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Therapeutic End Points for the Treatment of Atrioventricular Node Reentrant Tachycardia by Catheter-Guided Radiofrequency Current

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Objectives. **The purpose of this prospective study was to test the hypothesis that the elimination of inducible repetitive atrioventricular (AV) node reentry despite the persistence of slow AV pathway conduction is a valid end point for radiofrequency catheter ablation procedures in patients with supraventricular tachycardia due to AV node reentry.**

Background. **Although modification of AV node physiology by radiofrequency current can eliminate AV node reentrant tachycardia, therapeutic end points that are definitive of a satisfactory result in patients undergoing modification of the slow AV pathway have not been established. Applications of radiofrequency current at selected sites may eliminate all evidence of slow pathway conduction or sufficiently modify the refractory properties of the slow pathway to preclude sustained arrhythmias. Accordingly, total abolition of dual AV node physiology may not be necessary to prevent arrhythmia recurrence.**

Methods. **Radiofrequency catheter ablation of the slow AV pathway was attempted in 59 patients with typical AV node reentry. Tissue ablation was performed with a continuous wave of 500-kHz radiofrequency current. Twenty-five to 35 W was applied for 60 s at the site selected for tissue destruction.**

Results. **Dual AV node physiology was eliminated completely in 35 patients (59%), persisted without inducible AV node reentry in 13 patients (22%) and persisted with inducible single AV reentrant beats in 11 patients (19%). In patients with persistent dual AV node physiology, the maximal difference between the effective refractory period of the fast and slow pathways was reduced from 104 ± 62 ms before the procedure to 37 ± 37 ms after AV conduction had been modified ($p < 0.001$). During a mean follow-up interval of 15 months (range 4 to 28), only one patient (2%) had a recurrence of the tachycardia.**

Conclusions. **Results demonstrate that when complete elimination of dual AV node physiology is difficult, modification of slow pathway conduction to the extent that repetitive AV node reentry cannot be induced is a definitive end point that portends a good prognosis.**

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Modification of dual atrioventricular (AV) node physiology by the application of radiofrequency current to atrial tissue near the AV node provides an important therapeutic alternative to pharmacologic or surgical therapy for the treatment of supraventricular tachycardia due to AV node reentry (1–5). The major risk of this procedure is inadvertent AV conduction block, which may occur when radiofrequency current is applied too close to the AV node. The risk of heart block may be increased when attempts to completely eliminate dual AV node physiology become protracted and result in repetitive applications of radiofrequency current along the tricuspid annulus, especially when energy is delivered close to the junction of the AV node and His bundle. Moreover, difficult procedures often require prolonged use of fluoroscopy and reduce the efficiency of facilities in centers that perform a large number of electrophysiologic studies (6,7).

The therapeutic end points that portend a satisfactory result in patients undergoing modification of the slow AV pathway have not been established. Radiofrequency current applications along the tricuspid annulus at the site of presumed slow pathway potentials may eliminate all evidence of slow pathway conduction or sufficiently modify the refractory properties of the slow pathway to preclude sustained arrhythmias. Thus, modification of AV node physiology may be sufficient to prevent a recurrence of symptoms in patients in whom complete elimination of dual AV node physiology is difficult and in whom persistent attempts to abolish all evidence of slow AV conduction may be harmful. Accordingly, the purpose of this study was to prospectively determine the extent to which the inability to induce repetitive AV node reentry irrespective of the presence or absence of dual AV node physiology is a valid therapeutic end point for radiofrequency catheter ablation procedures in patients with AV node supraventricular tachycardia.

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Table 1. Characteristics of the 59 Study Patients

Male/female	18/41
Age (yr)	
Mean	44 ± 18
Range	12 to 77
Other heart disease	
None	51
Ischemic heart disease	6
Nonischemic heart disease	2
Spontaneous supraventricular tachycardia	
Every 1 to 7 days	24 (41)
Every 1 to 4 weeks	12 (20)
Every 1 to 3 months	15 (25)
Every 3 to 6 months	8 (14)

Data are expressed as number (%) of patients, mean value ± SD, or range.

Methods

Study patients. Fifty-nine patients with AV node reentry who were referred to Barnes Hospital for evaluation of supraventricular tachycardia underwent a diagnostic electrophysiologic study and modification of AV node physiology during a single procedure. The protocol was approved by the Human Studies Committee (September 21, 1990) and informed consent was obtained. The clinical characteristics of the patients studied are summarized in Table 1. All patients had typical AV node reentry with slow anterograde and fast retrograde conduction. There were 18 male and 41 female patients, ranging in age from 12 to 77 years. The majority of patients had no other evidence of heart disease. Two patients, who had both orthodromic supraventricular tachycardia and AV node reentry, underwent ablation of an accessory pathway and modification of the AV node during the same procedure. Before the ablation procedure, clinical episodes of supraventricular tachycardia occurred frequently in the majority of patients.

