Clinical, Electrocardiographic, and Electrophysiological Observations of Dual A-V Nodal Pathways

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In 45 (25%) of 182 patients with various cardiac arrhythmias, dual A-V nodal pathways (DPWs) were diagnosed with atrial extrastimulus technique at least at one or more basic cycle lengths and/or after intravenous administration of atropine (1 mg). The jump of discontinuous \( A_1 A_2, H_1 H_2 \) curve of these 45 ranged from 25 to 235 (92 ± 56) msec and the jump of \( A_1 A_2, A_2 H_2 \) curve ranged from 40 to 260 (107 ± 55) msec. The fast pathway FRP (functional refractory period), slow pathway FRP, fast pathway ERP (effective refractory period) and slow pathway ERP was 464 ± 87 msec, 532 ± 91 msec, 404 ± 96 msec and 328 ± 70 msec, respectively. DPWs were demonstrated in 10 (59%) of 17 patients with paroxysmal supraventricular tachycardia (PSVT), 3 (5%) of 55 with WPW syndrome, 2 (20%) of 10 with paroxysmal atrial fibrillation, 11 (29%) of 38 with sick sinus syndrome, 10 (38%) of 26 with first degree and/or second degree A-V (AH) block, none of 3 with second degree HV block, 3 (27%) of 11 with bundle branch block and 6 (27%) of 22 with the other cardiac arrhythmias. In 17 patients with PSVT, seven demonstrated A-V nodal reentrant tachycardia. Six of these 7 had evidence of DPWs. In the other 7 of the 17, concealed accessory pathway was demonstrated. Three of these 7 had DPWs, which did not constitute the reentrant circuit. Twenty-eight of 45 patients (62%) with DPWs had one or more electrophysiological abnormalities suggesting A-V nodal dysfunction: 1) prolonged AH interval (> 130 msec) during sinus rhythm (10 patients), 2) atrial pacing rates of 130 or less inducing A-V nodal Wenckebach periods (24 patients), 3) prolonged A-V nodal ERP or slow pathway ERP (> 400 msec) (8 patients), and 4) prolonged A-V nodal FRP or fast pathway FRP (> 500 msec) (16 patients). However, in most patients, atropine improved A-V nodal dysfunction.

We consider that DPWs are a common electrophysiological finding and have a strong association not only with PSVT but also with A-V nodal dysfunction.

PAROXYSMAL supraventricular tachycardia (PSVT) may be the result of reentry within the atrioventricular (A-V) node.\(^1-3\) The mechanism of A-V nodal reentry is still debated. Impulse reflection resulting from depressed conduction\(^4\) or dual A-V nodal pathways are usually invoked. Animal studies suggest that the A-V node itself may undergo longitudinal dissociation into two pathways demonstrable under appropriate conditions, and that a reciprocation within the A-V node can produce a tachycardia\(^5-9\).

Rosen et al. reported a patient with two sets

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Key Words:
- Dual A-V nodal pathways
- Two PR intervals
- Atrial extrastimulus technique
- Paroxysmal supraventricular tachycardia
- A-V nodal dysfunction

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of A-V nodal conduction times and refractory periods, suggesting dual A-V nodal pathways, by testing with the extrastimulus technique in the atrium.\textsuperscript{10,11} Denes and co-workers stressed the role of dual A-V nodal pathways in the genesis of A-V nodal reentrant tachycardia.\textsuperscript{12} Patients who were considered to have dual A-V nodal pathways had evidence suggestive of both a fast and a slow A-V nodal pathways, each with its own effective and functional refractory period. Failure of the fast pathway was often accompanied by occurrence of A-V nodal echoes, implying that this pathway was utilized for retrograde propagation during episodes of PSVT.\textsuperscript{13–17}

Since programmed electrical stimulation\textsuperscript{18} of the heart has become a widely used technique for the investigation of arrhythmias, considerable interest has been shown in the conduction patterns suggesting dual A-V nodal pathways in patients with or without PSVT. Recent reports have suggested that this type of A-V conduction is common, and may be seen in patients without a history of PSVT.\textsuperscript{19,20} However, there have been few studies to determine the frequency of occurrence of dual A-V nodal pathways in patients with various cardiac arrhythmias. Moreover, there are few studies which have shed a light on the clinical significance, one of the most intriguing questions, of dual A-V nodal pathways.

Although dual A-V nodal pathways are defined in the human heart with the atrial extrastimulus technique when an abrupt increase in A-V nodal delay is produced by a minimal decrease in the test atrial stimulus.\textsuperscript{11–13,16,17,19,20} there have been few reports of quantitative criteria for diagnosis of dual A-V nodal pathways.

Therefore, this study was performed to define the criteria for diagnosis of dual A-V nodal pathways, to analyze the electrophysiological properties of dual A-V nodal pathways, and to provide further information about the clinical significance of dual A-V nodal pathways.

