Selective Radiofrequency Ablation of the Slow Pathway for the Treatment of Atrioventricular Nodal Reentrant Tachycardia

Evidence for Involvement of Perinodal Myocardium Within the Reentrant Circuit

G. Neal Kay, MD; Andrew E. Epstein, MD; Sharon M. Dailey, MD; and Vance J. Plumb, MD

Background. The circuit of atrioventricular (AV) nodal reentrant tachycardia may include perinodal atrial myocardium. Furthermore, in patients with dual AV nodal pathways, the atrial insertion of the slow pathway is likely to be located near the ostium of the coronary sinus, caudal to the expected location of the AV node. The present study was designed to evaluate the safety and efficacy of selective catheter ablation of the slow pathway using radiofrequency energy applied along the tricuspid annulus near the coronary sinus ostium as definitive therapy for AV nodal reentrant tachycardia.

Methods and Results. Among 34 consecutive patients who were prospectively enrolled in the study, the slow pathway was selectively ablated in 30, and the fast pathway was ablated in four. Antegrade conduction over the fast pathway remained intact in all 30 patients after successful selective slow pathway ablation. There was no statistically significant change in the atrio-His interval (68.5±21.8 msec before and 69.6±23.9 msec after ablation) or AV Wenckebach rate (167±27 beats per minute before and 178±50 beats per minute after ablation) after selective ablation of the slow pathway. However, the antegrade effective refractory period of the fast pathway decreased from 348±94 msec before ablation to 309±79 msec after selective slow pathway ablation (p=0.005). Retrograde conduction remained intact in 26 of 30 patients after selective ablation of the slow pathway. The retrograde refractory period of the ventriculo-atrial conduction system was 285±55 msec before and 280±52 msec after slow pathway ablation in patients with intact retrograde conduction (p=NS). There were three complications in two patients, including an episode of pulmonary edema and the development of spontaneous AV Wenckebach block during sleep in one patient after slow pathway ablation and the late development of complete AV block in another patient after fast pathway ablation. Over a mean follow-up period of 322±73 days, AV nodal reentrant tachycardia recurred in three patients, all of whom were successfully treated in a second ablation session.

Conclusions. Radiofrequency ablation of the slow AV pathway is highly effective and is associated with a low rate of complications. (Circulation 1992;85:1675-1688)

Key Words • ablation • electrophysiology • atrioventricular node

Atrioventricular (AV) conduction in individuals with AV nodal reentrant tachycardia is characterized by several unusual features. These include a discontinuous response of the atrio-His (AH) interval to programmed atrial stimulation that is compatible with the presence of dual pathways of antegrade conduction,1-3 a short RP interval, and a long PR interval during tachycardia4-6 and the observation that initiation of the tachycardia is dependent on the induction of critical delay in the AH interval.14-7 These observations suggest that the mechanism of the tachycardia involves intranodal reentry with antegrade conduction via a slowly conducting AV pathway and retrograde conduction via a rapidly conducting pathway.8

This traditional concept has been supported by the demonstration that the tachycardia may continue in the presence of block below the His bundle,9,10 that premature ventricular extrastimuli introduced at the time of antegrade His bundle activation do not advance the atrial cycle length,10,11 and that premature atrial extrastimuli may capture portions of the atria without resetting the tachycardia.12

Despite these observations, several findings conflict with the traditional concept of this arrhythmia as a reentrant circuit that is confined to the AV node. First, in contrast to control subjects, retrograde conduction from the His bundle to the atria typically has a nondirectional character in patients with AV nodal reentrant tachycardia.13,14 Second, the sequence of retrograde atrial activation often shifts from the apex to the base of Koch's triangle simultaneously with sudden prolongation of the His-to-atrial conduction interval.15 This observation suggests that the atrial insertion of the slowly conducting AV pathway is located at the base of Koch's triangle, near the coronary sinus ostium,
whereas the more rapidly conducting pathway is located near the apex of the triangle. Third, intraoperative mapping studies in patients with AV nodal reentrant tachycardia have demonstrated that retrograde conduction can be interrupted by reversible cooling of atrial myocardium along the tendon of Todaro near the insertion to the central fibrous body, whereas antegrade conduction can be interrupted by cooling near the ostium of the coronary sinus.16 Fourth, atrial extrastimuli introduced at the base of Koch’s triangle during AV nodal tachycardia at a time when the fast pathway is refractory to antegrade penetration are capable of preexciting the slow pathway and advancing the cycle length of the tachycardia.17 Fifth, animal models of dual AV nodal pathways have indicated that atrial reentry may occur around the anatomic obstacle provided by the ostium of the coronary sinus.18,19 Last, several investigators have demonstrated that AV nodal reentrant tachycardia can be permanently cured by surgical procedures confined to perinodal tissue.20–23 These features have led to a revised concept of this arrhythmia as involving reentrant excitation that incorporates perinodal tissue within the circuit.21,24

