Selective Transcatheter Ablation of the Fast and Slow Pathways Using Radiofrequency Energy in Patients With Atrioventricular Nodal Reentrant Tachycardia

Mohammad R. Jazayeri, MD; Sandy L. Hempe, RN; Jasbir S. Sra, MD; Anwer A. Dhala, MD; Zalman Blanck, MD; Sanjay S. Deshpande, MD; Boaz Avitall, MD, PhD; David P. Krum, MS; Carol J. Gilbert, RN; and Masood Akhtar, MD

Background. The safety and efficacy of selective fast versus slow pathway ablation using radiofrequency energy and a transcatheter technique in patients with atrioventricular nodal reentrant tachycardia (AVNRT) were evaluated.

Methods and Results. Forty-nine consecutive patients with symptomatic AVNRT were included. There were 37 women and 12 men (mean age, 43±20 years). The first 16 patients underwent a fast pathway ablation with radiofrequency current applied in the anterior/superior aspect of the tricuspid annulus. The remaining 33 patients initially had their slow pathway targeted at the posterior/inferior aspect of the right interatrial septum. The fast pathway was successfully ablated in the initial 16 patients and in three additional patients after an unsuccessful slow pathway ablation. A mean of 10±8 radiofrequency pulses were delivered; the last (successful) pulse was at a power of 24±7 W for a duration of 22±15 seconds. Four of these 19 patients developed complete atrioventricular (AV) block. In the remaining 15 patients, the post-ablation atrio-His intervals prolonged from 89±30 to 138±43 msec (p<0.001), whereas the shortest 1:1 AV conduction and effective refractory period of the AV node remained unchanged. Ten patients lost their ventriculoatrial (VA) conduction, and the other five had a significant prolongation of the shortest cycle length of 1:1 VA conduction (280±35 versus 468±30 msec, p<0.0001). Slow pathway ablation was attempted initially in 33 patients and in another two who developed uncommon AVNRT after successful fast pathway ablation. Of these 35 patients, 32 had no AVNRT inducible after 6±4 radiofrequency pulses with the last (successful) pulse given at a power of 36±12 W for a duration of 35±15 seconds. After successful slow pathway ablation, the shortest cycle length of 1:1 AV conduction prolonged from 295±44 to 332±66 msec (p<0.0005), the AV nodal effective refractory period increased from 232±36 to 281±61 msec (p<0.0001), and the atrio-His interval as well as the shortest cycle length of 1:1 VA conduction remained unchanged. No patients developed AV block. Among the last 33 patients who underwent a slow pathway ablation as the initial attempt and a fast pathway ablation only when the former failed, 32 (97%) had successful AVNRT abolition with intact AV conduction. During a mean follow-up of 6.5±3.0 months, none of the 49 patients had recurrent tachycardia. Forty patients had repeat electrophysiological studies 4–8 weeks after their successful ablation, and AVNRT could not be induced in 39 patients.

Conclusions. These data suggest that both fast and slow pathways can be selectively ablated for control of AVNRT. Slow pathway ablation, however, by obviating the risk of AV block, appears to be safer and should be considered as the first approach. (Circulation 1992;85:1318–1328)

Key Words • ablation • atrioventricular node • tachycardia • radiofrequency

Atrioventricular nodal reentrant tachycardia (AVNRT) is one of the most common causes of paroxysmal supraventricular tachycardia. For many years, a pharmacological approach was the only therapeutic modality available for managing AVNRT. Because antiarrhythmic therapy has several limitations, including a long-term commitment to consuming medications, inefficacy, and intolerance, nonpharmacological approaches have become increasingly important therapeutic alternatives.

With the introduction of the surgical modification of the atrioventricular (AV) node, the possibility of a permanent cure in these patients was raised. There are now different surgical ablative techniques that can effectively modify the reentrant circuit and eradicate AVNRT yet preserve the antegrade AV conduction. These procedures require thoracotomy and cardiopulmonary bypass, however, and they are associated with morbidity as well as the risk of serious complications.

See p 1619
During the past decade, catheter ablation techniques have progressed dramatically and have become an alternative modality in providing a permanent cure. A high-energy electrical shock was initially used to ablate the His bundle and to achieve complete AV block in patients with various types of supraventricular tachyarrhythmias.\(^5\)\(^-\)\(^7\) Subsequently, modifications of this technique showed that it could selectively abolish the AV nodal fast pathway and eradicate the AVNRT circuit with a reasonably high success rate.\(^8\)\(^9\) More recently, radiofrequency current, by providing a safe and effective source of energy for the transcatheter ablative technique, has superseded high-energy electric shock.\(^10\)

Initial reports have dealt primarily with ablation of the AV nodal fast pathway.\(^11\)\(^12\) The preliminary report of successful transcatheter ablation of the slow pathway has offered a new approach for abolishing AVNRT.\(^13\)

The purposes of this report are 1) to describe our technique for ablating the slow pathway as a practical approach in eradicating AVNRT and 2) to compare the safety and efficacy of fast versus slow pathway ablation in our series.

