Radiofrequency Catheter Ablation for Atrioventricular Node Reentry Tachycardia with Multiple Slow Atrioventricular Node Pathways

Muneshige Kaibara, M.D., Atsushi Konoe, M.D., Shojiro Isomoto, M.D.
Osmar A. Centurion, M.D., Tetsuya Hirata, M.D., Osamu Hano, M.D.
Ryoji Sakamoto, M.D., Keiji Iwamoto, M.D., Zhigang Liu, M.D.
Motonobu Hayano, M.D. and Katsusuke Yano, M.D.

Catheter ablation was attempted in 2 patients with atrioventricular node reentry tachycardia which showed fast, intermediate and slow anterograde atrioventricular node pathways. Radiofrequency currents were applied within a restricted area of the tricuspid annulus between the His bundle and the ostium of the coronary sinus where presumed slow pathway potentials were identified. Elimination of both the intermediate and the slow pathways, with preservation of anterograde and retrograde fast pathway conduction, was achieved in both patients. (Jpn Circ J 1995; 59: 224—230)

THE atrioventricular (AV) node conduction curve in some patients with atrioventricular node reentry tachycardia (AVNRT) consists of multiple slow conduction pathways. Based on the results of selective slow pathway radiofrequency ablation in the common slow-fast form of AVNRT, several reports have suggested that the slow pathway is located in or near the perinodal zone posterior to the AV node. It has also been suggested that the input of the slow pathway to the AV node is inferior to the compact node. However, only Philippon et al. have noted a relationship between multiple slow pathways and the radiofrequency ablation site, and the anatomical location of multiple slow pathways remains unclear. We report here 2 cases of AVNRT with multiple slow pathways which were ablated within a restricted area. These results may suggest the anatomical location of multiple slow pathways to the AV node.

CASE REPORT

Two patients with AVNRT were referred to Nagasaki University Hospital for radiofrequency catheter ablation of the AV conduction system.

Case 1
A 68-year-old woman, who had previously suffered from myocarditis, had recurrent paroxysms of palpitation and dyspnea. She could not take antiarrhythmic drugs which have a negative inotropic effect, since an echocardiographic study showed reduced wall motion and an enlarged cavity in the left ventricle.

Case 2
An 82-year-old man had previously been treated with antiarrhythmic drugs, which did not control his arrhythmia. The antiar-

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The Third Department of Internal Medicine Nagasaki University School of Medicine Nagasaki, Japan
Mailing address: Muneshige Kaibara, M.D., The Third Department of Internal Medicine Nagasaki University School of Medicine Nagasaki, Japan

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rhythmic drugs previously used were verapamil hydrochloride, propranolol hydrochloride, procainamide hydrochloride, aprindine hydrochloride, disopyramide, and cibenzoline succinate. Electrophysiological studies and catheter ablation were approved by the institutional ethics committee and written informed consent was obtained. Our techniques for conducting an electrophysiological study have been described previously.\textsuperscript{5,14} Cardioactive drugs were discontinued for >4 half-lives. The power source used for catheter ablation was a NL-50-I (Central Industry, Chiba). A 7Fr quadrupolar steerable 4mm-tip ablation catheter (type-B, Mansfield-Webster Catheters, Watertown, MA) was inserted percutaneously into the right femoral vein, advanced into the heart and positioned across the tricuspid valve. Both the left anterior oblique view (LAO 60°) and the right anterior oblique view (RAO 30°) were used to verify the catheter's position. Radiofrequency energy, which was delivered through the distal catheter electrode and a cutaneous patch on the left posterior chest, was applied at 20 W for 30 sec during sinus rhythm while impedance, catheter stability, surface electrocardiogram (leads I, AVF and V1), and intracardiac electrograms were continuously monitored.