On the basis of these observations, several investigators have developed catheter ablative techniques that use direct current shocks25,26 or radiofrequency energy27–30 to destroy the fast AV pathway and permanently eliminate AV nodal reentrant tachycardia. Despite the generally excellent results achieved with fast pathway ablation, this procedure is complicated by a small but definite incidence of AV block requiring permanent pacemaker implantation.29,30 An alternative procedure, slow pathway ablation, has been proposed that may be associated with a lower risk of AV block because ablative energies can be delivered relatively distant from the compact AV node.31 The present study was performed to evaluate the safety and efficacy of selective slow AV pathway ablation using radiofrequency energy for the treatment of AV nodal reentrant tachycardia. In particular, a major goal of the study was to assess the effect of slow pathway ablation on residual AV nodal function.

Methods

Study Population

The study population consisted of 34 consecutive patients (26 women and eight men; mean age, 51±17 years) with typical slow antegrade pathway, fast retrograde pathway AV nodal reentrant tachycardia who were referred for modification of the AV conduction system (Table 1). All patients had previously been treated with antiarrhythmic drugs without control of their arrhythmia or had experienced adverse effects requiring drug withdrawal. The experimental protocol was approved by the Investigational Review Board for Human Subjects of the University of Alabama at Birmingham. All patients gave written, informed consent. The mean cycle length of AV nodal reentrant tachycardia was 385±79 msec (range, 280–565 msec). Both the usual and unusual (antegrade conduction over the fast pathway and retrograde conduction over the slow pathway) forms of AV nodal reentrant tachycardia were inducible in two patients. Dual antegrade AV conduction pathways were demonstrable in 28 of 34 patients in the baseline state. In six patients, demonstration of dual antegrade AV pathways required the use of isoproterenol (four patients), isoproterenol and atropine (one patient), or esmolol (one patient).

Electrophysiological Protocol

The diagnostic electrophysiological study and catheter ablation procedures were performed during a single session. After sedation with intravenous meperidine and midazolam and local anesthesia with 1% mepivacaine, 6F quadripolar catheters were introduced percutaneously into the femoral vein and advanced to the right ventricular apex, high right atrium, and across the tricuspid valve for recording of His bundle activation. A 6F hexapolar or octapolar catheter was introduced into the right internal jugular vein and positioned in the coronary sinus. Heparin was administered as an intravenous bolus of 2,500–5,000 IU immediately after placement of catheters, followed by an additional 1,000 IU each subsequent hour of the procedure.

Antegrade AV Nodal Functional Assessment

After sedation of the patient and catheter positioning, baseline intracardiac conduction intervals were measured. Programmed electrical stimulation was performed at the high right atrium using an eight-beat S1 drive at a cycle length of 500 msec with introduction of single atrial extrastimuli. The extrastimulus coupling interval was decremented by 10 msec until atrial refractoriness. The A1-A2 duration recorded with each extrastimulus coupling interval was plotted against the A1-A2 interval (measured in the His bundle electrogram) to construct an antegrade AV nodal function curve.1,2 An increase in the A1-H2 interval of >50 msec in response to a decrease in the A1-A2 coupling interval of 10 msec or less was defined as a discontinuous AV nodal function curve and taken as evidence of dual antegrade AV pathways.1,2 In the presence of a discontinuous AV nodal function curve, the effective refractory period of the antegrade fast pathway was defined as the longest A1-A2 coupling interval that conducted over the slow pathway. The antegrade effective refractory period of the slow pathway was defined as the longest A1-A2 interval that failed to propagate to the His bundle. When a reproducible discontinuous AV conduction curve was not demonstrable in the baseline state, dual antegrade pathways could be demonstrated with an intravenous infusion of isoproterenol in four patients, atropine plus isoproterenol in one patient, or esmolol in one patient. Rapid atrial pacing was performed by increasing the atrial pacing rate from just faster than the sinus rate until AV Wenckebach conduction was observed.

