Radiofrequency Ablation of Slow Pathway in Patients With Atrioventricular Nodal Reentrant Tachycardia

Do Arrhythmia Recurrences Correlate With Persistent Slow Pathway Conduction or Site of Successful Ablation?

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**Background** Residual slow pathway conduction in the form of persistent jump in the atrioventricular (AV) conduction time or atrial echo beats is a common finding after successful radiofrequency (RF) ablation of the slow pathway in patients with AV nodal reentrant tachycardia (AVNRT). Sites of successful RF ablation of the slow pathway may be located anteriorly in the tricuspid annulus (cephalad to the coronary sinus os) or posteriorly (at, within, or caudal to the coronary sinus os). The aim of this study was to investigate whether arrhythmia recurrences correlate with persistent slow pathway conduction or site of successful ablation.

**Methods and Results** Among 55 patients with symptomatic AVNRT having RF ablation, 23 patients (42%) (group 1) had evidence of persistent dual AV nodal pathway physiology and/or echo beats, whereas in 32 patients (group 2), slow pathway conduction had been completely eliminated. With regard to ablation sites, 14 patients (25%) (group A) had their slow pathway successfully ablated at an inferoposterior site, whereas in 41 patients (group B), the ablation site was located anteriorly to the coronary sinus os. The study patients included 17 men and 38 women, aged 37±18 years. The electrophysiological study and RF ablation were performed in a single session in 50 patients (91%). After the first session, the technique was successful in all patients (100%), with elimination of AVNRT and without affecting AV conduction. A mean of 9±6 lesions were applied. The total procedure time averaged 4±1 hours. Fluoroscopy time was 41±25 minutes. Except for transient AV block in 1 patient, no other complications occurred. Over 12±8 months, a total of 7 patients (13%) had recurrence of AVNRT, and 6 of them underwent successful repeat slow pathway RF ablation. Recurrence rate was 9% (2 patients) for group 1, with persistent jump or echo beats, and 16% (5 patients) for group 2, without residual slow pathway conduction (P=NS). Five of the recurrences (71%) were noted in group A and 2 in group B. Thus, the recurrence rate was 36% for group A (5 of 14 patients), with posterior ablations, and 5% for group B (2 of 41 patients), with anterior sites of successful RF ablation (P<.05).

**Conclusions** After successful RF ablation of the slow pathway in patients with AVNRT, residual slow pathway conduction does not correlate with clinical tachycardia recurrences. However, the site of successful RF ablation of the slow pathway does correlate with arrhythmia recurrences. More recurrences are observed when the site is located inferoposteriorly, or at below the os of the coronary sinus, as compared with medial and anterior locations of the ablation site. (Circulation. 1994;90:2815-2819.)

**Key Words** • radiofrequency • reentry • tachycardia • atrioventricular node

M odification of the atrioventricular (AV) node without creating complete heart block can now be successfully accomplished with percutaneous techniques by use of radiofrequency (RF) energy in patients with AV nodal reentrant tachycardia (AVNRT), resulting in cure of the tachycardia by nonsurgical methods.1-11 Of the two RF ablation techniques available for selective fast or slow pathway ablation,1,2 slow pathway ablation has been the preferred method due to lower risk of AV block.11 Residual slow pathway conduction in the form of persistent jump in the AV conduction time or atrial echo beats is a common finding after successful RF ablation of the slow pathway.2,4,5,8-10 It remains a controversial issue whether this is related to any higher incidence of arrhythmia recurrences. On the other hand, the site of successful ablation of the slow pathway may be located medially or anteriorly in the tricuspid annulus (cephalad to the coronary sinus os) or inferoposteriorly (at, within, or caudal to the coronary sinus os).2,3,5,6 It is not known whether arrhythmia recurrences correlate with the site of ablation. Thus, the aim of the present study was to investigate whether clinical tachycardia recurrences correlate with persistent slow pathway conduction or site of successful ablation.

