Characterization of Atrioventricular Nodal Reentry With Continuous Atrioventricular Node Conduction Curve by Double Atrial Extrastimulation

Chi-Tai Kuo, MD; Kuo-Hung Lin, MD; Nye-Jan Cheng, MD; Po-Hsien Chu, MD; Tsu-Shiu Hsu, MD; Cheng-Wen Chiang, MD; Ying-Shiung Lee, MD

Background—Characterization of typical atrioventricular nodal reentrant tachycardia (AVNRT) with continuous AVN conduction (A1A2/A2H2) curves by double atrial extrastimulation (A1A2A3) has never been systematically studied.

Methods and Results—This study was composed of 33 patients with typical AVNRT and continuous AVN conduction curves (group 1) and 103 patients with AVNRT and discontinuous AVN conduction curves (group 2). Using A1A2A3 with predefined fast pathway–conducted A2, we examined the effects of slow pathway ablation on the A2A3/A3H3 curves in both groups. In group 1, anterograde AVN effective refractory period (272±33 versus 277±47 ms, P>0.05) and AVN Wenckebach block cycle length (320±45 versus 343±59 ms, P>0.05) remained unchanged after ablation. A2H3,max was shorter in group 1 than group 2 (237±89 versus 395±72 ms, P<0.05) at baseline. It shortened in group 2 (395±72 versus 221±78 ms, P<0.001) but remained unchanged in group 1 (237±89 versus 214±59 ms, P>0.05) after ablation. A2A3 could further disclose discontinuous A2A3/A3H3 curves in 29 patients of group 1. A2H3,max shortened in both groups (375±81 versus 238±82 ms, P<0.001, and 419±104 versus 220±78 ms, P<0.001, respectively) in a similar fashion. Successful ablation resulted in loss of the left portion of the A2A3/A3H3 curves in the 4 patients of group 1 with continuous A2A3/A3H3 curves.

Conclusions—Use of A1A2A3 could expose discontinuous A2A3/A3H3 curves in most patients with continuous A2A3/A3H3 curves. Significant shortening of A2H3,max after ablation may be indicative of successful elimination of AVNRT.

Key Words: atrioventricular node • catheter ablation • tachycardia • electrophysiology

There is abundant evidence of a discontinuity (“jump”) in the atrioventricular node (AVN) conduction (A1A2/A2H2) curves that do not produce AV nodal reentrant tachycardia (AVNRT) and in AVNRT not associated with such jumps.1–3 Dual inputs “attached” to corresponding functionally different nodal pathways can explain the occurrence of sudden jumps in the AVN conduction curve.4–6 Using single atrial extrastimulation (A0A1A2), others2,7 have suggested that smooth A1A2/A2H2 curves might consist of 2 distinct components representing both fast and slow pathways, even when typical discontinuity is absent. Failure to show a discontinuous A1A2/H2 curve in AVNRT may be related to a “less than a jump” difference between slow pathway and fast pathway conduction.8–10 Sheahan et al2 showed that conventional slow pathway ablation in patients with otherwise typical AVNRT without evidence of dual pathways results in a clinical cure and loss of the terminal portion of the A1A2/A2H2 curve. However, characterization of AVNRT with a continuous A1A2/A2H2 curve by double atrial extrastimulation (A1A2A3) has never been systematically studied. Thus, the purpose of the present study was to define the therapeutic end points for successful slow pathway ablation in patients with a continuous AVN conduction curve by A1A2A3.

Methods

Patients

The study population consisted of 2 groups of patients. All patients underwent electrophysiological study and radiofrequency ablation at Chang Gung Memorial Hospital for typical AVNRT. Group 1 included 33 patients without clear-cut discontinuity of the AVN conduction curves defined by single atrial extrastimulation. Group 2 included 103 patients with discontinuous AVN conduction curves.

