posterior to the retrograde SP, the posterior wavefront from the CS orifice could have reset the tachycardia without advancing the LSRA electrogram at the His bundle recording site, which was never observed during extrastimulation from the CS orifice.

Two types of supraventricular tachycardia can mimic fast-slow AV nodal reentry (11–13): 1) orthodromic AV reentrant tachycardia using a slowly conducting posteroseptal accessory AV pathway capable only of retrograde conduction (8,14); and 2) intraatrial reentrant tachycardia arising in the region of the low interatrial septum (11–13). These two tachycardias were excluded using the following criteria.

Exclusion of orthodromic AV reentrant tachycardia (Table 2). Atrioventricular reentry using a slowly conducting posteroseptal accessory pathway was excluded by one of the following criteria.

Figure 2. Programmed atrial extrastimulation from the HRA and the CS orifice during the middle type of AVNRT. (A) An HRA single extrastimulus is delivered during AVNRT with a stable tachycardia cycle length of 300 to 305 ms. (B) A single extrastimulus from the CS orifice is delivered during AVNRT with a stable tachycardia cycle length of 285 to 290 ms. A0, H0 and V0 = atrial, His bundle and ventricular electrograms of ongoing tachycardia beats before the extrastimulus (S); A1, H1 and V1 = atrial, His bundle and ventricular electrograms immediately after the extrastimulus; A2 and A3 = atrial electrograms following A1; V2 and V3 = ventricular electrograms following V1; CS 3-4 (1-2) = coronary sinus (CS) electrogram recorded by the proximal (distal) pair of electrodes of a quadripolar electrode catheter positioned at the CS orifice; HBE = His bundle electrogram; V1, II = ECG leads V1 and II. Paper speed is 100 mm/s. See text for details.
Mechanism is not due to atrial reentry because the initiation of ventricle (Fig. 3). This strongly suggests that the tachycardia associated with an increased electrogram interval between the termination time (10 patients). Retrograde induction was always with the demonstration of a prolonged ventriculoatrial conduc-
ting atrial activity (13) (cases 1, 3, 6, 9, 10 and 12). Fourth, retrograde induction of tachycardia by ventricular stimulation or rapid pacing (13) (nine patients). Second, the were not conducted to the atria during programmed extrastimulation during AV reentry when the His bundle was refractory (15,16). We recognize that programmed ventricular extrastimulation during AV reentry using a slowly conducting accessory pathway may fortuitously fail to preexcite the atria because of an exact compensatory delay of conduction in the retrograde limb of the circuit (17). Second, tachycardia continued despite the occurrence of second-degree AV block in 10 of the 12 patients (the exceptions being cases 4 and 8). This finding excludes the possibility of a retrogradely conducting accessory AV pathway being a component of the reentry circuit, providing the strongest evidence supporting the AV node as the site of reentry (18).

**Exclusion of atrial tachycardia.** Atrial tachycardia was excluded by a combination of the following criteria (11–13). First, tachycardia could be terminated by ventricular stimuli that were not conducted to the atria during programmed extrastimulation or rapid pacing (13) (nine patients). Second, the atrial activation sequence during ventricular pacing in sinus rhythm was identical to that during tachycardia (nine patients, cases 3, 6 and 11 were the exceptions). This finding could be consistent with intraatrial reentry only if the latter occurred close to the exit from the AV node (13). Third, when atrial extrastimuli were delivered during tachycardia so that LSRA activity was advanced, the subsequent HA interval remained constant, indicating that atrial activation was linked to the preceding His–AV nodal activation rather than to the preceding atrial activity (13) (cases 1, 3, 6, 9, 10 and 12). Fourth, retrograde induction of tachycardia by ventricular stimulation was preceded by a ventricle-atrium-ventricle sequence (18), with the demonstration of a prolonged ventriculoatrial conduction time (10 patients). Retrograde induction was always associated with an increased electrogram interval between the two consecutive atrial beats conducting retrogradely from the ventricle (Fig. 3). This strongly suggests that the tachycardia mechanism is not due to atrial reentry because the initiation of atrial reentry should be associated with a shortening rather than a lengthening of atrial coupling intervals between two consecutive atrial beats, as well as with an increased stimulus–atrium activation delay (2,19). In the remaining two patients (cases 6 and 11) tachycardia was induced only by atrial stimulation.

