Hiroshima J. Med. Sci.
Vol. 47, No.1, 7~15, March, 1998
HIJM 47-2

Structural Characteristics of Koch's Triangle in Patients with Atrioventricular Node Reentrant Tachycardia

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ABSTRACT

The aim of this study is to investigate whether patients with atrioventricular node reentrant tachycardia (AVNRT) present different structural characteristics of Koch's triangle from patients with atrioventricular (AV) reentrant tachycardia and other control patients. Fifty-eight patients with arrhythmia or chest pain underwent selective coronary sinus angiography so that the diameter of the coronary sinus could be measured. The patients with arrhythmia also underwent electrophysiological study and measurement of the height of Koch's triangle. Patients with AVNRT had large coronary sinus ostial diameters compared with patients with AV reentrant tachycardia and those with chest pain (13.6 ± 2.2 mm vs. 10.6 ± 2.7 mm [$p < 0.005$] and 10.0 ± 2.1 mm [$p < 0.002$], respectively), while there were no differences in distal diameter. The ostial diameter in patients with dual AV node pathways but noninducible AVNRT (11.8 ± 1.5 mm) tended to be smaller than that in patients with AVNRT. No differences in the height of Koch's triangle and electrophysiological characteristics, including AV node properties, were found among the study groups. In conclusion, an increased size of the coronary sinus ostium (the base of Koch's triangle) is a structural characteristic in patients with AVNRT and may be the substrate needed for the appearance of AVNRT.

Key words: *Atrioventricular node reentrant tachycardia, Coronary sinus ostium, Dual atrioventricular node pathways*

Dual atrioventricular (AV) node pathways are well-known electrophysiological characteristics for the mechanism of atrioventricular node reentrant tachycardia (AVNRT)[1,4,11,12,22,30,34]. An abrupt increase in the AV node conduction curve led us to hypothesize that, in patients with AVNRT, two functionally distinct pathways were demonstrable within the AV node, namely, a fast pathway and a slow pathway, and that AVNRT resulted from this electrophysiological discrete substrate.

Recent studies have suggested that the substrate of the reentrant circuit in AVNRT involved the perinodal atrial tissue and that dual AV node pathways were not only functionally but also anatomically distinct[34]. In normal heart, the fast pathway is the electrophysiologically normal connection between the atrium and the ventricle in sinus rhythm. Its atrial insertion is located anteriorly in the septum close to the His bundle electrogram recording site, whereas the atrial insertion of the slow pathway is located posteriorly in the septum and the slow pathway runs posteroinferiorly surrounding the coronary sinus ostium[21,27,29]. Selective slow pathway ablation is now a well-established approach in abolishing AVNRT[2,17,21,23,25], and this procedure is based on the concept that dual AV node pathways provide a functionally and anatomically discrete substrate.

However, dual AV node pathways are frequently observed in patients undergoing electrophysiological study[4,12,18,24,32], and the presence of dual pathways appears to predispose to the development of AVNRT, some patients with electrophysiologically proven dual AV node pathways do not exhibit AVNRT. The aim of this study is to investigate whether patients with AVNRT have different structural characteristics of Koch's triangle (that related to AV node reentry) from patients with AV reentrant tachycardia and other control patients.

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This work was presented in part at the 68th Scientific Sessions of American Heart Association, Anaheim, California, United States of America, 1995, and the 60th Annual Scientific Meeting of Japanese Circulation Society, Osaka, Japan, 1996.

MATERIALS AND METHODS

Study patients. The study was performed in consecutive patients who underwent electrophysiological study for arrhythmia or coronary angiography for chest pain at Hiroshima University Hospital between June 1994 and March 1996. From the initial group, patients with any of the following characteristics were excluded from the study: moderate or severe tricuspid valve regurgitation; structural heart diseases such as old myocardial infarction and cardiomyopathy; angina pectoris with angiographically significant organic stenosis (≥75% narrowing); and cardiomegaly due to incessant or chronic tachycardia including atrial flutter, atrial fibrillation or atrial tachycardia. Two patients were also excluded because the mechanism of their tachycardia was unknown. The final study population comprised 58 patients (24 men and 34 women). Written and verbal informed consent for participation was obtained from each patient prior to study.

