JACC Vol. 28, No. 3
September 1996:732–7

Electrophysiologic Characteristics, Electropharmacologic Responses and Radiofrequency Ablation in Patients With Decremental Accessory Pathway

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

Taipei, Taiwan, Republic of China

Objectives. **This study sought to characterize the functional properties of decremental accessory atrioventricular (AV) pathways and to investigate their pharmacologic responses.**

Background. **Although decremental AV pathways associated with incessant reciprocating tachycardia have been studied extensively, information about the electrophysiologic characteristics and pharmacologic responses of anterograde and retrograde decremental AV pathways is limited.**

Methods. **Of 759 consecutive patients with accessory pathway-mediated tachyarrhythmia, 74 with decremental AV pathways were investigated (mean age 43 ± 18 years). After baseline electrophysiologic study, the serial drugs adenosine, verapamil and procainamide were tested during atrial and ventricular pacing. Finally, radiofrequency catheter ablation was performed.**

Results. **Five patients had anterograde decremental conduction over the accessory pathway but had no retrograde conduction. Of the 64 patients with retrograde decremental conduction over the accessory pathway, anterograde conduction over the pathway was absent in 41 (64%), intermittent in 5 (8%) and nondecremental in 18 (28%). In the remaining five patients, anterograde and retrograde decremental conduction over the same pathway was found. The anterograde and retrograde conduction properties and extent of decrement did not differ between anterograde and retrograde decremental pathways. Posteroseptal pathways had the highest incidences of anterograde and retrograde decremental conduction. Intravenous adenosine, procainamide and verapamil caused conduction delay or block, or both, in 10 of 10, 10 of 10 and 4 of 10 of the anterograde and 20 of 20, 20 of 20 and 8 of 20 of the retrograde decremental pathways, respectively. All patients had successful ablation of the decremental pathways without complications. During the follow-up period of 31 ± 19 months, only one patient experienced recurrence.**

Conclusions. **Decremental accessory pathways usually had functionally distinct conduction characteristics in the anterograde and retrograde directions. Their pharmacologic responses suggested the heterogeneous mechanisms of decremental conduction.**

(J Am Coll Cardiol 1996;28:732–7)

Anterograde and retrograde conduction at a constant interval during incremental pacing and programmed cardiac stimulation has been considered to be characteristic of conduction over an accessory atrioventricular (AV) pathway (1). Nevertheless, electrophysiologic studies have shown the presence of accessory pathways, including nodoventricular and atriofascicular pathways, with AV node-like properties and long conduction times in patients with incessant AV reciprocating tachycardia and Mahaim fibers (2–7). However, information about the electrophysiologic characteristics and pharmacologic responses of anterograde and retrograde decremental AV pathways was limited and unclear (8–12).

The purposes of this study were to characterize the functional properties of decremental accessory pathways and to investigate their responses to adenosine, verapamil and procainamide in a large series of patients with Wolff-Parkinson-White syndrome.

Methods

Patient characteristics. Of the 759 consecutive patients with accessory pathway-mediated tachyarrhythmia referred to Veterans General Hospital-Taipei for electrophysiologic study and radiofrequency catheter ablation, 74 (9.7%) had decremental AV pathways (without Mahaim fibers) (32 male, 42 female; mean age 43 ± 18 years, range 10 to 82). All were refractory to or intolerant of 2 ± 1 (range 1 to 4) antiarrhythmic drugs. The other 685 patients were designated as the control group for comparison.

Electrophysiologic study. All patients were studied in the postabsorptive, nonsedated state after written informed con-

From the Division of Cardiology, Department of Medicine, National Yang-Ming University 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, 85-2331-B-010-048 from the National Science Council, Taipei, Taiwan, Republic of China.

Manuscript received January 9, 1996; revised manuscript received April 3, 1996, accepted April 16, 1996.

