JACC Vol. 21, No. 7
June 1993:1612–21

A Simple Technique for Selective Radiofrequency Ablation of the Slow Pathway in Atrioventricular Node Reentrant Tachycardia

DELON WU, MD, FACC, SAN-JOU YEH, MD, CHUN-CHIEH WANG, MD, MING-SHIEN WEN, MD, FUN-CHUNG LIN, MD

Taipei, Taiwan

Objectives. **A simple technique was designed for radiofrequency ablation therapy of atrioventricular (AV) node reentrant tachycardia.**

Background. **This technique was based on the hypothesis that slow pathway conduction reflects conduction through the compact node and its posterior atrial input.**

Methods. **A total of 100 consecutive patients were studied; there were 37 men and 63 women, with a mean age of 48 ± 15 years. All 100 patients had induction of sustained tachycardia with (51 patients) or without (49 patients) administration of isoproterenol or atropine, or both. The ablation catheter was initially manipulated to record the largest His bundle deflection from the apex of Koch's triangle. It was then curved downward and clockwise to the area of the compact node when His deflection was no longer visible and the ratio of atrial to ventricular electrogram was <1. The radiofrequency current was delivered from the 4-mm tip electrode a mean of 5 ± 7 times at a power of 25 ± 4 W for a duration of 21 ± 4 s. The total fluoroscopic time was 19 ± 11 min.**

Results. **Selective ablation (56 patients) or modification (26 patients) of the slow pathway without affecting anterograde and retrograde fast pathway conduction was achieved in 82 patients. Ablation or modification of both the retrograde fast pathway and the slow pathway but with preservation of anterograde fast pathway conduction was noted in 12 patients. Ablation or modification of the retrograde fast pathway alone or both anterograde and retrograde fast pathway conduction was noted in three patients. Complete AV node block occurred in three patients. Seventy-three patients had no induction of echo beats or tachycardia and 24 patients had induction of a single echo beat after ablation. Follow-up study was performed in 62 patients 76 ± 18 days after ablation. Thirty-nine patients had no induction of echo beats or tachycardia, 22 had induction of echo beats alone and 1 patient had induction of sustained tachycardia.**

Conclusion. **Selective ablation of the slow AV node pathway can be achieved by a simple procedure with a high success rate and few complications.**

(J Am Coll Cardiol 1993;21:1612–21)

Catheter ablation using radiofrequency current has emerged as the therapy of choice in the management of the Wolff-Parkinson-White syndrome and atrioventricular (AV) node reentrant tachycardia (1–4). Preliminary results of attempts to ablate the retrograde fast pathway in AV node reentrant tachycardia, although promising, are usually followed by a high rate of recurrence of the retrograde fast pathway conduction. The ablation procedure is time consuming and is frequently complicated by the development of complete AV block requiring implantation of a permanent pacemaker. Thus, methods for selective ablation of the slow AV node pathway are currently being investigated in several laboratories (5–9). A simple technique for such ablation developed in our laboratory allows selective ablation of the slow pathway with preservation of anterograde AV conduction. Our initial experience with this technique in 100 consecutive patients suggests that it is an effective and safe procedure with a high success rate and few complications.

Methods

Study patients. In July 1991, a technique was developed in our laboratory for selective ablation of the slow pathway using radiofrequency current for curative treatment of AV node reentrant tachycardia. This study reports our experience of the initial 100 consecutive patients with the slow-fast form of AV node reentrant tachycardia who were referred to Chang Gung Memorial Hospital for modification of the AV node. The procedure was approved by the Institutional Review Board and was in accord with the local ethical standards. There were 37 men and 63 women, ranging in age from 18 to 84 years (mean ± SD 48 ± 15). Three patients had hypertension, 3 had bronchial asthma and the other 94 patients had no evidence of organic heart disease. All 100 patients had electrocardiographic (ECG) documentation of recurrent supraventricular tachycardia and all had induction of sustained AV node reentrant tachycardia (51 patients with

From the Section of Cardiology, Department of Medicine, Chang Gung Memorial Hospital, Chang Gung Medical College, Taipei, Taiwan. This study was supported in part by Grants NSC81-0412-B182-523 and NSC81-0412-B182-14 from the National Science Council and Grant DOH82-HR-C05 from the National Health Institutes of the Republic of China, Taipei.

Manuscript received July 8, 1992; revised manuscript received November 9, 1992, accepted December 22, 1992.

Address for correspondence: Delon Wu, MD, Chang Gung Memorial Hospital, 199 Tung Hwa North Road, Taipei, Taiwan.