The patients studied were classified into four groups, based on clinical findings and the results of electrophysiological study. Group A consisted of patients with AVNRT but no AV accessory pathway and no other tachyarrhythmia. Group B consisted of patients with paroxysmal supraventricular tachycardia (PSVT) corresponding to AV accessory pathways. All patients in group B underwent successful radiofrequency catheter ablation for the accessory pathways and none exibited dual AV node physiology after ablation under isoproterenol infusion. Group C, the control group, consisted of patients admitted for the evaluation of chest pain, ventricular tachycardia or AV block. All patients in group C except for those with chest pain received electrophysiological study and none exhibited dual AV node physiology. Group D consisted of patients with dual AV node physiology but noninducible AVNRT. This group included patients with AV accessory pathways and idiopathic ventricular tachycardia. Patients with tachycardia underwent successful radiofrequency catheter ablation for those arrhythmia. Following the ablation procedure, the electrophysiological study was repeated during the infusion of isoproterenol, and no inducible AVNRT was detected. During two-dimensional echocardiography and Doppler examination, we measured the left atrial dimension at end-systole from the parasternal long-axis view. Attention was given to the presence or absence of tricuspid valve regurgitation. Patients with moderate or severe tricuspid valve regurgitation were excluded from this study because of the dilatation of the right-side cardiac system including the coronary sinus. The frequency of tachycardia was compared in patients with tachycardia in groups A, B, and D. The maximum p wave duration was measured by baseline 12-lead electrocardiogram (ECG) in all study patients to evaluate the conduction time over the bilateral atrium which represents the size of the atrium.

Cardiac catheterization and coronary sinus angiography. Bilateral cardiac catheterization and coronary angiography were performed in fasting patients. Special attention was given to the venous phase of left coronary arteriography. The goal was to identify the coronary sinus orifice and major coronary sinus abnormalities, such as hypoplasia, angulation, localized narrowing, fistula and diverticulum. Then retrograde coronary sinus angiography was performed. The coronary sinus was cannulated with a 7Fr CSL™ 10-pole coronary sinus electrode with a lumen catheter (Daig Corporation, Minnetonka, MN), a 6Fr right Judkins catheter or a 6Fr Goodale-Lubin catheter from the right subclavian vein or the right femoral vein. About 10 ml of contrast medium was injected into the coronary sinus. Images of the coronary sinus were obtained during sinus rhythm and stored on cine film.

Electrophysiological study. Electrophysiological study was performed in patients with arrhythmia in the baseline, drug-free and unsedate state. Standard recording methods, stimulation techniques and definitions were used[24]. Antiarrhythmic drugs were discontinued at least 72 hours before the study. Under 1% local anesthesia, three 6Fr standard electrode catheters were introduced into both femoral veins and advanced to the high right atrium (HRA), His bundle electrogram recording position, and right ventricular apex, respectively. A 7Fr decapolar coronary sinus electrode catheter with a lumen or a 6Fr octapolar catheter was placed in the coronary sinus and the great cardiac vein via the right subclavian vein.

The following electrophysiological characteristics were defined: baseline atrio-His (AH) interval; the minimal cycle length in which 1:1 AV conduction was maintained during rapid HRA pacing; effective refractory period (ERP) of the atrium; anterograde and retrograde ERP of the AV node; anterograde ERP of the fast pathway; AH interval when the jumping phenomenon was observed during the series of single atrial extrastimulus tests; and the longest attainable AH interval during the series of single atrial extrastimulus tests. Whenever possible, ERPs were measured at a basic cycle of 600 msec in the baseline state. In addition, the interval between the atrial signal in the HRA catheter and that in the distal and proximal coronary sinus catheter (positioned at left lateral region and near the ostium, respectively) was measured in sinus rhythm [HRA-CSd interval and HRA-CSp interval, respectively]. The cycle length of the tachycardia for which the patient underwent electrophysiological study was also determined.