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

0735-1097/96/$15.00
PII S0735-1097(96)00219-7

sent was obtained. Details of the electrophysiologic study were described previously (13,14). In brief, baseline electrophysiologic studies were performed after antiarrhythmic drugs had been discontinued for at least five half-lives in all patients. The electrocardiogram (ECG) and femoral artery pressure were continuously monitored throughout the procedures. In brief, three multipolar catheters (interelectrode space 2 mm; Mansfield, Boston Scientific) were introduced from the femoral veins and placed in the right atrium, His bundle area and right ventricle for recording and stimulation. One or two orthogonal electrode catheters (Mansfield) were used for coronary sinus recording; these were percutaneously introduced into the internal jugular vein and placed in the coronary sinus to record left atrial activation. Intracardiac electrograms were simultaneously recorded with surface ECG leads I, II and V_1 on a multichannel oscilloscope recorder (Electronics for Medicine, PPG, VR-13 or MIDAS 2500) at a paper speed of 100 to 150 mm/s using a filter frequency setting of 30 to 500 Hz.

A programmed digital stimulator (DTU mode 210 or 215, Bloom Associates, Ltd.) was used to deliver electrical impulses of 2.0 ms at twice the late diastolic threshold. The standard protocol included 1) atrial and ventricular incremental pacing at cycle lengths ranging from just under that of sinus rhythm to the minimal cycle lengths maintaining AV and ventriculoatrial (VA) 1:1 conduction, respectively; 2) single and double atrial extrastimuli delivered during high right atrial pacing at one or two cycle lengths and during sinus rhythm; and 3) single and double ventricular extrastimuli delivered during right ventricular apical pacing at cycle lengths of 600 and 400 ms and during sinus rhythm. Left atrial stimulation was also performed. For induction of atrial flutter-fibrillation, atrial burst pacing was performed if atrial extrastimuli could not induce flutter-fibrillation. Intravenous isoproterenol (1 to 4 μg/min) or atropine (0.02 to 0.04 mg/kg body weight), or both, was used to facilitate induction of tachyarrhythmias.

Pharmacologic study. All patients with anterograde decremental pathways and 20 patients with retrograde decremental pathways received serial drug tests. On the first day of the study, adenosine and verapamil were given intravenously to each patient. On the third day, procainamide was administered intravenously. The effects of intravenous adenosine, verapamil and procainamide on anterograde decremental pathways were evaluated during atrial pacing at a cycle length 20 ms longer than the shortest paced cycle length with 1:1 accessory pathway conduction. The effects of these drugs on retrograde decremental pathway were evaluated during ventricular pacing at a cycle length 20 ms longer than the shortest paced cycle length with 1:1 accessory pathway conduction. After intravenous infusion of verapamil and procainamide, accessory pathway conduction properties were evaluated again, as in previous protocols.

Adenosine was injected into the femoral vein and flushed with 10 ml of saline. Additional incremental doses of adenosine (37.5 μg/kg body weight) were injected until block of accessory pathway conduction occurred (37.5 to 150 μg/kg). The dosage of intravenous verapamil was 0.15 mg/kg body weight over a period of 2 min. Procainamide (10 to 12 mg/kg) was given intravenously as a 50-mg bolus every 2 min (15).

Radiofrequency ablation. As described previously, a 7F quadripolar electrode catheter with a 4-mm distal electrode (interelectrode space 2 mm) and a deflectable curve was used for ablation (13,14). Radiofrequency current (generated from Radionic-3C) was delivered between the tip electrode and an indifferent patch electrode positioned on the left side of the posterior chest wall. The unit was coupled to a device that provided real-time monitoring of root-mean-square voltage, current and impedance. The ablation techniques and biophysical variables of radiofrequency energy used in various types of tachycardia have been described previously (13,14). In each patient a possible accessory pathway potential, local electrograms showing fusion of A and V waves with the V wave earlier than the delta wave (for anterograde conduction) or local electrograms showing the earliest A waves during ventricular pacing or reciprocating tachycardia (for retrograde conduction) were used to guide the ablation catheter. After the ablation procedure, isoproterenol (1 to 4 μg/min) was administered 20 to 30 min later to ensure successful results. If the delta wave or any tachycardia recurred, mapping and ablation procedures were repeated. The successful ablation sites were recorded in the cine films.