0735-1097/93/$6.00

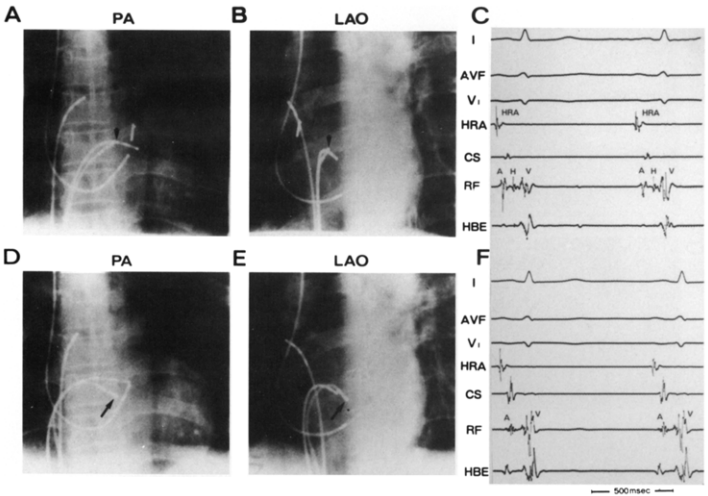

Figure 1. Catheter position and electrograms showing the approach for selective slow pathway ablation. Three quadripolar electrode catheters were positioned in the high right atrium, across the tricuspid valve and in the coronary sinus, respectively. A 7F steerable bulbous tip quadripolar electrode catheter with a 4-mm distal electrode and 2-mm interelectrode distance between the distal two electrodes was positioned at the apex of Koch's triangle (A and B) to record the largest His bundle deflection (C). The tip of the catheter was then curved inferiorly and clockwise (D and E) until the His bundle deflection was no longer visible and the atrial electrogram/ventricular electrogram ratio was <1 (F). A = atrial deflection; CS = atrial electrogram recorded from the coronary sinus; H = His deflection; HBE = His bundle electrogram recorded from the His bundle catheter; HRA = high right atrial electrogram; LAO = left anterior oblique view; I, AVF and V_1 = electrocardiographic leads I, aVF and V_1; PA = posteroanterior view; RF = His bundle electrogram recorded from the ablation catheter; V = ventricular deflection. The arrows indicate the tip of the steerable electrode catheter.

and 49 patients without administration of isoproterenol or atropine, or both) during electrophysiologic study. The diagnosis of AV node reentrant tachycardia was made according to previously described criteria (10–12); these criteria were summarized in a recent report from our laboratory (9).

Electrophysiologic study. All patients gave informed written consent for the electrophysiologic study, which was performed with the patient supine after cardioactive drugs had been discontinued for ≥3 half-lives. Four 6F quadripolar electrode catheters (USCI 002943) with an interelectrode distance of 1 cm were introduced percutaneously into the femoral or internal jugular vein and positioned under fluoroscopic guidance at various sites of the heart (across the tricuspid valve, in the high right atrium, in the right ventricular apex and in the coronary sinus) for recording of the intracardiac electrogram and pacing. Multiple surface ECG leads and intracardiac electrograms from different sites were simultaneously displayed and recorded on a multichannel oscilloscopic recorder (Electronics for Medicine, VR-16) at a paper speed of 100 or 150 mm/s. The pacing stimuli were provided by a digital programmable stimulator (Bloom Associates, DTU-200) and were approximately twice the diastolic threshold in strength and 2 ms in duration.

Catheter ablation. A 7F quadripolar steerable electrode catheter with a 4-mm distal electrode (Mansfield-Webster) was introduced percutaneously into the femoral vein, advanced to the right atrium and then positioned across the tricuspid valve. The radiofrequency current was provided by a commercially available electrosurgical generator (Radionics, RFG-3C) that delivered a continuous unmodulated sine wave energy at a frequency of 500 kHz with continuous digital display of the power strength and impedance. The radiofrequency current was delivered between the tip electrode and a cutaneous dispersive pad that was applied over the left posterior area of the chest.

JACC Vol. 21, No. 7
June 1993:1612-21

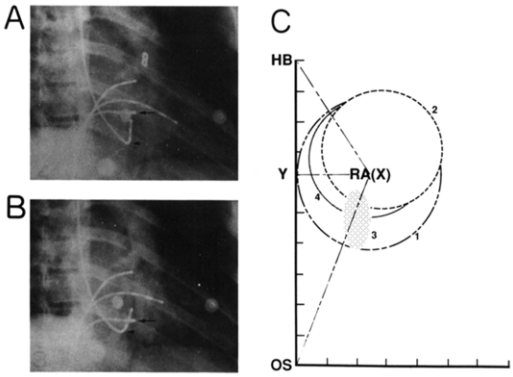

Figure 2. Radiographs and graphic presentation showing the slow pathway ablation site. **A** and **B**, Radiographs recorded in the 30° right anterior oblique view. Three 6F quadripolar electrode catheters with an interelectrode distance of 1 cm were positioned in the high right atrium within the coronary sinus and across the tricuspid valve. **A**, Site where the largest proximal His bundle deflection was recorded by the large-tip steerable electrode catheter at the apex of Koch's triangle. **B**, Slow pathway ablation site. **Arrow** indicates the tip of the steerable electrode catheter and **arrowhead** shows the ostium of the coronary sinus. **C**, Graphic representation of the ablation sites in 100 patients. HB = site where maximal proximal His bundle deflection was recorded; OS = ostium of the coronary sinus; RA (X) = ablation site; Y = point of the perpendicular projection of X point on His bundle deflection–ostium of the coronary sinus line; 1 = ablation site resulted in selective ablation or modification of the slow pathway; 2 = ablation site resulted in ablation or modification of both the slow pathway and the retrograde fast pathway; 3 = ablation site resulted in ablation or modification of the fast pathway; 4 = ablation site resulted in atrioventricular node block. See text for discussion.

The ablation catheter was initially manipulated to record the largest proximal His bundle deflection at the apex of Koch's triangle (Fig. 1, A to C). This was usually achieved at a site slightly inferior and posterior to the His bundle recording site of the regular quadripolar electrode catheter. The catheter tip was then slowly curved downward in a clockwise fashion until the His bundle deflection was no longer visible and the ratio of atrial to ventricular deflection was <1 (mean 0.46 ± 0.24) (Fig. 1, D to F). The radiofrequency current was delivered from the tip electrode during sinus rhythm under continuous ECG monitoring. Delivery of the radiofrequency current was discontinued immediately if PR prolongation or AV block was noted. The catheter tip was typically a few millimeters away from the site of maximal His bundle deflection and the ostium of the coronary sinus and was located at the inferior aspect of Koch's triangle along the tricuspid anulus. Meticulous care was taken to maintain a stable catheter position and a constant clockwise torque of the tip with good tissue contact during delivery of the radiofrequency current. If the attempted ablation failed, the same procedure was repeated. The ablation site was usually selected in an area at one-third anterior and two-thirds posterior between the His bundle and the ostium of the coronary sinus (Fig. 2).

