

## Multiple Anterograde Atrioventricular Node Pathways in Patients With Atrioventricular Node Reentrant Tachycardia

CHING-TAI TAI, MD, SHIH-ANN CHEN, MD, CHERN-EN CHIANG, MD,  
SHIH-HUANG LEE, MD,\* CHUEN-WANG CHIOU, MD, KWO-CHANG UENG, MD, ZU-CHI WEN, MD,  
YI-JEN CHEN, MD, MAU-SONG CHANG, MD

Taipei, Taiwan, Republic of China

**Objectives.** This study sought to investigate electrophysiologic characteristics and possible anatomic sites of multiple anterograde slow atrioventricular (AV) node pathways and to compare these findings with those in dual anterograde AV node pathways.

**Background.** Although multiple anterograde AV node pathways have been demonstrated by the presence of multiple discontinuities in the AV node conduction curve, the role of these pathways in the initiation and maintenance of AV node reentrant tachycardia (AVNRT) is still unclear, and possible anatomic sites of these pathways have not been reported.

**Methods.** This study included 500 consecutive patients with AVNRT who underwent electrophysiologic study and radiofrequency ablation. Twenty-six patients (5.2%) with triple or more anterograde AV node pathways were designated as Group I (16 female, 10 male, mean age  $48 \pm 14$  years), and the other 474 patients (including 451 with and 23 without dual anterograde AV node pathways) were designated as Group II (257 female, 217 male; mean age  $52 \pm 16$  years).

**Results.** Of the 21 patients with triple anterograde AV node pathways, AVNRT was initiated through the first slow pathway only in 3, through the second slow pathway only in 8 and through the two slow pathways in 9. Of the five patients with quadruple

anterograde AV node pathways, AVNRT was initiated through all three anterograde slow pathways in three and through the two slower pathways (the second and third slow pathways) in two. After radiofrequency catheter ablation, no patient had inducible AVNRT. Eleven patients (42.3%) in Group I had multiple anterograde slow pathways eliminated simultaneously at a single ablation site. Eight patients (30.7%) had these slow pathways eliminated at different ablation sites; the slow pathways with a longer conduction time were ablated more posteriorly in the Koch's triangle than those with a shorter conduction time. The remaining seven patients (27%) had a residual slow pathway after delivery of radiofrequency energy at a single or different ablation sites. The patients in Group I had a longer tachycardia cycle length, poorer retrograde conduction properties and a higher incidence of multiple types of AVNRT than those in Group II.

**Conclusions.** Multiple anterograde AV node pathways are not rare in patients with AVNRT. However, not all of the anterograde slow pathways were involved in the initiation and maintenance of tachycardia. Radiofrequency catheter ablation was safe and effective in eliminating critical slow pathways to cure AVNRT.

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Typical atrioventricular node reentrant tachycardia (AVNRT), one of the most common causes of paroxysmal supraventricular tachycardia in adults, has anterograde conduction through a "slow" pathway and retrograde conduction through a "fast" pathway (1). In contrast, atypical AVNRT has anterograde conduction through a "fast" or "slow" pathway and retrograde conduction through a "slow" pathway (2-4). Selective catheter ablation of the slow pathway using radiofrequency energy has become the treatment of choice for cure of this arrhythmia (5-9).

From the Division of Cardiology, Department of Medicine, National Yang-Ming University, School of Medicine and Veterans General Hospital-Taipei; and \*Shin-Kong Memorial Hospital, Taipei, Taiwan, Republic of China. This study was supported in part by Grants NSC 85-2331-B-075-071, 85-2331-B-010-047 and 85-2331-B-010-048 from the National Science Council, Taipei, Taiwan, Republic of China.

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Address for correspondence: Dr. Shih-Ann Chen, Division of Cardiology, Veterans General Hospital, 201 Sec 2, Shih-Pai Road, Taipei, Taiwan, Republic of China.

In the past, several investigators had reported the electrophysiologic characteristics of multiple anterograde AV node pathways and demonstrated multiple discontinuities in the anterograde AV node conduction curves during programmed electrical stimulation, but the patient numbers in these previous studies were very small and the role of these pathways in initiating AVNRT was still unclear (10-14). Furthermore, exploration of the anatomic sites of multiple anterograde slow AV node pathways using radiofrequency ablation has not been performed.