**MATERIALS AND METHODS**

The records of 182 patients who had undergone atrial extrastimulus studies in our department of the Nagasaki University Hospital between April 1975 and December 1979 were analyzed. Ages of these patients ranged from 8 to 78 years (mean ± SD 43 ± 19 years).

Electrophysiological studies were performed in the post absorptive, nonsedated state. All cardiac drugs were discontinued at least 5 days before the study.

Under local anesthesia, two electrode catheters were positioned to record the intracardiac electrograms from the His bundle and the high right atrium near its junction with the superior vena cava. The terminals of these electrode catheters were connected to the inputs of the ECG amplifiers of a Fukuda Electronics Recorder (MCM-8000). Frequencies below 40 and above 500 Hz were filtered. Three surface electrocardiographic leads (I, aVF, V\textsubscript{1}) were recorded simultaneously with the His bundle electrogram (HBE) and high right atrial electrogram (HRA) on a multichannel ink-jet recorder (Siemens-Mingograf 800) at a paper speed of 100 mm/sec.

Pacing stimuli were delivered to the right atrium and supplied by a programmable stimulator (Nihon Kodenh MSE-40) with a strength of approximately twice diastolic threshold and duration of 1.5 msec. Electrophysiological studies consisted of incremental atrial pacing and atrial extrastimulus technique. The right atrium was paced at rates slightly faster than spontaneous sinus rhythm. Pacing rates were increased in 10 beats/min increments until A-V nodal Wenckebach periods were observed. The atrial extrastimulus technique was similar to that described by Wit et al.\textsuperscript{18} Atrial extrastimulus was given after every tenth basic driven beat and the coupling interval was decreased in 10–20 msec steps until the refractory period of the right atrium was reached. The atrial extrastimulus technique was first performed at the longest basic cycle length assuring atrial capture, then, in most patients, was repeated at one or more basic cycle lengths which were shortened in 100–200 msec steps.

Electrophysiological studies were repeated after intravenous administration of atropine (1 mg) in 81 patients.

**Electrophysiological Definitions**

A\textsubscript{1}, H\textsubscript{1}, and V\textsubscript{1} symbolize the atrial, His bundle, and ventricular electrograms, respectively, of the basic driven beat preceding the premature stimuli. A\textsubscript{2}, H\textsubscript{2}, and V\textsubscript{2} are the atrial, His bundle, and ventricular electrograms, respectively, in response to the atrial extrastimulus. The response of the A-V node to the increasingly premature atrial stimulation was characterized by plotting H\textsubscript{1} H\textsubscript{2} intervals against A\textsubscript{1} A\textsubscript{2} coupling intervals. The A-V nodal effective refractory period (ERP) is defined as the longest A\textsubscript{1} A\textsubscript{2} interval not propagated to the His bundle. The A-V nodal functional refractory period (FRP) is

the shortest interval between two successive His bundle responses ($H_1, H_2$), both propagated from the atrium. Conduction velocities of the A-V node were also examined by plotting $A_2H_2$ intervals against $A_1A_2$ coupling intervals.

A smooth curve characterizes the normal physiologic response of an A-V node without functional longitudinal dissociation (Fig. 1). In patients with dual A-V nodal pathways, the curve is discontinuous. Dual A-V nodal pathways were
considered present if the $H_1H_2$ interval increased suddenly for a decrement of 10 msec or less in the $A_1A_2$ interval, with a concomitant sudden jump in the conduction time represented by the $A_2H_2$ interval, with no change in $H_2V_2$ intervals (Figs. 2 and 3). The portion of the curve to the right of the jump represents the fast pathway, and to the left, the slow pathway. The fast pathway ERP was defined as the longest $A_1A_2$ that failed to conduct via the fast pathway. The fast pathway FRP was defined as the shortest attainable $H_1H_2$ interval on the fast pathway curve. The ERP of the slow pathway was the longest $A_1A_2$ not conducted via the slow path-

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way. When atrial FRP limited slow pathway conduction, the slow pathway ERP could not be measured, and was considered to be less than the shortest conducted A1A2 interval. The slow pathway FRP was the shortest attainable H1H2 interval on the slow pathway curve.

RESULTS

1) The Maximal A2H2 and H1H2 Increase in the Smooth A-V Nodal Conduction Curve

In 86 of the 182 patients, A-V nodal conduction curves were smooth at every basic cycle length and after administration of atropine. The maximal A2H2 increase was defined as the maximal increment of A2H2 interval produced by 10 msec decrement of the A1A2 coupling interval in each smooth A1A2, A2H2 curve. In these 86 patients, the maximal A2H2 increase was measured at every basic cycle length and after administration of atropine. The maximal H1H2 increase was also measured.