Study protocol and definition. The study protocol and the definition of conduction intervals, refractory periods and dual AV node pathways were similar to those recently described by our laboratory (9). If sustained tachycardia was not induced, intravenous infusion of isoproterenol (1 to 4 μg/min) to achieve a 20% increase in sinus rate was performed and the electrophysiologic study was repeated. If sustained tachycardia was still not inducible, 1 mg of atropine was given intravenously and the electrophysiologic study was again repeated. Only those patients with induction of a sustained tachycardia underwent catheter ablation.

Radiofrequency ablation was performed at the completion of the control electrophysiologic study. Induction of AV node reentrant tachycardia with evaluation of anterograde and retrograde conduction was attempted immediately after each ablation attempt. Successful ablation was defined when 1) no induction or induction of only a single slow-fast AV node reentrant echo beat with no evidence or marked depression of the anterograde slow pathway conduction was noted, 2) no induction or induction of only a single slow-fast AV node reentrant echo beat with no evidence or marked depression of retrograde fast pathway conduction was noted, or 3) a combination of 1) and 2) was noted. The site where the largest His bundle deflection was recorded from the large tip catheter and the site of successful ablation were noted on cinefluoroscopic film in posteroanterior, right anterior oblique and left posterior oblique views. Subsequently, electrophysiologic studies were performed with isoproterenol infusion followed by intravenous administration of atropine (to a total of 0.04 mg/kg body weight) to ensure success of the ablation and of propranolol (0.2 mg/kg) to evaluate the effects of autonomic tone after ablation. The

Table 1. Electrophysiologic Findings in 82 Patients With Selective Ablation or Modification of the Slow Pathway

	Anterograde Properties (ms)				Retrograde Properties (ms)	
	CL With 1:1 FP Conduction (n = 82)	CL-AVB (n = 82)	ERP-FP (n = 52)	ERP-SP (n = 12)	CL With 1:1 FP Conduction (n = 82)	ERP-FP (n = 17)
Before	396 ± 82	330 ± 60	351 ± 76	297 ± 35	358 ± 63	366 ± 57
After	377 ± 89	369 ± 81	335 ± 72	338 ± 100	348 ± 60	349 ± 40
p value	0.027	0.0001	NS	NS	NS	NS

CL = cycle length; CL-AVB = longest atrial paced cycle length that induced second-degree atrioventricular node block; ERP-FP = effective refractory period of the fast pathway; ERP-SP = effective refractory period of the slow pathway; FP = fast pathway.

total study time and the fluoroscopic exposure time were recorded for each patient.

Patients were observed in the hospital for 3 days with multiple measurements of serum creatine kinase, a two-dimensional echocardiogram and 24-h ambulatory ECG monitoring. They were then followed up in the clinic with a repeat electrophysiologic study scheduled 2 to 3 months later. If symptoms of palpitation recurred during the follow-up period, 24-h ambulatory ECG recordings were performed to document the events.

Data analysis. Data were expressed as mean value ± SD and compared by the Student *t* test for paired data. A p value < 0.05 indicated statistical significance. Data before and after ablation were compared under similar physiologic conditions with or without administration of isoproterenol or atropine. Data before ablation and during the late follow-up period were compared in the basal state without administration of isoproterenol or atropine. Whenever possible, anterograde and retrograde refractory periods were compared at an identical driven cycle length.

The site of successful ablation was determined by analysis of cinefluoroscopic film in the right anterior oblique view (Fig. 2). The distance between the ablation site and the site where the largest His bundle deflection was recorded and the distance between the ablation site and the coronary sinus ostium were measured by using a quadripolar electrode catheter with an interelectrode distance of 1 cm as the reference. The site of successful ablation was also determined by drawing a line between the largest His bundle deflection and the coronary sinus ostium and a perpendicular line from the ablation site (X point) to the line between the His bundle deflection and the ostium of the coronary sinus (Y point). The distance between the His bundle deflection and the coronary sinus ostium was taken as 1 and the XY, His bundle-Y, and ostium-Y intervals relative to the His bundle deflection and the coronary sinus ostium interval were measured.

Results

Anterograde dual pathway physiology was noted in 97 patients. It was demonstrated as a discontinuous AA, AH curve during incremental pacing or as a discontinuous A_1A_2, A_2H_2 curve by atrial extrastimulus testing in 87 patients and as a discontinuous A_2A_3, A_3H_3 curve by double atrial extrastimuli in 10 patients. Nine of the 100 study patients showed a retrograde dual pathway physiology. Radiofrequency energy was applied a mean of 5 ± 7 times (median 2) at a power level of 25 ± 4 W for a duration of 21 ± 4 s. The total study duration was 121 ± 31 min and the fluoroscopic exposure time was 19 ± 11 min. Successful application of radiofrequency energy was associated with the occurrence of junctional tachycardia in all but two patients. Although low frequency deflections were occasionally observed at the site of successful ablation, they were not a consistent finding and their significance was difficult to validate. Prolongation of the PR interval was noted in four patients during the procedure. Immediate success was noted in 97 patients and complete AV block occurred in 3. There were no specific variables during the procedure that could predict AV block in the latter three patients who developed this complication.

