TREATMENT OF SUPRAVENTRICULAR TACHYCARDIA DUE TO ATRIOVENTRICULAR NODAL REENTRY BY RADIOFREQUENCY CATHETER ABLATION OF SLOW-PATHWAY CONDUCTION

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Abstract *Background.* Atrioventricular nodal reentrant tachycardia (AVNRT), the most common form of supraventricular tachycardia, results from conduction through a reentrant circuit comprising fast and slow atrioventricular nodal pathways. Antiarrhythmic-drug therapy is not consistently successful in controlling this rhythm disturbance. Catheter ablation of the fast pathway with radiofrequency current eliminates AVNRT, but it can produce heart block. We hypothesized that catheter ablation of the site of insertion of the slow pathway into the atrium would eliminate AVNRT while leaving normal (fast-pathway) atrioventricular nodal conduction intact.

Methods and Results. Eighty patients with symptomatic AVNRT were studied. Retrograde slow-pathway conduction (in which the earliest retrograde atrial potential was recorded at the posterior septum, close to the coronary sinus) was present in 33 patients. The retrograde atrial potential was preceded by a potential consistent with activation of the atrial end of the slow pathway (A_{SP}). In 46 of the 47 patients without retrograde slow-pathway conduction, a potential with the same characteristics as the A_{SP} potential was recorded during sinus rhythm. Radiofrequency current delivered through a catheter to the A_{SP} site (in the posteroseptal right atrium or coronary sinus) abolished or modified slow-pathway conduction in 78 patients, eliminating AVNRT without affecting normal atrioventricular nodal conduction. In the single patient without A_{SP}, the application of radiofrequency current to the proximal coronary sinus ablated the fast pathway and AVNRT. Atrioventricular block occurred in one patient (1.3 percent) with left bundle-branch block, after inadvertent ablation of the right bundle branch. AVNRT has not recurred in any patient during a mean (±SD) follow-up of 15.5±11.3 months. Electrophysiologic study 4.3±3.3 months after ablation in 32 patients demonstrated normal atrioventricular nodal conduction without AVNRT.

Conclusions. Catheter ablation of the atrial end of the slow pathway using radiofrequency current, guided by A_{SP} potentials, can eliminate AVNRT with very little risk of atrioventricular block. (N Engl J Med 1992;327:313-8.)

ATRIOVENTRICULAR nodal reentrant tachycardia (AVNRT) is the most common form of paroxysmal supraventricular tachycardia.[1] The reentrant circuit contains, as its critical components, a "fast" atrioventricular nodal pathway connecting the atrium to the His bundle and a "slow" pathway with a longer conduction time. In approximately 90 percent of patients, the slow pathway provides the antegrade limb of the circuit and the fast pathway the retrograde limb (the "slow–fast" or common form of AVNRT), but the circuit may be reversed (to the "fast–slow" or uncommon form). The fast pathway is thought to be the normal route of atrioventricular conduction, whereas the slow pathway has no known function.

Surgical procedures designed to interrupt the connections between the atrioventricular node and the atria eliminate AVNRT, with an incidence of heart block of less than 3 percent.[2-5] AVNRT can also be eliminated by modifying the atrioventricular node with destructive energy delivered through catheters near the junction of the node with the His bundle.[6-10] The atrioventricular nodal conduction time is prolonged after such procedures, indicating that the normal antegrade fast pathway has been destroyed or damaged. In some patients (up to 10 percent), atrioventricular conduction is totally eliminated, necessitating the implantation of a pacemaker.[6-10]

The junction of the slow pathway and the atrium is located posterior to the atrioventricular node, near the coronary sinus.[11] We postulated that slow-pathway conduction could be interrupted without affecting normal atrioventricular conduction through the fast pathway by ablating small segments of myocardium in the posterior septum near the coronary sinus with radiofrequency current delivered through catheter electrodes. Our objective was to abolish AVNRT with a low risk of heart block.

METHODS

Study Population

The study population consisted of the first 80 patients to undergo catheter ablation using radiofrequency current at the site of the atrial insertion of the slow pathway in order to eliminate AVNRT. There were 24 men and 56 women, ranging in age from 4 to 76 years (mean [±SD], 41.7±17.5). Fourteen had structural heart disease: coronary artery disease in three, mitral-valve prolapse in five, aortic valve disease in two, cardiomyopathy in two, pulmonic stenosis and ventricular septal defect in one, and situs inversus in one. The patients had had episodes of tachycardia for 14.9±11.9 years and had been treated unsuccessfully with 3.0±1.6 antiarrhythmic drugs. Tachycardia occurred every 1 to 7 days in 48 of the 80 patients, every 7 to 30 days in 17 patients, and at longer intervals in 15 patients. Thirty-one patients had syncope, 20 others had near-syncope, and 62 had substantial chest pain or dyspnea associated with the tachycardia.