Electrophysiologic studies. Patients were sedated with midazolam and morphine. Catheters were positioned in the right atrium, at the AV junction to record a His bundle electrogram, within the coronary sinus and at the right ventricular apex. The stimulation protocol included incremental atrial and ventricular pacing, assessment of AV conduction during the introduction of single atrial extrastimuli at paced cycle lengths of 700, 600, 500 and 400 ms and the use of single ventricular extrastimuli. The criterion for diagnosing dual AV node physiology was an increase in the AH interval of >50 ms in response to a 10-ms decrease in the coupling interval of the atrial extrastimulus (8). If sustained supraventricular tachycardia was not induced, isoproterenol (1 to 4 μg/min) was infused to achieve a heart rate of 120 to 130 beats/min.

Radiofrequency ablation procedure. Figure 1 illustrates the region along the tricuspid annulus between the os of the coronary sinus and the His bundle where presumed slow pathway potentials were identified and radiofrequency current was applied. In cases where slow pathway potentials were not identified, radiofrequency current was applied empirically within this region and the response to programmed stimulation was evaluated after each application.

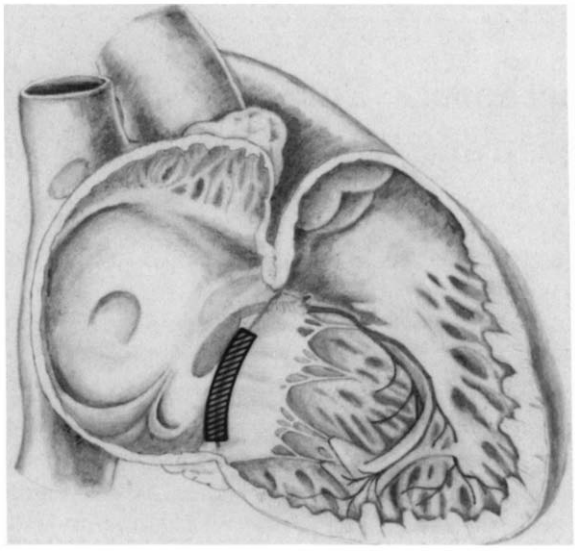

Figure 1. Schematic view of the heart demonstrating the region along the tricuspid annulus between the os of the coronary sinus and the His bundle where presumed slow pathway potentials were detected and radiofrequency current applied. Radiofrequency current was rarely applied in the upper third of the hatched region.

The objective of this procedure was to eliminate the slow pathway. Radiofrequency current was applied through a catheter with a 4- or 5-mm distal electrode. Tissue ablation was performed with a continuous wave of 500-kHz radiofrequency current. A control unit was used to select the duration and voltage of the application and monitor voltage, current and impedance. Twenty-five to 35 W was applied for approximately 60 s at the site selected for tissue destruction.

Immediately after the application of radiofrequency current, programmed atrial stimulation was performed to determine whether dual AV node physiology had been eliminated as evidenced by continuous AV node conduction curves and absence of AV node reentry. In patients who had persistent dual AV node physiology despite repeated applications of radiofrequency current, the procedure was concluded when repetitive AV node reentry had been eliminated. One hour after a therapeutic end point had been achieved, programmed stimulation was repeated to confirm that functional slow pathway physiology had not recovered. If repetitive AV node reentry could not be induced, isoproterenol was infused at a rate of 1 to 4 μg/min to achieve a heart rate at rest of 120 to 130 beats/min and programmed atrial stimulation was repeated.

Patients were generally discharged from the hospital the day after the electrophysiologic study and were seen for follow-up 1 to 3 months after the procedure. Late follow-up was obtained for each patient by a direct telephone interview.