Selective ablation or modification of the slow pathway (Table 1). The slow pathway conduction was selectively ablated with abolition of dual pathway physiology in 56 patients (Fig. 3, A and B and 4); it was modified in 26 patients (Fig. 5, A and B). The shortest atrial paced cycle length that sustained 1:1 fast pathway conduction was 396 ± 82 and 377 ± 89 ms, respectively, before and after ablation ($p = 0.027$). The longest atrial paced cycle length that induced second-degree AV block was 330 ± 60 and 369 ± 81 ms, respectively, before and after ablation ($p = 0.0001$). The anterograde fast pathway effective refractory period could be measured before and after ablation in 52 patients and was 351 ± 76 and 335 ± 72 ms, respectively, before and after ablation (p = NS). The anterograde slow pathway effective refractory period in the 12 patients with slow pathway modification was 297 ± 35 and 338 ± 100 ms, respectively, before and after ablation (p = NS).

All 82 patients had ventriculoatrial (VA) conduction through the fast pathway before and after ablation. The shortest ventricular paced cycle length that sustained 1:1 retrograde fast pathway conduction was 358 ± 63 and 348 ± 60 ms, respectively, before and after ablation (p = NS). Retrograde fast pathway effective refractory period was measured in 17 patients before and after ablation and was

JACC Vol. 21, No. 7
June 1993:1612–21

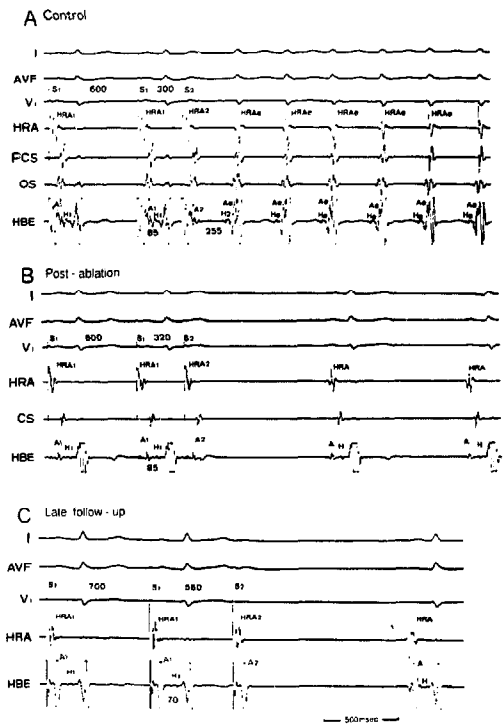

Figure 3. Recordings showing selective ablation of the slow pathway. A_1 Induction of sustained atrioventricular (AV) node reentrant tachycardia before ablation. The atrial driven cycle length was 600 ms and the A_1A_2 interval was 300 ms; A_2 was conducted through the slow pathway with induction of tachycardia. **B** and **C**, Abolition of slow pathway conduction immediately after delivery of radiofrequency current and during a late follow-up electrophysiologic study. In **B**, A_2 was blocked in the AV node at an A_1A_2 interval of 320 ms. In **C**, A_2 was coupled to a basic atrial driven cycle length of 700 ms at an A_1A_2 interval of 560 ms and was blocked in the AV node. HRA_1, A_1 and H_1 = high right atrial, low septal right atrial and His bundle response to basic driven stimulus, S_1; HRA_2, A_2 and H_2 = high right atrial, low septal right atrial and His bundle response to extrastimulus, S_2; HRAe, Ae and He = high right atrial, low-septal right atrial and His bundle response during tachycardia. OS = atrial electrogram recorded from the ostium of the coronary sinus; PCS = atrial electrogram recorded from the proximal coronary sinus; other abbreviations as in Figure 1.

366 ± 57 and 349 ± 40 ms, respectively (p = NS). Sustained AV node reentrant tachycardia was inducible in all 82 patients before ablation. After ablation, none had inducible sustained tachycardia and 24 patients had induction of a single AV node reentrant echo beat (in 10 patients with and in 14 without isoproterenol infusion).

Ablation or modification of both the slow pathway and the retrograde fast pathway (Table 2). Ablation or modification of both the slow pathway and the retrograde fast pathway conduction was noted in 12 patients. Eleven of these 12 patients had dual pathway physiology before ablation. After ablation, dual pathway physiology remained in four patients and was abolished in seven. The anterograde fast pathway conduction was not affected in 10 patients but was impaired in 2. The shortest atrial paced cycle length that sustained 1:1 fast pathway conduction was 396 ± 93 and 381 ± 98 ms, respectively, before and after ablation (p = NS). The longest atrial paced cycle length that induced second-degree AV block was 354 ± 78 and 363 ± 92 ms, respectively, before and after ablation (p = NS).