The basic cycle lengths (before and after administration of atropine) were divided into four ranges: 300 to 500 msec, 501 to 700 msec, 701 to 900 msec, and 901 to 1200 msec. There was no statistically significant difference in the maximal A2H2 increase between four ranges of basic cycle lengths (Fig. 4, left panel). The mean maximal A2H2 increase was 22 ± 11 msec (Fig. 4, right panel).

In 64 of the 86 patients who showed smooth A-V nodal conduction curves, the H1H2 curves continued to descend during the relative refractory period of the A-V node at a decreasing slope. At a critical A1A2 interval, H1H2 reached a minimum and then began to increase even though the A1A2 coupling interval shortened further. The H1H2 increase until complete block of conduction of the premature impulse occurred. In these 64, the maximal H1H2 increase was measured at every basic cycle length and after administration of atropine.

There was no statistically significant difference in the maximal H1H2 increase among the four ranges of basic cycle lengths (Fig. 5, left panel). The mean maximal H1H2 increase was 16 ± 10 msec (Fig. 5, right panel).

In patients who showed the smooth A-V nodal conduction curve, the mean maximal A2H2 increase was 22 ± 11 msec and mean
maximal H1H2 increase was 16 ± 10 msec. The mean maximal A2H2 and H1H2 increase plus 2 standard deviation from these values was 44 msec and 36 msec, respectively.

2) Electrophysiological Findings of Dual A-V Nodal Pathways

In 45 of the 182 patients, dual A-V nodal pathways were diagnosed with the atrial extrastimulus technique: thirty-seven of these 45 were considered to have dual A-V nodal pathways at least at one or more basic cycle lengths before administration of atropine. In 26 of 81 patients to whom atropine sulfate (1 mg) was given intravenously, dual A-V nodal pathways were diagnosed. In 8 of these 26, dual A-V nodal pathways were demonstrated only after atropine.

In 41 of 45 patients with dual A-V nodal pathways, the jump of A1A2, A2H2 curve and A1A2, H1H2 curve was larger than 44 msec (the mean maximal A2H2 + 2 SD) and 36 msec (the mean maximal H1H2 + 2 SD), respectively. In the total number of 11 A1A2, H1H2 curves of 3 patients of the remaining 4, the jump of 9 curves was below 36 msec. In the total number of 10 A1A2, A2H2 curves of 3 patients (2 of these 3 patients were the same patients of the above mentioned), the jump of 6 curves was below 44 msec. However, the A-V nodal conduction curves of these 4 patients were discontinuous and these 4 patients were considered to have dual A-V nodal pathways. The jump of A1A2, H1H2 curves of 3 typical patients was smaller than 36 msec (Fig. 6). Moreover, in one of these 3, the jump of A1A2, A2H2 curve was also smaller than 44 msec. However, the A-V nodal conduction curves of these 3 patients were discontinuous (Fig. 6).

Fig. 7 shows the jump in the A1A2, H1H2 and A1A2, A2H2 curves of the patients with
dual A-V nodal pathways. The jump of $H_1H_2$ interval of all the basic cycle lengths ranged from 25 to 235 msec (mean ± SD 92 ± 56 msec) in 37 patients. The jump of $A_2H_2$ interval ranged from 40 to 260 msec (107 ± 55 msec) (Fig. 7, left panels). In 26 patients who had the evidence of dual A-V nodal pathways after administration of atropine, the jump of $H_1H_2$ interval ranged from 30 to 210 msec (85 ± 47 msec) and the jump of $A_2H_2$ interval ranged from 40 to 215 msec (97 ± 50 msec) (Fig. 7, right panels).

In the 24 patients in whom dual pathway curves were obtained at two or more basic cycle lengths, the effect of decrease in basic cycle length on the jump of $H_1H_2$ and $A_2H_2$ intervals were determined (Fig. 7). After administration of atropine, the effect of decrease in basic cycle length on the jump were determined in only two patients. The effect of decrease in basic cycle length on the jump is represented as the slope. However, the slopes show no relationship of the jump of $H_1H_2$ and $A_2H_2$ to basic cycle lengths.

At all the basic cycle lengths, the fast pathway FRP ranged from 285 to 710 msec (mean ± SD 464 ± 87 msec) (Fig. 8). The slow pathway FRP ranged from 340 to 785 msec (532 ± 91 msec). As a whole, the slow pathway FRPs are longer than the fast pathway FRPs. The fast pathway ERP ranged from 250 to 670 msec (404 ± 96 msec). The slow pathway ERP were measured in most of patients with dual pathways. When the slow pathway ERP was atrial limited, the slow pathway ERP was not obtained, and was considered to be less than the atrial ERP. In these patients, the atrial ERP were depicted in Fig. 8 as the approximate values of the slow pathway ERPs. The slow pathway ERP ranged from 195 to 590 msec (328 ± 70 msec). Although the fast pathway ERP is longer than the slow pathway ERP in the individual patient with dual path-
ways, as a whole, the fast pathway ERPs are longer than the slow pathway ERPs.