Electrophysiological Study

Informed consent for the study and ablation was obtained from all patients. Each patient was studied in the postabsorptive state without sedation. All antiarrhythmic drugs were discontinued for at least 5 half-lives before the study.11,12 Multipolar electrode catheters were positioned in the coronary sinus, high right atrium. His bundle, and right ventricular apex. The decapolar electrode catheter in the coronary sinus had its most proximal electrode pair positioned just inside the ostium. Intracardiac electrograms from the high right atrium, His bundle, and coronary sinus electrodes, along with ECG leads I, aVF, and V1, were continuously recorded (EP Laboratory, EP Labware, Boston, MA).
Biomedical Instrumentation, Inc and Catheterization 2000 Crossover System, Gould, Inc). Programmed electrical stimulation (model DTU-215, Bloom Associates, Ltd) was delivered by use of 2-ms rectangular impulses at twice the late diastolic pacing threshold. All intracardiac electrograms were bandpass-filtered between 40 and 500 Hz. Briefly, the stimulation protocol consisted of high right atrial and right ventricular incremental pacing to block and extrastimulus testing with at least 2 driven cycle lengths (usually 600 and 400 ms). The initiation of typical AVNRT was generally associated with a discontinuous A2/AH curve.6,11,12 A2A3 was then performed by use of predetermined fast pathway–conducted A3 (A3 usually at 50 to 100 ms greater than the effective refractory period [ERP] of AVN or fast pathway with an AH <180 ms), namely fast pathway–coupled A2A3 in both groups. We always checked the atrial-His bundle (AH) during induction of single AVN echo or induction and maintenance of the AVNRT to determine that the predefined A1 and thus A1H did not fall into the slow pathway range. The fast of 2 premature beats was called the conditioning beat. The test beats, labeled A1, were introduced with progressively shorter A2A3, starting from 600 ms in steps of 10 ms, until the ERP of AVN or the atrium was reached in each stimulating cycle. The A2A3 was then constructed. The examination of a given patient was performed with stimulation from the same pacing sites during different protocols.

**Endocardial Mapping and Radiofrequency Ablation**

All patients underwent ablation through the posterior approach.6,13–17 Three zones within the triangle of Koch along the tricuspid annulus were arbitrarily defined, including the anterior third, middle third, and posterior third regions between the His-bundle recording site and coronary sinus ostium. These regions were further divided into 2 subsections, the anterior-2 (A2) and anterior-1 (A1), middle-2 (M2) and middle-1 (M1), and posterior-2 (P2) and posterior-1 (P1), respectively. The orifice of the coronary sinus was demarcated by coronary sinus venography. A 7F quadrupolar deflectable catheter (Mansfield Scientific) with a 4-mm tip electrode was used for mapping and ablation. A radiofrequency generator (RFG-3C, Radioc- onics) was used to deliver energy at a power setting of 30 W for 30 seconds during each attempt. Application of energy was interrupted if junctional tachycardia did not appear within 10 seconds or if impedance rise, PR prolongation, or AVN block occurred. Lesions were basically anatomically guided and directed to the posterior, then the middle, and finally to the anterior area if necessary. The presumed ablation site was considered optimal if the bipolar electrograms recorded from the distal electrodes showed an atrial-to-ventricular electrogram amplitude ratio of 0.1 to 0.5 (usually ≤0.25).6,11,13 After each application of energy, the presence or absence of slow pathway conduction and inducibility of AVNRT was assessed with programmed electrical stimulation. The end point of a successful ablation was defined as noninducibility of AVNRT with isoproterenol infusion at (graded doses from 1 to 4 µg/min IV) and/or atropine (0.01 to 0.02 mg/kg IV), even though the residual antegrade slow pathway might be present6,11,13,14 without or with a single AVN echo.

**Postablation Electrophysiological Evaluation**

All patients underwent repeat testing with single and double atrial extrastimuli before and during the administration of isoproterenol and/or atropine, 30 minutes after successful ablation, and during a later follow-up study at 3 months. All parameters were measured on the 3 occasions. Each time, measurements were done during the baseline states.