The study protocol was approved by the institutional review board. After providing written informed consent, each patient was studied in the fasting state under heavy sedation with fentanyl (50 to 150 μg per hour) and midazolam (2 to 6 mg per hour). Five multielectrode catheters (with a distance of 2 mm between electrodes) were inserted percutaneously into the right subclavian, right fem-

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oral, and left femoral veins.[12] Sustained tachycardia was induced by programmed stimulation, and the presence of mechanisms other than atrioventricular nodal reentry was excluded by standard techniques.[13] The antegrade and retrograde conduction properties of the fast and slow atrioventricular nodal pathways were assessed by programmed atrial and ventricular stimulation.

In patients who had retrograde conduction over the slow pathway, the atrial insertion of this pathway was defined as the site of earliest atrial activation during such conduction, as located by mapping the right atrial septum and the coronary sinus. Selective retrograde conduction over the slow pathway was obtained by inducing either the uncommon form of AVNRT or single echo beats that use the slow pathway for retrograde conduction, or by techniques of ventricular pacing that resulted in retrograde block in the fast pathway. Such a block was facilitated by heavy sedation and, in some patients, by the administration of adenosine or esmolol. Electrograms recorded near the sites of earliest atrial activation during retrograde slow-pathway conduction showed a characteristic pattern of dual potentials (Fig. 1A). Separate origins for the two potentials were suggested by the response to late atrial extrasystoles, which advanced the timing of the second potential (coincident with the advance of the atrial potential in the nearby His-bundle recording) without altering the timing or morphologic aspects of the first potential (Fig. 2C). This response suggests that the second potential represents local atrial activation. The first potential (A_{SP}) may represent the activation of the atrial connection (or insertion) with the slow pathway. Recordings at these sites during sinus rhythm also showed the A_{SP} and atrial potentials, but in reverse order, with the A_{SP} potential following the atrial potential (Fig. 1B and 2A). The A_{SP} potential in sinus rhythm was used to identify the atrial insertion of the slow pathway in the patients who did not have retrograde slow-pathway conduction (Fig. 3A).

Application of Radiofrequency Current

A quadripolar catheter with a distance of 2 mm between electrodes, a large-tip electrode (7 French; length, 4 mm), and a deflectable curve was used for ablation.[14] The catheter was inserted through a right femoral venous sheath, and the tip electrode was positioned firmly at the site from which the largest, sharpest, and earliest A_{SP} potential was recorded (from the distal pair of electrodes) during retrograde slow-pathway conduction (Fig. 1A and 2B), or from which the largest, sharpest, and latest A_{SP} potential was recorded during sinus rhythm (Fig. 3A). Radiofrequency current (550 to 750 kHz) was delivered at 45 to 70 V between the catheter-tip electrode and an adhesive electrosurgical dispersive pad applied to the left posterior chest.[12,14] Root-mean-square voltage, current, and impedance were monitored. Current was applied for 45 seconds or longer but was terminated immediately in the event of an increase in impedance or displacement of the catheter electrode.

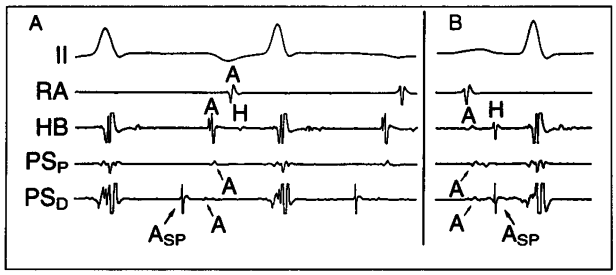

Figure 1. Characteristic Potentials Recorded from the Posterior Septum at the Site of Successful Slow-Pathway Ablation in a Patient with Both Common (Slow–Fast) and Uncommon (Fast–Slow) Forms of AVNRT.

From the top, the figure shows a tracing from Lead II and electrograms recorded from the right atrial appendage (RA), the His-bundle region (HB), and the proximal (PS_P) and distal (PS_D) pairs of electrodes of the mapping catheter, positioned against the posteroseptal right atrium between the coronary-sinus ostium and the tricuspid annulus. During fast–slow AVNRT (Panel A), retrograde conduction occurred over the slow pathway, and the earliest retrograde atrial potential was recorded from the PS_D electrodes, which showed a small atrial potential (A), nearly coincident with the atrial potentials in the PS_P and HB electrograms. It was preceded by a large A_{SP} potential (large arrow) recorded 60 msec before the onset of the P wave. During sinus rhythm (Panel B), when the slow pathway was activated in the antegrade direction, the order of the two potentials in PS_D was reversed, with the A potential still coincident with the other atrial potentials. The application of radiofrequency current at this site eliminated antegrade and retrograde slow-pathway conduction and both forms of AVNRT.
H denotes His-bundle potential.