Ventriculoatrial conduction using the fast pathway was noted in all 12 patients before ablation, but retrograde dual pathway physiology was noted in only 4. After ablation, four patients lost VA conduction and eight patients had a depressed retrograde fast pathway conduction without evidence of slow pathway conduction; retrograde dual pathway physiology was noted in only one patient. The shortest ventricular paced cycle length that sustained 1:1 fast path-

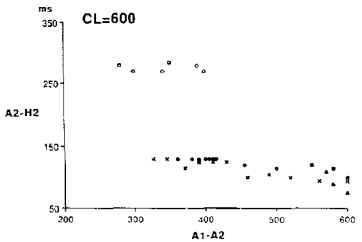

Figure 4. Atrioventricular conduction curve from the same patient as in Figure 3, showing abolition of dual pathway physiology after selective slow pathway ablation. The A_1A_2 coupling intervals are on the **abscissa** and A_2H_2 responses on the **ordinate**; the curve was generated at an atrial driven cycle length (CL) of 600 ms during the initial study and of 700 ms during the late follow-up study. Before ablation, the effective refractory period of the fast and slow pathway was 350 and <280 ms, respectively. After ablation, the effective refractory period of the fast pathway was 320 ms, whereas slow pathway conduction was abolished. During late follow-up study, there was an increase in the effective refractory period to 360 ms; slow pathway conduction was not seen. **Closed circles** = fast pathway conduction before ablation; **open circles** = slow pathway conduction with induction of tachycardia before ablation; **crosses** = fast pathway conduction immediately after delivery of the radiofrequency current; **closed triangles** = fast pathway conduction during the late follow-up study.

way conduction in the eight patients with depressed retrograde fast pathway conduction after ablation was 333 ± 57 and 498 ± 109 ms, respectively, before and after ablation ($p = 0.004$). The retrograde fast pathway effective refractory period could be compared before and after ablation in only four patients and was 440 ± 83 and 378 ± 95 ms, respectively (p = NS).

Sustained AV node reentrant tachycardia was inducible in all 12 patients before ablation. After ablation, none had induction of AV node reentrant echo beats or tachycardia.

Ablation or modification of the fast pathway. Ablation of the retrograde and modification of the anterograde fast pathway conduction were noted in one patient, whereas modification with marked depression of the retrograde with or without modification of the anterograde fast pathway conduction was noted in two patients. Each of these three patients had dual pathway physiology before ablation. After ablation, two patients continued to show dual pathway physiology and one patient had abolition of dual pathway physiology. The shortest atrial paced cycle length that sustained 1:1 fast pathway conduction was 300, 360 and 500 ms before and 370, 330 and 400 ms after ablation, respectively. The longest atrial paced cycle length that induced second-degree AV block was <280, 330 and 260 ms before and 350, 330 and 380 ms after ablation, respectively.

Ventriculoatrial conduction through the fast pathway was noted in all three patients before ablation, and retrograde dual pathway physiology was not noted in any patient. After ablation, one patient lost VA conduction and two patients had depressed retrograde fast pathway conduction without evidence of slow pathway conduction. The shortest ventricular paced cycle length that sustained 1:1 fast pathway conduction in the two patients with depressed retrograde fast pathway conduction after ablation was 300 and 330 ms before and 480 and 640 ms after ablation, respectively. The retrograde fast pathway effective refractory period could be compared before and after ablation in only one patient and was 360 ms before and 455 ms after ablation.

Sustained AV node reentrant tachycardia was inducible in all three patients before ablation. After ablation, none had induction of AV node reentrant echo beats or tachycardia.

Ablation site. The site that resulted in selective ablation or modification of the slow pathway was 1.6 ± 0.6 cm from the largest His bundle recording site and 2.1 ± 0.9 cm from the ostium of coronary sinus (Fig. 2). The XY, His bundle-Y and coronary sinus ostium-Y intervals relative to the His bundle deflection and the coronary sinus ostium were 0.24 ± 0.25, 0.37 ± 0.22 and 0.63 ± 0.23, respectively. The site that resulted in ablation or modification of both the slow pathway and the retrograde fast pathway was 1.7 ± 0.8 cm from the largest His bundle recording site and 2.9 ± 1.4 cm from the ostium of the coronary sinus. The XY, His bundle-Y and the coronary sinus ostium–Y intervals relative to the His bundle deflection and the coronary sinus ostium were 0.28 ± 0.22, 0.29 ± 0.19 and 0.71 ± 0.19, respectively. The site that resulted in ablation or modification of the fast pathway alone was 1.9 ± 0.1 cm from the largest His bundle recording site and 1.8 ± 0.7 cm from the ostium of the coronary sinus. The XY, His bundle-Y and the coronary sinus ostium–Y intervals relative to the His bundle deflection and the coronary sinus ostium were 0.20 ± 0.02, 0.52 ± 0.12 and 0.48 ± 0.11, respectively. The ablation site that resulted in AV node block was 1.4 ± 0.5 cm from the largest His bundle recording site and 3.1 ± 1.4 cm from the coronary sinus ostium. The XY, His bundle-Y and the coronary sinus ostium–Y intervals relative to the His bundle deflection and the coronary sinus ostium were 0.24 ± 0.20, 0.32 ± 0.19 and 0.68 ± 0.19, respectively. These variables were not statistically different among the four sites.