**Methods**

**Patient Population**

Between June 1990 and April 1991, 49 consecutive patients (37 women and 12 men ranging in age from 9 to 82 years; mean, 43±20 years) underwent catheter modification of the AV node for AVNRT. All patients had recurrent paroxysmal palpitations with a mean duration of 10±9 years. Structural heart disease was documented in only four patients. Patients with coexistent AV accessory pathways were excluded from this study.

**Baseline Electrophysiology Study**

Before catheter modification, each patient underwent a complete electrophysiological evaluation, which was performed in a postabsorptive state after informed consent was obtained. The investigational protocol was approved by the Investigational Review Board of Sinai Samaritan Medical Center. All antiarrhythmic medications had been discontinued for at least five half-lives before the study. By individual preference, patients were either sedated with intravenous midazolam hydrochloride or anesthetized with intravenous propofol. Three multipolar electrode catheters were introduced percutaneously via femoral veins and positioned under fluoroscopic guidance in the high right atrium, His bundle region, and right ventricular apex. A fourth 7F decapolar electrode catheter, with a center-to-center interelectrode spacing of 5.0 mm (Bard Electrophysiology, Billerica, Mass.), was inserted via a jugular vein and positioned in the coronary sinus. Attempts were made to position the most proximal two pairs of electrodes in close proximity to the coronary sinus ostium. Surface ECG leads (I, II, and V\(_1\)), intracardiac electrograms, and time lines were displayed simultaneously on a multichannel oscilloscope (EVR-180, PPG Biomedical, Pleasantville, N.Y.) and printed on a thermal recorder. Data were recorded on optical disks by the EP Lab System (Biomedical Instrumentation Inc., Markham, Ontario, Canada) for subsequent reproduction. Electrical stimulation was performed with a programmable digital stimulator (Bloom Associates, Reading, Pa.).

The stimulation protocol consisted of atrial and ventricular incremental pacing and extrastimulation. The induction of AVNRT was attempted repeatedly to determine the most reliable and reproducible method(s) of the tachycardia initiation. In cases where AVNRT could not be initiated or sustained at baseline, isoproterenol was infused and titrated to achieve a minimum of 20% increase in the heart rate. After this, the stimulation protocol was repeated. In patients requiring isoproterenol administration, all electrophysiological parameters (before and after ablation) were measured during isoproterenol infusion. Intravenous heparin was administered as an initial dose of 2,000 units at the onset of the procedure, and subsequent boluses of 1,000 units/hr were given throughout the procedure.

**Ablative Procedure**

Radiofrequency current was delivered between the distal electrode of the ablating catheter and an external adhesive patch electrode (Scotchplate 1149C, 3M Co.) placed on the chest wall. Two radiofrequency generators were used for the ablations. In the first 24 patients, a conventional electrosurgical generator (Valleylab Force 2, Boulder, Colo.) was the source of radiofrequency energy. The unmodulated bipolar output of this unit, which produced a radiofrequency energy at 750 kHz, was used. This generator was coupled to a controller unit (Mansfield Scientific, Mansfield, Mass.) that provided outputs for monitoring load impedance, voltage, current, and power. A LIZ-88 (American Cardiac Ablation Corporation, Foxboro, Mass.) radiofrequency ablation unit was used for the subsequent procedures. This unit generates a pure sine wave output at 540 kHz and integrates the power source and energy output monitoring circuitry into a single package. The LIZ-88 gives direct readouts of the voltage and load impedance. In addition, the power was monitored with a digital voltmeter attached to the output provided on the unit. The ablation catheter was a 7F deflectable quadrupolar catheter with a 4-mm bulbous tip electrode\(^14\) (Mansfield Scientific, Mansfield, Mass.). In the first 16 patients, the anterior/superior aspect of the tricuspid septal annulus was targeted for a fast pathway ablation. In the remaining patients (patients 17–49), the posterior/inferior aspect of the tricuspid septal annulus was targeted first for selective slow pathway ablation. Because the ablation technique varies in accordance with the target pathway, the description of ablative procedure for each pathway will be outlined separately.