Definitions. *Decremental conduction* was considered to be present in the accessory pathways if the following criteria were satisfied: 1) rate-dependent prolongation of VA or atrial-delta wave intervals by >30 ms as measured in the electrogram nearest the accessory pathway, or 2) Wenckebach anterograde or retrograde block (11). In the case of septal accessory pathways where retrograde conduction could be confused with retrograde AV node conduction, atrial pre-excitation or prolongation of the VA interval without change in the retrograde activation sequence after ventricular extrastimuli delivered during AV reciprocating tachycardia at a time when the His bundle was refractory was considered to occur over the accessory pathway (Fig. 1). For patients with intact retrograde conduction over the AV node, changes in the VA interval during ventricular pacing or extrastimulation might be misleading because of changing degrees of atrial fusion. VA conduction curves before and after successful ablation of the accessory pathway were compared to prove the decremental conduction of the accessory pathway.

Retrograde decremental pathways were divided into two types according to the clinical presentation. The retrograde pathways in patients with permanent junctional reciprocating tachycardia belonged to major decremental type, and those in other patients belonged to minor decremental type.

Statistical analysis. Data were expressed as mean value ± SD. Differences in continuous variables were analyzed by the paired and unpaired *t* test, and differences in categorical variables were analyzed by chi-square analysis with the Yates correction or the Fisher exact test. A p value <0.05 was considered statistically significant.