Anterograde dual AV node physiology was defined as a sudden increase of the AH interval of

Table 1. Baseline Clinical Characteristics by Study Group

	Group A (n = 13)	Group B (n = 18)	Group C (n = 15)	Group D (n = 12)
Age (yr)				
Mean	49 ± 12	42 ± 17	54 ± 19	42 ± 17
Range	23 – 65	16 – 70	16 – 77	18 – 75
Male/Female	1/12	8/10	10/5	5/7
Height (cm)	156.7 ± 8.2	161.2 ± 10.9	162.1 ± 6.4	160.5 ± 11.1
LAD (mm)	31.7 ± 4.1	33.1 ± 4.8	33.2 ± 4.7	31.7 ± 2.7
PD (msec)	102 ± 8	101 ± 17	95 ± 16	105 ± 10

Data are expressed as mean ± SD. LAD = left atrial dimension; PD = maximum p wave duration.

at least 50 msec with a 10-msec decrement during extrastimulus testing with at least two basic cycle lengths. Retrograde dual AV node physiology was defined as a discontinuity of the retrograde AV node conduction curve or an alteration of the retrograde atrial activation sequence from the His bundle electrogram (anteroseptal region) to the coronary sinus ostium (posteroseptal region) during ventricular extrastimulus testing with at least two basic cycle lengths[34].

Measurement of the dimensions of Koch's triangle. Later, another investigator, who did not know the diagnosis of the patients, measured the diameter of the coronary sinus ostium and of the lateral segment at the distal region of the coronary sinus (the 3 o'clock position) and the height of Koch's triangle. The measurements were made at the ventricular end-systolic phase of the angiogram, using a cinevideo edge detection method. The size of the catheter was used to calibrate the diameter of the coronary sinus and the height of Koch's triangle expressed as millimeters. Measurements of the coronary sinus diameter were obtained in the left anterior oblique view and those of the height of Koch's triangle were obtained both in the right and the left anterior oblique view.

The coronary sinus orifice was defined as the junctional point made by the atrial septum above and below the coronary sinus ostium, which was outlined by the contrast medium flowing out of the coronary sinus, and the long axis of the proximal portion of the coronary sinus as it entered the right atrium[10]. The height of Koch's triangle was defined as the distance from the largest His bundle electrogram recording site to the coronary sinus ostium on a coronary sinus electrode catheter. Because the dimensions may vary with body size, each measurement was also corrected by dividing it by the patient's height.

Statistical analysis. Data are expressed as mean ± SD. Values were analyzed with StatView 4.5 (Abacus Concepts Inc., Berkeley, CA). For continuous variables, comparisons were made with one-way analysis of variance in multiple groups. When significant differences were present, the post-hoc test of Scheffe's multiple comparison test was used to determine which means differed significantly. The unpaired *t*-test was used to compare two groups, as appropriate. A value of $p < 0.05$ was considered statistically significant.

RESULTS

Baseline findings. Fifty-eight patients (24 men, 34 women; mean age 47 ± 17 years, range 16 to 77) were eligible for this study. The baseline clinical characteristics of the four groups are presented in Table 1. All patients were in sinus rhythm.

In group A, 12 of the 13 patients had the common type (slow-fast) AVNRT, and one had the uncommon type (fast-slow). All patients in group A had dual AV node physiology. One patient had two discrete discontinuous AV node conduction curves, suggesting triple AV node pathways. Group B consisted of 18 patients, 9 of whom had left free wall accessory pathways, 1 left posterolateral, 1 left posteroseptum, 2 right free wall, 2 right anteroseptum, 1 right anterior, 1 right posteroseptum and 1 a double accessory pathway. Of the 15 patients in group C, 10 had chest pain syndrome, 3 had idiopathic ventricular tachycardia and two had idiopathic ventricular fibrillation and AV block. The patients with chest pain had no documented and symptomatic tachyarrhythmia. Group D consisted of 12 patients, 8 of whom had AV accessory pathways; 4 were left free wall accessory pathways, 2 left posterolateral, 1 right posterior and 1 right anteroseptum. One patient in group D had idiopathic left ventricular tachycardia, and another had abnormal sinus node function. Two patients in group D, who underwent electrophysiological study for palpitations, did not have clinical tachycardia or other abnormal findings except for dual AV node physiology.