The purposes of this study were 1) to investigate the electrophysiologic characteristics and possible anatomic sites of multiple anterograde slow AV node pathways, and 2) to compare these findings with those of typical anterograde dual pathways in a large series of patients with AVNRT.

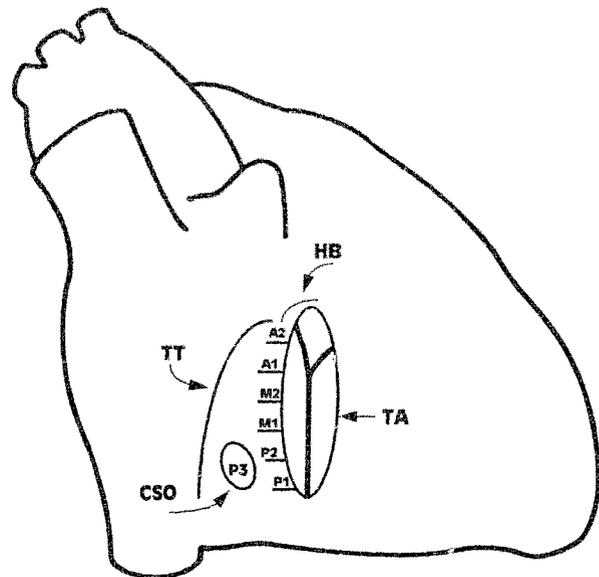
### Methods

**Patient characteristics.** Five hundred consecutive patients with AVNRT were referred to the Veterans General Hospital-

Taipei to receive electrophysiologic study and radiofrequency catheter ablation. Twenty-six patients with three or more anterograde AV node pathways were designated as Group I (10 male, 16 female; mean age  $48 \pm 14$  years, range 29 to 76); two patients had hypertensive cardiovascular diseases. The other 474 patients, including 451 with and 23 without dual anterograde AV node pathways) were designated as Group II (217 male, 257 female; mean age  $52 \pm 16$  years range 12 to 93).

**Baseline electrophysiologic study.** As described previously (9), each patient underwent a baseline electrophysiologic study in the fasting, unsedated state at least five half-lives after discontinuation of antiarrhythmic drugs. Written informed consent for the study and for ablation was obtained from each patient. Four multipolar, closely spaced (interelectrode space 2 mm) electrode catheters (Boston Scientific Inc.) were introduced from the right and left femoral veins and placed in the high right atrium, His bundle area, posteroseptal aspect of the tricuspid annulus and right ventricle for programmed electrical stimulation and recording. One orthogonal electrode catheter (distal 3 cm free of electrodes; Boston Scientific Inc.) was introduced from the right internal jugular vein and placed in the coronary sinus to record the electrical activity around the posteroseptal and proximal coronary sinus area. Intracardiac electrograms were displayed simultaneously with electrocardiographic (ECG) leads I, II and  $V_1$  on a multichannel oscilloscope recorder (model VR-13, PPG Biomedical Systems) and were recorded on paper at a speed of 100 to 150 mm/s. The filter was set from 30 to 500 Hz. A programmed digital stimulator (DTU-210 or 215, Bloom Associates Ltd.) was used to deliver electrical impulses of 2.0 ms in duration at approximately twice the diastolic threshold. The standard protocol included that 1) atrial or ventricular incremental pacing at cycle lengths, ranging from those slightly shorter than the sinus cycle length to the minimal cycle lengths, in which AV or ventriculoatrial (VA) 1:1 conduction was maintained; 2) a single atrial extrastimulus was delivered during high right atrial pacing at two cycle lengths. If dual AV node pathway could not be demonstrated, double atrial extrastimuli were delivered; 3) single and double ventricular extrastimuli were delivered during right ventricular apical pacing at two cycle lengths. If tachycardia was not induced under the baseline state, isoproterenol (at a graded dosage from 1 to 4  $\mu\text{g}/\text{min}$ ) or atropine (0.01 to 0.02 mg/kg body weight) was infused intravenously to facilitate its induction. AVNRT was defined according to the standard criteria (1,2).