Retrograde AV Nodal Functional Assessment

Programmed ventricular stimulation was performed from the right ventricular apex at a drive cycle length of 500 msec with introduction of single extrastimuli. A retrograde ventriculoatrial (VA) conduction curve was constructed by plotting the stimulus-to-atrial electrogram interval and the H2-A2 interval versus the V1-V2 coupling interval. The H2-A2 interval was measured during programmed ventricular stimulation and during AV nodal reentrant tachycardia. The H2-A2 interval during ventricular programmed stimulation was mea-
TABLE 1. Characteristics of 34 Consecutive Patients Referred for Modification

<table>
<thead>
<tr>
<th>Ablation/ Patient</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Antegrade</th>
<th>Retrograde</th>
<th>After ablation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Selective slow</td>
<td></td>
<td></td>
<td>ERP fast</td>
<td>ERP slow</td>
<td>ERP fast</td>
</tr>
<tr>
<td>pathway</td>
<td></td>
<td></td>
<td>AH</td>
<td>AV Wen</td>
<td>VA Wen</td>
</tr>
<tr>
<td>1 F 2F 72</td>
<td>1 F</td>
<td>72</td>
<td>530</td>
<td>342</td>
<td>140</td>
</tr>
<tr>
<td></td>
<td>2 F</td>
<td>51</td>
<td>318</td>
<td>&lt;270</td>
<td>170</td>
</tr>
<tr>
<td></td>
<td>3 F</td>
<td>16</td>
<td>410</td>
<td>300</td>
<td>162</td>
</tr>
<tr>
<td></td>
<td>4 F</td>
<td>46</td>
<td>365</td>
<td>&lt;230</td>
<td>150</td>
</tr>
<tr>
<td></td>
<td>5 F</td>
<td>56</td>
<td>310</td>
<td>280</td>
<td>180</td>
</tr>
<tr>
<td></td>
<td>8 M</td>
<td>27</td>
<td>46</td>
<td>350</td>
<td>300</td>
</tr>
<tr>
<td></td>
<td>9 F</td>
<td>36</td>
<td>65</td>
<td>270</td>
<td>250</td>
</tr>
<tr>
<td></td>
<td>10 F</td>
<td>37</td>
<td>55</td>
<td>&lt;225</td>
<td>215</td>
</tr>
<tr>
<td></td>
<td>11 F</td>
<td>64</td>
<td>82</td>
<td>280</td>
<td>260</td>
</tr>
<tr>
<td></td>
<td>12 M</td>
<td>58</td>
<td>70</td>
<td>270</td>
<td>255</td>
</tr>
<tr>
<td></td>
<td>13 F</td>
<td>19</td>
<td>72</td>
<td>302</td>
<td>265</td>
</tr>
<tr>
<td></td>
<td>14 M</td>
<td>72</td>
<td>135</td>
<td>445</td>
<td>360</td>
</tr>
<tr>
<td></td>
<td>15 F</td>
<td>60</td>
<td>90</td>
<td>372</td>
<td>338</td>
</tr>
<tr>
<td></td>
<td>16 F</td>
<td>74</td>
<td>115</td>
<td>620</td>
<td>380</td>
</tr>
<tr>
<td></td>
<td>17 M</td>
<td>45</td>
<td>75</td>
<td>320</td>
<td>&lt;280</td>
</tr>
<tr>
<td></td>
<td>19 M</td>
<td>71</td>
<td>52</td>
<td>490</td>
<td>300</td>
</tr>
<tr>
<td></td>
<td>21 F</td>
<td>29</td>
<td>45</td>
<td>265</td>
<td>&lt;225</td>
</tr>
<tr>
<td></td>
<td>22 F</td>
<td>54</td>
<td>58</td>
<td>&lt;240</td>
<td>&lt;260</td>
</tr>
<tr>
<td></td>
<td>23 F</td>
<td>60</td>
<td>50</td>
<td>270</td>
<td>245</td>
</tr>
<tr>
<td></td>
<td>24 F</td>
<td>46</td>
<td>50</td>
<td>310</td>
<td>265</td>
</tr>
<tr>
<td></td>
<td>25 F</td>
<td>34</td>
<td>60</td>
<td>300</td>
<td>260</td>
</tr>
<tr>
<td></td>
<td>26 M</td>
<td>71</td>
<td>110</td>
<td>550</td>
<td>265</td>
</tr>
<tr>
<td></td>
<td>27 F</td>
<td>58</td>
<td>77</td>
<td>320</td>
<td>240</td>
</tr>
<tr>
<td></td>
<td>28 F</td>
<td>63</td>
<td>82</td>
<td>330</td>
<td>280</td>
</tr>
<tr>
<td></td>
<td>29 F</td>
<td>59</td>
<td>75</td>
<td>280</td>
<td>260</td>
</tr>
<tr>
<td></td>
<td>30 F</td>
<td>68</td>
<td>52</td>
<td>330</td>
<td>300</td>
</tr>
<tr>
<td></td>
<td>31 F</td>
<td>31</td>
<td>55</td>
<td>280</td>
<td>220</td>
</tr>
<tr>
<td></td>
<td>32 F</td>
<td>66</td>
<td>52</td>
<td>290</td>
<td>260</td>
</tr>
<tr>
<td></td>
<td>33 M</td>
<td>53</td>
<td>62</td>
<td>380</td>
<td>330</td>
</tr>
<tr>
<td></td>
<td>34 F</td>
<td>67</td>
<td>55</td>
<td>380</td>
<td>280</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>No. of radiofrequency applications</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
</tr>
<tr>
<td>23</td>
</tr>
<tr>
<td>6</td>
</tr>
<tr>
<td>15</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>4</td>
</tr>
</tbody>
</table>