Follow-up and late electrophysiologic study (Table 3). The average follow-up duration was 10 ± 2 months (range 7 to 13). Of the three patients with AV block, one received a permanent implantable pacemaker and the other two were asymptomatic with stable junctional rhythm over a period of 10 and 9 months, respectively. In the remaining 97 patients, there was no inappropriate sinus tachycardia or second- or third-degree AV block; however, PR prolongation was noted in 2 patients. Two patients reported episodic palpitation; one

JACC Vol. 21, No. 7
June 1993:1612–21

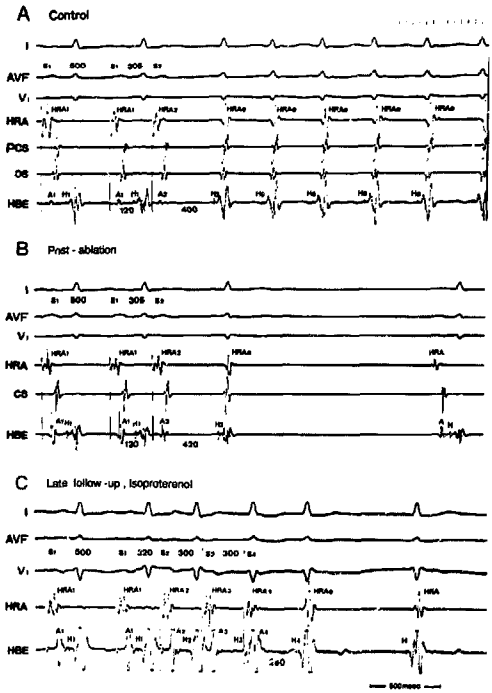

Figure 5. Recordings from a patient showing modification of the slow pathway with radiofrequency ablation. **A**, Induction of sustained atrioventricular (AV) node reentrant tachycardia when A_2 was coupled to an atrial driven cycle length of 500 ms at an A_1A_2 interval of 305 ms; A_2 was blocked in the fast pathway and conducted through the slow pathway at an A_2H_2 interval of 400 ms. **B**, Induction of only a single AV node reentrant atrial echo beat immediately after delivery of radiofrequency current. **C**, Induction of only a single AV node reentrant atrial echo beat by triple atrial extrastimuli (A_1A_2, A_2A_3 and A_3A_4, respectively, of 320, 300 and 300 ms) with isoproterenol infusion during the late follow-up study. Note that in both **B** and **C**, the slow pathway was still conducting but was unable to conduct repetitively. Abbreviations as in Figures 1 and 3.

of them had ECG documentation of paroxysmal atrial fibrillation. Follow-up electrophysiologic study was performed in 62 patients 76 ± 18 days after ablation. In these 62 patients, the sinus cycle length was 829 ± 148 and 812 ± 130 ms, the AH interval 77 ± 18 and 84 ± 18 ms and the HV interval 43 ± 6 and 43 ± 7 ms, respectively, during the control electrophysiologic study before ablation and the late electrophysiologic study (p = NS). In 38 patients, there was no evidence of anterograde slow pathway conduction during incremental atrial pacing and single- or double-atrial extrastimulus testing with or without isoproterenol infusion during the late study (Fig. 3C and 4). In one patient, only slow pathway conduction was noted. In the remaining 23 patients, anterograde dual pathway physiology was demonstrated. The shortest atrial paced cycle length that sustained 1:1 fast pathway conduction in the 61 patients with anterograde fast pathway conduction was 453 ± 95 and 458 ± 11[illegible] ms, respectively, during the control electrophysiologic study before ablation and the late electrophysiologic study (p =

Table 2. Electrophysiologic Findings in 12 Patients With Ablation or Modification of Both the Slow Pathway and the Retrograde Fast Pathway

	Anterograde Properties (ms)		Retrograde Properties (ms)	
	CL With 1:1 FP Conduction (n = 12)	CL-AVB (n = 12)	CL With 1:1 FP Conduction (n = 8)	ERP-FP (n = 4)
Before	396 ± 93	354 ± 78	333 ± 57	440 ± 83
After	381 ± 98	363 ± 92	398 ± 109	378 ± 95
p value	NS	NS	0.004	NS

Abbreviations as in Table 1.

Table 3. Electrophysiologic Findings Before Ablation and During Late Follow-Up in 62 Patients

	Anterograde Properties (ms)							Retrograde Properties (ms)	
	Sinus CL (n = 62)	AH (n = 62)	HV (n = 62)	CL With 1:1 FP Conduction (n = 61)	CL-AVB (n = 62)	ERP-FP (n = 48)	ERP-SP (n = 12)	CL With 1:1 FP Conduction (n = 44)	ERP-FP (n = 20)
Before	839 ± 148	77 ± 18	43 ± 6	453 ± 95	362 ± 68	320 ± 67	285 ± 25	368 ± 92	418 ± 76
Follow-up	812 ± 130	84 ± 18	43 ± 7	458 ± 116	432 ± 109	361 ± 72	320 ± 67	400 ± 94	380 ± 81
p value	NS	NS	NS	NS	0.0001	NS	NS	0.006	NS

AH = interval between the atrial and His deflections; HV = interval between the His and ventricular deflections; other abbreviations as in Table 1.

NS). The longest atrial paced cycle length that induced second-degree AV block in the 62 patients was 362 ± 68 and 432 ± 109 ms, respectively, during the control and the late electrophysiologic study (p = 0.0001). The anterograde fast pathway effective refractory period could be compared in 48 patients during the control and the late electrophysiologic study and was 320 ± 67 and 361 ± 72 ms, respectively (p = NS). The anterograde slow pathway effective refractory period could be compared in 12 patients who had slow pathway conduction at the late electrophysiologic study and was 285 ± 25 and 320 ± 67 ms, respectively, during the control and the late study (p = NS).

Retrograde study showed absence of VA conduction in 4 patients and the presence of VA conduction through the fast pathway in 58 patients, 2 of whom also had VA conduction through the slow pathway. The shortest ventricular paced cycle length that sustained 1:1 fast pathway conduction in the 44 patients who had VA conduction through the fast pathway was 368 ± 92 and 400 ± 94 ms, respectively, during the control and the late electrophysiologic study (p = 0.006). The retrograde fast pathway effective refractory period was measured in 20 patients and was 418 ± 76 and 380 ± 81 ms, respectively, during the control and the late electrophysiologic study in these 20 patients (p = NS).