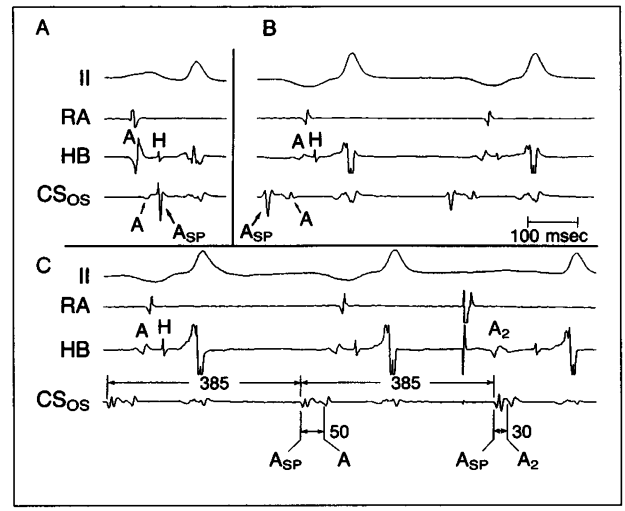

Figure 2. Evidence of Separate Origins of the A_{SP} and Atrial Potentials.

The bottom tracing in each panel (CS_{OS}) was recorded from the posterior margin of the coronary-sinus ostium. During sinus rhythm (Panel A), a large A_{SP} potential followed a small atrial potential (A) in the CS_{OS} electrogram. During fast–slow AVNRT, with retrograde conduction over the slow pathway (Panel B), the large A_{SP} potential preceded the smaller A potential. An atrial extrastimulus during fast–slow AVNRT (Panel C) advanced the timing of atrial activation (A_2) in the CS_{OS} electrogram by 20 msec (so that A_2 followed A_{SP} by 30 msec, as compared with 50 msec before the extrastimulus) without altering the timing or morphology of the A_{SP} potential (the interval between A_{SP} potentials remained constant at 385 msec), suggesting that the A_{SP} potential does not result from local atrial activation and may represent activation of the atrial insertion of the slow pathway. RA denotes right atrium, HB His bundle, and H His-bundle potential.

Radiofrequency current was applied during AVNRT or right ventricular pacing with retrograde slow-pathway conduction in 18 patients. In the remaining 62 patients, radiofrequency current was applied during sinus rhythm for improved electrode–tissue contact because of the slower heart rate and to monitor antegrade fast-pathway conduction. After the application of current, programmed atrial and ventricular stimulation was repeated before and during the administration of isoproterenol (1 to 2 μg per minute). If AVNRT or 1:1 slow-pathway conduction (antegrade or retrograde) was still present during decremental atrial or ventricular pacing, radiofrequency current was reapplied at the same site after electrode–tissue contact had been improved or at another site if there was a change in the site of earliest atrial activation during retrograde slow-pathway conduction or in the site from which a large, sharp A_{SP} potential was recorded. The procedure was terminated when AVNRT could not be induced and 1:1 slow-pathway conduction was absent for at least one hour.

Treatment after Ablation

The patients were monitored in an ambulatory unit and discharged two days after ablation. Transesophageal echocardiography was performed 18 to 72 hours after ablation in 70 patients.

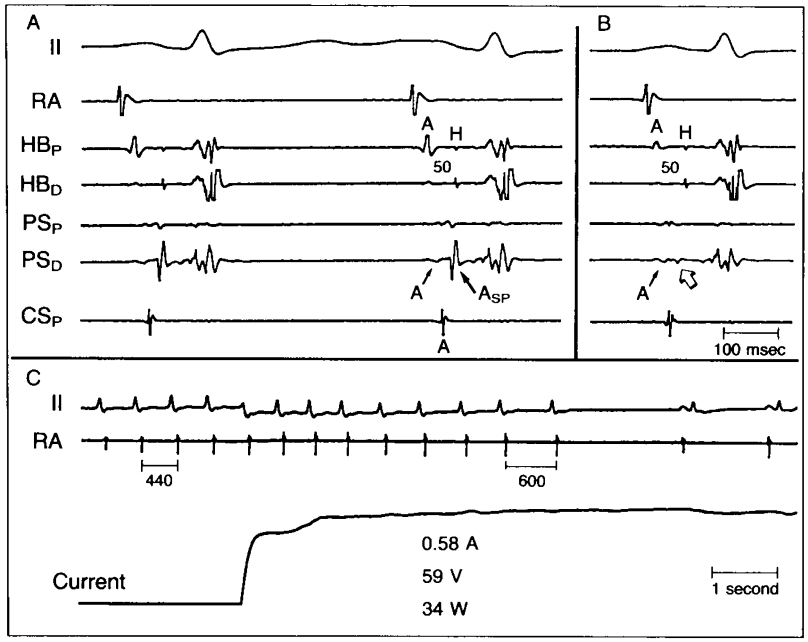

Figure 3. Effects of Slow-Pathway Ablation on the A_{SP} Potential and Fast-Pathway Conduction.