For all V1 V2 coupling intervals that demonstrated a clear retrograde His bundle deflection. A discontinuous retrograde conduction curve was defined as an increase in the H2A2 interval of >50 msec observed with a decrease in the V1 V2 coupling interval of ≤10 msec. The retrograde AV nodal function curve was categorized as nondecremental if the H2A2 interval did not increase by more than 10 msec from the longest to the shortest V1 V2 coupling intervals achieved. The site of earliest retrograde atrial activation was recorded both during programmed ventricular stimulation and during AV nodal reentrant tachycardia. Rapid ventricular pacing was performed by increasing the pacing rate until VA Wenckebach conduction was observed. Single ventricular extrastimuli were introduced during tachycardia simultaneously with antegrade His bundle activation, and the response of the atrial cycle length was assessed.

Diagnostic Criteria

AV nodal reentrant tachycardia was diagnosed by the following criteria: 1) Initiation of the tachycardia was dependent on achievement of a critical delay in the AH interval; 2) retrograde atrial activation during tachycar-
radiofrequency energy at the same site was delivered at 50–60 V for 60 seconds. If selective slow pathway ablation was not successful by application of radiofrequency energy in this region, the catheter was repositioned higher along the tricuspid annulus (more cephalad) in a continued attempt to ablate the slow pathway, recognizing that the fast pathway might be ablated. After delivery of radiofrequency energy and achievement of a stable sinus rate, programmed atrial and ventricular stimulation using single extrastimuli was performed to construct antegrade and retrograde AV nodal function curves. Incremental atrial and ventricular pacing were used to determine antegrade and retrograde Wenckebach rates. Isoproterenol was then infused, and atrial and ventricular pacing were used in an attempt to induce AV nodal reentrant tachycardia or identify the presence of slow pathway conduction. The ablation session was ended when there was no evidence of slow pathway conduction, including the elimination of AV nodal echo beats.

Follow-up Evaluation

All patients had continuous ECG monitoring for 24 hours after ablation, with 12-lead ECGs recorded at approximately 6 and 24 hours after the procedure. Serum creatine kinase–MB fraction was measured immediately after the procedure and every 6 hours for the next 24 hours. All patients were discharged without antiarrhythmic drugs and were evaluated at 1 month in clinic with a repeat 12-lead ECG. If patients developed any evidence of tachycardia recurrence, such as palpitations or by ECG recordings, repeat electrophysiological testing was performed.

Statistical Analysis

Continuous variables are expressed as mean±SD. Variables were compared before and after ablation of the slow pathway using the Wilcoxon sign rank test; a value of p<0.05 was considered to indicate statistical significance.

Results

Catheater Ablation

Radiofrequency ablation was successful in eliminating inducible AV nodal reentrant tachycardia in all 34 patients. In four patients who were treated early in our experience, selective slow pathway ablation could not be accomplished, and these patients developed impairment of antegrade fast pathway conduction as the catheter was moved higher (more cephalad) in Koch’s triangle. AV nodal reentrant tachycardia was eliminated in each of these patients without complications. This subgroup of patients was considered separately in the analysis of the effects of selective slow pathway ablation on residual AV conduction. These were the only patients who developed prolongation of the AH interval with delivery of radiofrequency energy.

In 30 patients, the antegrade slow pathway was ablated. The median number of radiofrequency applications required for slow pathway ablation was 10 (range, three to 59), with a mean of 28.7±6.6 W for the apparently successful application. The catheter locations required for successful ablation of the slow pathway are shown in Figure 2. The most common site of
successful slow pathway ablation was along the tricuspid annulus anterior to the ostium of the coronary sinus (24 patients). In four patients, the slow pathway was ablated along the tricuspid annulus inferior to the coronary sinus ostium and in two patients within the ostium of the coronary sinus. The peak serum creatine kinase–MB fraction was a mean of 3.6±4.1 IU/l.