**Definitions**

Dual pathway physiology was defined as discontinuous AVN conduction curves during single atrial extrastimulation. It was characterized by a ≤50-ms jump in AH at a critical range of A2 coupling intervals (10-ms decrease) during 2 different paced cycle lengths,6,11,14 resulting in a discontinuity between the curve to the right of the jump in AH (fast pathway) and the portion with the jump (slow pathway). The ERP of the fast pathway was defined on the basis of discontinuous A2A3 curves. The ERP of the AVN was defined as the longest A1A3 that failed to result in an H1-H2 response. In patients with discontinuous A2A3 curves, the AVN ERP therefore reflects the ERP of the slow pathway; in those with continuous A2A3 curves, the AVN ERP refers to the shortest ERP. For each patient, the driven cycle lengths and the coupling intervals of the A1A3 at which ERP measurements and A2A3 curves were obtained before ablation were matched and repeated after ablation. The A1Hmax and A2Hmax were defined as the maximal AH measured during A1A3 and A2A3A3, respectively.

**Statistical Analysis**

Data were expressed as mean±SD. A repeated-measures analysis was applied to compare the continuous variables among 3 consecutive data points before and after ablation. Multiple-comparison analyses were performed to test the significance of continuous variables between 2 different groups. A χ2 test with Yates’ correction or Fisher’s exact test was used to compare the categorical data, and a Student’s t test was performed to compare continuous variables between groups. A value of P<0.05 was considered statistically significant.

**Results**

**Baseline**

Group 1 (continuous A2A3 curve) included 33 patients, 22 women and 11 men, 55±13 years old. Group 2 (discontinuous A2A3 curve) included 103 patients, 60 women and 43 men, 48±15 years old. The 2 groups did not differ with respect to age, sex, AH interval, the AVN ERP, and retrograde AVN Wenkebach block cycle length (WCL) at the baseline. The cycle length of the induced tachycardias did not differ between groups 1 and 2. The Table summarizes the electrophysiological properties of the AVN in the 2 groups before and after ablation. Details of each patient are shown in Figure 1.

**Effects of Ablation With the Posterior Approach on AVN Conduction in Group 1**

Sustained AVNRT of the slow-fast form was induced in all patients before ablation. Isoproterenol infusion was required for its initiation in 12 patients (36%). Definite evidence of dual AVN physiology as shown by discontinuity of the A1A2A3 curve was not present in all patients before ablation.

The effects of ablation with the posterior approach on the refractory period and conduction properties of the AVN are shown in the Table. The sinus cycle length (714±191 versus 688±118 ms), AH (77±18 versus 78±15 ms), AVNCL (320±45 versus 343±59 ms), and retrograde AVNCL (369±125 versus 375±115 ms) remained unchanged after ablation (P>0.05). Of note, the antegrade AVN ERP (272±33 versus 277±47 ms, P>0.05) also remained unchanged after ablation. A1Hmax showed little change after ablation (237±89 versus 214±59 ms, P>0.05). Using a predetermined fast pathway–conducted A1A2A3, was able to further disclose discontinuous A1A2A3 curves with an AH jump (≥50 ms) in 29 patients (Figures 2 and 3). Successful ablation resulted in the loss or marked diminution (Figures 2B and 3B) of the discontinuous A1A2A3 curve on the left in the former. A lack of discontinuity in the A1A3/
The ERP of the slow pathway increased (274 ± 19 ms) during both A1A2 and A1A2A3. Atrial burst pacing and isoproterenol were required for the induction of AVNRT in 3 of these patients before ablation. The atrial wavefronts were either terminated or deflected around the slow pathway during both A1A2 and A1A2A3.

A3H3 curve was seen during 1 A2A3 study in the other 4 patients of group 1 (Figure 4), in whom ablation resulted in the loss of the terminal portion of the A3H3 curve on the left. Atrial burst pacing and isoproterenol were required for the initiation of AVNRT in 3 of these patients. One other patient required A1A2A3 to initiate AVNRT. The only parameter that clearly demonstrated significant change after ablation was the A3H3max obtained during A1A2A3 (375 ± 81 ms versus 238 ± 82 ms, P < 0.0001).