**Methods**

**Study Patients**

From June 1991 through November 1993, 55 consecutive patients with symptomatic AVNRT underwent RF ablation at our center. These were 17 male and 38 female patients, aged 37±18 years (range, 8 to 73 years), who presented with palpitations (n=33), presyncope (n=14), or syncope (n=8). The clinical arrhythmia was narrow QRS tachycardia (n=54) or wide QRS tachycardia (n=1). In 50 patients (91%), elec-
trophy physiological study and RF ablation were performed at a single session. There was no evidence of underlying structural heart disease in 50 patients, while 2 patients had coronary artery disease, 1 had dilated cardiomyopathy, and 2 had mitral valve prolapse. All patients had normal systolic left ventricular function (ejection fraction >50%) except for 1 patient with cardiomyopathy, who had a significantly reduced (25%) left ventricular ejection fraction.

Electrophysiological Study

The electrophysiological study was performed in the fasting state after all antiarrhythmic agents had been discontinued for at least 5 drug elimination half-lives. All patients gave written informed consent. Routinely, three 6F quadripolar electrode catheters were introduced from the femoral vein and positioned under fluoroscopy at the high right atrium, across the tricuspid valve for His bundle recording and at the right ventricular apex. Programmed stimulation was carried out with the use of a constant current stimulator (Medtronic 5328) at 2-millisecond pulse duration and output twice diastolic threshold. Standard recording methods, stimulation techniques, and definitions were used. The interval for the radiofrequency (RF) ablation was determined, and the mechanism of inducible tachycardia was defined. Anterograde and retrograde AV nodal function curves were constructed to assess dual AV nodal pathway physiology. If a discontinuous curve was not demonstrable at baseline or AVNRT could not be reproducibly induced, intravenous atropine or infusion of isoproterenol was administered. Most patients received heavy sedation with intravenous fentanyl and/or midazolam for the entire duration of the procedure.

Ablation Procedure

After the mechanism of tachycardia had been determined to be AVNRT during the initial part of the procedure, a 7F steerable, quadripolar, deflectable-tip catheter with a 4-mm distal electrode and 2-5-2-mm interelectrode spacing (Mansfield-Webster or EP Technologies) was used for ablation of the slow pathway with delivery of RF current. The RF current was generated at a frequency of 500 kHz by a commercially available electrosurgical unit (Radionics) and was delivered between the distal electrode and a cutaneous, indifferent, dispersive pad positioned on the posterior thorax.

The ablation site was identified by local bipolar electrograms showing a small atrial and a large ventricular deflection during sinus rhythm and fluoroscopically using the right anterior oblique projection to position the ablation catheter at the tricuspid annulus (Fig 1) and to maneuver it to posterior, medial, and anterior anatomic locations, as suggested by other groups. Slow pathway potentials were sought but not verified with pacing techniques. For the purpose of this study, the ablation site was characterized as posterior for positions at or inferior to the coronary sinus ostium (P1, P2, and posterior to P1 positions) and anterior for medial and anterior positions superior to the coronary sinus ostium (M1, M2, A1, and A2 positions).

Once the target site was identified, 20 to 35 W of RF energy was delivered by the distal tip of the ablation catheter for 30 seconds. If impedance rose during ablation, RF application was interrupted and the catheter was removed and cleaned before reinsertion. After each RF lesion, programmed stimulation was repeated to assess the presence of slow pathway conduction and the inducibility of AVNRT. The end point of ablation was the elimination of inducible AVNRT with and without isoproterenol infusion. Half an hour to 1 hour after ablation, programmed stimulation was performed with and without isoproterenol to confirm the efficacy of ablation and to construct AV nodal conduction curves. The patients were then monitored for 24 to 48 hours before discharge. During this period, serial ECGs were obtained, and an echocardiogram was performed to evaluate cardiac complications.

Follow-up

After discharge from the hospital, patients were followed up at our arrhythmia clinic or by their referring cardiologists every 3 to 6 months for the first year and annually thereafter.

Statistics

Data are presented as mean±SD. Comparisons were made using a two-tailed Student's t test for quantitative data or χ² statistic for qualitative data. A mean difference was considered statistically significant at a value of P<.05.