Functional Characteristics of the Fast and Slow AV Pathways

The baseline AH interval was a mean of 68.5±21.8 msec (range, 45–135 msec). The antegrade effective refractory period of the fast AV pathway before ablation was 348±94 msec, and the antegrade effective refractory period of the slow pathway was 282±44 msec. Before ablation, retrograde conduction over the fast pathway was limited by refractoriness in the His-Purkinje system in 32 patients, with block in the fast AV nodal pathway in two patients. The mean retrograde effective refractory period of the VA conduction system was 285±55 msec. The effective refractory period of the slow pathway in the retrograde direction was measurable in only two of the 34 patients, both of whom had inducible AV nodal reentrant tachycardia of the unusual form. In these individuals, the retrograde fast pathway effective refractory period (mean, 435 msec) was longer than that of the retrograde slow pathway (mean, 400 msec), and earliest retrograde atrial activation shifted from the apex (His bundle electrogram) to the base (coronary sinus ostium) of Koch’s triangle simultaneously with the change from retrograde fast to retrograde slow pathway conduction. The retrograde fast pathway was characterized by nondecremental conduction from the His bundle to the atrium (maximum increment in HA interval during programmed ventricular stimulation <10 msec) in 31 of 34 patients. The site of earliest retrograde atrial activation during the usual form of AV nodal reentrant tachycardia was at the His bundle electrogram in 32 patients and occurred simultaneously at the His bundle electrogram and coronary sinus ostium in two patients. In the two patients who also had the unusual form of AV nodal reentrant tachycardia, the site of earliest retrograde atrial activation during this arrhythmia was at the ostium of the coronary sinus in one patient and 1 cm within the coronary sinus ostium in the other.

Effect of Slow Pathway Ablation on Residual Antegrade Conduction (30 Patients)

After ablation of the slow pathway, there was no statistically significant change in the AH interval during sinus rhythm (68.5±21.8 msec before and 69.6±23.9 msec after ablation). The AV Wenckebach rate was also unchanged after ablation of the slow pathway (AV Wenckebach rate, 167±27 beats per minute before and 178±50 beats per minute after ablation, p=0.31). In contrast, the antegrade effective refractory period of the fast pathway shortened from 348±94 msec before to 309±79 msec after slow pathway ablation (p=0.005) (Figures 3 and 4). Although patients were kept heavily sedated throughout the procedure, the sinus cycle length decreased from 744±189 msec before ablation to 642±148 msec afterward (p=0.06). The change in the antegrade effective refractory period of the fast pathway observed after ablation of the slow pathway correlated moderately with the change in sinus cycle length (r=0.51).

Effect of Slow Pathway Ablation on Retrograde Conduction

In the 26 patients with intact retrograde conduction after slow pathway ablation, the retrograde effective refractory period of the VA conduction system demonstrated no significant change after slow pathway ablation (285±55 msec before versus 280±52 msec after, p=0.61). Similarly, the VA Wenckebach rate (175±30 beats per minute before) was unaffected by slow pathway ablation in these patients (182±38 beats per minute after, p=0.49). The H2A2 interval during programmed ventricular stimulation at similar coupling intervals was 86.3±25.3 msec before and 89.3±22.5 msec after ablation (p=0.73) (Figure 5). The nondecremental nature of retrograde His bundle–to–atrial conduction was not changed after slow pathway ablation. Retrograde conduction was abolished in four of the 30 patients who demonstrated evidence of antegrade slow pathway ablation. These individuals demonstrated no impairment of antegrade fast pathway conduction as a result of ablation. In each of these patients, a retrograde His bundle deflection could not be recorded after application of radiofrequency energy along the tricuspid annulus, suggesting that block was likely to have occurred in the His-Purkinje system. It seems likely that this observation is related to the effect of changing autonomic tone.

Ablation of Antegrade Fast Pathway Conduction (Four Patients)

Apparent ablation of antegrade conduction in the fast pathway was observed in four of 34 patients.

**FIGURE 2.** Diagram of distribution of successful slow pathway ablation sites. The triangle of Koch is represented, bounded anteriorly by the septal leaflet of the tricuspid valve (TV), posteriorly by the tendon of Todaro, and caudally by the coronary sinus ostium. The slow pathway was ablated by the application of radiofrequency energy along the tricuspid annulus anterior to the coronary sinus ostium in 24 patients. The site of successful slow pathway ablation was caudal to the coronary sinus along the tricuspid annulus in four patients (4) and within the coronary sinus itself in two patients (2).
Application of radiofrequency energy along the tricuspid valve annulus anterior to the coronary sinus was not successful in ablating slow pathway conduction in these individuals. The ablation catheter was then moved higher along the tricuspid annulus, closer to the expected location of the AV node. After application of radiofrequency current at this site, there was prolongation of the AH interval (from a mean of 55 to 154 msec), and the antegrade AV conduction curve became continuous, suggesting that the antegrade fast pathway had been ablated. The antegrade effective refractory period of the slow pathway (mean, 281 msec before and 291 msec after) and the AV Wenckebach rate (mean, 208 beats per minute before and 208 beats per minute after) were not significantly changed after ablation in this subgroup of patients. Although the antegrade AV nodal conduction curve suggested that the antegrade fast pathway had been eliminated, retrograde conduction remained intact in three of these four patients, with continued nondecremental His–to–atrial conduction properties. This observation may suggest that the application of radiofrequency energy in these patients resulted in impairment of conduction within a portion of the AV node rather than selectively ablating an anatomically distinct fast pathway.