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The endpoint was elimination of the induction of AVNRT as evaluated by atrial extra-stimuli or incremental atrial pacing. The total applied energy of the radiofrequency current was 1135 J in case 1 and 613 J in case 2. The effects of ablation on the electrophysiological properties of the AV node were evaluated 20 min after the last delivery of radiofrequency energy. The radiofrequency currents were applied at sites along the tricuspid annulus between the His bundle and the ostium of the coronary sinus, where presumed slow pathway potentials were identified, as shown in Fig. 1.

In both cases, anterograde triple AV node pathways were demonstrated before ablation as a discontinuous A1—A2, A2—H2 curve by atrial extrastimulus testing, as shown in Figs. 2 and 3. The AV nodal conduction times and refractory periods were associated with three distinct pathways, i.e., fast, intermediate, and slow. In both cases, during the application of ventricular extrastimuli, the retrograde conduction curve was continuous and an atrial activation was first inscribed at the low septal right atrium from the His bundle recording site, suggesting retrograde fast pathway conduction (data not shown). In case 1, as A1—A2 intervals decreased from 410 ms to 400 ms, the A2—H2 interval increased from 170 ms to 235 ms (Fig. 4 A and B). As the A1—A2 intervals decreased further, a second jump of 105 ms occurred at an A1—A2 interval of 380 ms (Fig. 4 C and D). AVNRT was induced at an A1—A2
interval of 360 ms (Fig. 2). The second application of radiofrequency current resulted in elimination of the induction of AVNRT. After ablation, both the intermediate pathway conduction and the slow pathway conduction were eliminated, as shown in Figs. 2 and 4 E, and the effective refractory period of the fast pathway (ERP_F) was reduced from 400 ms to 320 ms. The minimum cycle length which maintained anterograde 1:1 conduction over the AV node (1:1 Ant) was decreased from 353 ms to 333 ms (Table I). Ablation did not affect the AH interval, the minimum cycle length which maintained retrograde 1:1 conduction over the AV node (1:1 Ret) or the retrograde effective refractory period of the AV node (Ret ERP_{AVN}) (Table I).

In case 2, at a drive cycle length of 700 ms, as A1-A2 intervals decreased from 370 ms to 360 ms, the A2-H2 interval increased from 190 ms to 250 ms. AVNRT was induced at an A1-A2 interval of between 350 ms and 300 ms. As the A1-A2 interval decreased further, a second jump of 130 ms occurred at an A1-A2 interval of 290 ms (Fig. 3A). At a drive cycle length of 500 ms, as A1-A2 intervals decreased from 350 ms to 340 ms, the A2-H2 interval increased from 230 ms to 315 ms (Fig. 5 A and B). AVNRT was induced at an A1-A2 interval of between 340 ms and 300 ms. As the A1-A2 interval decreased further, a second jump of 90 ms occurred at an A1-A2 interval of 280 ms (Fig. 5 C and D). A single application of radiofrequency current resulted in the elimination of the induction of AVNRT. After ablation, both the inter-
mediate pathway conduction and the slow pathway conduction were eliminated, as shown in Figs. 3 B and 5 E. ERP_F was reduced from 340 ms to 240 ms and 1:1 Ret was reduced from 400 ms to 333 ms. Ablation did not affect the resting AH interval (Table I).

Follow-up observation over 10 and 6 months in cases 1 and 2, respectively, showed no recurrence of paroxysmal supraventricular tachycardia and no appearance of AV block.

DISCUSSION

In this study, the three ranges of AH conduction times and effective refractory periods in each case suggested the presence of triple antegrade AV node pathways. Spurrell et al.\textsuperscript{15} reported that changes in the AH interval during tachycardia suggested the presence of multiple antegrade pathways. Wu et al.\textsuperscript{16} reported 7 patients with multiple AV node reentry tachycardias. Although the properties of antegrade intermediate pathways were unclear in their report, three types of retrograde pathways were differentiated based on conduction times and the sites of the earliest retrograde atrial activation during AVNRT. During AVNRT in the present cases, antegrade conduction passed through the slow pathway and the intermediate pathway in cases 1 and 2, respectively, and retrograde conduction passed through the fast pathway in both cases. Alternation was not observed. It could be argued that the jumpy slow pathway conduction curves could be due to an unstable autonomic tone. Nevertheless, the presence of triple antegrade AV node
TABLE I ELECTROPHYSIOLOGICAL EFFECTS OF CATHETER ABLATION