Sustained AV node reentrant tachycardia was induced in one (2%) of the two patients with episodic palpitation. This patient subsequently had successful ablation with elimination of the slow pathway conduction by a second trial using a similar procedure. Induction of a single AV node reentrant echo beat alone was noted in 22 patients (13 with and 9 without isoproterenol infusion) (Fig. 5C). Thirty-nine patients (63%) (including the patient with documented atrial fibrillation) continued to have no induction of AV node reentrant echo beats or tachycardia.

Discussion

Modification of the AV node reentrant circuit by surgery. Therapy in AV node reentrant tachycardia was largely dependent on drugs until 1979 when Pritchett et al. (13) fortuitously cured the tachycardia by surgical dissection of the AV junctional area with preservation of AV conduction. Subsequently, Ross et al. (14) innovated surgical techniques for dissecting the perinodal tissue and Cox et al. (15) developed cryosurgical techniques for modifying this tissue. These procedures abolished AV node reentrant tachycardia with preservation of AV conduction primarily by interruption of slow pathway conduction. Anterograde fast pathway conduction was minimally affected, although retrograde fast pathway conduction was sometimes interrupted.

Modification of the AV node reentrant circuit by catheter techniques. *Modification of the AV node by closed chest technique.* Use of a closed chest technique with delivery of large direct current shocks through an electrode catheter to the site of earliest atrial activation during tachycardia was first reported by Haissaguerre et al. (16) and later by Epstein et al. (17) in 1989. This technique preferentially abolished or impaired retrograde fast pathway conduction but was frequently associated with various degrees of damage to anterograde fast pathway and slow pathway conduction. Dual pathway physiology was frequently abolished. Although most patients were free of arrhythmia after the shocks, there were several disadvantages. These included the requirement for general anesthesia, occurrence of high grade AV block and the potential risks of serious side effects due to arcing and gas formation with the delivery of the high voltage current. Goy et al. (3) in 1990 and Lee et al. (4) in 1991 reported successful ablation of tachycardia by radiofrequency current. With this technique, a large-tip steerable electrode catheter was first positioned to record the largest His bundle deflection during sinus rhythm, and the catheter was then withdrawn to obtain the largest atrial potential and the smallest His bundle deflection. Radiofrequency current was delivered through the tip electrode. It did not require general anesthesia and could avoid the side effects caused by arcing and gas formation with delivery of high voltage shocks. However, complete heart block still occurred in a substantial number of patients.

Selective ablation or modification of the slow pathway using radiofrequency energy. This technique has been investigated in several laboratories (5–9). Kay et al. (6) delivered the radiofrequency current to the base of Koch's triangle anterior or inferior to the ostium of the coronary sinus along the tricuspid anulus or within the coronary sinus. However, the ablation site shown in their representative illustration was ≥2 cm away from the coronary sinus and was closer to the His bundle. Jackman et al. (5) delivered the radiofrequency current to the right posteroseptal region

where a discrete sharp deflection was recorded. The mean duration of the procedure was 8.0 ± 2.6 h. Jazayeri et al. (7) delivered the radiofrequency current to the posteroinferior area of Koch's triangle anterior to the ostium of the coronary sinus along the tricuspid anulus. If it failed, the ablation site was moved in a stepwise fashion toward the His bundle. Haissaguerre et al. (8) delivered the radiofrequency current to a site where low amplitude slow potentials were recorded. The site was projected at the two-thirds anterior one-third posterior of the area between the His bundle to the coronary sinus ostium (8). Preliminary reports indicated that these techniques appeared to be promising, although they were difficult and the procedure was time consuming.

Present study. In our study, the radiofrequency current was delivered to the area of the inferior aspect of Koch's triangle along the tricuspid anulus, which was several millimeters away from the apex of the triangle and the ostium of the coronary sinus but was closer to the apex of the triangle. It was projected at the area one-third anterior two-thirds posterior between the His bundle and the ostium of the coronary sinus and was slightly anterior to the site selected by Haissaguerre et al. (8). The procedure was simple and could be accomplished in a single study session with minimal fluoroscopic exposure time. It resulted in selective ablation or modification of slow pathway conduction with preservation of anterograde fast pathway conduction in 82% of patients. Ablation or modification of both retrograde fast pathway and slow pathway conduction but with preservation of anterograde fast pathway conduction was achieved in 12% of patients. Ablation or modification of retrograde fast pathway alone or of both anterograde and retrograde fast pathway conduction occurred in 3%. The immediate success rate was 97% and the incidence of complete AV block was 3%. Inappropriate sinus tachycardia reported by others (18) after catheter ablation was not noted in our study. Late follow-up electrophysiologic study showed that the effect was persistent. Sixty-three percent of the patients had no induction of AV node reentrant echo beats or tachycardia during late electrophysiologic study even with isoproterenol infusion, and 35% of the patients had induction of only a single AV node reentrant echo beat. Only one patient had induction of sustained tachycardia and subsequently underwent successful ablation with the same procedure. This study also found that complete elimination of AV node reentrant echo beats appeared to be unnecessary to guarantee a late success; impairment of the slow pathway or retrograde fast pathway conduction, or both, allowing induction of only a single AV node reentrant echo beat was also associated with late success during the follow-up period.