The fast and slow pathway FRPs have a tendency to decrease as basic cycle length is shortened, whereas the fast and slow pathway ERPs have a tendency to increase as basic cycle length is shortened (Fig. 8). However, slopes show no significant relationship of the functional and effective refractory periods of dual pathways to basic cycle lengths.

The acute effect of atropine on the functional and effective refractory periods of dual A-V nodal pathways were observed in 13 patients, in whom refractory periods at similar basic cycle lengths could be obtained before and after administration of atropine (the difference among these basic cycle lengths was within 100 msec). In these 13, the fast and slow pathway refractory periods decreased after administration of atropine as follows: fast pathway FRP from 429 ± 79 to 338 ± 36 msec, slow pathway FRP from 462 ± 63 to 412 ± 55 msec, fast pathway ERP from 364 ± 82 to 287 ± 33 msec, and slow pathway ERP from 291 ± 43 to 238 ± 39 msec.

In 35 of 45 patients with dual A-V nodal pathways, the atrial extrastimulus technique was performed at two or more basic cycle lengths. In 27 of these 35, the difference of two basic cycle lengths was 200 msec or more. In 19 of these 27, dual pathways were demonstrable at both two basic cycle lengths. In the other 3, conduction curves were smooth at longest cycle length, and dual pathways were demonstrated at shorter basic cycle length. However, in 5 patients, dual pathways were demonstrable only at longest basic cycle length, and conduction curves were smooth at shorter basic cycle length.

In 36 of 45 patients with dual A-V nodal pathways, the atrial extrastimulus technique was performed after administration of atropine. In 21 of these 36, refractory periods at similar basic cycle lengths could be obtained before and after administration of atropine (the difference of these basic cycle lengths was within 100 msec). In 13 of these 21, dual pathways were demonstrable prior to and following atropine. In 5 patients, conduction curves were smooth prior to atropine administration, and discontinuous after the drug. In the remaining 3, dual pathways were demonstrable only before atropine, and
conduction curves were smooth after the drug.

3) Incidence of Dual A-V Nodal Pathways

In 45 (25%) of the 182 patients, dual A-V nodal pathways were diagnosed with atrial extrastimulus technique. Eighty-six patients were considered to have smooth A-V nodal conduction curves. In the remaining 51, the A-V nodal conduction curve was classified into neither the smooth curve nor the discontinuous curve. In 6 patients of these 51, the deviation of each dot of the A-V nodal conduction curve from the predicted curve was so large and irregular that the diagnosis of the curve was impossible. There were 45 patients with WPW syndrome in these

51.

There were 26 males and 19 females among 45 patients with dual A-V nodal pathways whose mean age was 41 ± 19 years (range 14—73 years). Dual A-V nodal pathways were demonstrated in 10 of 17 patients (59%) with PSVT, 3 of 55 (5%) with WPW syndrome, 2 of 10 (20%) with paroxysmal atrial fibrillation, 11 of 38 (29%) with sick sinus syndrome, 10 of 26 (38%) with first degree and/or second degree A-V block (AH block in His bundle electrogram), none of 3 with second degree HV block and 3 of 11 (27%) with bundle branch block (right bundle branch block, left bundle branch block, or right bundle branch block with left axis deviation, etc.) (Table I).
Patients with documented first degree and/or second degree A-V block in our present series maintained 1:1 A-V conduction during the period of electrophysiological study using atrial extrastimulus technique. In the remaining 22, 6 (27%) had the evidence of dual A-V nodal pathways. Eleven patients of these 22 had paroxysmal palpitation without documentation of spontaneous or induced PSVT. Dual A-V nodal pathways were demonstrated in 2 of these 11 (18%). There were 2 patients with ectopic atrial tachycardia and 2 with history of syncopal attack in the remaining 4 with dual A-V nodal pathways.

4) PSVT and Dual A-V Nodal Pathways

Seventeen of the 182 patients had electrocardiographic documentation of paroxysmal supraventricular tachycardia (Table II). In these 17, PSVT was induced during electrophysiological studies. Patients with electrocardiographic documentation of delta wave and patients without electrocardiographic or electrophysiological evidence of PSVT were excluded from the PSVT group. In 7 of these 17 patients, A-V nodal reentrant tachycardia was demonstrated during electrophysiological studies. In the other 7, concealed accessory pathways were demonstrated. In the remaining 3, the mechanism of the reent-
TABLE I  INCIDENCE OF DUAL A-V NODAL PATHWAYS IN VARIOUS CARDIAC ARRHYTHMIAS