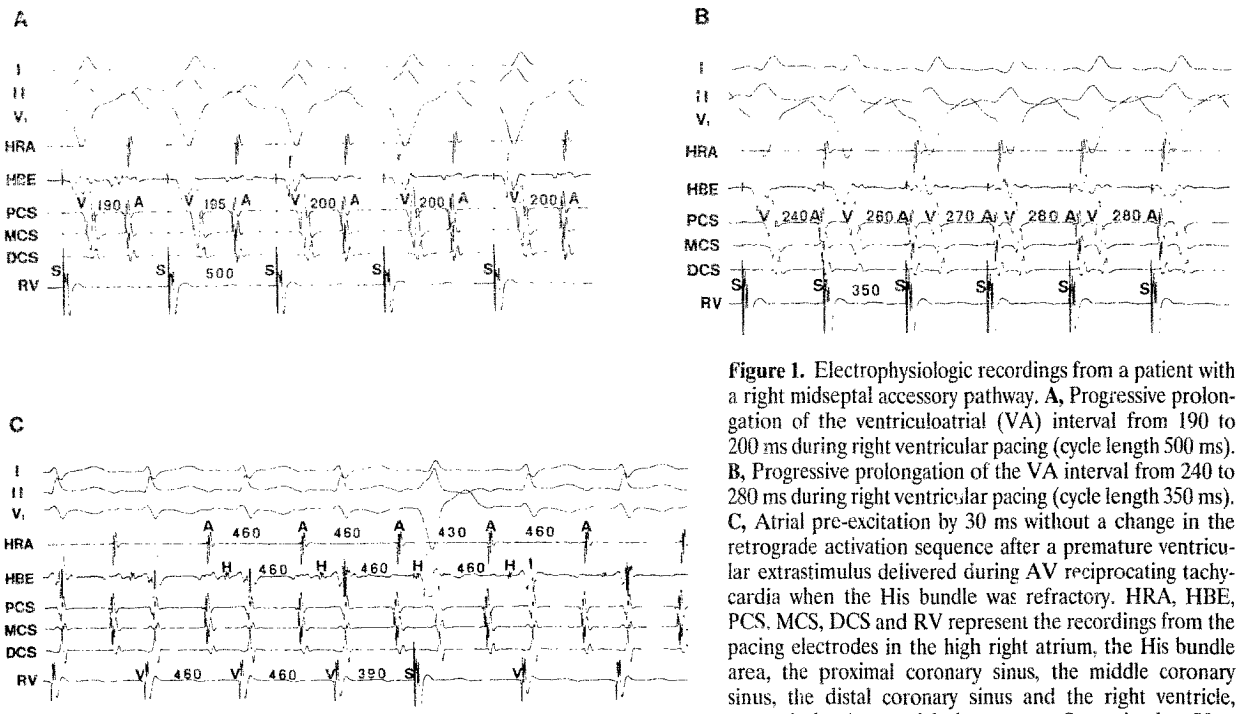

Figure 1. Electrophysiologic recordings from a patient with a right midseptal accessory pathway. **A,** Progressive prolongation of the ventriculoatrial (VA) interval from 190 to 200 ms during right ventricular pacing (cycle length 500 ms). **B,** Progressive prolongation of the VA interval from 240 to 280 ms during right ventricular pacing (cycle length 350 ms). **C,** Atrial pre-excitation by 30 ms without a change in the retrograde activation sequence after a premature ventricular extrastimulus delivered during AV reciprocating tachycardia when the His bundle was refractory. HRA, HBE, PCS, MCS, DCS and RV represent the recordings from the pacing electrodes in the high right atrium, the His bundle area, the proximal coronary sinus, the middle coronary sinus, the distal coronary sinus and the right ventricle, respectively. A = atrial electrogram; S = stimulus; V = ventricular electrogram.

Results

Clinical and electrophysiologic characteristics. Decremental conduction was present in the anterograde direction exclusively in 5 patients (5 pathways), retrograde direction exclusively in 64 patients (64 pathways) and in both directions in 5 patients (5 pathways). The documented tachyarrhythmias in these patients included AV reciprocating tachycardia (45 patients), atrial fibrillation (5 patients), atrial fibrillation and AV reciprocating tachycardia (15 patients) and the permanent form of junctional reciprocating tachycardia (9 patients). The clinical tachycardia was induced in all patients during electrophysiologic study.

Anterograde decremental conduction (five patients). The shortest atrial paced cycle length with 1:1 anterograde conduction over the accessory pathway was 306 ± 49 ms, and the anterograde accessory pathway effective refractory period was 266 ± 46 ms. Retrograde conduction over the accessory pathway could not be demonstrated in these five patients. Anterograde accessory pathway conduction curves were continuous in all pathways. The maximal prolongation of atrial electrogram to delta wave interval was 62 ± 31 ms.

Retrograde decremental conduction (64 patients). The shortest ventricular paced cycle length with 1:1 retrograde conduction over the accessory pathway was 332 ± 93 ms, and the retrograde accessory pathway effective refractory period was 272 ± 54 ms. Retrograde accessory pathway conduction curves during ventricular extrastimulus testing were continuous in all cases. Anterograde conduction over the accessory pathway was absent in 41 patients (64%), and intermittent pre-excitation was present in 5 patients (8%). In the remaining 18 patients (28%) with nondecremental anterograde conduction, the shortest atrial paced cycle length with 1:1 anterograde conduction over the pathways was 332 ± 96 ms, and the anterograde accessory pathway effective refractory period was 292 ± 57 ms. Anterograde and retrograde conduction properties, including 1:1 conduction and effective refractory period, were not significantly different ($p > 0.05$). The maximal prolongation of the VA interval was 64 ± 30 ms. The anterograde conduction properties, retrograde conduction properties and extent of decrement did not differ between anterograde and retrograde decremental pathways (Table 1).

Table 1. Electrophysiologic Characteristics in Patients With Decremental Accessory Pathways

	Anterograde Decrement (n = 10)	Retrograde Decrement (n = 64)	p Value
Anterograde conduction			
AP 1:1 (ms)	308 ± 72	332 ± 96	0.387
APERP (ms)	277 ± 69	292 ± 57	0.268
Retrograde conduction			
AP 1:1 (ms)	272 ± 49	332 ± 93	0.189
APERP (ms)	252 ± 45	272 ± 54	0.591
Extent of decrement (ms)	63 ± 22	64 ± 30	0.642

Data are expressed as mean value ± SD. AP = accessory pathway; AP 1:1 = shortest paced cycle length maintaining 1:1 conduction over the accessory pathway; APERP = accessory pathway effective refractory period.

Table 2. Decremental Accessory Pathway Location

	Anterograde Conduction		Retrograde Conduction	
	Decrement (n = 10)	Control (n = 400)	Decrement (n = 69)	Control (n = 616)
LFW	2 (20%)	245 (61%)	13 (19%)	386 (63%)
RFW	2 (20%)	81 (20%)	21 (30%)	105 (17%)
PS	5 (50%)	56 (14%)	30 (43%)	94 (15%)
AMS	1 (10%)	18 (5%)	5 (8%)	31 (5%)
p value	0.043		<0.001	

AMS = anteromidseptal; LFW = left free wall; PS = posteroseptal; RFW = right free wall.