**Electrocardiographic study.** Twelve-lead ECGs during fast–slow AVNRT were obtained in all patients. The polarity and the depth of the retrograde P waves in the inferior leads during the tachycardia were analyzed. The P wave configuration during tachycardia was independently reviewed by two of the authors (H.N. and F.S.) without knowledge of the electrophysiologic data, and conflicting interpretations were resolved by consensus.

**Statistical analysis.** All values are expressed as mean ± SD. The electrophysiologic data among each type of fast–slow AVNRT were compared using the Kruskal–Wallis test. A p value <0.05 was considered statistically significant.

**Results**

**Tachycardia characteristics.** All patients had an inducible, sustained fast–slow form of AVNRT with a mean tachycardia cycle length of 385 ± 68 ms (range, 305 to 535 ms). The AH interval during tachycardia was 96 ± 30 ms (range, 45 to 155 ms) and the HA interval was 288 ± 78 ms (range, 200 to 440 ms). One patient (case 3) also had inducible orthodromic AV reentrant tachycardia using a slowly conducting accessory pathway. The posterior, middle and anterior types of fast–slow AVNRT were induced in six (cases 1 to 6), three (cases 7 to 9) and three patients (cases 10 to 12), respectively. Typical tracings of the three types of fast–slow AVNRT are shown in Figure 4. The mean tachycardia cycle lengths were 398 ± 73 ms, 342 ± 33 ms and 402 ± 85 ms, respectively, for posterior, middle and anterior types of AVNRT. The mean AH intervals were 77 ± 21 ms, 112 ± 8 ms and 120 ± 38 ms,

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<th>Patient No.</th>
<th>Reset by PVS During His Refractoriness</th>
<th>Site of AV Block</th>
<th>Termination by PVS Without Atrial Capture</th>
<th>Identical Atrial Sequence During Ventricular Pacing</th>
<th>Constant HA Interval While LSRA Electrogram Is Advanced by PAS</th>
<th>V-A-V Sequence During Tachycardia Induction</th>
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A = atrium; AHB = atrium-His block; AV = atrioventricular; HVB = His-ventricular block; LSRA = low septal right atrium; PAS = programmed atrial stimulation; PVS = programmed ventricular stimulation; V = ventricle; + = present; – = absent.
respectively, and the mean HA intervals, 321 ± 61 ms, 230 ± 30 ms and 282 ± 120 ms, respectively, for the posterior, middle and anterior types of AVNRT. The tachycardia cycle length and HA interval were similar among the three groups. The AH interval was significantly shorter in the posterior group as compared with the middle and the anterior groups (p = 0.045).

Tachycardia induction. Sustained fast–slow AVNRT was inducible by incremental atrial pacing in 10 patients (cases 1 to 5, 7 to 11), atrial extrastimulus testing in 10 patients (cases 1, 2, 4 to 10, 12), incremental ventricular pacing in 8 patients (cases 1, 2, 4, 7 to 10, 12) and ventricular extrastimulus testing in 6 patients (cases 1, 2, 8 to 10, 12). Nonsustained slow–fast