<table>
<thead>
<tr>
<th></th>
<th>Total No. of patients</th>
<th>No. of patients with dual A-V nodal pathways</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.</td>
</tr>
<tr>
<td>P S V T</td>
<td>17</td>
<td>10</td>
</tr>
<tr>
<td>WPW syndrome</td>
<td>55</td>
<td>3</td>
</tr>
<tr>
<td>Paroxysmal atrial fibrillation</td>
<td>10</td>
<td>2</td>
</tr>
<tr>
<td>Sick sinus syndrome</td>
<td>38</td>
<td>11</td>
</tr>
<tr>
<td>1°−2° AH block</td>
<td>26</td>
<td>10</td>
</tr>
<tr>
<td>2° HV block</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Bundle branch block</td>
<td>11</td>
<td>3</td>
</tr>
<tr>
<td>Other cardiac arrhythmias</td>
<td>22</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td>182</td>
<td>45</td>
</tr>
</tbody>
</table>

PSVT = Paroxysmal supraventricular tachycardia.
Patients with other cardiac arrhythmias include patients with ectopic atrial tachycardia, paroxysmal palpitation without documentation of spontaneous or induced PSVT, history of syncopal attack, and Romano-Ward syndrome.

TABLE II  MECHANISM OF TACHYCARDIA IN PATIENTS WITH PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

<table>
<thead>
<tr>
<th>Mechanism of PSVT</th>
<th>Total No. of patients</th>
<th>Evidence of DPWs</th>
<th>PSVT using DPWs</th>
</tr>
</thead>
<tbody>
<tr>
<td>A-V nodal reentrant tachycardia</td>
<td>7</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Concealed accessory pathway</td>
<td>7</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Undeterminate</td>
<td>3</td>
<td>1</td>
<td>?</td>
</tr>
<tr>
<td>Total</td>
<td>17</td>
<td>10</td>
<td>6 + ?</td>
</tr>
</tbody>
</table>

DPWs = dual A-V nodal pathways
rant tachycardia could not be determined.

In 7 patients with A-V nodal reentrant tachycardia, 6 (86%) had the evidence of dual A-V nodal pathways (Table II). In these 6, it was demonstrated that the reentrant tachycardia used a slow pathway for anterograde conduction and a fast pathway for retrograde conduction. In the remaining one, the mechanism of A-V nodal reentry was considered as reflection.

A-V nodal reentrant tachycardia using dual pathways was induced with atrial extrastimulus (Fig. 9). The high right atrium is driven at a basic cycle length of 700 msec. An atrial premature beat at a coupling interval (A1 A2) of 370 msec lengthens the A2 H2 interval to 170 msec (Fig. 9). An atrial premature beat at a coupling interval (A1 A2) of 360 msec suddenly prolongs the A2 H2 interval to 300 msec (a shift of conduction from the fast pathway to the slow pathway) and induces sustained A-V nodal reent-

rant tachycardia of the slow-fast form (Fig. 9).

In 7 patients with concealed accessory pathways, the reentrant tachycardia used the normal A-V conduction pathway for anterograde conduction and the concealed bypass for retrograde conduction. However, in these 7 with concealed accessory pathways, 3 (43%) had the evidence of dual A-V nodal pathways, which did not constitute the reentrant circuit (Table II).

In the remaining 3 patients in whom the mechanism of PSVT could not be determined, one had evidence of dual A-V nodal pathways.

Evidence of A-V nodal reentrant tachycardia using dual pathways was found in 6 of 17 patients (35%) with PSVT. In 45 patients with dual A-V nodal pathways, it was demonstrated in only these 6 (13%).

5) Two Non-overlapping Ranges of PR (AH) Intervals

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In 4 patients, two non-overlapping ranges of PR intervals were documented in surface electrocardiograms (routine electrocardiograms and/or 24-hour, long-term ambulatory electrocardiographic recordings). In one electrocardiogram a sudden shift from 0.20 sec of PR interval (fast pathway) to 0.36 sec or more of PR intervals (slow pathway) without significant change of P-P intervals occurred (Fig. 10).

In 45 patients with dual A-V nodal pathways, 15 patients (33%) demonstrated the shift of conduction from the fast pathway to the slow pathway by continuous atrial pacing. In one case, two AH intervals at an atrial pacing rate of 90/min was found (Fig. 11). Progressive lengthening of the AH interval from 155 to 175 msec was followed by a sudden jump of the AH interval to 295 msec, suggesting block in the fast pathway.
pathway with shift of conduction to the slow pathway (Fig. 11).

6) Atypical Wenckebach Periods

In 2 patients, atypical Wenckebach periods were documented in surface electrocardiograms. Sudden increases develop in PR intervals in the middle or terminal portions of Wenckebach periods (Fig. 12).

In 45 patients with dual A-V nodal pathways, 37 patients (82%) manifested a somewhat characteristic form of atypical Wenckebach periodicity with atrial pacing. These patients developed sudden increases in AH interval in the middle or terminal portions of pacing induced Wenckebach periods (Fig. 13). In Fig. 13, the first three beats engage both the fast and slow pathways and arrive at the His bundle via fast pathway. The fourth beat is blocked in the fast pathway and conducted anterogradely through the slow pathway. Therefore, the fourth beat shows sudden AH prolongation. The fifth beat is blocked in both pathways.