The tracings shown were recorded from a patient with slow–fast AVNRT and no retrograde slow-pathway conduction. Before ablation (Panel A), the largest, sharpest, and latest A_{SP} potential during sinus rhythm was recorded from the posteroseptal catheter (PS_D electrogram), positioned near the tricuspid annulus, posterior to the coronary-sinus ostium. The A_{SP} potential was recorded after atrial activation in the proximal coronary sinus (the A potential in the CS_P electrogram). Radiofrequency current applied at this site (Panel C) eliminated slow-pathway conduction. Immediately after ablation (Panel B), the A_{SP} potential (open arrow) was markedly attenuated. The antegrade fast atrioventricular nodal pathway conduction time, as measured by the atrial–His bundle (AH) interval in the HB_P electrogram, was unchanged from the preablation value of 50 msec. During slow–fast AVNRT, 59 V of radiofrequency current (current, 0.58 A; power, 34 W) was applied to the large-tip electrode on the PS catheter, which recorded the large A_{SP} potential in Panel A. The bottom tracing in Panel C indicates the root-mean-square output of current from the radiofrequency generator. Beginning two seconds after the start of delivery of current, the cycle length of the tachycardia increased progressively from 440 to 600 msec, and tachycardia ended at five seconds. The increase in cycle length and the termination of tachycardia resulted from the prolongation of conduction time and then from conduction block in the slow pathway. After the termination of tachycardia, the PR interval was not prolonged during sinus rhythm, indicating that ablation did not affect antegrade fast-pathway conduction. RA denotes right atrium, and HB His bundle.

The last 31 patients to be studied received aspirin (325 mg daily) for six weeks. Anticoagulant therapy was not used routinely in the other patients. None of the patients received antiarrhythmic drugs after ablation.

The patients were followed by the investigators or the referring physicians. Electrocardiographic monitoring was not performed routinely on an outpatient basis. A follow-up electrophysiologic study two to three months after ablation was recommended. Follow-up information was obtained by telephone or written questionnaire.

Statistical Analysis

Electrophysiologic measurements obtained before and after ablation, as well as those obtained before ablation and during follow-up, were compared by a two-tailed, paired t-test using the means procedure provided by an SAS statistical package.

Results

Programmed atrial and ventricular stimulation induced slow–fast AVNRT in 69 of the 80 patients (86 percent), fast–slow AVNRT in 4 patients (5 percent), and both forms in 7 patients (9 percent). The mean cycle length of the tachycardia was 338±70 msec.

Retrograde fast-pathway conduction was present in all patients. The site of earliest atrial activation during retrograde fast-pathway conduction was located in the anterior septum, close to the bundle of His (shaded region in Fig. 4). Selective retrograde conduction over the slow pathway (for at least one complex) was produced in 33 patients (41 percent), including the 11 patients with fast–slow AVNRT and 22 of the 69 patients who had only slow–fast AVNRT. In all 33 patients, the shift from retrograde fast-pathway conduction to retrograde slow-pathway conduction was associated with a shift in the site of earliest atrial activation to the posterior septum, either in the region between the coronary-sinus ostium and the tricuspid annulus or in the coronary sinus (Fig. 4). Bipolar electrograms recorded near the site of the earliest atrial activation during retrograde slow-pathway conduction showed a distinct A_{SP} potential preceding the retrograde atrial potential in all 33 patients (Fig. 1A and 2B). The A_{SP} potential was also recorded during sinus rhythm, in which case it followed the atrial potential (Fig. 1B and 2A).

In 46 of the 47 patients without retrograde slow-pathway conduction, a potential with the same characteristics as the A_{SP} potential was recorded during sinus rhythm in the posteroseptal region (Fig. 3A). The largest, sharpest, and latest A_{SP} potential in sinus rhythm was most often recorded along the posteroseptal right atrium, close to the tricuspid annulus (Fig. 4), where the atrial potential was small and the ventricular potential was large (Fig. 1B and 3A). The A_{SP} potential was often recorded after atrial activation in the proximal coronary sinus (Fig. 3A). The region in which a large, sharp A_{SP} potential was recorded was usually less than 5 mm in diameter, and its location varied among the patients (Fig. 4). The interval between the local atrial potential and the A_{SP} potential during sinus rhythm ranged from 10 to 40 msec (mean, 24.3±7.3).