**Anterior/Superior Ablation**

See Figures 1 and 2. The catheter was positioned at the His bundle region to obtain a bipolar recording of the His bundle deflection. This catheter was then withdrawn until it recorded an atrial (A) deflection that was at least as large as the ventricular (V) deflection (A/V≥1) along with the smallest (<0.1 mV) or no His bundle potential. The site was considered optimal for ablation when 1) the retrograde atrial activation via the fast pathway occurred earlier than or simultaneously with that in the His bundle electrogram and 2) pacing of the atria at 5.0 mA via the distal pair of electrodes demonstrated adequate contact between the catheter tip and the atrial wall. During applications of radiofre-
Figure 1. Schematic representation of anatomic ablation sites. This figure shows right anterior oblique view of tricuspid annulus. The atrioventricular junction of the septal cusp, extending from the coronary sinus ostium to the most proximal His bundle recording site, is arbitrarily divided into three sites: posterior (P), medial (M), and anterior (A). These sites were attempted for the ablation of the slow pathway. For ablation of the fast pathway, the usual site was located anteriorly and superiorly (S) adjacent to the His bundle recording site.

Frequency energy, the surface ECG was continuously monitored for the PR interval prolongation (>30%) and/or occurrence of AV block. Whenever accelerated junctional rhythm was noted, atrial pacing at a faster rate was attempted to ensure the integrity of the AV conduction during application of radiofrequency energy. Initially, the radiofrequency power was set at 15–20 W and was applied for 10–15 seconds. If this attempt did not abolish the retrograde fast pathway, the power and duration were gradually titrated (by 5–10 W and 5–10 seconds, respectively) up to 35 W and 60 seconds, respectively, before the catheter was repositioned at a new site. During each ablation attempt, the application of radiofrequency current was immediately terminated if complete AV block, junctional tachycardia, or impedance rise was noted. After delivery of each radiofrequency pulse, ventricular pacing was performed to evaluate the retrograde conduction. The end point of the procedure was to achieve a complete retrograde block over the fast pathway during ventricular pacing and noninducibility of the AVNRT.

Posterior/Inferior Ablation

We assumed that in each patient, antegrade and retrograde slow pathways were either the same or anatomically in close proximity to each other. Therefore, attempts were made to disclose the retrograde conduction over the slow pathway for mapping and ablating purposes. These attempts included incremental ventricular pacing (before and after isoproterenol infusion) and premature ventricular stimulation at basic drive cycle lengths of 400 and 350 msec to block the retrograde impulses in the fast pathway and unmask the retrograde conduction over the slow pathway. In the majority of patients, however, this could not be accomplished; thus, a slow pathway ablation was attempted in a stepwise fashion (Figures 1 and 2) as outlined below.

As shown in Figure 1, in the right anterior oblique (35–45°) radiographic view, the septal annulus of the tricuspid valve, extending from the most posterior region of the annulus adjacent to the coronary sinus ostium (posterior) to the His bundle recording site (anterior), was divided into posterior (P), medial (M), and anterior (A) sites. In each case, the following consecutive steps were taken to position the ablation catheter. 1) The catheter was placed at the His bundle region to record either the most distal His bundle or right bundle potential. 2) The deflectable tip was fully bent. 3) The catheter was slowly withdrawn along the tricuspid septal annulus down to the most posterior/inferior aspect of the interatrial septum adjacent to the coronary sinus ostium (site P). 4) This site was considered optimal if the bipolar recording obtained from the distal electrodes showed an A/V electrogram ratio of 0.1 to 0.5. If the ablation attempt at this site was unsuccessful, then, while an optimal A/V ratio was maintained, the catheter tip was moved anteriorly (with or without slight advancement of the catheter itself) to reach sites M and then A if necessary. In each site, at least two pulses of radiofrequency energy (30–35 W for 20–60 seconds) were delivered at two separate positions (in close proximity) before the catheter was placed in a new site. After delivery of each radiofrequency pulse and before any further attempt, pacing protocols were repeated to reassess AVNRT inducibility and the AV nodal conduction properties. The end point of the procedure was determined by noninducibility of AVNRT.

Follow-up

Atrial and ventricular pacing protocols were repeated at 30 minutes and 24–48 hours after ablation. Complete electrophysiological studies were performed 4–8 weeks later in patients who had initial successful results. Isoproterenol infusion was routinely used when atrial and ventricular pacing protocols alone could not elicit AVNRT. Patients underwent two-dimensional echocardiography/Doppler studies 1–2 days and 4–8 weeks after the procedure to evaluate specifically the integrity of the tricuspid valve and to search for mural thrombi at the site(s) of radiofrequency energy delivery.