Figure 3. Induction of supraventricular tachycardia during right ventricular extrastimulus testing with a basic drive (S1S1) cycle length of 800 ms (case 2). (A) (S1S2 = 610 ms) The ventricular extrastimulus (S2) is conducted to the atria through an FP, with the earliest atrial activation being registered on the His bundle electrogram (HBE); the resultant A1A2 interval on the CS3-4 electrogram is 720 ms. (B) (S1S2 = 600 ms) The ventricular extrastimulus (S2) is conducted to the atria through an SP, with the earliest atrial activation being registered on the CS3-4 electrogram; the resultant A1A2 interval on the CS3-4 electrogram is 750 ms. Initiation of supraventricular tachycardia is associated with a ventricle-atrium-ventricle sequence and a prolonged A1A2 interval (720 ms → 750 ms). A1, A2, Ve1 and Ve2 = atrial, His bundle and ventricular electrograms during the induced supraventricular tachycardia. Other abbreviations as in Figure 2. Paper speed is 100 mm/s.

Figure 4. Typical tracings of intracardiac electrograms of three types of fast–slow AVNRT. In posterior type AVNRT, the CS3-4 (CS orifice) electrogram precedes the LSRA electrogram at the His bundle recording site by 20 ms. In the middle type, the LSRA electrogram precedes the CS3-4 electrogram by 5 ms (see the Definition of terms section). In the anterior type, the LSRA electrogram precedes the CS3-4 electrogram by 30 ms. Abbreviations as in Figure 2. Paper speed is 100 mm/s.
During sinus rhythm, anterograde and retrograde conduction. A form of AV nodal reentry (reversed form of reentry) was also induced in five patients (cases 1, 6, 8, 10, 12).

### Characteristics of atrioventricular and ventriculoatrial conduction.

During sinus rhythm, anterograde and retrograde AV nodal function were assessed using both extrastimulus testing and the incremental pacing technique.

**Atrioventricular conduction.** Anterograde SP conduction was manifested in seven patients (cases 1, 3, 5, 6, 8, 10, 12). In the remaining five patients neither a sudden increase nor a sudden jump in the AH interval was demonstrated.

**Ventriculoatrial conduction** (Table 3).

1. **Posterior type AVNRT** (cases 1 to 6): Four of the six patients exhibited a discontinuous ventriculoatrial conduction curve. During retrograde FP conduction, earliest activation occurred at the His bundle recording site, whereas activation of the CS orifice was earliest during retrograde SP conduction (Fig. 3). In the remaining two patients (cases 3, 6) intact retrograde conduction was not demonstrated in sinus rhythm (20), but was demonstrable during entrainment pacing of tachycardia from the ventricle.

2. **Middle type AVNRT** (cases 7 to 9): Two (cases 8 and 9) of the three patients exhibited a discontinuous ventriculoatrial conduction curve, consistent with retrograde dual AV nodal pathways. In case 7 retrograde conduction was only through an SP, with simultaneous retrograde activation at the LSRA and the CS orifice recording site. In case 8 right ventricular extrastimulus testing revealed a similar atrial activation sequence during FP and SP conduction, with the LSRA electrogram preceding the CS orifice electrogram by 15 ms; however, during tachycardia the LSRA electrogram preceded the CS orifice electrogram only by 5 ms, indicating that a different SP was used as the retrograde limb of the tachycardia circuit (i.e., retrograde triple AV nodal pathways). In case 9 the retrograde activation sequence was similar during FP and SP conduction, with the LSRA electrogram always preceding the CS orifice electrogram by 5 ms.

3. **Anterior type AVNRT** (cases 10 to 12): In two patients (cases 11 and 12) retrograde conduction during ventricular extrastimulus testing was only through the FP with a continuous ventriculoatrial conduction curve. During FP conduction the CS orifice electrogram preceded the LSRA electrogram by 10 ms in case 11, and in case 12 retrograde atrial activation occurred simultaneously at the LSRA and CS orifice recording sites. In case 12, retrograde SP conduction was demonstrated only by the use of rapid ventricular pacing in sinus rhythm, and during retrograde SP conduction the LSRA electrogram preceded the CS orifice electrogram by 30 ms. In case 10, retrograde conduction during ventricular extrastimulus testing was only through the SP, with the same atrial activation sequence as during the tachycardia.