Statistical analysis. Data are presented as the mean value ± SD and were compared between groups by an unpaired

Table 2. Results of Electrophysiologic Studies

Electrophysiologic Characteristics	Preablation	Postablation
AH interval (ms)	85 ± 16	82 ± 16
HV interval (ms)	49 ± 8	50 ± 8
Wenckebach CL (ms)	356 ± 56	362 ± 59
AV node conduction		
Discontinuous	55 (93)	24 (41)
Continuous	4 (7)	35 (59)
Single AV node echo beat	59 (100)	11 (19)
Sustained AV node reentrant tachycardia	56 (95)	0

Data are presented as mean value ± SD or number (%) of patients. AV = atrioventricular; CL = cycle length.

t test and within groups with a paired *t* test. The Fisher exact test was used to compare the percent of conduction curves that exhibited evidence of dual AV node physiology before compared with after the ablation procedure. A significant difference was considered to exist when $p < 0.05$.

Regression analysis of AV node conduction was performed using data acquired before and after the procedure at a paced cycle length of 600 ms. Conduction through the fast AV pathway typically exhibited little change in the AH interval at long coupling intervals, but at shorter coupling intervals the AH interval prolonged before block occurred in the fast pathway. For each patient, the slope of the regression line of fast pathway conduction was determined over the range of coupling intervals that exhibited decremental conduction. Results obtained before the procedure were compared with those obtained after modification of AV node physiology using *t* tests. The slope of the regression line of the slow pathway was also determined over the range of coupling intervals that exhibited conduction through the slow pathway with a linear increment in the AH interval.

Results

Observed changes in dual AV node physiology. A mean of 18 ± 12 applications of radiofrequency current was delivered during the procedure. Table 2 summarizes the observed changes in AV node physiology. For the entire group of patients, there was no significant change in the AH interval, the cycle length at which AV node Wenckebach conduction was first observed or the HV interval. On the basis of the response to programmed atrial extrastimuli, AV node conduction was discontinuous in 55 patients (93%) at the beginning of the study. Discontinuous AV node conduction was detected in 24 (41%) of the patients after all attempts to ablate the slow pathway. Single AV node echo beats were induced in all 59 patients at the beginning of the study and in 11 patients (19%) after the ablation procedure was performed. Sustained AV node reentrant tachycardia was induced in 56 patients (95%) before the application of radiofrequency current. Six patients required an infusion of isoproterenol to induce sustained AV node reentry.

A mean of 8 ± 7 episodes of sustained AV node reentry (range 0 to 35) was induced before attempted ablation of the slow pathway. No patient had an inducible tachycardia or more than a single AV node reentrant beat after the slow pathway had been ablated or modified ($p < 0.0001$).

Analysis of the response to programmed atrial extrastimuli was based on data acquired at the beginning and conclusion of the study at paced cycle lengths of 700, 600, 500 or 400 ms. Not all cycle lengths could be evaluated in each patient because of individual variability in rest heart rate, exclusive conduction through the fast or slow pathway at all coupling intervals and the cycle length at which AV node Wenckebach conduction was observed. Accordingly, the number and percent of paced cycle lengths tested in patient groups that demonstrated dual AV node physiology before the ablation procedure were compared with results obtained after the procedure. Patients in whom dual AV node physiology was eliminated had evidence of dual AV node physiology in 68 (86%) of 79 paced cycle lengths tested in the initial portion of the study compared with 0 of 82 paced cycle lengths tested at the conclusion of the study ($p < 0.0001$). In the patients with residual dual AV node physiology, evidence of dual AV node physiology was detected in 53 (92%) of 57 paced cycle lengths tested at the initial portion of the study compared with 40 (68%) of 59 cycle lengths tested at the conclusion (p = NS).

Table 3 summarizes the response to programmed atrial stimulation before and after the ablation procedure in patients in whom dual AV node physiology was completely eliminated and in those in whom dual AV node physiology persisted. Initial recordings were made before the application of radiofrequency current or infusion of isoproterenol. Final recordings were made 1 h after the therapeutic end point had been reached and before isoproterenol was infused. The maximal increment in the AH interval in response to a 10-ms decrement in the atrial coupling interval, the AH jump, was determined at several paced cycle lengths in each patient. The maximal AH jump was used for patient to patient comparison. There was no significant difference in the magnitude of the AH jump before the ablation procedure in patients who ultimately had complete elimination of dual AV node physiology (94 ± 55 ms) compared with those who had persistent dual AV node physiology (95 ± 44 ms). Moreover, for the latter patients, there was no significant difference in the magnitude of the AH jump before (95 ± 44 ms) compared with after (100 ± 60 ms) modification of the slow AV node pathway.