Definition of Terms

All intervals were measured between the onsets of two successive local electrograms.

\[ A_v - A_w \text{ interval. This interval was measured during AVNRT and ventricular pacing from the onset of local atrial deflection in the His bundle electrograms to the onset of local atrial deflection in the proximal coronary sinus adjacent to the coronary sinus ostium.} \]

\[ AV \text{ nodal reentrant tachycardia. The presence of AV nodal dual-pathway physiology and a critical atrio-His (AH) interval at tachycardia initiation are frequently present in patients with AVNRT. These findings, however, cannot completely exclude the possibility of other reentrant supraventricular tachycardias. Therefore, the following criteria were used to verify AVNRT (common variety) as the underlying mechanism of reentry in these patients. 1) The occurrence of atrial activation simultaneously with or before the ventricular activation. 2) An} \]
inability to preexcite the atria by a premature ventricular impulse introduced when the His bundle was refractory from the antegrade impulse. These two criteria argue against the participation of an accessory pathway as the retrograde limb of the reentry. 3) Identical sequence of retrograde atrial activation during tachycardia and ventricular pacing. 4) Constant His-atrial interval of the return cycle after introduction of a premature atrial impulse with a wide range of coupling intervals during tachycardia. The latter two criteria argue against atrial tachycardia.

**Statistical Analysis**

All data are expressed as mean±SD. Statistical comparisons were performed by Student’s t test for paired and unpaired values. In patients who underwent a fast pathway ablation, the association of $A_r-A_s$ interval and the outcome was assessed by Fisher’s exact test. A probability value of $p<0.05$ was considered significant.

**Results**

Sustained AVNRT of the common variety (mean cycle length of 328±53 msec) was induced in 47 patients (Tables 1 and 2) either during the baseline study (31 patients) or after isoproterenol infusion (16 patients). Sustained uncommon AVNRT was induced in two patients who also had nonsustained common AVNRT during the baseline study.

**Fast Pathway Ablation**

Fast pathway ablation was attempted in the first 16 consecutive patients and in three additional patients (patients 17–19) who had unsuccessful attempts at a slow pathway ablation. The mean procedure time, including the initial electrophysiological evaluation followed immediately by ablation, was 165±61 minutes. These 19 patients received 10±9 radiofrequency pulses, the last of which (successful pulse) was given at a power of 24±7 W for a mean duration of 22±15 seconds. Eighteen of 19 (95%) became noninducible for AVNRT of the common variety. In the remaining patient (patient 18), who had previously undergone an unsuccessful slow pathway ablation, AVNRT remained inducible despite dramatic prolongation of the cycle lengths of 1:1 AV and ventriculoatrial (VA) conduction after the fast
pathway ablation. Twenty hours after the procedure, this patient developed Wenckebach periodicity, which gradually progressed to a higher-degree AV block and finally complete AV block. This patient, in addition to the other three patients who developed immediate AV block during the procedure, brought the total number of patients with complete AV block to 4 of 19 (21%). These patients were all women with a mean age of 63 years. Dual-chamber pacemakers were implanted in three of these patients. The fourth patient had previously had a permanent pacemaker implanted in another institution for her sick sinus syndrome.

In 15 patients with successful fast pathway ablation and intact AV conduction (Figure 3), the AH interval prolonged from $89 \pm 30$ to $136 \pm 43$ msec ($p < 0.0001$), and the shortest cycle length of 1:1 AV conduction and the effective refractory period of the AV node remained unchanged. Ten patients did not have VA conduction (at ventricular pacing cycle lengths of 50–100 msec shorter than the sinus cycle lengths) after ablation, and the remaining five patients demonstrated a significant prolongation of the shortest cycle length of 1:1 VA conduction ($280 \pm 35$ versus $468 \pm 30$ msec, $p < 0.0001$).

Of these five patients with residual VA conduction, three developed AVNRT of uncommon variety (Figure 4), which was sustained in two patients (patients 8 and 16) without isoproterenol infusion. These patients did not have uncommon AVNRT inducible before the ablation. The earliest retrograde atrial activation during tachycardia was adjacent to the coronary sinus ostium. Further attempts at ablation of the antegrade limb of uncommon AVNRT at the anterior aspect of the tricuspid annulus were unsuccessful in these two patients.

One patient underwent the slow pathway ablation immediately after successful ablation of the retrograde fast pathway, and the other patient required a slow pathway ablation 4 weeks after successful fast pathway ablation and after recurrent episodes of uncommon AVNRT. The remaining two patients with residual VA conduction did not have any inducible AVNRT (common or uncommon). Although the earliest retrograde atrial activation was close to the His bundle recording site, the electrophysiological properties (i.e., VA conduction time and shortest cycle length of 1:1 VA conduction) of these residual retrograde pathways were entirely different from those of the “fast” pathways targeted for ablation.