Complications

Three complications were associated with the slow pathway ablation procedure in two patients. In one patient who developed fast pathway impairment as the ablation site was moved more cephalad along the tricuspid annulus (patient 7), asymptomatic complete AV block with a narrow QRS escape at a rate of 60 beats per minute was found at a routine clinic visit 1 month after the procedure, and a permanent pacemaker was implanted. One elderly patient (patient 17) with a dilated cardiomyopathy related to incessant AV nodal reentrant tachycardia developed pulmonary edema after the ablation procedure, probably as a result of intravenous fluid administration. This patient also had a prolonged antegrade refractory period of the fast pathway (620 msec) before ablation of the slow pathway with AV Wenckebach conduction at a pacing rate of 111 beats per minute. The day after ablation of the slow pathway, the patient was observed to have spontaneous AV Wenckebach block during sleep that was asymptomatic and did not require pacing. This observation may or may not have been a complication of the procedure. In long-term follow-up, there has been no further AV block in this patient, and the left ventricular function has returned to normal with control of the tachycardia.

Follow-up

During a mean follow-up period of 322±73 days, AV nodal reentrant tachycardia recurred in three patients after slow pathway ablation (at intervals of 1 day to 6 months). Each of these patients underwent a second

FIGURE 3. Graphs showing atrioventricular (AV) nodal function curves. Top panel: The A2H2 interval in msec is plotted on the ordinate versus the A1A2 coupling interval (in msec) during programmed atrial stimulation in patient 3. Before slow pathway ablation (Pre RF), there is a discontinuous curve with a marked increase in the A2H2 interval from 160 to 320 msec between A1A2 coupling intervals of 420 and 410 msec, respectively (open circles). After ablation (Post RF), the AV nodal function curve (closed squares) is characterized by a smooth and gradual prolongation of the A2H2 interval until the refractory period of the AV node is encountered (340 msec). Also note that the effective refractory period of the fast AV pathway has decreased from 410 msec before ablation of the slow pathway to 330 msec after slow pathway ablation. Bottom panel: atrioventricular nodal conduction curves before (open circles) and after (closed squares) ablation of the slow pathway are demonstrated for patient 4.
FIGURE 4. Tracings of typical response of antegrade conduction to slow atrioventricular pathway ablation. Surface ECG leads I, II, III, V₁, and simultaneously recorded bipolar intracardiac electrograms from the high right atrium proximal (HRAp) and distal (HRAd) electrode pairs, His bundle electrogram proximal (HBEp) and distal (HBEd) electrode pairs, coronary sinus proximal (CSp), mid (CSm), and distal (CSd) electrode pairs, and right ventricular apex (RVA). S₁, drive stimulus; S₂, extrastimulus. Panel A: Before ablation (Pre RF). Programmed atrial stimulation with basic drive cycle length of 500 msec before slow pathway ablation with an A₁A₂ coupling interval of 430 msec and A₂H₂ interval of 160 msec. Panel B: Before ablation (Pre RF). The A₁A₂ coupling interval is...
shortened to 420 msec with an increase in the $A_2H_2$ interval to 320 msec. Note that two atrioventricular nodal echo beats (Ae) are observed simultaneously with the shift from antegrade fast to antegrade slow pathway conduction. Panel C: After ablation of the slow pathway (Post RF). The $A_1A_2$ coupling interval is 430 msec and the $A_2H_2$ interval is 130 msec with conduction over the fast pathway. Panel D: After ablation of the slow pathway (Post RF). The $A_1A_2$ interval is shortened to 330 msec. Note that the $A_2H_2$ interval remains short (150 msec), indicating that there is continued conduction over the fast pathway. There was complete atrioventricular block at an $A_1A_2$ coupling interval of 320 msec without evidence of slow pathway conduction.
electrophysiological study with successful ablation of the slow pathway, and all have remained asymptomatic. All other patients have done well without recurrent palpitations or arrhythmia recurrence. No patient with successful slow pathway ablation has developed PR interval prolongation on follow-up ECG or has required pacemaker implantation.