Decremental conduction in both anterograde and retrograde directions (five patients). The shortest atrial paced cycle length with 1:1 anterograde conduction over the accessory pathway was 310 ± 97 ms, and the anterograde accessory pathway effective refractory period was 288 ± 91 ms. The shortest ventricular paced cycle length maintaining 1:1 retrograde conduction over the accessory pathway was 272 ± 49 ms, and the retrograde accessory pathway effective refractory period was 252 ± 45 ms. The maximal prolongation of atrial electrogram to delta wave interval and VA interval was 58 ± 18 ms and 72 ± 23 ms, respectively.

Comparisons with the control group. The locations of anterograde and retrograde decremental pathways were significantly different from the locations of the pathways in the control group (Table 2). Anterograde decremental conduction occurred mostly in the posteroseptal pathways. Retrograde decremental conduction occurred mostly in the posteroseptal and right free wall pathways.

Pharmacologic responses. *Anterograde decremental pathways.* After adenosine administration (minimal dose 4.2 ± 1.5 mg), anterograde Wenckebach conduction block of decremental pathways during atrial pacing could be demonstrated in all 10 patients (Fig. 2). After procainamide infusion (750 ± 18 mg), progressive prolongation of the atrial electrogram to delta wave interval was found in eight patients, and Wenckebach conduction block was noted in two patients. After verapamil administration (10 ± 2 mg), anterograde Wenckebach conduction block of decremental pathways was found in four patients, and in the remaining six patients, neither prolongation of the atrial electrogram to delta wave interval nor conduction block was found. The shortest atrial paced cycle length with 1:1 accessory pathway conduction and effective refractory period increased significantly after verapamil and procainamide infusion.

Retrograde decremental pathways. After adenosine administration (minimal dose 4.5 ± 1.3 mg), retrograde Wenckebach conduction block during ventricular pacing was found in 18 patients and sudden conduction block in 2 patients. After procainamide infusion (762 ± 14 mg), progressive prolongation of the VA interval was found in 14 patients and Wenckebach conduction block in 6 patients. After verapamil administration (9.6 ± 1.8 mg), retrograde Wenckebach conduction block was demonstrated in 8 patients, whereas in the remaining 12 patients, the VA interval did not change. The shortest ventricular paced cycle length with 1:1 accessory pathway conduction and effective refractory period increased significantly after verapamil and procainamide infusion. Comparisons of electropharmacologic characteristics between major and minor decremental pathways showed a larger extent of decrement, a longer VA interval during tachycardia and a higher incidence of verapamil-induced conduction block in the major decremental pathway (Table 3).

Radiofrequency ablation and clinical follow-up. All patients had successful ablation of the decremental accessory pathways. No complication occurred during each ablation session. A suspected accessory pathway potential was recorded at the successful target site in 23 (82%) of 28 patients with a manifest pathway and in 35 (76%) of 46 patients with a concealed pathway or intermittent pre-excitation. In the remaining 16 patients without registered accessory pathway potential, the A/V ratio at the successful ablation site was 0.28 ± 0.16 for manifest pathways, and the V/A ratio was 3.54 ± 2.62 for concealed pathways. The mean pulse number was 5 ± 4, the fluoroscopy time was 36 ± 21 min and the total procedure time was 2.8 ± 1.4 h. After ablation, no reciprocating tachycardia was induced, but atrial fibrillation was still inducible in 6 of 20 patients with a clinical history of this arrhythmia.

During the follow-up period of 31 ± 19 months (range 4 to 68), only one patient experienced recurrence of accessory pathway conduction with reciprocating tachycardia. She had a successful result during the second ablation procedure.

Discussion

Major findings. The incidence of decremental accessory pathways in patients with Wolff-Parkinson-White syndrome was 9.7%. However, decremental conduction rarely occurred in both the anterograde and retrograde directions within the same pathway (0.7%). Posteroseptal pathways had the highest incidences of anterograde and retrograde decremental conduction. Adenosine and procainamide had a direct inhibitory effect on decremental pathway conduction, whereas verapamil's inhibitory effect was less.