The four groups did not differ in their mean left atrial dimension or maximum p wave duration. No

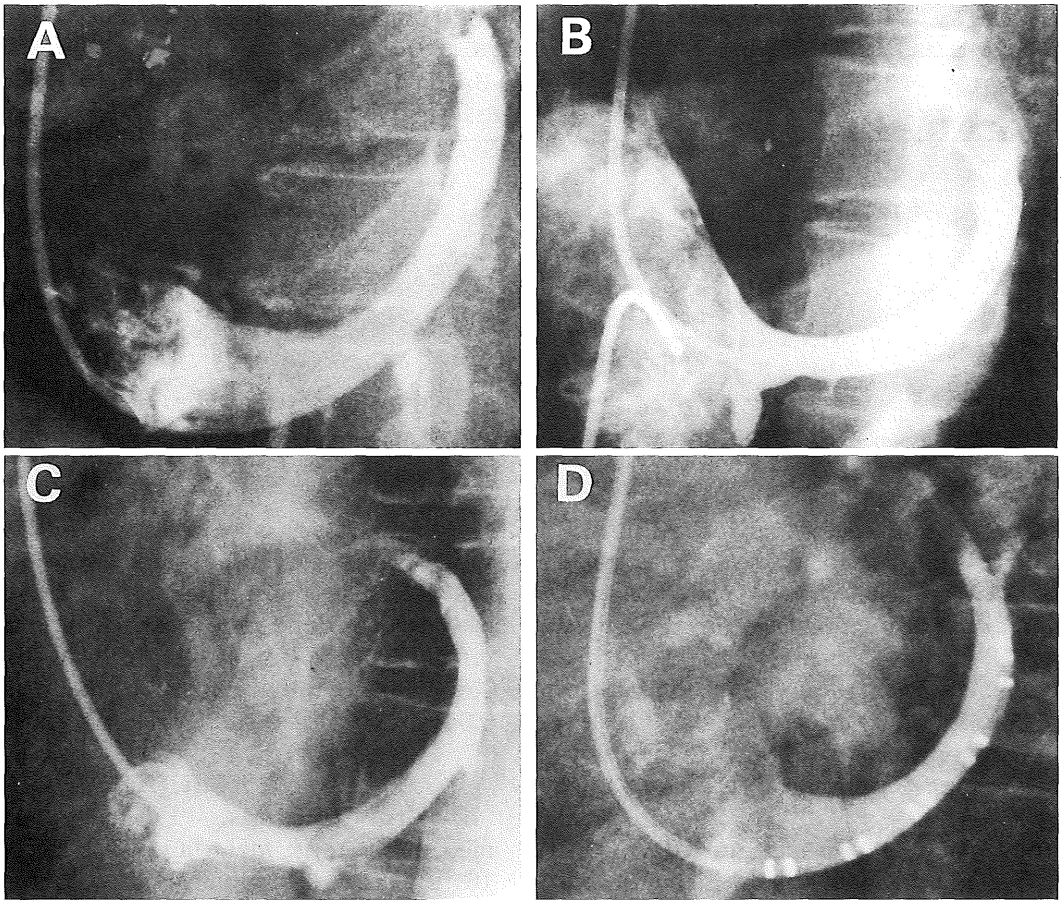

Fig. 1. Coronary sinus angiograms obtained in the left anterior oblique view in representative patients of each group. **A:** Patient with atrioventricular node reentrant tachycardia (group A). **B:** Patient with Wolff-Parkinson-White syndrome. The accessory pathway was located in the left lateral atrioventricular ring (group B). **C:** Patient with chest pain (group C). **D:** Patient with dual atrioventricular node pathways admitted for evaluation of paroxysmal supraven-tricular tachycardia due to atrioventricular accessory pathway (group D). The accessory pathway was located in the left lateral atrioventricular ring. Atrioventricular node reentrant tachycardia could not be induced in the electrophysiological laboratory after successful radiofrequency catheter ablation of the accessory pathway.