Effective refractory periods. The difference between the effective refractory period of the slow and fast pathways was also compared. This difference represents the interval during which AV node reentry is likely to be induced. For each patient, the maximal difference between effective refractory periods of the fast and slow pathways measured at several paced cycle lengths was used for patient comparisons. Table 3 compares the maximal difference in the effective refractory periods of the fast and slow AV pathways at all cycle lengths tested, as well as the difference at specific paced cycle

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Table 3. Effects of Procedure on Dual Atrioventricular Node Physiology

Electrophysiologic Characteristics	Dual Physiology Eliminated	Dual Physiology Persisted	p Value
Maximal AH jump (ms)			
Initial	94 ± 55	95 ± 44	NS
Final	—	100 ± 60	—
p value		NS	
Difference between ERP of fast and slow AV pathways (ms)			
Maximal			
Initial	123 ± 85	104 ± 62	NS
Final	—	37 ± 37	—
p value	—	< 0.001	—
PCL 700 ms			
Initial	164 ± 80	143 ± 73	NS
Final	—	40 ± 20	—
p value	—	0.01	—
PCL 600 ms			
Initial	111 ± 93	105 ± 64	NS
Final	—	38 ± 28	—
p value	—	0.019	—
PCL 500 ms			
Initial	88 ± 58	81 ± 28	NS
Final	—	45 ± 33	—
p value	—	0.113	—
PCL 400 ms			
Initial	60 ± 29	52 ± 31	NS
Final	—	22 ± 25	—
p value	—	0.23	

p values listed in the left column compare initial with final results; p values in the right column compare results in patients who had dual atrioventricular (AV) node physiology eliminated with those of patients in whom it persisted. Data are presented as mean value ± SD. ERP = effective refractory period; PCL = paced cycle length.

Table 4. Effect of Procedure on Effective Refractory Periods of the Fast and Slow Pathways

	Dual Physiology Eliminated	Dual Physiology Persisted	p Value
Fast pathway (ms)			
PCL 700 ms			
Initial	466 ± 91	466 ± 79	NS
Final	352 ± 42	357 ± 36	NS
p value	0.001	0.003	
PCL 600 ms			
Initial	393 ± 102	411 ± 96	NS
Final	355 ± 68	353 ± 66	NS
p value	0.001	0.038	
PCL 500 ms			
Initial	364 ± 72	356 ± 63	NS
Final	304 ± 53	295 ± 72	NS
p value	0.043	0.086	—
PCL 400 ms			
Initial	310 ± 49	325 ± 28	NS
Final	274 ± 56	303 ± 15	NS
p value	0.015	0.2	—
Slow pathway (ms)			
PCL 700 ms			
Initial	281 ± 52	311 ± 27	NS
Final	—	311 ± 18	—
p value	—	NS	
PCL 600 ms			
Initial	280 ± 50	278 ± 44	NS
Final	—	311 ± 62	—
p value	—	0.045	
PCL 500 ms			
Initial	304 ± 29	287 ± 47	NS
Final	—	260 ± 49	
p value	—	0.131	
PCL 400 ms			
Initial	259 ± 41	268 ± 8	NS
Final	—	276 ± 31	
p value	—	0.578	

p values listed in the left column compare initial with final results; p values in the right column compare results in patients who had dual atrioventricular (AV) node physiology eliminated with those of patients in whom it persisted. Data are presented as mean value ± SD. Abbreviations as in Table 3.

lengths before and after the procedure. At paced cycle lengths of 700 and 400 ms, there was a significant decrease in this difference at the conclusion of the procedure compared with initial results in patients with residual dual AV node physiology. There was a similar trend at paced cycle lengths of 500 and 400 ms. There were no significant differences in these values before the ablation procedure between patients in whom dual AV node physiology was eliminated and those in whom it persisted.

The effective refractory periods of the fast and slow AV pathways were determined, when possible, at several paced cycle lengths before and after attempted ablation of the slow pathway. Results of analysis (Table 4) demonstrated that the effective refractory period of the fast pathway was significantly shorter at the conclusion of the procedure in patients without persistence of dual AV node physiology. In patients with residual dual AV nodal physiology, the effective refractory period was significantly shorter at paced cycle lengths of 700 and 600 ms, and this trend was present at paced cycle lengths of 500 and 400 ms. In patients with persistent dual AV node physiology, the effective refractory period of the slow pathway was significantly longer at a paced cycle length of 600 ms but not at the other cycle lengths tested.