Discussion
Anatomy of Discontinuous AV Conduction

AV conduction in patients with AV nodal reentrant tachycardia is usually discontinuous in nature, suggesting that there may be at least two pathways of conduction. It is not known whether these functional properties are related to distinct anatomic pathways, longitudinal dissociation within the AV node, or functional properties of the perinodal atrial myocardium. Although the precise anatomy associated with dual AV conduction characteristics has not been clearly defined, the concepts of fast and slow conduction pathways have clinical relevance and were used in the interpretation of our findings. Although we have referred to fast and slow AV conduction pathways, these terms are meant to characterize functional properties rather than necessarily implying that these are distinct anatomic structures.

Clinical Efficacy of Slow Pathway Ablation

This study demonstrates that the application of radiofrequency energy along the tricuspid annulus near the coronary sinus ostium is an effective therapy for the definitive management of AV nodal reentrant tachycardia. Indeed, immediately after the procedure, AV nodal reentrant tachycardia was noninducible in all 34 patients. Elimination of this tachycardia was achieved by apparent ablation of the slow AV nodal pathway in 30 patients (88%) and by apparent ablation of the fast pathway in four patients (12%). In follow-up, AV nodal reentrant tachycardia recurred in three patients after slow pathway ablation (9%), each of whom was successfully treated during a second session. Thus, the use of
catheter ablation with radiofrequency energy resulted in the control of this arrhythmia without the use of antiarrhythmic drugs in all 34 patients studied and was associated with a low incidence of complications.

Limitations and Technical Considerations Related to Slow Pathway Ablation

An important potential limitation of this study is the lack of long-term electrophysiological study data (except in patients with clinical symptoms suggesting tachycardia recurrence). However, the mean follow-up period (322 days) may be sufficiently long to conclude that the technique provided genuine clinical benefit in this study population with recurrent supraventricular tachycardia. In addition, the inability to precisely regulate the autonomic tone of patients during the course of the procedure limits conclusions regarding the effect of slow pathway ablation on residual AV conduction.

Several clinical observations regarding the practical application of this technique should be emphasized. First, there was a small degree of variability in the anatomic location of radiofrequency energy applications that resulted in elimination of slow pathway conduction. The most common site for successful slow pathway ablation was along the tricuspid annulus immediately anterior to the coronary sinus ostium (80%) (Figures 1 and 2). The site of successful slow pathway ablation was caudal to the coronary sinus ostium along the tricuspid annulus in 13% and within the ostium of the coronary sinus in 7%. Although slow pathway ablation was not achieved in four patients who were treated early in our experience, radiofrequency applications were delivered along the tricuspid annulus near the ostium of the coronary sinus before the catheter was moved more cephalad. Although AV nodal reentrant tachycardia was eliminated in each of these patients after apparent elimination of antegrade fast pathway conduction, it is likely that selective slow pathway ablation could have been accomplished in these individuals had radiofrequency energy been applied along the tricuspid annulus caudal to or within the ostium of the coronary sinus. Judging from our later experience, if slow pathway ablation cannot be achieved by radiofrequency applications anterior to the coronary sinus, the catheter should be moved caudal to the coronary sinus ostium along the tricuspid annulus or positioned within the coronary sinus ostium itself. It is likely that this strategy will result in an even higher probability of selective slow pathway ablation and reduce the chances of impairing fast pathway conduction.

Second, slow pathway ablation is most feasible when dual AV pathways are demonstrable with reproducible evidence of slow pathway conduction. Although dual AV pathways cannot be demonstrated in up to 25% of individuals with AV nodal reentrant tachycardia, the ability to discriminate slow pathway conduction from that of fast pathway conduction can be enhanced by the use of pharmacological manipulations such as the infusion of isoproterenol, atropine, or esmolol. Thus, slow pathway ablation may become technically feasible in some patients without evidence of discontinuous AV conduction characteristics by the use of these pharmacological manipulations. Third, although ablation of the slow pathway is generally associated with preserved AV conduction, the status of the fast pathway should be carefully considered before ablation of the slow pathway. Indeed, in patients with a very long antegrade refractory period of the fast pathway, residual AV conduction may be impaired after ablation of the slow pathway. Fast pathway ablation may be the preferred treatment for these individuals.

Effect of Slow Pathway Ablation on Residual AV Conduction

This study demonstrates that ablation of the antegrade slow AV pathway does not impair antegrade AV nodal function when radiofrequency current is applied along the tricuspid annulus near the coronary sinus ostium. The AH interval during sinus rhythm and the AV and VA Wenckebach rates (in patients with intact retrograde conduction) were unchanged after apparent slow pathway ablation compared with the baseline values. Of interest is the finding that the antegrade effective refractory period of the fast pathway tended to decrease after elimination of slow pathway conduction. The most likely explanation for this finding is enhanced sympathetic tone coincident with the conduct of the procedure. The observation of a decrease in the sinus cycle length after ablation of the slow pathway compared with baseline suggests that the autonomic milieu was likely to have changed during the course of the procedure. Supporting this explanation is the moderate correlation observed between the change in effective refractory period of the fast pathway after slow pathway ablation with the change in sinus cycle length (r=0.51).