Table 2. Coronary Sinus Angiographic Measurements by Study Group

	Group A (n = 13)	Group B (n = 18)	Group C (n = 15)	Group D (n = 12)
CSO (mm)	13.6 ± 2.2*†	10.6 ± 2.7	10.0 ± 2.1	11.8 ± 1.5
Range	10.4 – 18.0	6.1 – 15.5	7.2 – 15.0	9.7 – 14.8
CSO/H	0.087 ± 0.015‡§	0.065 ± 0.015	0.062 ± 0.013	0.076 ± 0.015
Range	0.065 – 0.116	0.040 – 0.091	0.044 – 0.091	0.060 – 0.117
CSL (mm)	4.8 ± 1.4	5.2 ± 1.3	4.6 ± 0.8	5.0 ± 1.2
Range	3.0 – 7.4	3.4 – 8.0	3.4 – 6.1	3.1 – 7.9
CSL/H	0.031 ± 0.01	0.033 ± 0.009	0.029 ± 0.005	0.032 ± 0.009
Range	0.020 – 0.051	0.021 – 0.049	0.021 – 0.038	0.019 – 0.051

Data are expressed as mean ± SD. *$p < 0.005$ versus group B. †$p < 0.002$ versus group C. ‡$p < 0.002$ versus group B. §$p < 0.0005$ versus group C. CSL = diameter of the coronary sinus in lateral segment; CSO = diameter of the coronary sinus ostium; CSL/H = CSL/patient's height; CSO/H = CSO/patient's height.

Table 3. Height of Koch's Triangle by Study Group

	Group A (n = 13)	Group B (n = 18)	Group C (n = 5)	Group D (n = 12)
His-CSO in RAO (mm)	30.7 ± 4.1	30.6 ± 6.9	30.6 ± 2.4	31.7 ± 4.6
Range	24.2 – 38.6	18.1 – 43.5	28.3 – 33.8	21.6 – 38.0
His-CSO/H in RAO	0.196 ± 0.024	0.188 ± 0.038	0.188 ± 0.014	0.198 ± 0.030
Range	0.169 – 0.247	0.123 – 0.256	0.170 – 0.200	0.237 – 0.142
His-CSO in LAO (mm)	32.7 ± 5.8	31.6 ± 6.1	31.6 ± 1.9	31.7 ± 4.2
Range	20.1 – 42.9	25.1 – 42.7	29.2 – 33.5	23.2 – 37.6
His-CSO/H in LAO	0.210 ± 0.038	0.196 ± 0.034	0.192 ± 0.006	0.198 ± 0.025
Range	0.128 – 0.286	0.139 – 0.267	0.186 – 0.199	0.154 – 0.243

Data are expressed as mean ± SD. His-CSO in LAO = Distance from the largest His bundle electrogram recording site to the coronary sinus ostium in the left anterior oblique view; His-CSO/H in LAO = His-CSO/patient's height in the left anterior oblique view; His-CSO in RAO = Distance from the largest His bundle electrogram recording site to the coronary sinus ostium in the right anterior oblique view; His-CSO/H in RAO = His-CSO/patient's height in the right anterior oblique view.

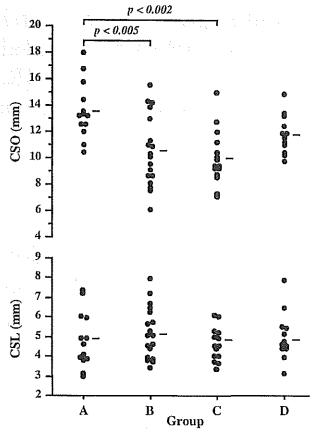

Fig. 2. Top: Diameter of the coronary sinus ostium (CSO), and **bottom:** diameter of coronary sinus in lateral segment (CSL) in each patient in the four study groups. Small horizontal bars represent mean values.

evidence of volume and pressure overloading in the right-side cardiac system was found on transthoracic echocardiography and cardiac catheterization.

Coronary sinus cannulation and morphology. Coronary sinus catheterization was performed with a 7Fr 10-pole catheter with a lumen in 36 cases, a 6Fr right Judkins catheter in 12 cases, or a 6Fr Goodale-Lubin catheter in 10 cases. There were no complications during the procedure. Figure 1 displays findings on coronary sinus angiography that are representative of each of the groups.