To identify predictors of complete AV block during fast pathway ablation, we examined a number of electrophysiological parameters (i.e., AH interval during sinus rhythm, His-atrial interval during common AVNRT, A/V ratio at ablation site, amplitude of His bundle potential, and $A_N-A_A$ interval) in patients who had and in those who did not have this complication. The only identifiable parameter was the $A_N-A_A$ interval (Table 1), which was significantly shorter in patients who developed AV block (Figure 5) than in those who did not ($6 \pm 5$ versus $26 \pm 6$ msec, respectively, $p < 0.0001$). An $A_N-A_A$ interval $\leq 15$ msec was a significant predictor of complete AV block ($p < 0.001$) in this series with 100% specificity and 100% sensitivity. The mean power and duration of the last (successful) radiofrequency pulse in patients with and without complete AV block were $22 \pm 7$ versus $24 \pm 7$ W ($p = NS$), respectively, and $19 \pm 13$ versus $23 \pm 15$ seconds ($p = NS$), respectively.

### Table 1. Patients Undergoing Fast Pathway Ablation

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<th>1:1 VA ERP (msec)</th>
<th>AVNRT ERP (msec)</th>
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AH, atrio-His; AV, atrioventricular; VA, ventriculoatrial; AVN, atrioventricular node; ERP, effective refractory period; AVNRT, atrioventricular nodal reentrant tachycardia; CL, cycle length; ABLA, ablation; S, superior; AVB, atrioventricular block.

*Patients who had undergone an unsuccessful slow pathway ablation earlier.

†Electrophysiological data immediately after ablation attempts.
### Table 2. Patients Undergoing Slow Pathway Ablation

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AH, atrio-His; AV, atrioventricular; VA, ventriculoatrial; AVN, atrioventricular node; ERP, effective refractory period; AVNRT, atrioventricular nodal reentrant tachycardia; CL, cycle length; ABLA, ablation; AVB, atrioventricular block.

*Patients who had undergone a successful fast pathway ablation earlier.

†Sustained AVNRT of uncommon variety.

‡Electrophysiological data immediately following ablation attempts.

### Slow Pathway Ablation

Thirty-five patients underwent a slow pathway ablation, including the two patients (patients 8 and 16) in whom uncommon AVNRT was unmasked after successful fast pathway ablation. The mean procedure time, including the initial electrophysiological assessment followed immediately by ablation attempts, was 151 ± 50 minutes. A mean of 6 ± 4 radiofrequency pulses was delivered. The last pulse, which led to a successful ablation, was delivered at a mean power of 36 ± 12 W for 35 ± 15 seconds. The mean A/V electrogram ratio at the effective sites was 0.27 ± 0.10. No patients developed AV block during or after the slow pathway ablation. In eight patients (Table 2), the retrograde conduction over the slow pathway could be identified during sustained (four patients) and nonsustained (four patients) uncommon AVNRT. After precise mapping along the atrial side of the tricuspid annulus and localization of the earliest retrograde atrial activation (34 ± 10 msec earlier than atrial activation in the His bundle recording), a single radiofrequency pulse successfully abolished the slow pathway in both antegrade and retrograde directions in all eight patients. The remaining 27 patients underwent the “stepwise” approach. Before the radiofrequency application, attempts were made in 15 consecutive patients to identify any potentials (i.e., discrete, high-frequency spikes) between the atrial and ventricular deflections that could be associated with the antegrade propagation of impulses over the slow pathway. This was done to detect the optimal site for ablation, specifically for patients without any discernible retrograde conduction over the slow pathway. Nevertheless, no such reliable markers could be identified in any of these 15 patients (Figure 6). An extra potential, unrelated to
the His bundle activation, was observed in one patient and initially appeared to be related to a slowly conducting pathway with decremental conduction properties. Further electrophysiological analysis, however, could dissociate that pathway from the AVNRT circuit. Furthermore, ablation of the pathway accountable for the extra potential did not alter AVNRT inducibility or AV nodal conduction characteristics (Figure 7).

Of 35 patients, 32 (91%) had no AVNRT inducible after the slow pathway ablation. The other three patients with persistent AVNRT subsequently underwent a fast pathway ablation, as mentioned earlier. Ablation of the slow pathway was accomplished within the posterior site (Figure 1) in 16 patients, the medial site in 15 patients, and the anterior site in one patient. In patients with a slow pathway ablation achievable at medial or anterior sites, several radiofrequency pulses delivered at sites located more posterior than the effective site did not alter the AV nodal conduction properties at all. After successful slow pathway ablation, the shortest cycle length of 1:1 VA conduction remained unchanged. Of the two patients (patients 8 and 16) undergoing successful fast and slow pathway ablations, only one (patient 16) continued to have residual VA conduction. Eleven patients continued to have single AV nodal reentrant echo beats (of common variety) without sustained AVNRT.