**Definitions.** Anterograde AV node conduction curves were drawn from the results of programmed atrial extrastimulus testing. Dual pathway physiology was characterized by a jump ( $\geq 50$  ms) in  $H_1$  to  $H_2$  at a critical range of  $A_1$  to  $A_2$  coupling intervals (10-ms decrease), resulting in a discontinuity between the portion of the curve to the right of the jump in  $H_1$  to  $H_2$  (fast pathway conduction) and the portion of the curve to the left of the jump (slow pathway conduction). Comparable discontinuous  $A_1$  to  $A_2$  and  $A_2$  to  $H_2$  curves were also demonstrated. Three or more anterograde AV node pathways were defined by AV node conduction curves with two or more



**Figure 1.** Schematic representation of the right anterior oblique view of Koch's triangle showing the anatomic ablation sites. The right atrial septum adjacent to the septal leaflet of the tricuspid valve, extending from the ostium of the coronary sinus to the recording site of the proximal His bundle, was arbitrarily divided into posterior 1 (P1), posterior 2 (P2), medial 1 (M1), medial 2 (M2), anterior 1 (A1) and anterior 2 (A2). The posterior 3 (P3) subsection indicates the site around and inside the ostium of the coronary sinus. CSO = coronary sinus ostium; HB = His bundle; TA = tricuspid annulus; TT = tendon of Todaro.

discontinuities. Each discontinuity was the result of a jump in  $H_1$  to  $H_2$  at a critical range of  $A_1$  to  $A_2$  coupling intervals during two different paced cycle lengths. The portion of the curve to the right of the first discontinuity reflected fast pathway conduction. The portion of the curve to the left of the first discontinuity represented the first slow pathway conduction, and that to the left of the second discontinuity reflected the second slow pathway conduction. Thus, the two or more portions of the curve to the left of each discontinuity reflected different slow pathways with different refractory periods and conduction times (1,11,14).

**Mapping and ablation.** To define the possible anatomic sites of the multiple slow pathways, the stepwise upward method was used for mapping and ablation. The right atrial septum adjacent to the septal leaflet of the tricuspid valve, extending from the ostium of the coronary sinus to the recording site at the His bundle area, was divided into posterior (P), medial (M) and anterior (A) regions (Fig. 1). These regions were further divided into three, two and two subsections, respectively: posterior 1 (P1), posterior 2 (P2) and posterior 3 (P3) (around the coronary sinus ostium); medial 1 (M1) and medial 2 (M2); and anterior 1 (A1) and anterior 2 (A2). The ostium of the coronary sinus was demarcated by coronary sinus venography (15).

A multipolar, closely spaced (2 mm), deflectable, large-tipped (4 mm) electrode catheter (Mansfield Scientific) was

used for mapping and ablation. To determine the possible anatomic sites of multiple anterograde slow pathways, the mapping and ablation catheter tip was positioned initially in the posterior area, then to the medial and finally the anterior areas, if necessary. The presumed ablation site was considered optimal if bipolar electrograms obtained from the distal electrodes showed an atrial/ventricular ratio of 0.1 to 0.5 with a possible slow pathway potential (5,8). A radiofrequency generator (Radionic-3C, Radionics, Inc.) was used to deliver energy at a power setting of 30 to 40 W for 20 to 60 s. Energy was terminated immediately in the event of an increase in impedance, dislocation of the catheter, prolongation of the PR interval or occurrence of AV block (second-degree or complete AV block). An attempt to induce AVNRT with evaluation of AV node conduction properties was conducted immediately after each application of the radiofrequency current. The end point of the procedure was modification of anterograde or retrograde slow pathways with noninducibility of AVNRT under intravenous infusion of isoproterenol (2 to 4  $\mu\text{g}/\text{min}$ ).

**Postablation electrophysiologic study.** Immediate electrophysiologic study was performed in each patient after the ablation procedure. After hospital discharge, all patients were followed up closely and came back to the outpatient clinic in the second week, the first month and the second month after ablation, and then every 3 months. Long-term efficacy was assessed clinically on the basis of the rest surface ECG, 24-h Holter monitoring and clinical symptoms.