Catheter ablation using radiofrequency current eliminated AVNRT in all 80 patients in a single procedure. This resulted from the elimination or depression of slow-pathway conduction in 78 of the 80 patients. In 27 of these 78 patients (35 percent), both antegrade and retrograde slow-pathway conduction were completely eliminated. Some degree of slow-pathway con-

duction remained in 51 patients (65 percent), but the residual slow pathway could not sustain 1:1 conduction, and only single echo beats occurred with programmed stimulation during the administration of isoproterenol.

The sites of successful application of radiofrequency current were distributed broadly in the posteroseptal region, with most located along the posteroseptal right atrium between the coronary-sinus ostium and the tricuspid annulus (Fig. 4). In 14 of the 78 patients, energy applied at each of two to four separate sites altered slow-pathway conduction substantially, as reflected by an increase in conduction time or a shift in the site of earliest atrial activation during retrograde slow-pathway conduction.

The selective effect on slow-pathway conduction of energy applied in the posteroseptal region is shown in Figure 3. Successful applications of current were often associated with attenuation or alteration of the A_{SP} potential (Fig. 3B). Fast-pathway conduction was not significantly depressed in either the antegrade or the retrograde direction in these 78 patients (Table 1).

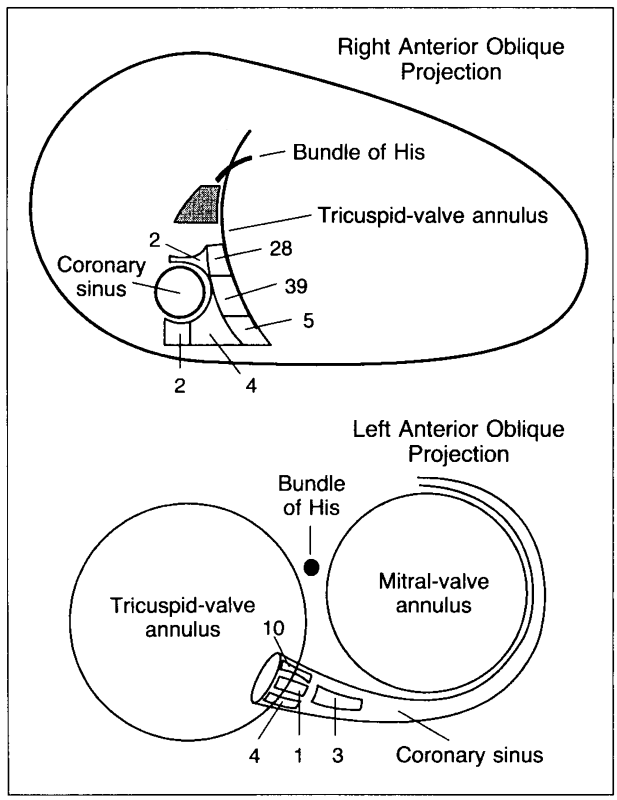

Figure 4. Schematic Representation of the Septum as Viewed Fluoroscopically in the Right and Left Anterior Oblique Projections, Showing the 98 Sites of Successful Slow-Pathway Ablation in the 78 Patients.

Numbers indicate the number of sites of successful ablation in each region. During selective retrograde fast-pathway conduction, the earliest atrial activation was recorded in the anterior septum, near the bundle of His (shaded region). During selective retrograde slow-pathway conduction, the earliest atrial potential was recorded in the posterior septum or proximal coronary sinus (unshaded regions).

Table 1. Effects of Slow-Pathway Ablation on Fast Atrioventricular Nodal Pathway Conduction.*

Variable†	Immediate Effects (N = 78)		Effects after 4.3±3.3 Mo of Follow-up (N = 22)	
	Before Ablation	After Ablation	Before Ablation	After Follow-up
	milliseconds			
AH interval	60±16	59±15	60±14	65±17
Fast-pathway conduction				
1:1 CL antegrade	378±90	360±80	343±77	364±84
ERP antegrade	334±76‡	314±69‡	323±61	321±78
1:1 CL retrograde	321±76	314±65	342±84	342±101

*Plus–minus values are means ±SD.

†AH interval denotes the atrial–His bundle interval, 1:1 CL antegrade the shortest atrial pacing cycle length that maintains 1:1 antegrade fast atrioventricular nodal pathway conduction, ERP antegrade the antegrade effective refractory period of the fast atrioventricular nodal pathway measured at the same atrial pacing cycle length before and after ablation (usually 500 msec), and 1:1 CL retrograde the shortest ventricular pacing-cycle length that maintains 1:1 retrograde fast atrioventricular nodal pathway conduction.