A major coronary sinus abnormality was identified in only two cases. One, a 16-year-old patient, had a coronary sinus diverticulum corresponding to an AV accessory pathway in the left posteroseptal region near the coronary sinus ostium. In this case, AV reentrant tachycardia was associated with the diverticulum. The other in group B had a localized narrowing coronary sinus in the left posterolateral region.

Coronary sinus diameters and the heights of Koch's triangle. The diameters of the ostial and distal coronary sinus in each group are shown in Table 2. The mean diameter of the coronary sinus ostium in group A, the patients with AVNRT, was 13.6 ± 2.2 mm. This was significantly larger compared with the mean ostial diameters in group B (10.6 ± 2.7 mm, $p < 0.005$) and in group C (10.0 ± 2.1 mm, $p < 0.002$). Individual measurements are shown in Figure 2. The ostial diameter corrected by height was also significantly larger in group A than in groups B and C (0.087 ± 0.015 in group A vs. 0.065 ± 0.015 in group B and 0.062 ± 0.013 in group C). The four groups did not differ with respect to the mean diameters of the lateral segment in the distal coronary sinus and the heights of Koch's triangle (Table 2, 3 and Fig. 2).

Electrophysiological findings. The clinical electrophysiological characteristics, including the refractory and conduction properties of the AV node, are summarized for each group in Table 4. Groups A, B, and D showed no differences in the baseline AH interval, the minimal cycle length in which 1:1 AV conduction was maintained during rapid HRA pacing, ERP of the atrium, anterograde or retrograde ERP of the AV node, and the longest attainable AH interval. The HRA-CSd interval and HRA-CSp interval also did not differ significantly among these groups. The cycle length and the frequency of the tachycardia for which the patient underwent electrophysiological study were also similar in the three groups.

The electrophysiological properties of the AV node in patients with dual AV node physiology were compared between groups A and D. No dif-

Table 4. Electrophysiological Characteristics by Study Group

	Group A (n = 13)	Group B (n = 18)	Group D (n = 12)
AH interval (msec)	76 ± 19	77 ± 17	84 ± 15
1:1 AV (msec)	400 ± 38	362 ± 66	396 ± 66
ERP of the atrium (msec)	222 ± 32	232 ± 29	221 ± 31
ant. ERP of the AVN (msec)	247 ± 53	269 ± 60	269 ± 43
ant. ERP of the fast pathway (msec)	310 ± 60	—	323 ± 71
ret. ERP of the AVN (msec)	326 ± 112	340 ± 100	435 ± 170
AH interval of the slow pathway (msec)	261 ± 73	—	234 ± 45
Longest attainable AH interval (msec)	293 ± 110	236 ± 89	295 ± 74
HRA-CSd interval (msec)	44 ± 13	47 ± 14	55 ± 21
HRA-CSp interval (msec)	60 ± 13	61 ± 13	64 ± 15
TCL (msec)	344 ± 41	328 ± 28	367 ± 61
Frequency (per year)	22 ± 25	14 ± 13	12 ± 16

Data are expressed as mean ± SD. 1:1 AV = the minimal cycle length in which 1:1 AV conduction was maintained during rapid HRA pacing; AH interval = atrio-His interval; AH interval of the slow pathway = AH interval when the jumping phenomenon was observed during atrial extrastimulus testing; ant. = anterograde; AVN = atrioventricular node; ERP = effective refractory period; Frequency = frequency of tachycardia per year; HRA-CSd interval = the interval between the atrial signal in the high right atrium and that in the distal coronary sinus catheter; HRA-CSp interval = the interval between the atrial signal in the high right atrium and that in the proximal coronary sinus catheter; Longest attainable AH interval = the longest AH interval during the series of atrial extrastimulus testing, ret. = retrograde; TCL = tachycardia cycle length.

ferences in AV node properties, in patients with or without AVNRT, were found. Ten of the 13 patients in group A (77%) and eleven of the 12 patients in group D (92%) demonstrated anterograde dual AV node pathways in the baseline state. Five patients in group A (38%) and six patients in group D (50%) demonstrated retrograde dual AV node pathways at baseline.