**Statistical analysis.** All data are expressed as mean value  $\pm$  SD. The Student *t* test was used to compare the continuous data. The chi-square test with a Yates correction or the Fisher exact test was used to compare the categorical data. A *p* value  $<0.05$  was considered statistically significant.

## Results

**Electrophysiologic characteristics of multiple AV node pathways.** Triple anterograde pathways were present in 21 patients and quadruple anterograde pathways in 5 patients during atrial extrastimulus testing at two atrial paced cycle lengths (range 450 to 650 ms). The magnitude of the discontinuity in the  $H_1$  to  $H_2$  curve was  $106 \pm 52$  ms (range 50 to 300) for the first discontinuity,  $105 \pm 58$  ms (range 50 to 250) for the second discontinuity and  $101 \pm 17$  ms (range 75 to 120) for the third discontinuity. The minimal cycle length with 1:1 anterograde conduction was  $360 \pm 62$  ms (range 280 to 540). The effective and functional refractory periods of the anterograde fast pathway were  $343 \pm 62$  ms (range 250 to 510) and  $414 \pm 64$  ms (range 300 to 540), respectively. The effective refractory periods of the first, second and third anterograde slow pathways were  $301 \pm 46$ ,  $272 \pm 45$  and  $234 \pm 54$  ms, respectively; the functional refractory periods of these anterograde slow pathways were  $520 \pm 81$ ,  $624 \pm 82$  and  $676 \pm 91$  ms, respectively. A retrograde fast pathway was present in all patients, and a retrograde slow pathway was demonstrated in eight patients. One patient had retrograde conduction only

after intravenous infusion of isoproterenol. The minimal cycle length with 1:1 VA conduction during incremental ventricular pacing was  $397 \pm 90$  ms. The effective and functional refractory periods of the retrograde VA conduction were  $306 \pm 80$  and  $412 \pm 95$  ms, respectively.

**Induction of AVNRT.** All 26 patients had clinically documented paroxysmal supraventricular tachycardia; three of them had two different types of tachycardias. The sustained slow-fast form of AVNRT was inducible in 25 patients, but 11 needed intravenous infusion of isoproterenol to facilitate induction. Only one patient did not have inducible AVNRT in the electrophysiology laboratory. Two patients also had the sustained fast-slow form of AVNRT.

During the slow-fast form of AVNRT, 23 patients had only one tachycardia cycle length; two patients had two different tachycardia cycle lengths with different AH intervals and identical HA intervals, suggesting that different slow pathways were used for anterograde conduction and a fast pathway for retrograde conduction.

Table 1 summarizes the different patterns of tachycardia induction in Group I patients. For patients with three anterograde pathways (one fast and two slow), three patterns of tachycardia induction were found (Fig. 2, A to C): 1) *pattern 1* (three patients) = both initiation and maintenance of tachycardia using the first slow pathway (the second slow pathway was not used for initiation or maintenance of tachycardia); 2) *pattern 2* (eight patients) = both initiation and maintenance of tachycardia using the second slow pathway; 3) *pattern 3* (nine patients) = initiation of tachycardia using either the first or the second slow pathway, and maintenance of tachycardia using the first slow pathway only (five patients), the second slow pathway only (three patients) or the first and the second slow pathways alternately (one patient).

For patients with four anterograde pathways (one fast and three slow), two patterns of tachycardia induction were found (Fig. 2, D and E): 1) *pattern 4* (two patients) = initiation of tachycardia using any of the three anterograde slow pathways and maintenance of tachycardia using the first or the second slow pathway; 2) *pattern 5* (three patients) = initiation of tachycardia using either the second or the third slow pathway, and maintenance of tachycardia using the second slow pathway only (one patient), the third slow pathway only (one patient) or the second and the third slow pathways alternately (one patient).