‡P = 0.01 for this comparison. None of the other comparisons shown were statistically significant at the 0.05 level.

Slow-pathway conduction was not ablated in two patients. In one patient no A_{SP} potential was recorded, and applications of current at the ablation sites usually used were ineffective. Applying radiofrequency current to the anterior wall of the proximal coronary sinus selectively eliminated fast-pathway conduction and AVNRT. Antegrade slow-pathway conduction persisted. The other patient, one of the first studied, had preexisting left bundle-branch block. Inadvertent movement of the catheter to the region of the proximal right bundle branch during the application of radiofrequency current produced right bundle-branch block, causing complete atrioventricular block with the persistence of AVNRT. Radiofrequency current was then intentionally delivered over the atrioventricular node, eliminating the tachycardia. A dual-chamber pacemaker was implanted.

The elimination of AVNRT required a median of 2 (range, 1 to 33) applications of radiofrequency current. A median of only one application was required in the last 55 patients in the series. The successful applications of radiofrequency current were delivered for 45±25 seconds at 59±7 V, 0.63±0.10 A, and 37.1±9.8 W. The mean duration of the procedure was 8.0±2.6 hours (5.6±1.4 hours in the last 10 patients studied).

Follow-up

The 80 patients have been followed for a range of 1 to 40 months (mean, 15.5±11.3) without a recurrence of tachycardia in any patient. Follow-up electrophysiologic studies were performed in 32 patients (40 percent) 4.3±3.3 months after ablation. Programmed atrial and ventricular stimulation at base line and during isoproterenol administration failed to induce AVNRT in all 32 patients. Quantitative analysis of fast-pathway and slow-pathway conduction was available for the 22 patients who underwent follow-up studies at our institution. The characteristics of antegrade and retrograde conduction over the fast pathway were not significantly different from the preablation values

(Table 1). Slow-pathway conduction was completely absent in 12 of the 22 patients. During decremental atrial pacing, 1:1 slow-pathway conduction was present in only two patients and was maintained only to pacing-cycle lengths of 380 and 420 msec. In the remaining eight patients, programmed atrial stimulation induced only single ventricular complexes conducted over the slow pathway.

Forty-eight patients did not undergo repeat electrophysiologic study, 27 of whom have been followed for more than six months. Of the 27 patients, 19 had weekly or monthly (13 and 6 patients, respectively) episodes of tachycardia before ablation.

Complications

Complications of the procedure occurred in five patients (6.3 percent). In one patient (1.3 percent), complete atrioventricular block developed as described above. The second patient had a small pulmonary embolus three days after ablation. Because of a previous pulmonary embolus, this patient received heparin during the procedure and for 24 hours thereafter. In three asymptomatic patients, thrombi were identified by transesophageal echocardiography: in the right atrial appendage in one, in the superior vena cava near the right atrial junction in another, and in the inferior vena cava near the right atrial junction in the third. The thrombi produced no overt clinical manifestations. Transesophageal echocardiography did not identify a thrombus at the site of ablation, a tricuspid-valve injury, or a pericardial effusion in any patient. There were no late complications.

Discussion

This report describes a new approach to the treatment of AVNRT in which radiofrequency energy is delivered through a catheter to the atrial insertion of the slow pathway in the posterior septum, remote from the atrioventricular node and the atrial insertion of the fast pathway. The fast pathway is the normal route of atrioventricular conduction in sinus rhythm. AVNRT was eliminated in all patients with a single procedure, without atrioventricular nodal block in any patient. Atrioventricular block within the His–Purkinje system occurred in one patient with preexisting left bundle-branch block after an inadvertent application of current to the proximal right bundle branch. The elimination of slow-pathway conduction had no effect on normal fast-pathway conduction. It is important to note that this approach was equally effective for the common (slow–fast) and uncommon (fast–slow) forms of AVNRT.