### Determination of the location of the anterogradely conducting fast pathway.

During tachycardia, attempts were made to determine, electrophysiologically, the location of an anterogradely conducting FP by the use of programmed atrial stimulation from the anterior HRA and the CS orifice (in the manner as described in the Methods section [Figs. 1 and 2]).

Atrial dissociation during tachycardia between the CS orifice and the LSRA site was demonstrated in all 12 patients (Table 4). In six patients with posterior type (cases 1 to 6) and three patients with middle type (cases 7 to 9) fast–slow AVNRT, anterior HRA extrastimulation demonstrated atrial dissociation between the CS orifice and the LSRA site, denoting that the anterogradely conducting FP is located anterior to the retrograde SP in the confines of the triangle of Koch.

### Table 3. Retrograde AV Nodal Conduction Time During Ventricular Pacing Versus Tachycardia

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>(CSOS–LSRA)* Interval During Ventricular Pacing in Sinus Rhythm (ms)</th>
<th>(CSOS–LSRA)* Interval During AVNRT (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Via Fast Pathway</td>
<td>Via Slow Pathway</td>
</tr>
<tr>
<td>1</td>
<td>15</td>
<td>20</td>
</tr>
<tr>
<td>2</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>3</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>4</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>5</td>
<td>10</td>
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</tr>
<tr>
<td>6</td>
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<td>8</td>
<td>15</td>
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</tr>
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<td>9</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>10</td>
<td>NA</td>
<td>25</td>
</tr>
<tr>
<td>11</td>
<td>10</td>
<td>NA</td>
</tr>
<tr>
<td>12</td>
<td>0</td>
<td>30</td>
</tr>
</tbody>
</table>

AVNRT = ativoventricular nodal reentrant tachycardia; CSOS = orifice of coronary sinus; LSRA = low septal right atrium; NA = not available because of absent retrograde conduction. *Minus value of (CSOS–LSRA) indicates that the retrograde activation of LSRA is earlier than that of CSOS.

### Table 4. Demonstration of Atrial Dissociation Between the CS Orifice and the LSRA

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Tachycardia Resetting With Extrastimulation</th>
<th>Atrial Dissociation With Extrastimulation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>From HRA</td>
<td>From CSOS</td>
</tr>
<tr>
<td>1</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
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<td>6</td>
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<td>+</td>
<td>+</td>
</tr>
<tr>
<td>12</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

CSOS = orifice of the coronary sinus; HRA = high right atrium; LSRA = low septal right atrium. *HRA extrastimulation did not reset the tachycardia because of local atrial refractoriness.
contrast, in three patients with anterior type AVNRT (cases 10 to 12), extrastimulation from the CS orifice demonstrated atrial dissociation between the CS orifice and the LSRA site, indicating that the anterogradely conducting FP is located posterior to the retrograde SP.

P wave morphologies in the inferior leads. The polarity and the depth of the retrograde P waves in the inferior leads during fast–slow AVNRT were analyzed. Typical ECG tracings are shown in Figure 5. The retrograde P waves are deeply negative in patients with posterior and middle type AVNRT, whereas they are shallow in patients with anterior type AVNRT. The depth of the retrograde P waves in lead III differed significantly among the three groups, $-2.7 \pm 0.4$ mm, $-2.3 \pm 1.0$ mm and $-0.7 \pm 0.3$ mm, respectively, in the groups with posterior, middle and anterior types of AVNRT ($p = 0.039$).