**Results of radiofrequency ablation.** All 26 patients had successful ablation. Of the 21 patients with three anterograde pathways, 10 had two anterograde slow pathways eliminated simultaneously at a single ablation site, 5 had two anterograde slow pathways eliminated separately at different ablation sites and the remaining 6 had one residual slow pathway (the first slow pathway) after delivering radiofrequency energy at a single or different ablation sites (3 of these 6 patients had one slow-fast echobeat). Of the five patients with four anterograde pathways, one had three anterograde slow pathways eliminated simultaneously at a single ablation site, three had three anterograde slow pathways eliminated separately at different

**Table 1.** Induction of Tachycardia and Effects of Radiofrequency Catheter Ablation in Group I Patients With Multiple Atrioventricular Node Pathways

Patient No.	Before ABL		After ABL				
	SP for T (Pattern)	SP for Echobeat	ABL Site(s)	Ant-SPs		Ret-SP	
				Residual SP	Residual Echobeat	ABL Site	Ant/Ret Same
Two Ant-SP + Ret-FP							
1	S1 (I)	S1	P2	+	-	-	-
2	S2 (II)	S2	M1	-	-	-	-
3	S2 (II)	S2	M1	-	-	-	-
4	S2 (II)	S2	M2*	-	-	-	-
5	S2 (II)	S2	P2	-	-	-	-
6	S2 (II)	S2	P1	+	-	-	-
7	S2 (II)	S2	M1	-	-	-	-
8	S2 (III)	S1, S2	P2	-	-	-	-
9	S2 (III)	S1, S2	P2	-	-	-	-
10	S2 (III)	S1, S2	P2	+	+	-	-
11	S1 (III)	S1, S2	P2, M1	+	+	-	-
12	S1 (III)	S1, S2	M1, M2*	-	-	-	-
13	S1 (III)	S1, S2	P2	+	+	-	-
14	S1 (III)	S1, S2	M1, M2	-	-	-	-
15	S1 (III)	S1, S2	P2, M1	-	-	-	-
16	S1, S2 (III)	S1, S2	M1, M2	-	-	-	-
Two Ant-SP + Ret-DP							
17	S1 (I)	S1	P2	+	-	P2	+
18	S1 (I)	S1	P2, M1	-	-	M1	+
19	S2 (II)	S2	P2	-	-	P2	+
20	S2 (II)	S2	M1	-	-	M1	+
21	-	-	P1	-	-	P1	+
Three Ant-SP + Ret-FP							
22	S1 (IV)	S1, S2, S3	P1, P2	-	-	-	-
23	S2 (IV)	S1, S2, S3	P1, P2	-	-	-	-
Three Ant-SP + Ret-DP							
24	S3 (V)	S2, S3	P2	-	-	P2	+
25	S2 (V)	S2, S3	P2, M1	-	-	P3	-
26	S2, S3 (V)	S2, S3	P1, P2	+	-	P3	-

\*Ventriculoatrial conduction block after radiofrequency ablation. ABL = ablation; Ant = anterograde; DP = dual pathway; FP = fast pathway; Ret = retrograde; SP = slow pathway; S1 = first slow pathway; S2 = second slow pathway; S3 = third slow pathway; T = tachycardia.