Other recently developed methods of eliminating AVNRT by catheter ablation, referred to as atrioventricular nodal modification, have directed the ablation to the retrograde fast pathway by positioning the ablation electrode at the site that recorded the earliest retrograde atrial potential. Slow–fast AVNRT was eliminated in 76 to 95 percent of the patients, but there was an 8 to 9.5 percent incidence of complete atrioventricular block.[7-9] With refinement of this technique, slow–fast AVNRT was eliminated in 84 percent of patients in a single procedure and in 95 percent of patients in two sessions, with an incidence of heart block of 2.2 percent at the level of the atrioventricular node.[10] Attempted ablation of fast–slow AVNRT in two patients was unsuccessful.[10]

Our observation that the A_{SP} potential was always recorded at sites of successful slow-pathway ablation has practical and functional importance. In patients without retrograde slow-pathway conduction, the A_{SP} potential recorded during sinus rhythm provided guidance to sites where slow-pathway ablation was successful. Several observations from this study suggest that the A_{SP} potential may represent the activation of the atrial end of the slow pathway. First, the sequence of atrial and A_{SP} potentials is reversed during antegrade (sinus-rhythm) and retrograde slow-pathway conduction; in the latter, the A_{SP} potential precedes the retrograde atrial potential. Second, the retrograde A_{SP} potential can be dissociated from the atrial potential. Finally, energy delivered at the sites from which the largest, sharpest A_{SP} potential was recorded is associated with the successful ablation of slow-pathway conduction without alteration of fast-pathway conduction. The fibers generating the A_{SP} potentials are unknown, but they may be located in the plane of connective tissue that separates the thin layer of right atrial myocardium between the coronary-sinus ostium and the tricuspid annulus from the underlying ventricular septum.

The sites of successful ablation of slow-pathway conduction were distributed over a wide area in the posteroseptal region. In addition, conduction over the slow pathway was affected by energy delivered at two to four separate sites in some patients, suggesting the presence either of multiple atrial insertions of the slow pathway or of multiple slow pathways.

It was not necessary to eliminate all slow-pathway conduction to eliminate AVNRT. In 51 patients, programmed atrial stimulation at the end of the procedure still induced single atrioventricular nodal reentrant atrial echo complexes, indicating that some slow-pathway conduction persisted. The absence of AVNRT in all patients over a mean follow-up period of 15.5 months and the inability to induce tachycardia in follow-up electrophysiologic studies performed in 40 percent of patients suggest that the procedure can be terminated successfully when the residual slow pathway cannot sustain 1:1 conduction during atrial or ventricular pacing and when only single echo complexes can be induced despite the administration of isoproterenol.

Complications occurred in only five patients (6.3 percent). One complication, atrioventricular block, resulted directly from the application of radiofrequency current. The other four complications, pulmonary embolus in one patient and thrombi in the right atrial appendage, the superior vena cava, and the inferior vena cava detected by transesophageal echocardiography in three asymptomatic patients,

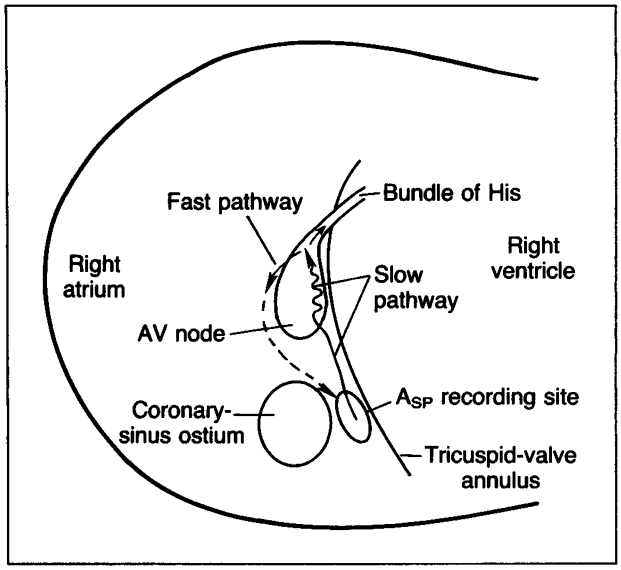

Figure 5. Schematic Representation of a Proposed Model of the Reentrant Circuit Used in the Common (Slow–Fast) Form of AVNRT.

The slow pathway is shown adjacent to the tricuspid annulus. Activation of its proximal portion in the posterior septum generates the A_{SP} potential. Its distal portion, near the bundle of His, activates the distal portion of the fast pathway. The fast pathway activates the anterior atrial septum, close to the bundle of His. The impulse then propagates posteriorly through the atrial septum and activates the proximal portion of the slow pathway, completing the reentrant circuit. The course of the hypothetical atrial portion of the reentrant circuit (dashed line) is unknown. AV denotes atrioventricular.

were probably related to the insertion and manipulation of the catheters. We currently treat all patients with aspirin for six weeks after ablation, but further study may indicate a role for short-term warfarin therapy.