Discussion

Major findings. The major findings of the present study were as follows. First, the fast–slow form of AVNRT could be categorized as posterior, middle and anterior types, according to the site of the retrograde SP within the confines of the triangle of Koch. Thus, we were able to identify the patients with fast–slow AVNRT in which an anteriorly located SP comprised the retrograde limb of the reentry circuit—a rare occurrence in AV nodal reentry. Second, using extrastimulation from the HRA and the CS orifice, we were able to present electrophysiologic evidence (i.e., atrial dissociation between the CS orifice and the LSRA site; see Fig. 2, A), suggesting that the anterogradely conducting FP during tachycardia was located anterior to the posteriorly located SP (posterior and middle type AVNRT), or posterior to the anteriorly located SP (anterior type AVNRT). Thus, anterior type AVNRT, where an anteriorly located SP is used in the retrograde direction and a posteriorly located FP is operative in the anterograde direction, is likely to represent an anatomic reversal of the posterior type AVNRT, which uses a posterior SP for retrograde and an anterior FP for anterograde conduction. Third, the retrograde P waves in the inferior leads were deeply negative in patients with posterior and middle type AVNRT, whereas they were shallow in patients with anterior type AVNRT.

Site of the fast and slow AV nodal pathways. Determination of the location of the retrograde AV nodal pathways. The location of the retrograde FP or SP can be determined electrophysiologically by identifying the site of earliest atrial activation during retrograde impulse conduction through the AV node (10). During retrograde FP conduction, earliest atrial excitation occurs near the His bundle recording site in the anterior septum, whereas during retrograde SP conduction earliest excitation is near the CS orifice in the posterior septum. Rare cases have been reported in which the site of earliest atrial excitation through the FP has been posteroinferior, close to the CS orifice (4,21).

Determination of the location of the anterograde AV nodal pathways. Unlike the retrograde AV nodal pathways, it is generally not feasible during clinical electrophysiologic study to determine the location (i.e., anterior or posterior) of the anterograde AV nodal pathways. Only surgical or ablative techniques seem to localize anterogradely conducting AV nodal pathways. In an animal study, McGuire et al. (22) demonstrated that during sinus rhythm in the normal heart, atrial impulses enter the AV node by way of the anterior atrionodal connections, because interruption of these connections prolonged the AH interval. This observation strongly suggests that the anterogradely functioning FP in sinus rhythm is located anterior to the compact AV node in the anterior septum. In humans with the slow–fast form of AVNRT, intraoperative ice mapping during tachycardia was able to localize discrete areas of the atrial septum critical to the function of the anterogradely conducting SP (23); cooling with a cryoprobe in the area posterior and inferior to the compact AV node along the tricuspid annulus terminated the tachycardia, with an atrial electrogram not followed by a reentrant ventricular electrogram. This finding strongly suggests that the anterogradely conducting SP during tachycardia is located in the posterior septum. It has also been shown that the sites of anterogradely conducting pathways can be determined after successful catheter ablation by identifying the site of successful FP or SP ablation (24).

In the current study we have proposed a hypothesis that the
site of an anterograde FP can be determined electrophysiologically by use of programmed atrial stimulation from the CS orifice and the HRA, based on the hypothetical model of the reentry circuit illustrated schematically in Figure 1. Although we believe that the analysis based on the hypothetical model in Figure 1 is valid, only a relative anatomic relation of the FP to the SP can be determined. For instance, the anterograde FP may be localized to a site which is “anterior” to the posterior SP site; however, the exact anatomic site (either in the mid septum or anterior septum) is unknown.

Configuration of the retrograde P waves during supraventricular tachycardia. There are certain morphologic characteristics of P waves which, if observed during supraventricular tachycardia, can aid in the diagnosis of arrhythmia (11,25,26).