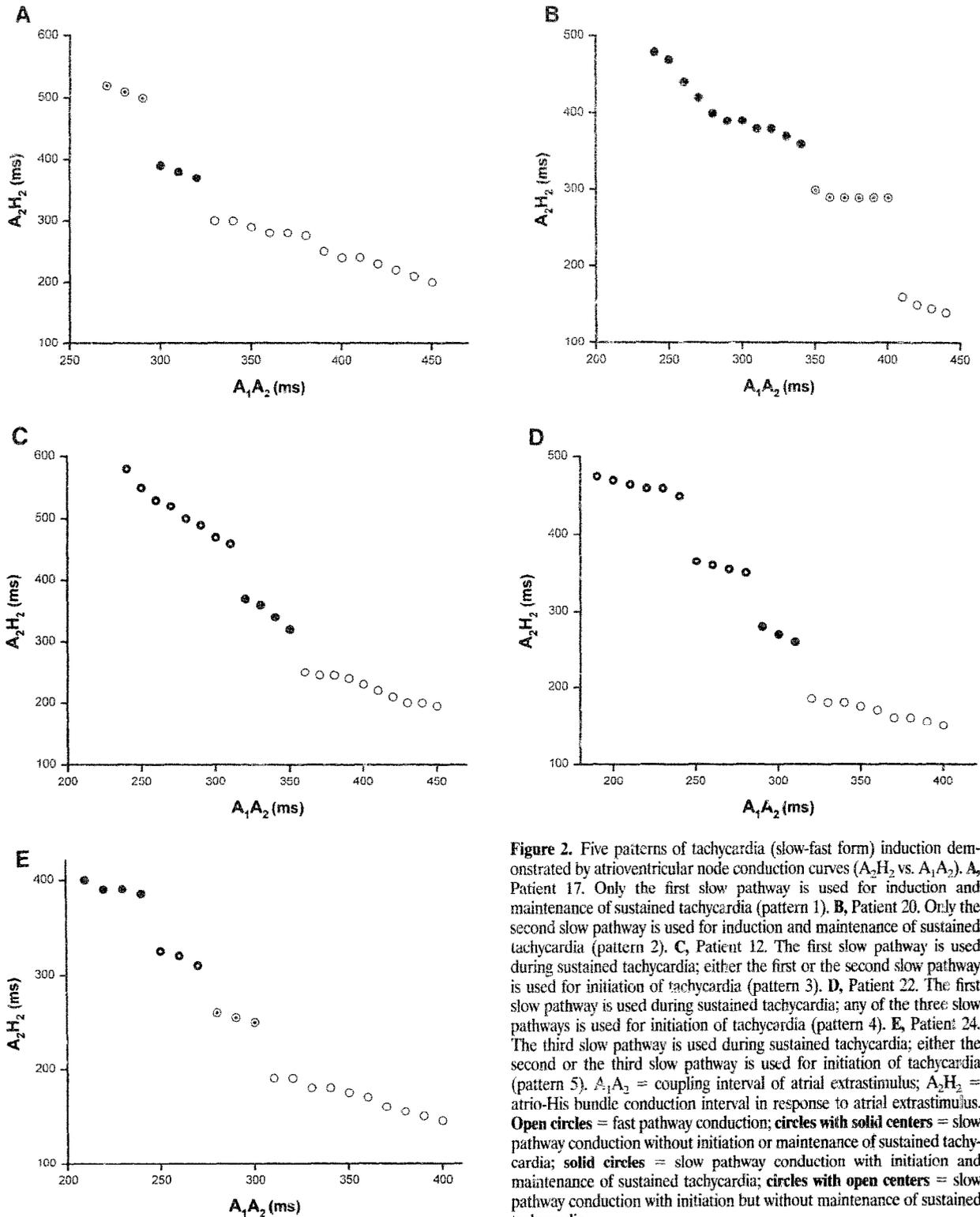
ablation sites and one had a residual slow pathway (the first slow pathway) without any echobeat after delivering radiofrequency energy at different ablation sites. Of the eight patients with retrograde dual pathways, six had anterograde and retrograde slow pathways eliminated simultaneously at a single ablation site, and two had anterograde and retrograde slow pathways eliminated separately at different ablation sites (Table 1).

Analysis of the ablation sites showed that the successful sites were located in the posterior zone in 14, the medial zone in 8 and both the posterior and medial zones in 4 patients. The longest functional refractory period of the slow pathways was significantly longer in the patients with a successful ablation site in the posterior zone than in the medial zone ( $676 \pm 83$  vs.  $591 \pm 30$  ms,  $p = 0.005$ ). In the eight patients who had successful ablation of different anterograde slow pathways at different locations, the slow pathways with a longer conduction time were located more posteriorly in the Koch's triangle than those with a shorter conduction time.

**Postablation electrophysiologic study.** Electrophysiologic study 20 to 30 min after successful ablation showed that the minimal atrial paced cycle length with 1:1 anterograde fast pathway conduction did not have significant change compared with the paced cycle length before ablation ( $401 \pm 69$  vs.  $398 \pm 52$  ms,  $p > 0.05$ ), nor did the minimal ventricular paced cycle length with 1:1 VA conduction ( $397 \pm 90$  vs.  $386 \pm 78$  ms,  $p > 0.05$ ).

**Clinical follow-up.** During a mean follow-up period of  $14 \pm 12$  months (range 3 to 47), tachycardia recurred in one patient who had successful ablation during the second session. The other patients remained free of tachycardia without any antiarrhythmic drug.