Results of regression analysis demonstrated that as a consequence of the procedure, the slope of the fast pathway increased significantly in patients who had complete elimination of dual AV node physiology ($p = 0.017$), as well as those in whom AV node conduction remained discontinuous without reentry ($p = 0.034$) and those with residual single echo beats ($p = 0.024$). The slopes of the fast AV pathway conduction did not differ significantly among these groups, nor was there any significant difference among groups in the magnitude of change of slope before compared with after the procedure. A similar analysis was performed for the slope of conduction through the slow pathway. There was no significant difference in the slope of slow pathway conduction before compared with after the procedure in patients with

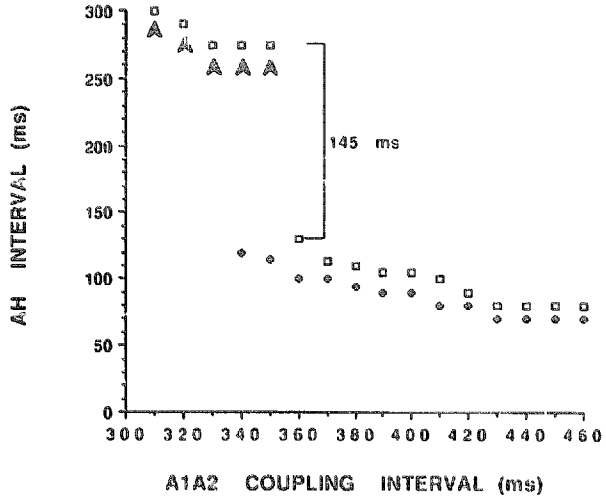


Figure 2. Atrioventricular node conduction curves demonstrating complete elimination of dual AV node physiology. The response to programmed atrial stimulation before (**open squares**) the ablation procedure demonstrated a discontinuity of 145 ms and conduction through the slow AV pathway was associated with reproducible induction of AV node reentrant supraventricular tachycardia (**arrows**). After the ablation procedure (**closed diamonds**), AV node conduction was continuous and reentry was not induced.

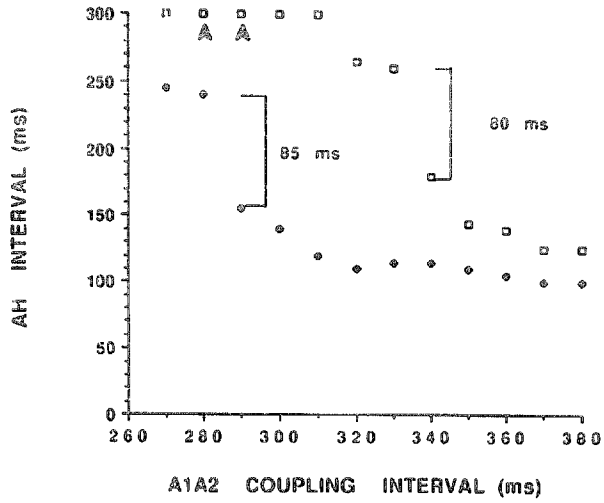


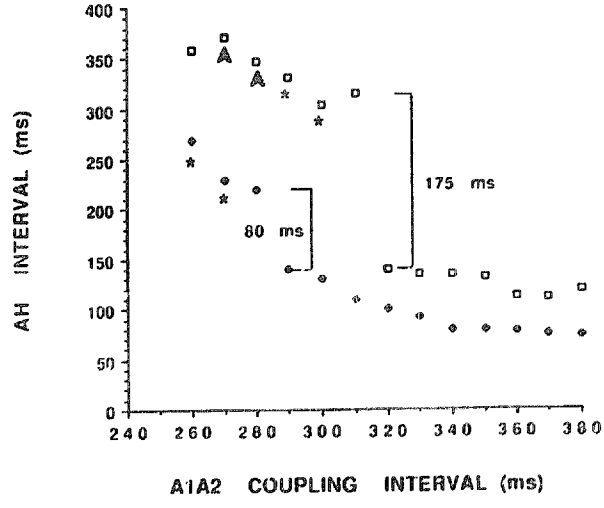
Figure 3. Persistence of discontinuous AV node conduction without reentry. The response to programmed atrial stimulation before (**open squares**) the ablation procedure exhibited a discontinuity of 80 ms and conduction through the slow AV pathway resulted in sustained AV node reentrant supraventricular tachycardia (**arrows**). After modification of the slow pathway (**closed diamonds**), AV node conduction exhibited a discontinuity of 85 ms, the difference between fast and slow pathway refractoriness was reduced from 70 to 20 ms and reentry was not induced.

residual dual AV node physiology. At the conclusion of the procedure, the slopes of slow pathway conduction did not differ between the group that had residual dual AV node physiology and the group without single reentrant echo beats, nor was there any difference between these groups in the magnitude of the change in slope.