7) A-V Nodal Dysfunction and Dual A-V Nodal Pathways

Twenty-eight patients (62%) of the 45 patients with dual A-V nodal pathways had one or more electrophysiological abnormalities suggesting depressed A-V nodal function.

Electrophysiological evidence for A-V nodal dysfunction was considered to be present on the
basis of one or more of the following: 1) prolonged AH interval (>130 msec) during sinus rhythm, which was found in 10 patients (22%), 2) atrial pacing rates of 130/min or less inducing A-V nodal Wenckebach periods, which was found in 24 patients (53%) (Fig. 14), 3) prolonged A-V nodal ERP or slow pathway ERP (>400 msec) at the longest basic cycle length slightly shorter than sinus, which was found in 8 patients (18%), and 4) prolonged A-V nodal FRP or fast pathway FRP (>500 msec) at the longest basic cycle length, which was found in 16 patients (36%).

However, in most patients, Wenckebach type AH block occurred at higher atrial pacing rates after administration of atropine than before (Fig. 14). The fast and slow pathway refractory periods decreased after administration of atropine.

**DISCUSSION**

1) *Electrophysiological Diagnosis of Dual A-V Nodal Pathways*

Dual pathway curves are characterized by a sudden jump in H1, H2 at a critical range of A1, A2 coupling intervals. However, there have been few reports regarding the definite criteria of a jump for diagnosis of dual A-V nodal pathways.

Rosen, Denes, Wu et al. diagnosed dual A-V nodal pathways only by above mentioned discontinuous A1, A2, H1, H2 curve.11,12,19 There was no quantitative criteria in their reports. However, in some of the cases where the diagnosis of dual

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pathways was equivocal, they used curve fitting analysis. Thapar and Gillette considered that dual A-V nodal pathways were present if the curve was discontinuous and the $H_1 H_2$ interval increased by 40 msec for a decrement of 10 msec or less in the $A_1 A_2$ interval. Bissett et al. measured the maximal $A_2 H_2$ and $H_1 H_2$ increase produced by a single premature atrial contraction at 10 msec interval (mean maximal $A_2 H_2$ increase $26 \pm 17$ msec and mean maximal $H_1 H_2$ increase $20 \pm 15$ msec). They considered that patients having a maximal $A_2 H_2$ and $H_1 H_2$ increase greater than 3 standard deviation from these values (77 and 65 msec, respectively) had dual A-V nodal pathways.

In our study, measurements of the maximal $A_2 H_2$ and $H_1 H_2$ increase were performed in 86 patients who showed the apparent smooth A-V nodal conduction curve. From our results, we considered that discontinuous A-V nodal conduction curve was diagnosed as dual A-V nodal pathways if the $H_1 H_2$ interval increased 40 msec or more for a decrement of 10 msec in the $A_1 A_2$ coupling interval. However, some patients with a smaller jump were detected to have dual A-V nodal pathways because of an apparently discontinuous A-V nodal conduction curve (Fig. 6). We therefore conclude that the essential for diagnosis of dual A-V nodal pathways is to detect a discontinuity of the A-V nodal conduction curve. Even if the $H_1 H_2$ increase was slightly smaller than 40 msec, patients having a concomitant $A_2 H_2$ increase greater than 40 msec and showing the discontinuous A-V nodal conduction curve were considered to have dual A-V nodal pathways.

2) Electrophysiological Findings of Dual A-V Nodal Pathways

Forty-five out of the 182 patients were considered to have dual A-V nodal pathways according to our criteria. Of these 45 patients, 8 have been the subjects of a previous report.2

(a) Jump

There have been few reports regarding the value and range of the jump of A-V nodal conduction curves in many patients with dual A-V nodal pathways. In our present series, 66 jumps of $H_1 H_2$ intervals in 37 patients ranged from 25 to 235 msec (mean $\pm$ SD 92 $\pm$ 56 msec). The jump of $A_2 H_2$ intervals ranged from 40 to 260 msec (107 $\pm$ 55 msec).

(b) Effects of basic cycle length

Denes et al. reported that both the ERP and FRP of the atrium shortened with decreasing basic cycle lengths, however, A-V nodal ERP lengthened while A-V nodal FRP shortened slightly. Patients with dual A-V nodal pathways had evidence suggestive of both a fast and a slow A-V nodal pathway, each with its own effective and functional refractory period. Denes et al. determined the effect of decreasing basic cycle lengths on fast and slow pathway refractory periods. They observed a significant increase in fast pathway ERP with shortening of basic cycle length.

In our study, the fast and slow pathway FRPs appeared to have a tendency to decrease with
shortening of basic cycle length. The fast and slow pathway ERPs appeared to have a tendency to increase with shortening of basic cycle length. However, the effect of decreasing basic cycle lengths on fast and slow pathway refractory periods was not uniform (Fig. 8).