Follow-up

Of 49 patients, two patients who underwent fast pathway ablations (including one who developed uncommon AVNRT after successful fast pathway ablation) and one patient who underwent a slow pathway ablation required repeat ablative procedures within 1–14 days after their initial unsuccessful attempts. During a mean period of 6.5±3.0 months, no patients developed symptoms suggestive of AVNRT recurrence or complete AV block (except those mentioned earlier who developed complete AV block within 36 hours after the procedure). Forty patients underwent 4–8-week follow-up electrophysiological evaluations and were found to have no inducible AVNRT, except for one patient in whom AVNRT was induced only after isoproterenol infusion and rapid ventricular pacing. This patient, however, has remained free of any spontaneous recurrence during a 6-month follow-up period. Echo-cardiographic studies performed immediately (49 patients) and 4–8 weeks (40 patients) after the procedure did not show any anatomic or functional abnormalities related to the site of ablation.

Discussion

In this study, 45 of 49 patients (92%) underwent a successful modification of the AV node without interrupting AV conduction. Of the 19 patients undergoing ablation of the fast pathway, the abolition of spontaneous or inducible AVNRT was achieved in all. Four patients (21%), however, developed complete AV block. The higher incidence of this complication in our series compared with 8% reported recently12 may be related to the differences in patient populations, operators’ technical skills, degree of aggressiveness in pursuing successful results, location of ablation sites, total energy delivered at the site (i.e., power, duration, and the number of pulses), size of the ablation catheter tips (4 versus 2–3 mm), or other factors. The occurrence of AV block remains the major concern and drawback during fast pathway ablation. Therefore, it seems critical to identify patients who may potentially be at risk for AV block when their fast pathways are targeted for the ablation. Thus far, no clinical or electrophysiological parameters have been reported to distinguish patients with and without a susceptibility to this complication. During the course of this study, we noticed that patients who developed AV block had a significantly shorter A_r-A_s interval than those who maintained the AV conduction integrity.

The exact reason for the high incidence of AV block in this series among patients with a short A_r-A_s interval (i.e., ≤15 msec) is unclear. It seems conceivable, however, that in this subgroup of patients, the fast pathway may be situated toward the posterior portion of the interatrial septum rather than in the usual anterior location. Consequently, ablation lesions at an anterior

![Figure 3. Tracings of successful fast pathway ablation (patient 11). Tracings from top to bottom are ECG leads (I, II, and V1), ostial coronary sinus (CSp, panels A, B, and C) or high right atrial (HRA, panel D), proximal and distal His bundle electrogams (HBp and HBd, respectively), and time lines (T). A similar format is used in subsequent tracings. Panel A depicts a sinus beat with the atrio-His (AH) interval of 70 msec before ablation. During premature atrial stimulation at 400 msec and a premature coupling interval of 270 msec, sustained atrioventricular nodal reentrant tachycardia (AVNRT) of common variety is induced (panel B). Note that the A_r_A_s interval during AVNRT is 35 msec. Panel C shows a sinus beat after successful fast pathway ablation. The AH interval (90 msec) is prolonged by 20 msec. During ventricular pacing at 500 msec, there is no retrograde conduction (ventriculoatrial dissociation, panel D).](http://circ.ahajournals.org/doi/abs/10.1161/01.CIR.88.4.281?journalCode=cir)
site might be more likely to damage the AV node or its lower common pathway. Further studies are needed to verify this observation.

Of 15 patients with successful fast pathway ablation and intact AV conduction, two (13%) exhibited sustained AVNRT of uncommon variety. Others have also reported the occurrence of uncommon AVNRT after successful abolition of the fast pathway. In both of our patients, the AH interval during sinus rhythm was markedly prolonged, indicating that the fast pathway was probably successfully ablated in the antegrade direction. Further radiofrequency applications at the anterior/superior aspect of the tricuspid annulus could not disrupt this antegrade pathway, suggesting that it had a different location or that it was situated deeper within the AV junctional structure. It seems reasonable, therefore, to infer that the antegrade limb of uncommon AVNRT in these two patients was not the same pathway as the retrograde limb of common AVNRT.