Results

In 50 patients (90%), the initial electrophysiological study and the RF ablation were performed at a single session. The induced AVNRT had a cycle length of 340±60 milliseconds. With the use of the anatomic approach, successful RF ablation was accomplished in all 55 patients (100%) after the initial session with elimination of AVNRT without affecting AV conduction, except transiently in 1 patient. This patient developed transient complete heart block that resolved over 2 hours without subsequent problems or need for a pacemaker. No other complications occurred. A mean of 9±6 RF lesions was applied. The total procedure time averaged 4±1 hours. Fluoroscopy time was 41±25 minutes. Nonsustained junctional tachycardia occurring during RF current application was observed in 50 patients (91%) at the successful site (Fig 2).

Of the 55 patients undergoing slow pathway ablation, 23 patients (42%) constituted group 1, who had evidence of persistent dual AV nodal pathway physiology and/or echo beats at the end of the RF ablation procedure; group 2 comprised 32 patients in whom slow pathway conduction was completely eliminated. After slow pathway ablation, there was a significant increase in the AV block cycle length (from 346±38 to 390±47 milliseconds) and in the AV node effective refractory
period (from 271±51 to 303±62 milliseconds), whereas the ventriculoatrial (VA) block cycle length did not change significantly (from 298±70 to 293±65 milliseconds). According to the site of successful ablation of the slow pathway, 14 patients assigned to group A (25%) had their slow pathway successfully ablated at an inferoposterior site (at or below the os of the coronary sinus); in 41 patients, constituting group B, the RF ablation was successful at a site located medially or anteriorly and superior to the coronary sinus os (Fig 1).

During long-term clinical follow-up of 12±9 months, a total of 7 patients had recurrence of AVNRT. Clinical tachycardia recurrences were reported at 1 week (2 patients), 3 months (3 patients), and 6 months (2 patients) after the ablation. Six of these patients underwent successful repeat slow pathway ablation. The electrophysiological parameters before and after ablation were not significantly different in patients with and without recurrences. Before the ablation, the AV block cycle length was 340±58 milliseconds in patients with recurrences and 345±40 milliseconds in patients without recurrences; similarly, the VA block cycle length was 307±102 milliseconds in those with recurrences and 300±80 milliseconds in those without recurrences. The effective refractory period of the AV node was 263±111 milliseconds in the former and 270±55 milliseconds in the latter group. After ablation, the AV block cycle length was 373±53 and 380±50 milliseconds, respectively; the VA block cycle length was 312±105 and 295±70 milliseconds, and the effective refractory period was 317±95 and 305±60 milliseconds in the respective groups. The cycle length of the recurring AVNRT (328±47 milliseconds) was similar to that of the initial tachycardia (343±70 milliseconds).

There was no significant difference in the number of recurrences between groups 1 and 2, with a 9% recurrence rate (2 of 23 patients) for group 1, with persistent jump or echo beats, and 16% for group 2 (5 of 32 patients), without residual slow pathway conduction (P=NS) (Table 1). However, a significant difference was noted in the recurrence rate between groups A and B (Table 2). Five of the recurrences (71%) were noted in group A and 2 in group B. Thus, the recurrence rate was 36% for group A (5 of 14 patients), with posterior ablations, and 5% for group B (2 of 41 patients), with anterior sites of successful RF ablation (P<.05). With regard to the relation among the four groups, it was noted that among group 1 patients, 2 of 8 having their successful ablation at a posterior site and none of 15 patients having ablation at an anterior site had

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**Table 1. Recurrence Rate of Atrioventricular Nodal Reentrant Tachycardia In Patients With or Without Residual Slow Pathway Conduction**

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<tr>
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<th>Group 1, Persistent Dual Pathways</th>
<th>Group 2, Loss of Dual Pathways</th>
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<tbody>
<tr>
<td>No. of patients</td>
<td>23</td>
<td>32</td>
</tr>
<tr>
<td>Persistent jump or echo beats</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>No. of patients with recurrence (%)</td>
<td>2 (9%)*</td>
<td>5 (16%)*</td>
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*P=NS.
Table 2. Recurrence Rate of Atrioventricular Nodal Reentrant Tachycardia According to Site of Successful Slow Pathway Ablation