Denes et al. demonstrated the unmasking of dual A-V nodal curves with decreasing basic cycle length. They reported that dual A-V nodal pathways were only demonstrated at shorter basic cycle length in many patients with smooth A-V nodal conduction curves at longer basic cycle length. They postulated that at longer basic cycle lengths, slow pathway ERP was longer than fast pathway ERP, thus preventing demonstration of dual pathways with the extrastimulus technique. With shortening of basic cycle length, lengthening of the fast pathway ERP relative to the slow pathway ERP could allow demonstration of dual pathways. Also, the FRP of the atrium must be shorter than the slow pathway ERP, otherwise, dual pathways would be masked. In this situation, atrial pacing at a fast rate would shorten the FRP of the atrium, perhaps helping to unmask the presence of dual A-V nodal pathways.

In some of our patients, the unmasking of dual A-V nodal pathways with decreasing basic cycle length was observed. On the other hand, dual pathways were masked in some other patients. Our result was compatible with the diversity of responses of fast and slow pathway refractory periods to alteration of basic cycle lengths.

(c) Effects of atropine

In our study, atropine shortened the FRP and ERP of both the fast and slow pathways. Bisset et al. reported that conduction through the slow pathway might be concealed by atropine. However, in 5 patients of our series, dual pathways developed after administration of atropine.

The responses of fast and slow pathways to shortening of basic cycle lengths were not uniform. The mechanism of unmasking of dual A-V nodal pathways are not as simple as previously believed. However, the ability to demonstrate dual A-V nodal pathways may be increased by changing the basic cycle length or administration of atropine.

3) Incidence of Dual A-V Nodal Pathways

In a series of Denes et al., 10% of the adults undergoing electrophysiologic studies had dual A-V nodal pathways. Thapar and Gillette reported that the incidence of dual A-V nodal pathways in children and young adults was 46%. The results of these studies demonstrated that dual A-V nodal pathways were a common electrophysiologic response in adults and in children.

Our present study also suggests that dual A-V nodal pathways are a common electrophysiologic finding, being found in 25% of 182 patients with and without PSVT undergoing atrial extrastimulus technique.

On the other hand, Bisset et al. reported that dual A-V nodal pathways were common only in patients with PSVT (7 of 13 patients) and were significantly less frequent in patients without PSVT (9 of 135 patients). However, in their study, the atrial extrastimulus technique were performed only at similar basic cycle lengths. Moreover, their quantitative criteria for diagnosis of dual A-V nodal pathways may have a possibility of underestimation.

4) PSVT and Dual A-V Nodal Pathways

One of the most important clinical manifestations of dual A-V nodal pathways is paroxysmal supraventricular tachycardia.

Bisset et al. reported that evidence of dual A-V nodal pathways was found in seven (54%) of 13 patients with documented PSVT, and suggested a strong association between dual A-V nodal pathways and PSVT. Thapar and Gillette demonstrated that thirteen (62%) of the 21 children and young adults with PSVT had evidence of dual A-V nodal pathways. Rosen et al. also reported that approximately 50% of the patients studied with recurrent PSVT had dual A-V nodal pathways.

In our 17 patients with PSVT, ten (59%) had evidence of dual A-V nodal pathways. Comparison of our results with those of other investigators showed a similar incidence of dual A-V nodal pathways.

Thapar and Gillette reported that in 9 of 12 children with PSVT and dual A-V nodal pathways, the mechanism of tachycardia was due to reentry within the A-V node. However, atrial muscle reentry tachycardia was present in one and concealed accessory ventriculoatrial pathway in two. In our ten patients with documented PSVT and dual A-V nodal pathways, six patients demonstrated A-V nodal reentrant tachycardia using dual pathways. However, the evidence of reentrant tachycardia through concealed accessory ventriculoatrial pathway was observed in three. The mechanism of PSVT in the remaining...
one was undeterminate.

These results showed that dual A-V nodal pathways were a common electrophysiologic response, particularly in patients with PSVT. The most common mechanism of PSVT in the presence of dual A-V nodal pathways was reentry in the A-V node. However, it was demonstrated that PSVT might be due to reentry at other sites, despite the presence of dual A-V nodal pathways. One of these was PSVT using concealed anomalous A-V bypass tracts. In a series of Denes et al., seventeen of the 41 patients with dual A-V nodal pathways had previously documented PSVT. In 15 of these 17, A-V nodal reentrance with echo zones was demonstrated during electrophysiological studies. On the other hand, in our 45 patients with dual A-V nodal pathways, only 6 (13%) demonstrated the evidence of A-V nodal reentrant tachycardia. For the induction of A-V nodal reentrant tachycardia, the following conditions seem to be necessary: 1) an anterograde fast pathway ERP longer than the slow pathway anterograde ERP, 2) a slow pathway anterograde conduction time long enough for the pathway to recover for retrograde conduction, 3) presence of a final common pathway distal to the fast and slow pathways, and 4) ability for the fast pathway to conduct in retrograde direction. In order to know if the presence of dual A-V nodal pathways in a patient would predispose to A-V nodal reentrant tachycardia, a longer follow-up is necessary.