**Comparisons between Group I and Group II patients.** In Group II patients, the effective and functional refractory periods of the anterograde fast pathway were  $318 \pm 61$  and  $389 \pm 62$  ms, respectively; the effective and functional refractory periods of the anterograde slow pathway were  $273 \pm 45$  and  $501 \pm 83$  ms, respectively. Compared with Group II



**Figure 2.** Five patterns of tachycardia (slow-fast form) induction demonstrated by atrioventricular node conduction curves ( $A_2H_2$  vs.  $A_1A_2$ ). **A**, Patient 17. Only the first slow pathway is used for induction and maintenance of sustained tachycardia (pattern 1). **B**, Patient 20. Only the second slow pathway is used for induction and maintenance of sustained tachycardia (pattern 2). **C**, Patient 12. The first slow pathway is used during sustained tachycardia; either the first or the second slow pathway is used for initiation of tachycardia (pattern 3). **D**, Patient 22. The first slow pathway is used during sustained tachycardia; any of the three slow pathways is used for initiation of tachycardia (pattern 4). **E**, Patient 24. The third slow pathway is used during sustained tachycardia; either the second or the third slow pathway is used for initiation of tachycardia (pattern 5).  $A_1A_2$  = coupling interval of atrial extrastimulus;  $A_2H_2$  = atrio-His bundle conduction interval in response to atrial extrastimulus. **Open circles** = fast pathway conduction; **circles with solid centers** = slow pathway conduction without initiation or maintenance of sustained tachycardia; **solid circles** = slow pathway conduction with initiation and maintenance of sustained tachycardia; **circles with open centers** = slow pathway conduction with initiation but without maintenance of sustained tachycardia.

**Table 2.** Comparisons Between Patients With (Group I) and Without (Group II) Multiple Atrioventricular Node Pathways

	Group I (n = 26)	Group II (n = 474)	p Value
Age (yr)	48 ± 14	52 ± 16	0.580
Associated CV diseases	2 (7.7)	108 (22.8)	0.117
Retrograde dual pathways	8 (30.7)	99 (20.9)	0.342
Inducibility of tachycardia	25 (96.2)	445 (93.9)	1.000
AV node reentry			
S-F type	19 (73.1)	409 (86.2)	0.557
F-S type	0	12 (2.5)	1.000
Variant type	0	6 (1.3)	1.000
Multiple types	7 (26.9)	47 (9.9)	0.011
AV 1:1 conduction (ms)			
Fast pathway	401 ± 69	389 ± 73	0.819
Slow pathway	360 ± 62	347 ± 63	0.262
VA 1:1 conduction (ms)	397 ± 90	360 ± 88	0.045
TCL of S-F type (ms)	447 ± 95	396 ± 88	0.013
Catecholamine sensitive	11 (42.3)	167 (35.3)	0.601
RF ablation variables			
Pulse (no.)	4 ± 3	5 ± 5	0.564
Pulse duration (s)	43 ± 6	45 ± 8	0.150
Power (W)	35 ± 5	33 ± 7	0.112
Initial success (%)	26 (100)	472 (99.5)	1.000
Recurrent SVT (%)	1 (3.8)	6 (1.3)	0.314
Complications (%)	0	3 (0.6)	1.000

Data are expressed as mean value ± SD or number (%) of patients. AV = atrioventricular; CV = cardiovascular; F-S = fast-slow; RF = radiofrequency; S-F = slow-fast; SVT = supraventricular tachycardia; TCL = tachycardia cycle length; VA = ventriculoatrial.

patients, Group I patients had a longer tachycardia cycle length (447 ± 95 vs. 396 ± 88 ms,  $p = 0.013$ ), longer effective (343 ± 62 vs. 318 ± 61 ms,  $p = 0.015$ ) and functional (414 ± 64 vs. 389 ± 62 ms,  $p = 0.016$ ) refractory periods of the anterograde fast pathway, poorer retrograde VA conduction properties (397 ± 90 vs. 360 ± 88 ms,  $p = 0.04$ ) and a higher incidence of multiple types of AVNRT (26.9% vs. 9.9%,  $p = 0.011$ ). Age, prevalence of associated cardiovascular diseases, incidence of retrograde dual pathways, inducibility of sustained tachycardia, percentage of patients requiring isoproterenol for facilitating induction of tachycardia and minimal cycle length with 1:1 AV conduction during incremental atrial pacing did not show significant differences. Radiofrequency pulse number, pulse duration, radiofrequency power energy, initial success rate and late recurrence rate were also similar between the two groups (Table 2).