Electrophysiologic characteristics and pathway locations. Decremental conduction over an accessory pathway had typically been associated with the presence of Mahaim fibers and the permanent form of junctional reciprocating tachycardia, but it was reported infrequently in patients with paroxysmal AV reciprocating tachycardia. Gillette et al. (9) first reported four patients who had right anterior accessory connections with long conduction times and decremental conduction and recurrent wide QRS tachycardia. These workers suggested that these pathways represent the remnants of anterior AV node tissue (9). Centurion et al. (10) have demonstrated that in left-sided AV pathways, 7% of anterograde conduction and 15% of retrograde conduction had decremental conduction (maximal decrement ≥30 ms). Murdork et al. (11) have shown that the incidence of decremental accessory pathways was

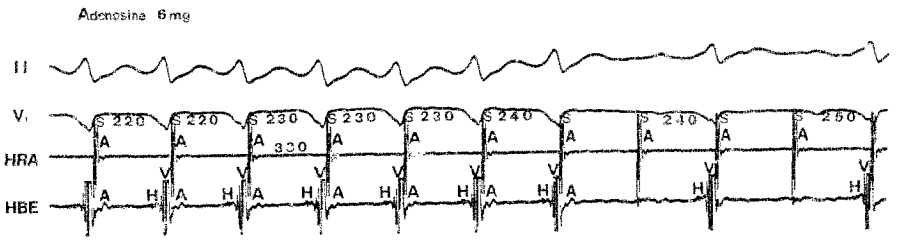

Figure 2. Anterograde Wenckebach conduction block in a right free-wall accessory pathway by intravenous adenosine. Abbreviations as in Figure 1.

7.6%; right parietal pathways had the highest incidence of anterograde decremental conduction, and left parietal pathways had the highest incidence of retrograde decremental conduction.

In the present study, a clinical pattern consistent with the permanent form of junctional reciprocating tachycardia was present in 9 patients (12%). In the remaining 65 patients, paroxysmal AV reciprocating tachycardia or atrial fibrillation, or both, was found. The former had a larger extent of decrement and a longer VA interval during reciprocating tachycardia. Posteroseptal pathways had the highest incidences of anterograde and retrograde decremental conduction. However, decremental conduction rarely occurred in both the anterograde and retrograde directions within the same pathway. Although decremental conduction was present in one direction, conduction was often absent in the other. These findings suggest that conduction over the accessory pathway in anterograde and retrograde directions may be functionally distinct.

Electropharmacologic characteristics. In the present study, adenosine infusion produced conduction block in all decremental pathways, and procainamide infusion prolonged the effective refractory periods and conduction times of these pathways, whereas verapamil had a direct inhibitory effect on these pathways in only 12 of 30 patients (40%). The direct effects of adenosine have been demonstrated to result in activation of the potassium current, which hyperpolarizes the cell membrane to near E_K and depresses the action potential in the AV node cells (16). Because adenosine produced conduction block in all decremental pathways in this study, it was proposed that decremental conduction is due to partial depolarization in the anomalous atrial fascicles (12). This hypothesis is also consistent with previous observations in which adenosine was found to have no effect on normally conducting, nondecremental accessory pathways with rest membrane potential near E_K (17). Previous reports have demonstrated that procainamide may impair or abolish anterograde conduction over accessory pathways and has more marked effects on anterograde than on retrograde accessory pathway refractoriness (15,18–20). In the present study, procainamide was equally effective in impairing or blocking anterograde and retrograde decremental pathways and major and minor decremental pathways. These adenosine-sensitive and procainamide-sensitive characteristics suggest that the decremental AV pathways were mainly composed of depressed fast-channel fibers. In contrast, only some of the decremental AV pathways, especially those with permanent junctional reciprocating tachycardia (major decremental pathways), had responses to verapamil, which is a calcium channel blocking agent. This finding might suggest that major decremental AV pathways consist of more slow-channel fibers (AV node-like cells) than do minor decremental pathways (21–24). Furthermore, this may explain why the major decremental pathways had a larger extent of decrement and a longer VA interval during reciprocating tachycardia.