In our 55 patients with WPW syndrome in whom delta waves were documented at sinus rhythm, three (5%) had the evidence of dual A-V nodal pathways. In patients with WPW syndrome, a diagnosis of dual A-V nodal pathways can only be made when accessory A-V pathway had an ERP in the anterograde direction that exceeds the fast pathway ERP. Sung et al. reported that in 67 patients with reciprocating tachycardia using an anomalous A-V bypass tract, electrophysiological findings suggested the co-existence of dual A-V nodal pathways in 8 patients. Three of these 8 had concealed accessory pathways. Pritchett et al. also reported that dual A-V nodal pathways were found in five patients who had the WPW syndrome. All these patients had a reentrant tachycardia that used the A-V node for anterograde conduction and an accessory A-V pathway for retrograde conduction.

5) Two Non-overlapping Ranges of PR (AH)

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Intervals

The first case with dual A-V nodal pathways who Rosen et al. reported had electrocardiographic documentation of two PR intervals, and electrophysiological evidence of two sets of A-V nodal conduction times and refractory periods. In our 45 patients with dual A-V nodal pathways, four patients had documented two non-overlapping ranges of PR intervals in surface electrocardiograms. The electrocardiographic findings of two non-overlapping ranges of PR intervals is one of the important signs for diagnosis of dual A-V nodal pathways if it is documented.

Continuous atrial pacing demonstrated the shift of conduction from the fast pathway to the slow pathway in 15 of our 45 patients with dual A-V nodal pathways. Shift of conduction to the slow pathway can occur at pacing rates which induce Wenckebach type block in the fast pathway. This finding with atrial pacing is also considered to be useful for diagnosis of dual A-V nodal pathways.

6) Atypical Wenckebach Periods

In our present series, atypical Wenckebach periods were documented in surface electrocardiograms in 2 of 45 patients. However, thirty-seven of these 45 manifested a characteristic form of atypical Wenckebach periodicity with atrial pacing. These patients demonstrated sudden increases in AH in the middle or terminal portions of pacing induced Wenckebach periods. Atypical Wenckebach periods in surface electrocardiograms or with atrial pacing are useful findings for diagnosis of dual A-V nodal pathways.

7) A-V Nodal Dysfunction and Dual A-V Nodal Pathways

In a study of Denes et al., 22 of 41 patients (54%) with dual A-V nodal pathways had A-V nodal dysfunction as manifested by abnormal electrophysiological findings (prolonged conduction times or refractory periods) and/or etiological factors directly associated with A-V nodal injury (previous diaphragmatic infarction or intracardiac surgery). Injury to the A-V node secondary to diaphragmatic myocardial infarction or intracardiac surgery may manifest itself as A-V nodal dysfunction. A-V nodal dysfunction may be due to functional and/or anatomic disintegration of the A-V node. This may result in a pathological background of dual A-V nodal
pathways. Both fast and slow pathways may reflect either functional longitudinal dissociation or anatomical septation of the A-V node. Denes et al. considered that in some of the patients, pathological lesions might be responsible for anatomical division of the A-V node into two pathways. On the other hand, Bisset et al. reported that the incidence of dual A-V nodal pathways in patients with short or long PR intervals was studied at similar basic cycle lengths and that dual A-V nodal pathways did not appear with increased frequency in patients with A-V nodal dysfunction. In a study group of Thapar and Gillette, dual A-V nodal pathways were equally prevalent in children with corrected and uncorrected congenital cardiac defects. Their data suggested that intracardiac surgical procedures did not alter the frequency of dual A-V nodal pathways.

Twenty-eight of the 45 patients (62%) with dual A-V nodal pathways in our study had one or more electrophysiological abnormalities suggesting A-V nodal dysfunction (prolonged conduction times or refractory periods). In our 26 with first degree and/or second degree A-V (AH) block, ten patients (38%) had the evidence of dual A-V nodal pathways. Our results showed a strong association between dual A-V nodal pathways and A-V nodal dysfunction. However, in our study group, no patients had etiological factors directly associated with A-V nodal injury such as previous diaphragmatic myocardial infarction or intracardiac surgery. Moreover, in most of our patients, the administration of atropine was effective to improve electrophysiologically findings of A-V nodal dysfunction. These results suggest that most etiology of A-V nodal dysfunction in our study group may be due to functional causes under autonomic nervous influences. Although, dual A-V nodal pathways may be due to either functional longitudinal dissociation or anatomic septation of the A-V node, it is possible to consider that, in most patients, functional longitudinal dissociation is responsible for division of the A-V node into two pathways, with and without underlying pathological lesions of the A-V node.

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