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Ablation</th>
<th></th>
<th>Control</th>
<th>Ablation</th>
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</thead>
<tbody>
<tr>
<td>(AH)</td>
<td>110</td>
<td>110</td>
<td>(1:1) Ant</td>
<td>95</td>
<td>95</td>
</tr>
<tr>
<td>(1:1) Ret</td>
<td>353</td>
<td>333</td>
<td>(1:1) Ret</td>
<td>400</td>
<td>ND</td>
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<tr>
<td>(ERP_F)</td>
<td>400</td>
<td>400</td>
<td>(ERP_{INT})</td>
<td>400</td>
<td>333</td>
</tr>
<tr>
<td>(ERP_S)</td>
<td>380</td>
<td>320</td>
<td>(ERP_S)</td>
<td>340</td>
<td>240</td>
</tr>
<tr>
<td>Ret (ERP_{AVN})</td>
<td>280</td>
<td>-</td>
<td>Ret (ERP_{AVN})</td>
<td>270</td>
<td>-</td>
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<tr>
<td></td>
<td>260&gt;</td>
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<td>250&gt;</td>
<td>230&gt;</td>
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\(AH=AH\) interval; \(1:1\) Ant=minimum cycle length maintaining \(1:1\) anterograde AV conduction; \(1:1\) Ret=minimum cycle length maintaining \(1:1\) retrograde AV conduction; \(ERP_F\)=effective refractory period of the fast pathway measured at a drive cycle length of 500 ms; \(ERP_{INT}\)=effective refractory period of the intermediate pathway measured at a drive cycle length of 500 ms; \(ERP_S\)=effective refractory period of the slow pathway measured at a drive cycle length of 500 ms; Ret \(ERP_{AVN}\)=effective refractory period of retrograde AV conduction measured at a drive cycle length of 500 ms, except in the control study in case 2. In the control study in case 2, Ret \(ERP_{AVN}\) was measured at a drive cycle length of 600 ms; ND=not documented.

Radiofrequency catheter ablation of multiple anterograde slow AV node pathways in individuals with AVNRT was reported by Philippon et al\(^\text{13}\) in 1993. They reported that the slow pathway was usually ablated more inferiorly in Koch's triangle than the intermediate pathway, while in some cases both pathways could be ablated at a single site. They suggested that multiple slow pathways might represent different approaches to the AV node. In our cases, both the anterograde intermediate and slow pathways were eliminated by the application of radiofrequency current within a restricted area of the tricuspid annulus between the His bundle and the coronary sinus ostium. Since the sites for selective ablation of the slow pathway in our patients were closer to the compact node than the sites reported by Philippon's co-workers; the intermediate and slow pathways may gather in a narrow space which leads to the compact node. Along these lines, Wu et al\(^\text{16}\) reported that the inferior approach, which was designed to selectively ablate the slow pathway at the site of the compact node, was used in 5 patients with multiple AV node reentry tachycardias which showed triple retrograde AV node pathways. Their procedures affected both the retrograde intermediate pathway and the anterograde slow pathway in 3 of 5 cases, and selectively affected the retrograde intermediate pathway in 2 of 5 cases. These results indicate that the approach of the anterograde and/or retrograde intermediate pathway to the compact node may be anatomically similar to that of the anterograde slow pathway. Preservation of the anterograde and retrograde fast pathway conduction after ablation suggests that this fast pathway is anatomically distant from the multiple slow pathways.

Radiofrequency current was applied relatively near the compact node. Although both the anterograde and retrograde conduction of the fast pathway were apparently preserved after the selective ablation of the slow pathways, a careful follow-up study is necessary to assess the function of the fast pathway.

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