Characteristic responses to atrial stimulation. Three patterns of responses of the AV node to programmed atrial stimulation were observed after the ablation procedure. All evidence of dual AV node physiology was eliminated in 59% of the patients. Representative AV node conduction curves before and after the procedure from a patient having this pattern of response are shown in Figure 2. In the second pattern, dual AV node physiology persisted, but reentry was not induced. This pattern was observed in 22% of the patients studied (Fig. 3). Moreover, in this group, the difference between the effective refractory periods of the fast and slow pathways decreased after the ablation procedure. In the third pattern, observed in 19% of the patients, dual AV node physiology persisted and single AV node reentrant (echo) beats were induced (Fig. 4). In patients with residual echo beats, the response to a sufficiently premature atrial extrastimulus was characterized by anterograde conduction through the slow pathway and retrograde conduction through the fast pathway with a short HA interval, followed by AV block in the slow pathway (Fig. 5). This response suggests that radiofrequency current modified the refractory properties of the slow AV pathway to an extent that precluded repetitive conduction, which would be required to sustain AV node reentry.

Follow-up. Table 5 summarizes the clinical course of the 59 patients during a mean follow-up period of 15 ± 6 months (range 4 to 28). No patient had a documented recurrence of supraventricular tachycardia. One patient, in whom dual AV

Figure 4. Persistence of discontinuous AV node conduction with single reentrant beats. Before ablation, AV node conduction (**open squares**) showed a discontinuity of 175 ms and conduction through the slow pathway resulted in sustained supraventricular tachycardia (**arrows**). After the slow pathway had been modified (**closed diamonds**), a discontinuity of 80 ms was observed and single AV node echo beats were induced (**star**). The difference between the refractory periods of the fast and slow pathways was reduced from 60 to 30 ms.



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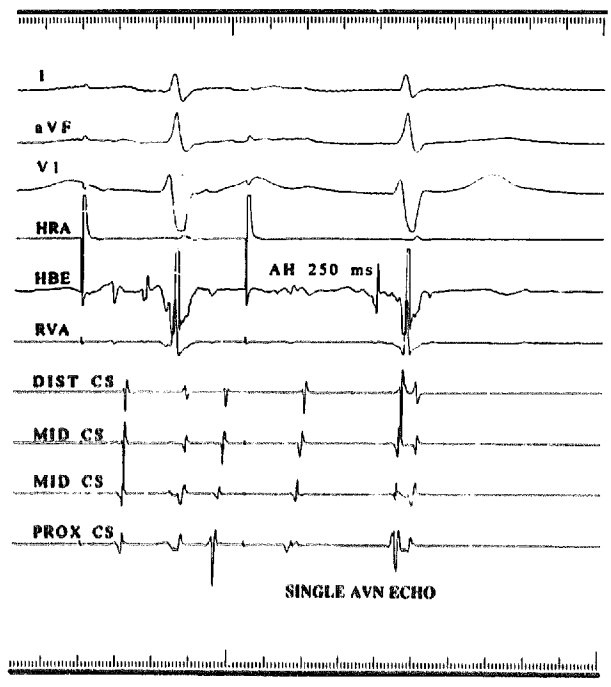

Figure 5. Representative recording of residual dual AV node physiology with a single retrograde atrial echo beat (AVN ECHO) at the conclusion of the procedure. Anterograde AV conduction occurs through the fast AV pathway at a paced cycle length of 600 ms. Anterograde conduction occurs through the slow AV pathway in response to a premature atrial extrastimulus and is followed by retrograde conduction over the fast AV node pathway. CS = coronary sinus; DIST = distal; HBE = His bundle electrogram; HRA = high right atrium; PROX = proximal; RVA = right ventricular apex.

node physiology had been eliminated, has experienced over a period of 1 year three episodes of palpitation, lasting up to 10 min, that were very similar to the symptoms she experienced daily before the ablation procedure. This patient declined to undergo follow-up study. If one assumes that these symptoms represented AV node reentry, the recurrence rate for the entire group was 2%. None of the patients with persistent dual AV node physiology have had a recurrence of supraventricular tachycardia. There was no difference between the recurrence rate for patients with (0 of 24) and that of patients without (1 of 35) residual dual AV node physiology. There was no significant difference in the length of the follow-up interval for patients who had complete elimination of dual AV node physiology and that of patients with persistent evidence of dual AV node physiology.