The accepted concept regarding the mechanism of AVNRT has been that the reentrant circuit is confined entirely to the atrioventricular node. This hypothesis was supported by reports of AVNRT continuing undisturbed despite the dissociation of portions of right and left atrial myocardium close to the atrioventricular node as a result of atrial pacing, ventriculoatrial block, or atrial fibrillation.[15-17] This model does not easily explain the observation by Sung et al.[11] that the site of the earliest retrograde atrial activation differs in the case of retrograde conduction over the fast and slow pathways. The concept of a completely intranodal reentrant circuit was also challenged by the observations that surgical procedures have cured AVNRT by creating perinodal lesions to separate some of the atrial connections from the node.[4,5,18,19] Our study provides strong evidence that the atrial insertions of the fast and slow pathways are anatomically distinct: the atrial insertion of the fast pathway is located anteriorly in the septum (close to the bundle of His), whereas the atrial insertion of the slow pathway is located posteriorly in the septum, often as much as 3 cm from the bundle of His. These observations should lead to a reconsideration of the possibility that a portion of atrial myocardium between the atrial insertions of the two pathways forms part of the reentrant circuit in AVNRT. One proposed model of such a circuit in slow–fast AVNRT is shown in Figure 5.

Selective catheter ablation of the atrial insertion of the slow pathway with radiofrequency current safely and reliably eliminates the common and uncommon forms of AVNRT without affecting normal atrioventricular nodal conduction. This procedure may prove to be the preferred initial therapy in patients with AVNRT.

References

1. 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.
2. Pritchett ELC, Anderson RW, Benditt DG, et al. Reentry within the atrioventricular node: surgical cure with preservation of atrioventricular conduction. Circulation 1979;60:440-6.
3. Marquez-Montes J, Rufilanchas JJ, Esteve JJ, et al. Paroxysmal nodal reentrant tachycardia: surgical cure with preservation of atrioventricular conduction. Chest 1983;83:690-4.
4. Ross DL, Johnson DC, Denniss AR, Cooper MJ, Richards DA, Uther JB. Curative surgery for atrioventricular junctional ("AV nodal") reentrant tachycardia. J Am Coll Cardiol 1985;6:1383-92.
5. Cox JL, Holman WL, Cain ME. Cryosurgical treatment of atrioventricular node reentrant tachycardia. Circulation 1987;76:1329-36.
6. Kunze K-P, Geiger M, Schlüter M, Kuck K-H. Katheter-induzierte Modulation der elektrischen Leitfähigkeit des Atrioventrikularknotens mittels Hochfrequenzstroms. Dtsch Med Wochenschr 1988;113:1343-8.
7. Epstein LM, Scheinman MM, Langberg JJ, Chilson D, Goldberg HR, Griffin JC. Percutaneous catheter modification of the atrioventricular node: a potential cure for atrioventricular nodal reentrant tachycardia. Circulation 1989;80:757-68.
8. Haissaguerre M, Warin JF, Lematayer 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.
9. 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.
10. Calkins H, Sousa J, El-Atassi R, 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.
11. Sung RJ, Waxman HL, Saksena S, Juma Z. Sequence of retrograde atrial activation in patients with dual atrioventricular nodal pathways. Circulation 1981;64:1059-67.
12. 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.
13. Sung RJ, Huycke EC, Keung EC, Tseng C-D, Lai WT. Atrioventricular node reentry: evidence of reentry and functional properties of fast and slow pathways. In: Zipes D, Jalife J. Cardiac electrophysiology: from cell to bedside. Philadelphia: W.B. Saunders, 1990:513-25.
14. Jackman WM, Wang XZ, Friday KJ, et al. Catheter ablation of atrioventricular junction using radiofrequency current in 17 patients: comparison of standard and large-tip catheter electrodes. Circulation 1991;83:1562-76.
15. Josephson ME, Kastor JA. Paroxysmal supraventricular tachycardia: is the atrium a necessary link? Circulation 1976;54:430-5.
16. Bauernfeind RA, Wu D, Denes PO, Rosen KM. Retrograde block during dual pathway atrioventricular nodal reentrant paroxysmal tachycardia. Am J Cardiol 1978;42:499-505.
17. Portillo B, Mejias J, Leon-Portillo N, Zaman L, Myerburg RJ, Castellanos A. Entrainment of atrioventricular nodal reentrant tachycardias during overdrive pacing from high right atrium and coronary sinus: with special reference to atrioventricular dissociation and 2:1 retrograde block during tachycardias. Am J Cardiol 1984;53:1570-6.
18. Guiraudon GM, Klein GJ, Sharma AD, Yee R, Kaushik RR, Fujimura O. Skeletonization of the atrioventricular node for AV node reentrant tachycardia: experience with 32 patients. Ann Thorac Surg 1990;49:565-73.
19. Johnson DC, Nunn GR, Richards DA, Uther JB, Ross DL. Surgical therapy for supraventricular tachycardia, a potentially curable disorder. J Thorac Cardiovasc Surg 1987;93:913-8.