DISCUSSION

This appears to be the first report on the structural characteristics of the coronary sinus in patients with AVNRT as compared with patients with other types of PSVT, normal subjects, and patients with dual AV node physiology but noninducible AVNRT. This study also investigated the relationship between the diameter of the coronary sinus and the size of the atrium, as well as electrophysiological characteristics.

Morphological characteristics of the coronary sinus and the pathogenesis of the arrhythmia. Recent studies have reported the relationship between the pathogenesis of arrhythmia and structural characteristics of the human heart[6,16,31]. In patients with Wolff-Parkinson-White syndrome, who have posteroseptal accessory pathways, coronary sinus diverticula are frequently observed in coronary sinus angiography. In such cases, radiofrequency energy can be successfully delivered in the neck of the coronary sinus diverticulum or the midcardiac vein[6,31,36].

Chiang et al[7] reported that the incidence of major coronary sinus abnormalities in 408 patients with PSVT was 2.9%, and such abnormalities were more common in patients with accessory pathways than in patients with AVNRT. The abnormalities, including hypoplasia, angulation, localized narrowing, fistula and diverticulum, were seen in close relation to left-sided accessory pathways. This is logical, because the coronary sinus is a remnant of the left superior vena cava. The coronary sinus itself and its afferent branches may be the anatomic substrate for accessory AV connections. On the other hand, in patients with AVNRT, the incidence of such coronary sinus abnormalities was rare. Two of our 58 patients (3.4%) had similar major coronary sinus abnormalities. One was a coronary sinus diverticulum corresponding to an accessory pathway in the left posteroseptal region, and the other was a localized narrowing in the left posterolateral region.

Relationship between increased ostial diameter of the coronary sinus and the pathogenesis of AVNRT. McGuire et al[28] determined the dimensions of Koch's triangle in 65 human adult hearts during surgery for arrhythmia or postmortem examination. The mean length of the triangle (measured from the central fibrous body to the nearest edge of the coronary sinus) was 17 ± 3 mm; the mean height (measured from the tricuspid annulus to the nearest edge of the coronary sinus) was 13 ± 3 mm. These investigators did not

measure the dimension of the coronary sinus, or assess it in relation to arrhythmia.

Doig et al[13] performed direct coronary sinus angiography in patients with AVNRT and a control population with other tachyarrhythmias. The ostium in their patients with AVNRT was 44% larger than that in the control population, which included patients with AV reentrant tachycardia, paroxysmal atrial fibrillation, atrial tachycardia and ventricular tachycardia. The coronary sinus resembled a wind sock in patients with AVNRT and was tubular in patients with other forms of supraventricular tachycardia. In this study, furthermore, we investigated the diameter of the coronary sinus and the height of Koch's triangle in patients without PSVT, who served as normal controls, and in patients with dual AV node physiology but noninducible AVNRT. In our study, the diameter of the distal coronary sinus and the height of Koch's triangle were similar among all groups, but the ostial diameter was larger in patients with AVNRT compared with patients with other forms of PSVT and patients without tachyarrhythmia.

We measured the p wave duration and the interval between the atrial signal in the HRA catheter and that in the distal and proximal coronary sinus catheter. These were electrophysiological markers reflecting the size of the supraventricular cardiac system. Cycle length and the frequency of tachycardia were also compared. There were no differences in these parameters among our groups of study subjects. The results indicate that there is regional stretch of the base of Koch's triangle only in patients with AVNRT. The increase in ostial diameter was not correlated with the size of the atrium nor with tachycardia itself. Our study subjects were limited to patients with structurally normal hearts because the coronary venous circulation system and the ostial diameter may be affected by cardiomegaly due to chronic or incessant tachycardia, moderate or severe tricuspid valve regurgitation, cardiomyopathy or severe ischemic heart disease.

Although AVNRT can present at any age, the incidence of AVNRT is low in young children and infants[8,9,26]. Ko et al[26] found tachycardia due to reentry within the AV node in 13% of children who underwent electrophysiological study. On the other hand, AVNRT is the most common form of regular narrow QRS tachycardia in older children and adults. The propensity to tachycardia due to AV node reentry develops postnatally[5,8,9,26] and we speculate that there may be an age-dependent structural, functional or autonomic property in patients with the potential substrate of AV node reentry.