In fast–slow AVNRT in which retrograde P waves are readily seen, P waves are usually associated with a midline, cuffed, atrial activation pattern; inverted negative P waves in the inferior leads, isoelectric P waves in lead I and nearly equal upright P waves in aVR and aVL leads (11). The present study has shown that retrograde P waves in the inferior leads were deeply negative in posterior and middle type AVNRT, whereas negative P waves were shallow in the anterior type (Fig. 5). Waldo et al. (27,28) have demonstrated in their experimental and human (intraoperative) studies that atrial endocardial pacing near the CS orifice was associated with deeply negative retrograde P waves, whereas pacing from an atrial site near the compact AV node was associated with positive P waves. Their studies suggest that retrograde P waves resulting from retrograde AV nodal conduction could be positive in the inferior leads. The results of this study are in agreement with more traditional electrocardiographic concepts that retrograde atrial activation through the AV node results in negative P waves in the inferior leads (29). Pacing from an atrial site near the compact AV node (27,28) may not exactly represent a true infranodal rhythm (30). In the latter rhythm, retrograde impulses exiting from the “entire” AV node and spreading to both atria may consistently result in inverted, negative P waves. Retrograde positive P waves in the inferior leads are exceedingly rare in clinical electrocardiography. Further study may be necessary in the setting of clinical 12-lead ECG recording to definitively determine whether positive or negative retrograde P waves will result from ventriculoatrial conduction through the AV node.

Study limitations. The present study has several limitations. First, electrograms from the region of Koch’s triangle were recorded simultaneously only from two atrial sites (i.e., the His bundle recording site and the CS orifice), and simple broad anatomic terms (anterior, middle and posterior) were used to define the anatomic sites of the atrial septum. More detailed endocardial mapping over the triangle of Koch or along the tendon of Todaro (31) may have yielded more information about the exact FP and SP location. However, unlike intraoperative computerized mapping (32), fluoroscopy cannot identify precise endocardial anatomy and, therefore, the presumed FP or SP location has only been inferential, even with the use of decapolar electrode catheters—an inherent limitation to clinical electrophysiologic study. In addition, mapping restricted over the right endocardial surface can only provide limited information about the excitation of the AV node and the atrial septum, which are three-dimensional subendocardial structures. McGuire et al. (22) postulated that the atrial exit site from the AV node may be at a distance from the right atrial endocardial surface. Subendocardial or left atrial endocardial recording may allow us to better define the excitation of these structures (32). Such an approach, however, may be infeasible with the conventional clinical electrophysiologic techniques. More recently, by use of a decapolar CS catheter, Hwang et al. (33) demonstrated atypical AV nodal reentry with eccentric retrograde left-sided activation in 6% of patients with AVNRT. It is possible that in these patients the left-sided perinodal transitional cells were not electrically silent, and could serve as an atrial input to the compact AV node, thus forming an essential component of AVNRT. The left-sided perinodal transitional cells, however, were not considered to be critically involved in AVNRT circuit (i.e., “bystander role”), because the SP ablation at the posterior aspects of the right atrial septum successfully eliminated the AVNRT, whereas ablation near the lateral CS leads that registered the earliest retrograde atrial activation failed to abolish the tachycardia (33). Thus, the role of left-sided perinodal transitional cells in AV nodal reentry still remains unclear.

Second, the results of radiofrequency ablation of AVNRT, if available, may provide some supportive evidence regarding the FP or SP location in the atrial septum; however, none of the studied patients was a candidate for radiofrequency ablation before December 1993.

Clinical implications. P wave configuration and polarity during supraventricular tachycardia may be of use to properly predict the tachycardia mechanism (AVNRT vs. AV reciprocating tachycardia) or the location of concealed accessory pathways (11,25,26). The present study has demonstrated that retrograde P waves in the inferior leads were deeply negative in patients with posterior and middle type AVNRT, whereas they were shallow in patients with anterior type AVNRT. Recognition of different morphologic characteristics of retrograde P waves between anterior versus posterior type AVNRT may help avoid incorrect estimation of possible ablation sites, thus minimizing the requisite time for endocardial mapping in patients with fast–slow AVNRT. Anterior type AVNRT, where an anteriorly located SP is used in the retrograde direction and a posteriorly located FP is operative in the anterograde direction, should be considered in the differential diagnosis of long RP tachycardias (8) with shallow negative P waves in the inferior leads.

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References