## Discussion

**Main findings.** The present study showed that 1) the incidence (5.2%) of multiple anterograde AV node pathways was not rare in patients with AVNRT; 2) five patterns of tachycardia induction through preferred slow pathways were found; the presence or absence of tachycardia based on any given pathway may be related to intact connections between the involved pathways of a potential circuit, and to conduction and refractory properties over the respective pathways condu-

cive to reentry; 3) 42% of the patients with multiple anterograde slow pathways had simultaneous elimination of all these pathways at a single ablation site; and 4) the slow pathways with a longer conduction time (the second or third slow pathway, or both) were located at a more inferoposterior position than those with a shorter conduction time.

**Lessons from radiofrequency catheter ablation.** Whether multiple anterograde AV node pathways originate from anatomically different pathways or anisotropic conduction-induced functional pathways is still controversial. Regarding the anatomically different pathways, the proximal AV node may include several pathways with varying lengths and electrophysiologic properties when one considers its complex histologic findings (16-19). Thus, premature atrial depolarizations might tranverse some different slow pathways preferentially to arrive at the distal AV node and His bundle; in which case, a wide difference of AH interval was found. In contrast, the marked heterogeneity of the transitional cells and their connections produce a nonuniform structure. It is reasonable to suggest that the nonuniform properties of the AV node can produce anisotropic conduction and longitudinal dissociation (20,21).

In the present study, all AVNRTs were eliminated by delivering radiofrequency energy in the medial and posterior zones without impairment of anterograde fast pathway conduction. This finding suggests that the fast pathway was anatomically distant from the multiple slow pathways. For the eight patients (30.7%) who had successful ablation of the different slow pathways at different sites, and for the seven patients (27%) who had a residual slow pathway after successful ablation, the possibility of anatomically different slow pathways was considered. Furthermore, the slow pathways with much longer functional refractory periods were located at a more inferoposterior area in the Koch's triangle in these patients. This finding resembled the report by Philippon et al. (22), who demonstrated that the slow pathway with the longest conduction interval was ablated more inferiorly in the Koch's triangle than the slow pathway with an intermediate conduction interval, suggesting that the slow pathway with a longer conduction time needs to tranverse a longer anatomic distance. For the 11 patients (42.3%) who had successful ablation of multiple slow pathways at a single site, anisotropic conduction over the low septal area of the right atrium is a possible explanation for the presence of multiple anterograde slow pathways.

**Study limitations.** The protocol in this study did not include autonomic blockade. It may be argued that the presence of multiple discontinuities in the AV node conduction curve might result from unstable autonomic tone. However, several studies have demonstrated that unstable autonomic tone did not really affect AV node functional properties (23-25). Although the radiologic division of Koch's triangle into seven sections may be rough to some extent, a more detailed grid is not factual because the mapping/ablation catheter would be moved along with the cardiac and respiratory cycles, and it is

difficult to fix the catheter tip at the narrower zone. The region of Koch's triangle is rather small, and the size of the potential ablation lesion is relatively large in this area; thus, a single burn at a more superior portion may well eliminate two or three slow pathways, even if they are anatomically distinct. It is possible that radiofrequency energy applied in the very inferior portions of Koch's triangle will allow separation of the atrial insertion of the multiple AV node pathways. At last, the reproducibility of multiple AV node pathway patterns is unknown; therefore, the electrophysiologic findings observed after ablation cannot be definitely attributed to the procedure, and further study will be necessary.

**Conclusions.** This study demonstrated that multiple anterograde AV node pathways were not rare in patients with AVNRT. These functional and possibly anatomic slow pathways led to various patterns of tachycardia induction. Radiofrequency catheter ablation was safe and effective in eliminating critical slow pathways to cure AVNRT.

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