<table>
<thead>
<tr>
<th>Group A, Posterior Site</th>
<th>Group B, Anterior Site</th>
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<tbody>
<tr>
<td>No. of patients</td>
<td>14</td>
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<tr>
<td>No. of patients with recurrence (%)</td>
<td>5 (36%)*</td>
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<tr>
<td></td>
<td>41</td>
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<td>2 (5%)*</td>
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*P < .05.

recurrences during follow-up; among group 2 patients, 3 of 6 with posterior ablation (50%) and 2 of 26 with anterior ablation (8%) had recurrences (P < .05). Finally, there was no difference in the number of lesions or energy amount delivered in patients with and without recurrences.

Discussion

Slow pathway ablation has become the most commonly and successfully used method of AV nodal modification offering a cure to patients with AVNRT with excellent efficacy and safety; this is a consistent finding in the present study and in other recent reports.3,5,11 Although a couple of reports have proposed the use of the electrophysiological technique with recordings of slow pathway potentials to guide the ablation,4,5 most studies, including the present one, have relied on an anatomically guided approach, with excellent results.2,3,8,12 Recurrences of AVNRT after initially successful RF ablation of the slow pathway have been reported at a rate of about 10%.3,8,9 However, no previous study has yet examined the relation of persistent slow pathway conduction or site of successful ablation with the rate of arrhythmia recurrence in these patients. The present study established a relation of tachycardia recurrences with the site of successful ablation but not with persistent slow pathway conduction (Tables 1 and 2). Significantly more recurrences were observed when the ablation site was located posteriorly (at or below the os of the coronary sinus) as compared with anterior locations. On the other hand, residual slow pathway conduction in the form of persistent jump in the AV conduction time or atrial echo beats did not correlate with tachycardia recurrences.

A degree of variability in the anatomic location of successful RF ablation of the slow pathway has been reported, with the most common site of successful slow pathway ablation described along the tricuspid annulus immediately anterior to the coronary sinus os (70% to 80%).3,5 Although anterior anatomic locations have been chosen by some investigators as the initial sites of slow pathway ablation,9 most prefer to start at posterior sites in an attempt to minimize the risk of injuring the AV node and causing AV block.4,6,11 However, according to the results of the present study, RF ablation at posterior sites is associated with a higher recurrence rate. It would therefore be preferable to choose more anterior sites as the initial targets and only if unsuccessful to move to inferoposterior locations. Because RF ablation was initiated posteriorly in the present study and was ended when successful, one cannot exclude the presence of other successful sites more anteriorly (cephalad to the coronary sinus os), which if identified first may confer a lower recurrence rate, as suggested by the results of the present study, a possibility that needs to be explored further. Only a prospective, randomized comparison of posterior versus anterior sites selected as initial targets of slow pathway ablation would clarify this issue. Such a study should also examine whether ablation at anterior sites is safe and does not increase the incidence of AV block. In the present study, the patient who developed transient AV block did have successful ablation at an anterior site.

In most studies, residual slow pathway conduction in the form of persistent jump in the AV conduction time or atrial echo beats is common after successful slow pathway RF ablation.2,4,5,8,10 It has been intuitively believed that it is not necessary to eliminate all slow pathway conduction to successfully treat AVNRT, but one can end the procedure when AVNRT cannot be induced rather than aiming at eliminating all slow pathway conduction.5,9 Conversely, others target all slow pathway conduction and aim at eliminating any jump or AV nodal echo beats.3,6 The results of the present study support the view that it is not necessary to eliminate all slow pathway conduction for successful treatment of AVNRT and effective prevention of any recurrences. There was no higher incidence of tachycardia recurrences in patients with residual slow pathway conduction. On the contrary, more recurrences, although not at a statistically significant degree, were observed in patients with no residual slow pathway conduction. These results contrast with those of a recent report,14 whereby the tachycardia recurrence rate was higher in patients with residual slow pathway conduction. The routine use of isoproterenol infusion after ablation in our study may account for this discrepancy, as it probably facilitated the induction of AVNRT, which otherwise would have falsely denoted a successful ablation.

References


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