Table 5. Results of Follow-Up Study

Group	No. of Patients	Months of Follow-Up (mean; range)	Patients With Arrhythmia Recurrence
Dual AV node physiology eliminated	35 (59)	15 ± 6; 4 to 28	1 (3)
Dual AV node physiology persisted			
No reentry	13 (22)	17 ± 7; 4 to 28	0
Single AV node echo beat	11 (19)	15 ± 5; 6 to 21	0
Combined	59	15 ± 6; 4 to 28	1 (2)

Data are expressed as number (%) of patients, mean value ± SD or range.

Complications. Three patients developed symptomatic AV node Wenckebach block within 24 h of the ablation procedure. We conclude that the fast pathway was damaged; however, this effect was not apparent at the end of the procedure. None of these patients exhibited prolongation of the effective refractory period of the fast pathway; the paced cycle length at which AV Wenckebach block occurred was unchanged and retrograde conduction remained intact. In each case, radiofrequency current had been applied relatively close to the AV node–His bundle junction. Conduction returned to normal in two of the patients within a few days and no intervention was required. In the third patient, symptomatic AV node Wenckebach block at rest persisted even though AV conduction was intact during exercise. A permanent pacemaker was implanted 3 months after the ablation procedure. Two other patients have had inappropriate sinus tachycardia at rest. These patients had normal findings on electrocardiogram, echocardiogram, erythrocyte sedimentation rate and chest X-ray study. Neither patient had chest pain or objective evidence of pericarditis. Inappropriate sinus tachycardia has persisted for months and has required treatment with low doses of beta-adrenergic antagonists. The patients who developed AV conduction abnormalities or inappropriate sinus tachycardia had each received >20 applications of radiofrequency current.

Discussion

Development of procedures to eliminate AV node reentry. In 1979, Pritchett et al. (9) reported that during attempted surgical dissection of the His bundle in a patient with incessant AV node reentry, the tachycardia was abolished and sinus rhythm with normal AV conduction was preserved. This fortuitous result demonstrated the feasibility of modifying the substrate required for AV node reentry as a curative procedure. Subsequent studies in experimental animals and the surgical experience in humans established that at least a portion of the tissue that mediates AV node reentry resides outside the anatomic confines of the AV node (10–15). The success of cryothermal lesions around the triangle of Koch in preventing AV node reentry was often associated with ablation of the slow AV pathway (14,15). Although the precise location of tissue that mediated slow AV conduction was not identified intraoperatively, results of clinical electrophysiologic studies (2–5,16,17) have suggested that atrial tissue near the os of the coronary sinus is responsible for the phenomenon of slow AV pathway conduction.

Results obtained intraoperatively and in the electrophysiology laboratory have provided the basis for modification of AV node physiology by energy delivered from catheters

introduced percutaneously with electrodes positioned near the AV node. The early experience with modification of AV node physiology by delivering direct current shocks proximal to the region where optimal His bundle electrograms were recorded resulted in apparent ablation or modification of the fast AV node pathway and was associated with a 10% risk of inducing complete heart block (18,19). Additional experience with radiofrequency current has improved the efficacy of this approach and the risk of inducing complete heart block has decreased (20).

The possibility that slow AV pathway conduction could be altered by ablating tissue remote from the anatomic AV node–His bundle junction attracted interest because it offered promise for reduced risk of inducing AV block. Several groups (2–5) have established that selective ablation of the slow AV pathway can be accomplished by applying radiofrequency current near the tricuspid annulus between the os of the coronary sinus and the His bundle, where presumed slow pathway potentials are recorded. In some patients, this effect is achieved easily, but in others complete elimination of dual AV node physiology is difficult. Repeated attempts to abolish all evidence of dual AV node physiology may result in a large number of applications of radiofrequency current near the AV node, which increases the risk of inducing second- or third-degree AV block or inappropriate sinus tachycardia, increases fluoroscopy time and prolongs the entire procedure.

In the present study, we attempted to eliminate slow AV conduction, but hypothesized that modification of the refractory properties of this tissue might suffice to prevent a clinical recurrence of the tachycardia. Results of electrophysiologic testing conducted 1 h after completion of the ablation procedure demonstrated discontinuous AV node conduction curves without evidence of reentry in 22% of patients. Patients who exhibited this response were categorized as having residual dual AV node physiology based on the established criterion of an increment in the AH interval of 50 ms in response to a 10-ms decrement in the A_1A_2 coupling interval. There are no established criteria that differentiate this response from decremental conduction through the fast pathway. The absence of AV node echo beats in patients who exhibited this response raises the possibility that the slow pathway had been ablated. However, the magnitude of the increment in the AH interval (AH jump) before compared with after the procedure was not significantly changed in these patients. The difference between the effective refractory periods of the fast and slow pathways, which represented the range of coupling intervals at which reentry could occur, was significantly decreased. These changes may have altered the properties of the reentrant circuit sufficiently to prevent retrograde conduction through the fast pathway at coupling intervals when anterograde conduction through the slow pathway could be demonstrated.