Limitations of the study. This study has several limitations. First, a complication of AV block was noted in 3% of the patients. This complication was not noted in the 35 patients studied by Jazayeri et al. (7), but it occurred in 1 of the 34 patients studied by Kay et al. (6) 1 month after ablation and required implantation of a permanent pacemaker. It occurred transiently in 1 of the 64 patients studied by Haissaguerre et al. (8) and required a permanent implantable pacemaker in 1 of the 80 patients studied by Jackman et al. (5). Jackman et al. attributed this complication to the result of an inadvertent movement of the catheter to the region of proximal right bundle during application of radiofrequency current. In the present study and that of Haissaguerre et al. (8), the current was applied more anteriorly than in the study of Jazayeri et al. (7). Thus, not delivering the current near the His bundle (nodo-Hisian area or NH zone) and checking the catheter position with fluoroscopy during application of radiofrequency current may be warranted. However, even with such precautions, all AV node modification procedures carry a potential risk for the development of heart block. Second, the autonomic tone may be changed especially after application of radiofrequency current, making comparison of data less valid. However, the effects of slow pathway ablation persisted during the follow-up electrophysiologic studies in our patients. Third, the follow-up period is still not long enough to evaluate the long-term results of this procedure in this and other studies.

Addendum

We have extended our experience in radiofrequency ablation therapy for AV node reentrant tachycardia using the same approach in a total of 189 patients. The immediate success rate was 97.9 (185/189). One additional patient had the complication of transient AV node block that lasted for several weeks before normal AV conduction resumed. This patient was asymptomatic without pacemaker implantation during a follow-up period of 5 months. Thus, the risk of developing heart block was 2.1% for the total group of 189 patients.

References

1. Jackman WM, Wang X, Friday KJ, et al. Catheter ablation of accessory atrioventricular pathways (Wolff-Parkinson-White syndrome) by radiofrequency current. N Engl J Med 1991;324:1605–11.
2. Calkins H, Sousa J, Rosenheck S, et al. Diagnosis and cure of the Wolff-Parkinson-White syndrome or paroxysmal supraventricular tachycardias during a single electrophysiologic test. N Engl J Med 1991;324: 1612–8.
3. Goy JJ, Fromer M, Schlaepfer J, Kappenberger L. Clinical efficacy of radiofrequency current in the treatment of patients with atrioventricular node reentrant tachycardia. J Am Coll Cardiol 1990;16:418–23.
4. Lee MA, Morady F, Kadish A, et al. Catheter modification of the atrioventricular junction with radiofrequency energy for control of atrioventricular nodal reentry tachycardia. Circulation 1991;83:827–35.
5. Jackman WM, Beckman KJ, McClelland JH, et al. Treatment of supraventricular tachycardia due to atrioventricular nodal reentry by radiofrequency ablation of slow-pathway conduction. N Engl J Med 1992;327: 313–8.
6. Kay GN, Epstein AE, Dailey SM, Plumb VJ. Selective radiofrequency ablation of the slow pathway for the treatment of atrioventricular nodal reentrant tachycardia: evidence for involvement of perinodal myocardium within the reentrant circuit. Circulation 1992;85:1675–88.
7. Jazayeri M, Hempe SL, Sra J, et al. Selective transcatheter ablation of fast and slow pathways using radiofrequency energy in patients with

atrioventricular nodal reentrant tachycardia. Circulation 1992;85:1318–28.

8. Haissaguerre M, Gaita F, Fischer B, et al. Elimination of atrioventricular nodal reentrant tachycardia using discrete slow potentials to guide application of radiofrequency energy. Circulation 1992;85:2162–75.
9. Wu D, Yeh SJ, Wang CC, Wen MS, Chang HC, Lin FC. Nature of dual atrioventricular nodal pathways and the tachycardia circuit as defined by radiofrequency ablation technique. J Am Coll Cardiol 1992;20:889–95.
10. Wu D. Dual atrioventricular nodal pathways: a reappraisal. PACE 1982;5:72–89.
11. Wu D, Denes P, Amat-y-Leon F, et al. Clinical, electrocardiographic and electrophysiologic observations in patients with paroxysmal supraventricular tachycardia. Am J Cardiol 1978;41:1045–51.
12. Wu D. Electrophysiologic diagnosis of cardiac arrhythmias. In: Cheng TO, ed. The International Textbook of Cardiology. New York: Pergamon, 1986:309–33.
13. Pritchett ELC, Anderson RW, Benditt DG, et al. Reentry within the atrioventricular node: surgical cure with preservation of atrioventricular conduction. Circulation 1979;60:440–50.
14. Ross DL, Johnson DC, Denniss R, Cooper MJ, Richards DA, Uther JB. Curative surgery for atrioventricular junctional ("AV nodal") reentrant tachycardia. J Am Coll Cardiol 1985;6:1383–92.
15. Cox JL, Holman WL, Cain ME. Cryosurgical treatment of atrioventricular node reentrant tachycardia. Circulation 1987;76:1829–36.
16. Haissaguerre M, Warin JF, Lemetayer P, Saoudi N, Guillem JP, Blanchot P. Closed-chest ablation of retrograde conduction in patients with atrioventricular nodal reentrant tachycardia. N Engl J Med 1989;320:426–33.
17. Epstein LM, Scheinman MM, Langberg JJ, Chilson D, Goldberg HR, Griffin JC. Percutaneous catheter modification of the atrioventricular nodal reentrant tachycardia. Circulation 1989;80:757–68.
18. Ehlert FA, Goldberger J, Brooks R, Kadish A. Persistent inappropriate sinus tachycardia following radiofrequency modification for AV node reentrant tachycardia (abstr). PACE 1991;14(suppl II):II-164.