The following reasons encouraged us to attempt a slow pathway ablation: 1) the relatively high incidence of complete AV block in patients undergoing a fast pathway ablation, 2) successful mapping and ablation of the slow pathway in two patients who developed uncommon AVNRT after successful fast pathway ablation, and 3) the result of a preliminary report of successful slow pathway ablation that did not jeopardize the integrity of AV conduction. Of 35 patients undergoing a slow pathway ablation, 32 (91%) had successful results. The earliest atrial activation during retrograde conduction over the slow pathway could be precisely mapped and readily ablated in eight patients, including two in whom the fast pathway had been ablated earlier. Although the demonstration of the retrograde atrial breakthrough of the slow pathway is ideal for precise localization and optimal ablation, this cannot be done in the majority of patients for two main reasons. First, the conduction properties of the fast pathway in the retrograde direction (i.e., shortest cycle length of 1:1 conduction and effective refractory period) may conceal those of the slow pathway. Second, in many patients the slow pathway may not conduct retrogradely to the atrium. The most convincing piece of evidence supporting the latter issue is our observation that among the first 13 patients who had successful fast pathway ablation, only three patients (23%) had demonstrable retrograde conduction over the slow pathway.

In patients without any discernible retrograde conduction over the slow pathway (before and after isoproterenol infusion), no specific potentials could be identified for a precise localization and ablation of the slow pathway. Therefore, on the assumption that the slow pathway is located in the posterior/inferior aspect of the tricuspid annulus in close proximity to the coronary sinus ostium, a stepwise approach was used that led us to successfully abolish the slow pathway and eliminate AVNRT in 24 patients. Of 24 patients with successful ablation, the abolition of the slow pathway was achieved in the posterior and medial sites in 23 and in the anterior site in one. These data suggest that the slow pathway indeed has a distinctly different anatomic location from the fast pathway. In the remaining three patients, the slow pathway could not be ablated with this technique. The reasons for this are unclear; it is conceivable, however, that the slow pathway in these patients may have had anomalous locations: farther from the tricuspid annulus; in the left side of the septum, close to the mitral annulus; anteriorly and close to the
fast pathway; or deep within the AV junctional tissue, not readily accessible by radiofrequency pulses. Nevertheless, these three patients underwent a fast pathway ablation that resulted in complete abolition of AVNRT in two patients and complete AV block in one. Therefore, with the slow pathway ablation technique as the initial approach and the fast pathway ablation only if the former was unsuccessful, we could completely abolish AVNRT and yet maintain AV conduction integrity in 32 of our last 33 patients (97%).

Persistence of the AV nodal reentry after a slow pathway ablation is not uncommon, as 11 patients (34%) continued to have a single AV nodal reentrant echo complex. There are two possible explanations. First, the slow pathway may have been partially damaged by radiofrequency energy and made incapable of maintaining AVNRT. Second, the critical slow pathway necessary for sustained AVNRT may have been completely ablated and another slow pathway manifested that was incapable of maintaining AVNRT.

Although coronary sinus electrograms are not usually required to distinguish AVNRT of common variety from other supraventricular tachycardia, in our experience, placement of the coronary sinus catheter has been helpful 1) to identify and properly localize any residual

FIGURE 5. Tracings of atrioventricular block during fast pathway ablation (Patient 12). Panel A shows a sinus beat with the atrio-His (AH) interval of 80 msec (not labeled). Premature ventricular stimulation at a basic drive cycle length of 600 msec and a coupling interval of 280 msec is shown in panel B. Note that the retrograde conduction of V₂ occurs via the fast pathway with an H₂A₂ interval of 50 msec and an A₃H₂ interval of 0. A similar A₃A₃, interval is depicted during atrioventricular nodal reentrant tachycardia (panel C). The ablation catheter (AC) is positioned at the earliest retrograde atrial activation (panel D). The atrial activation at this site is 5 msec earlier than that in the His bundle electrogram. However, a single radiofrequency pulse at 25 W for 12 seconds resulted in complete atrioventricular block (panel E). HRA, high right atrium; CSₚ, ostial coronary sinus; HB, His bundle; T, time lines.