A previous study reported that anterograde slow pathway conduction time depends on length rather than functional properties[15]. In the process of postnatal cardiac development, the regional stretch of Koch's triangle may provide a potential substrate for AV node reentry. A wave front passing through the "slow pathway" might run a more extended course. Animal models of dual AV node pathways indicate that atrial reentry may occur around the anatomic obstacle provided by the ostium of the coronary sinus[20,30]. Our study provides strong evidence that the base of Koch's triangle is enlarged in patients with AVNRT. It suggests that the mechanism for AVNRT is related to the available length of the perinodal loop.

Provocation of the dual pathway physiology by atrial extrastimulus in patients without AVNRT or other forms of PSVT[4,18,24] supports the hypothesis that the structural and functional substrate of dual pathway physiology is in fact potentially present in everyone. Slow pathway physiology may exist in the normal heart as the second input of the AV node; it may not be a specific electrophysiological phenomenon in patients with AVNRT. In some of our patients in group B, the longest attainable AH interval was as great as that in patients with dual AV node pathways. Whether our patients had dual pathways was determined only by the standard electrophysiological definition[24]. There may have been two distinct pathways, representing fast and slow pathways, in our study patients. Some patients with smooth AV node function curves had initiation of AVNRT and a loss of the tail of the curve after a successful slow pathway ablation[33,35]. Dual AV node pathways may be made manifest by manipulating the autonomic tone in such patients[2,3,19].

Recent studies have revealed that the reentrant circuit in AVNRT is comprised of not only the compact AV node but also the perinodal myocardium[14,21,27]. Despite excellent success in abolishing AVNRT by radiofrequency catheter ablation of the slow pathway[17,21,23,25], the precise pathophysiology of the arrhythmia and the structural and electrophysiological substrate of dual AV node physiology are still unclear. Why the diameter of the coronary sinus ostium becomes larger in patients with AVNRT is not known. The findings of this study suggest implications for the mechanism and pathogenesis of AVNRT, that is, patients with clinical AVNRT have a larger coronary sinus ostial diameter compared with patients without AVNRT and this may become a potential substrate for AVNRT. As one grows older, if the ostial diameter increases so that the prolonged pathway becomes an obstacle, relative differences in conduction properties and refractoriness between the fast and the slow pathways may become adequate to maintain the stable reentrant circuit with alteration of autonomic nervous system input into the AV node. In that case, AVNRT may be observed.

Study Limitations. This study has several limitations: 1) The height of Koch's triangle is indi-

rectly estimated because the His amplitude does not necessarily determine the apex of the triangle of Koch. 2) Although in some of our study patients the ostial diameter of the coronary sinus was as large as that in group A patients, they did not exhibit AVNRT nor dual AV node pathway. In these cases, increased ostial diameter alone may not be enough to discriminate patients that present AVNRT and dual AV node pathways. 3) The patients with chest pain syndrome in group C did not undergo electrophysiological study, because such examinations are unethical in patients with no arrhythmia. Thus, whether some patients in group C had dual AV node pathways is unknown. 4) Because the patients were not heavily sedated in our study protocol, heightened sympathetic tone in unsedated patients undergoing electrophysiological study may obscure anterograde and retrograde slow pathway conduction[22]. 5) Group D included patients with spontaneous episodes of PSVT who had evidence of electrophysiologically proven dual AV node pathways but noninducible AVNRT in the electrophysiological laboratory. It is uncertain whether spontaneously initiated PSVT is a form of AVNRT.

ACKNOWLEDGMENTS

We gratefully thank Dr. Hiroshi Nakagawa, University of Oklahoma Health Sciences Center, Oklahoma city, Oklahoma, for his advice and critical review of the manuscript. We also thank Drs. Hitoshi Fujiwara, Yuji Muraoka, Masaya Kato, Norio Ishioka and Yukiko Nakano for their excellent technical assistance and Yuko Ohmura for her secretarial assistance.

(Received November 19, 1997)
(Accepted December 24, 1997)

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