Table 3. Comparisons of Electrophysiologic Chracteristics and Pharmacologic Responses Between Major and Minor Decremental Pathways

	Major (n = 9)	Minor (n = 60)	p Value
Tachycardia CL (ms)	410 ± 35	406 ± 32	0.642
Retrograde AP 1:1 (ms)	320 ± 92	335 ± 96	0.856
Extent of decrement (ms)	82 ± 26	60 ± 24	0.015
VA interval during tachycardia (ms)	302 ± 17	275 ± 20	0.001
VA prolongation or VA block			
Adenosine	5/5 (100)	15/15 (100)	0.109
Procainamide	5/5 (100)	15/15 (100)	0.109
Verapamil	4/5 (80)	4/15 (26)	0.033

Data are expressed as mean value ± SD or number (%) of patients. AP = accessory pathway; CL = cycle length; VA = ventriculoatrial.

Possible mechanisms of decremental conduction. The explanation for the occurrence of decrement over an accessory pathway is not known. Several possible mechanisms have been proposed: 1) In the present study and in previously reported cases, posteroseptal pathways located in close proximity to the AV node had the highest incidence of decremental conduction, raising the possibility that the pathway is an AV node-like structure (22,23). Furthermore, previous pathologic studies in patients with decrementally conducting accessory pathways showed that AV node-like cells were identified in some cases (24). 2) Decremental conduction over the accessory pathways in one direction but not in the other may be due to accessory pathway geometry or accessory pathway fiber orientation. Pathologic examination of septal accessory pathways in patients with the permanent form of junctional reciprocating tachycardia has demonstrated that the accessory pathway took a tortuous course with a concomitant change in axial resistance, providing further support that accessory pathway geom-

etry may contribute to decremental conduction (5). 3) A role for impedance mismatch between the accessory pathway and the atrium or ventricle has also been suggested (25,26).

Study limitations. Because the rate of administration of procainamide was cautiously slow in the present study, this minimized the opportunity for causing block in the accessory pathway. With rapid infusion rate (e.g., ≥100 mg/min), conduction block might occur at the time of peak plasma levels (27). The present study did not determine the site of conduction delay; atrial and ventricular pacing maneuvers could be helpful when pathway potentials were registered.

Conclusions. Decremental accessory pathways were not common in patients with Wolff-Parkinson-White syndrome. These pathways usually had functionally distinct conduction characteristics in the anterograde and retrograde directions. Most decremental pathways were located in the posteroseptal area. Adenosine and procainamide had a direct inhibitory effect on these pathways, but verapamil's inhibitory effect was less.