FIGURE 6. Mapping of the posterior/inferior aspect of the tricuspid annulus for a slow pathway ablation (patient 22). During sinus rhythm, the tricuspid septal annulus was mapped by use of the ablation catheter (AC). The local electrogram (ACd) obtained from the most posterior site of the annulus (site P₁) is shown in Panel A. The catheter tip was gradually positioned toward the anterior aspect of the annulus at the sites P₂ (panel B), M₁ (panel C), M₂ (panel D), and A₁ (panel E). The site A₂ (not shown) was actually the His bundle recording site. Subsequently, radiofrequency energy was applied at site P₁ and, if unsuccessful, it was reapplied at sites situated more anteriorly by a "stepwise approach." Radiofrequency energy delivered at the site M₂ completely abolished the slow pathway and the atrioventricular nodal reentrant tachycardia (AVNRT). Note that there are no discernible potentials or atrial electrogram characteristics during sinus rhythm or AVNRT (panel F) that could unequivocally distinguish the effective site (M₂) from other sites. CSₚ, ostial coronary sinus; HB, His bundle; T, time line.
FIGURE 7. Tracings of extra potential unrelated to the His bundle activation (patient 30). During ventricular pacing at 330 msec (panel A), the retrograde atrial activation (A) is via the fast pathway. An extra potential (E) follows each atrial deflection in the proximal coronary sinus electrogram (CSp). Ventricular pacing at a shorter cycle length (320 msec, panel B) results in a 2:1 appearance of E deflections despite a 1:1 retrograde conduction over the fast pathway. During sustained atrioventricular nodal reentrant tachycardia (AVNRT) (common variety) at a cycle length of 315 msec, the 1:1 relation between atrial deflections and E potentials is preserved (panel C). With ventricular pacing at 270 msec, a typical Wenckebach periodicity in the retrograde AV nodal pathway(s) is demonstrated (panel D). Note that the pattern of E activation follows that of atrial activation (i.e., A), and when the retrograde block occurs in the AV nodal pathway, no E deflection is seen. This suggests that the pathway accountable for these E potentials is activated antegrade after each ventricular impulse being conducted via the retrograde AV nodal pathway. On the basis of these phenomena shown in Panels A–D, one could assume that these E potentials are originated from antegrade activation of the AV nodal slow pathway. However, panel E shows a 2:1 relation between the atrial and E deflections during another episode of AVNRT of common variety with a shorter cycle length (305 msec), which argues against that assumption. The ablation catheter (AC) is positioned at the posterior aspect of the tricuspid annulus, close to the CS ostium to record this potential (during sinus rhythm, panel F). Panel G shows that during ventricular pacing, the E potential recorded in the AC is 35 msec earlier than that recorded in CSp. A single application of the radiofrequency current resulted in abolition of this “extra pathway” with no further E potentials recordable during ventricular pacing (panel H). However, ablation of that pathway did not abolish AVNRT (panel I). Further attempts at ablation were required to finally abolish the slow pathway at a medial site (see Figure 1). Panel J shows prolongation of the AV nodal effective refractory period after ablation. HRA, high right atrium; HB, His bundle; T, time lines.
reentrant supraventricular tachycardia that may exist after successful AV nodal modification, 2) to assist proper recognition of the posterior/inferior aspect of the tricuspid annulus as a reliable marker for the slow pathway ablation, and 3) to determine the A2-A3 interval in those undergoing a fast pathway ablation (after unsuccessful attempts at a slow pathway ablation) and possibly to detect patients who might be at risk for complete AV block.

Limitations of Study

This study was not designed to determine specifically the minimal energy required to abolish AVNRT successfully. Although we titrated the power and duration of each radiofrequency application in patients undergoing a fast pathway ablation, the initial parameters used in patients undergoing a slow pathway ablation were selected arbitrarily. It is possible, therefore, that radiofrequency applications delivered at a lower power and/or for a shorter duration than those used during this study could also have produced successful results. Also, a higher success rate and better outcome for ablation of the slow pathway versus the fast pathway could be partially related to the level of experience of the operators rather than the approach per se.

Summary and Conclusions

First, selective ablation of the fast and slow pathways can be accomplished at the anterior/superior and posterior/inferior aspects of the tricuspid annulus, respectively. Second, the fast pathway ablation is associated with a potential risk of complete AV block, whereas the slow pathway is not accompanied by this risk. Because complete AV block can occur after completion of the procedure and within the first 48 hours after a fast pathway ablation (as occurred in patient 18), it seems advisable to monitor these patients in the hospital for 48–72 hours. In contrast, patients undergoing successful slow pathway ablation may be discharged from the hospital after a shorter monitoring period. Third, although a fast pathway ablation can successfully abolish common AVNRT, in some cases (13% in this series) it might unmask uncommon AVNRT, which may require a slow pathway ablation. Finally, AV nodal reentry with single AV nodal reentrant echo complexes may persist after successful slow pathway ablation, but this does not appear to have any clinical significance.

Although within a comparable length of time both ablative techniques could effectively modify the AV node and eradicate AVNRT, a slow pathway ablation, because it maintains the integrity of the AV conduction system, provides a more favorable outcome. Thus, for modifying the AV node in patients with AVNRT, it seems a reasonable strategy to target the slow pathway first and to contemplate a fast pathway ablation and the potential risk associated with this approach only if a slow pathway ablation cannot be accomplished.

Acknowledgments

We express our appreciation to Jane Wellenstein for her excellent secretarial assistance and Brian Miller for his invaluable help in preparation of the illustrations. We are also indebted to Randy Nest, Larry Hanson, Kitty Koch, Ann Dwyer, Patricia Strommen, and John Makai for their technical assistance.

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Circulation. 1992;85:1318-1328
doi: 10.1161/01.CIR.85.4.1318

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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