Another potential mechanism for shortening of fast pathway refractoriness is that catheter ablation may have resulted in selective destruction of parasympathetic innervation to the AV node. Although innervation of the human AV node is less well understood than in animals, the parasympathetic fibers that innervate the AV node of canines are clustered near the crux of the heart and traverse the triangle of Koch. It is possible that application of radiofrequency energy in this region selectively destroyed these parasympathetic fibers.

In most patients, ablation of the slow pathway produced no effect on retrograde AV conduction. In patients with intact retrograde conduction after slow pathway ablation, there was no change in the retrograde effective refractory period of the fast pathway or the VA Wenckebach rate. Retrograde conduction was abolished in four of 30 patients, however, probably related to block in the His-Purkinje system, as no retrograde His bundle deflection was recorded during programmed stimulation. It is likely that this effect was related to a change in autonomic tone over the course of the procedure.

Observations From Patients Who Developed Apparent Impairment of Antegrade Fast Pathway Conduction

Apparent impairment of antegrade fast pathway function developed in four of 34 patients. The technique used in these patients differed considerably from that proposed by Lee et al, as the AV node was approached from sites inferior to the apex of Koch's triangle in our series. In addition, radiofrequency energy had previously been applied along the tricuspid annulus near the ostium of the coronary sinus in these patients. Although AV reentrant
tachycardia was eliminated in each of these individuals, retrograde conduction persisted in three of the four patients. Furthermore, there was persistence of nondecremental retrograde His–to-atrial conduction properties in these patients after impairment of antegrade fast pathway conduction. This observation differs from the results reported for fast pathway ablation using the technique of Lee and colleagues (as reported by Kunze et al,29 Lee et al,29 and Calkins et al30), which usually abolishes retrograde conduction. It is quite likely that the function of the AV node was altered in a nonspecific way rather than selective ablation of a specialized fast pathway in our patients.

Implications Regarding the Mechanism of AV Nodal Reentrant Tachycardia

The consistent ability to eliminate slow AV pathway conduction by the application of radiofrequency energy along the tricuspid annulus near the coronary sinus ostium may add support to the concept that the reentrant circuit of AV nodal reentrant tachycardia includes perinodal tissue.16,17,20,21,23 As suggested by animal models of this arrhythmia,18,19 the slow pathway may involve atrial myocardium near the coronary sinus ostium that provides posterior input to the zone of transitional cells in the AV junction. Our observations that radiofrequency applications anterior to, caudal to, or within the coronary sinus ostium that abolished retrograde conduction properties that respond to sodium channel blocking drugs34,35 Our usual finding of persistent nondecremental retrograde conduction from the His bundle to the atrium that is characteristic of individuals with AV nodal reentrant tachycardia has been suggested to indicate that the fast pathway may not be composed of typical AV nodal tissue.13,14 The retrograde fast pathway is further characterized by “all or none” conduction properties that respond to sodium channel blocking drugs.34,35 Our usual finding of persistent nondecremental retrograde conduction from the His bundle to the atrium that is characteristic of individuals with AV nodal reentrant tachycardia has been suggested to indicate that the fast pathway may not be composed of typical AV nodal tissue.13,14 The retrograde fast pathway is further characterized by “all or none” conduction properties that respond to sodium channel blocking drugs.34,35

Conclusions

Ablation of the slow AV pathway and elimination of AV nodal reentrant tachycardia by the application of radiofrequency energy along the tricuspid annulus near the coronary sinus ostium is a highly effective procedure that is associated with a relatively low risk of complications. Selective ablation of the slow pathway preserves residual AV conduction and is likely to minimize the risk of AV block compared with the previously reported fast pathway ablation technique. The successful ablation of AV nodal reentrant tachycardia by the application of radiofrequency energy along the tricuspid annulus near the ostium of the coronary sinus supports the concept that extranodal tissue is involved within the reentrant circuit.

References

Selective radiofrequency ablation of the slow pathway for the treatment of atrioventricular nodal reentrant tachycardia. Evidence for involvement of perinodal myocardium within the reentrant circuit.

G N Kay, A E Epstein, S M Dailey and V J Plumb

Circulation. 1992;85:1675-1688
doi: 10.1161/01.CIR.85.5.1675

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1992 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/85/5/1675

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/