References

1. Gallagher JJ, Pritchett ELC, Sealy WC, Kasell J, Wallace AG. The preexcitation syndrome. Prog Cardiovasc Dis 1978;20:285–327.
2. Gallagher JJ, Sealy WC. The permanent form of junctional reciprocating tachycardia: further elucidation of the underlying mechanism. Eur Heart J 1978;8:413–30.
3. Epstein ML, Stone FM, Benditt DG. Incessant atrial tachycardia in childhood: association with rate-dependent conduction in an accessory atrioventricular pathway. Am J Cardiol 1979;44:498–504.
4. Brugada P, Vanagt EJ, Bar FW, Wellens HJJ. Incessant reciprocating atrioventricular tachycardia. Factors playing a role in the mechanism of the arrhythmia. PACE 1980;3:670–7.
5. Critelli G, Gallagher JJ, Monda V, Coltorti F, Scherillo M, Rossi L. Anatomic and electrophysiologic substrate of the permanent form of junctional reciprocating tachycardia. J Am Coll Cardiol 1984;4:601–6.
6. Gallagher JJ, Smith WM, Kasell JH, Benson DW, Sterba R, Grant AO. Role of Mahaim fibers in cardiac arrhythmias in man. Circulation 1981;64:176–89.
7. Tchou P, Lehmann MH, Jazayeri M, Akhtar M. Atriofascicular connection or a nodoventricular Mahaim fiber? Electrophysiologic elucidation of the pathway and associated reentrant circuit. Circulation 1988;77:837–48.
8. Farre J, Ross D, Wiener I, Bar FW, Vanagt EJ, Wellens HJJ. Reciprocal tachycardia using accessory pathways with long conduction time. Am J Cardiol 1979;44:1099–1109.
9. Gillette PC, Garson A, Cooley D, McNamara DG. Prolonged and decremental antegrade conduction properties in right anterior accessory connections: wide QRS antidromic tachycardia of left bundle branch block pattern without Wolff-Parkinson-White configuration in sinus rhythm. Am Heart J 1982;103:66–74.
10. Centurion OA, Fukatani M, Shimizu A, et al. Anterograde and retrograde decremental conduction over left-sided accessory atrioventricular pathways in the Wolff-Parkinson-White syndrome [abstract]. Am Heart J 1993;125:1038.
11. Murdock CT, Leitch JW, Teo WS, Sharma AD, Yee R, Klein G. Characteristics of accessory pathways exhibiting decremental conduction. Am J Cardiol 1991;67:506–10.
12. Lerman BB, Greenmerg M, Overholt ED, et al. Differential electrophysiologic properties of decremental retrograde pathways in long RP tachycardia. Circulation 1978;76:21–31.
13. Chen SA, Tsang WP, Hsia CP, et al. Catheter ablation of accessory atrioventricular pathways in 114 symptomatic patients with Wolff-Parkinson-White syndrome: a comparative study of direct-current and radiofrequency ablation. Am Heart J 1992;124:356–65.
14. Chen SA, Chiang CE, Yang CJ, et al. Accessory pathway and atrioventricular nodal reentrant tachycardia in elder patients: clinical features, electrophysiological characteristics and results of radiofrequency ablation. J Am Coll Cardiol 1994;23:702–8.
15. Sellers TD, Campbell RFW, Bashmore TM, Gallagher JJ. Effects of procainamide and quinidine sulphate in the Wolff-Parkinson-White syndrome. Circulation 1977;55:15–22.
16. Lerman BB, Belardinelli L. Cardiac electrophysiology of adenosine: basic and clinical concepts. Circulation 1991;83:1499–1509.
17. DiMarco JP, Sellers TD, Lerman BB, Greenberg ML, Berne RM, Belardinelli L. Diagnostic and therapeutic use of adenosine in patients with supraventricular tachyarrhythmias [abstract]. J Am Coll Cardiol 1985;6:417.
18. Boahene KA, Klein GJ, Yee R, Sharma AD, Fujimura S. Termination of acute atrial fibrillation in the Wolff-Parkinson-White syndrome: importance of atrial fibrillatory cycle length. J Am Coll Cardiol 1990;16:1408–14.
19. Wellens HJJ, Durre D. Effect of procainamide, quinidine and ajmaline in the Wolff-Parkinson-White syndrome. Circulation 1974;50:114–20.
20. Leitch JW, Klein GJ, Yee R, Feldman RD, Brown J. Differential effect of intravenous procainamide on antegrade and retrograde accessory pathway refractoriness. J Am Coll Cardiol 1992;19:118–24.
21. Tai DY, Chang MS, Svinarich JT, Chiang BN, Sung RJ. Mechanisms of verapamil-induced conduction block in anomalous atrioventricular bypass tracts. J Am Coll Cardiol 1985;5:311–7.
22. Klein GJ, Prystowski EN, Pritchett ELC, Davis D, Gallagher JJ. Atypical patterns of retrograde conduction over accessory atrioventricular pathways in the Wolff-Parkinson-White syndrome. Circulation 1979;60:1477–86.
23. Denes P, Kehoe R, Rosen KM. Multiple reentrant tachycardias due to retrograde conduction of dual atrioventricular bundles with atrioventricular nodal-like properties. Am J Cardiol 1979;44:162–70.
24. Becker AE, Anderson RH, Durrer D, Wellens HJJ. The anatomical substrates of Wolff-Parkinson-White syndrome. A clinicopathologic correlation in seven patients. Circulation 1978;57:870–9.
25. de la Fuente, Sayniuk B, Moe GK. Conduction through a narrow isthmus in isolated atrial tissue: a model of the WPW syndrome. Circulation 1971;44:803–9.
26. Inoue H, Zipes DP. Conduction over an isthmus of atrial myocardium in vivo: a possible model of Wolff-Parkinson-White syndrome. Circulation 1987;76:637–47.
27. Wellens HJJ, Bar FW, Dassen WRM, Brugada P, Vanagt EJ, Farre J. Effect of drugs in the Wolff-Parkinson-White syndrome: importance of initial length of effective refractory period of the accessory pathway. Am J Cardiol 1980;46:665–9.