Discontinuous AV node conduction curves with single reentrant beats that blocked anterogradely in the slow AV pathway were observed in 19% of the patients. In the patients who exhibited single AV node reentrant beats, the difference between slow and fast pathway refractoriness was reduced. This difference, which corresponds to the range of coupling intervals during which spontaneous or programmed atrial extrastimuli are likely to induce AV node reentry, was primarily attributable to shortening of the effective refractory period of the fast pathway. Although the effective refractory period of the slow pathway was not changed by the procedure, the protocol assessed the response to single atrial extrastimuli. Further study might evaluate the response to multiple atrial extrastimuli. We conclude that repetitive conduction through the slow pathway was affected and that this was the reason that repetitive AV node reentry was not induced.

Although the objective of this study was to selectively ablate or modify the slow AV pathway, the response to programmed atrial stimulation at the conclusion of the study demonstrated shortening of the effective refractory period of the fast pathway at several paced cycle lengths. There was no significant change in the AH interval at rest before compared with after the procedure, nor was there a change in the paced cycle length at which AV node Wenckebach block was observed. Moreover, retrograde conduction remained intact in all patients studied. At the conclusion of the study, however, the response to programmed atrial stimulation demonstrated shortening of the effective refractory period of the fast pathway at several paced cycle lengths. These results suggest that the function of the fast pathway is affected by the presence of the slow pathway. Shortening of the effective refractory period and increased slope of the conduction curve of the fast pathway may reflect an alteration of parasympathetic or sympathetic innervation of the AV node or functional interactions may exist between the two pathways.

A mean of 18 ± 12 applications of radiofrequency current was applied in this study. One limitation of the technology employed at the time these patients were studied was that we could not determine whether ineffective applications were not optimally positioned with respect to the location of the slow pathway or whether tissue contact was inadequate. The use of thermistor catheters may resolve this issue in the near future. One reason that the number of lesions applied was relatively high is that we attempted to eliminate all evidence of dual AV node physiology. Our impression is that repetitive AV node reentry can often be eliminated by relatively few applications of radiofrequency current at the site of presumed slow pathway potentials. Attempted elimination of all evidence of dual AV node physiology increases the number of lesions and appears to be unnecessary. We are concerned that the risk of complications may increase with the number of radiofrequency current applications, but the results of this study do not define a minimal or maximal number of applications that should be attempted during an ablation procedure.

A salient feature of the protocol employed in this study

was that final testing was deferred for 1 h after the therapeutic end point had been achieved. The time course of recovery of slow pathway function was not studied systematically, but it was frequently observed during the 1st 30 min after applications of radiofrequency current and often prompted additional attempts to eliminate or modify the slow AV pathway. We also observed that dual AV node physiology was occasionally evident at one but not at other paced cycle lengths. Protocols that restrict the interval between applications of radiofrequency current and testing or evaluate the response to programmed atrial extrastimuli at only a single paced cycle length might obtain different results.

Limitations of the study. One limitation of this investigation was that a follow-up study was not performed. It would have been of interest to determine whether the immediate effects of the procedure on dual AV node physiology were concordant with the long-term effects. We chose not to perform a follow-up study, however, because the primary objective of the procedure was to eliminate symptoms. This study, which includes a mean follow-up interval of 15 months, demonstrates a successful long-term result. It is highly unlikely that results of a follow-up study would alter that conclusion. Moreover, other investigators (1,2) have reported little change in AV node physiology during follow-up, and it is difficult to justify an invasive follow-up procedure for asymptomatic patients whose prior arrhythmias were not life-threatening.

Clinical implications. The results of this study have important implications for electrophysiologists who perform ablation procedures for patients with AV node reentry. Protracted attempts to eliminate single AV node reentrant beats are unnecessary. Moreover, we are concerned that the risk of complications is related not only to the site at which radiofrequency current is applied, but also to the cumulative energy employed during the procedure. When complete elimination of dual AV node physiology is not easily accomplished, single AV node reentrant beats in response to programmed atrial stimulation represent a satisfactory therapeutic end point and portend a low risk for recurrence of the clinical tachycardia in patients whose arrhythmia was easily inducible before radiofrequency current was applied.

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