**Comparison of Groups 1 and 2**

Before ablation, the sinus cycle length, the anterograde AVNWCL, and the A2H2max and A3H3max showed significant differences (P < 0.05) between groups 1 and 2 (Figure 1). Immediately after ablation, the sinus cycle length, AVN ERP, and AVNWCL showed significant differences (P < 0.05) between the 2 groups.

**Late Electrophysiological Follow-Up at 3 Months or Later**

At late follow-up, the sinus cycle length, anterograde AVNWCL, and AVN ERP increased from immediate postablation studies in a similar trend (Figure 5) in both groups (Table). In group 2, the ERP of the slow pathway increased immediately after ablation (274 ± 45 ms versus 316 ± 72 ms, P = 0.0001) and increased further (368 ± 78 ms, P = 0.0001) at 3-month follow-up. Conversely, the ERP of the fast pathway in group 2 shortened (366 ± 82 ms versus 339 ± 73 ms, P < 0.0001) immediately after ablation but increased in the long run (406 ± 100 ms, P = 0.0001) during the late follow-up.

The baseline AH, retrograde AVNWCL, A2H2max, and A3H3max remained unchanged between the 2 postablation studies in both groups. As a whole, there were significant differences in the tendency of the series of changes in the electrophysiological variables, including the sinus cycle length, AVN ERP, anterograde AVNWCL, and A2H2max among 3 measurements between the 2 groups (P < 0.05, adjusted by age and time).

**Discussion**

**Major Findings**

In those with discontinuous AVN function curves during A1A2 (group 2), the anterograde AVN ERP increased. The ERP of the residual slow pathway increased, whereas that of
the fast pathway decreased,\textsuperscript{2,14} thus narrowing the slow pathway window after ablation. In those without manifestation of discontinuous curves during A1A2 (group 1), the anterograde AVN ERP and WCL remained unchanged after ablation. The A2H2max was shorter in group 1 than group 2 at baseline. After ablation, A2H2max shortened in group 2 but remained unchanged in group 1. With a predetermined fast pathway–conducted A2, A1A2A3 could further disclose discontinuous A2A3/H3 curves in 29 patients of group 1. The A3H3max decreased in a parallel fashion after ablation in those with and without discontinuous A1A2/H2 curves. Successful ablation resulted in the loss of the tail of the A2A3/H3 curve in the 4 patients with a continuous A1A2/H2 curve.

A strong correlation between the sinus cycle length, anterograde AVNWCL, and AVN ERP was noted between immediate postablation and late studies for both groups.

Mechanisms of Failure to Demonstrate a Distinct Discontinuity

Several potential mechanisms may account for the failure to demonstrate distinct discontinuity in the AVN conduction (A1A2/A3H2) curves in otherwise typical AVNRT. Differences in the refractory periods and conduction properties between the fast and slow pathways may not be sufficiently distinct at baseline study to yield discontinuity in the A1A2/A3H2 curve.\textsuperscript{1,2,7} It is well known in the electrophysiology laboratory that discontinuous AVN conduction curves may become continuous during isoproterenol infusion,\textsuperscript{3,15–20} because the refractory period of the fast pathway could be abbreviated to the extent that the slow pathway conduction could not be achieved with single or even double atrial extrastimuli or with incremental atrial pacing.

In group 1 patients, A1A2A3 failed to expose the discontinuity of the A2A3/H3 curve in a minority of patients. Successful ablation with the posterior approach in these patients resulted in the loss of the tail of the A3H3 curve representing the slow pathway portion of the conduction curve. However, one cannot conclude from this
result that the ablation eliminated the slow pathway. It is quite logical to say that the ablation may have now rendered the slow pathway not detectable in the A2 A3/H3 format as well. In other words, the elimination of the AVNRT may not necessarily be equaled with elimination of the slow pathway; thus the dual pathway electrophysiology. These findings suggest that the smooth A2 A3/H3 curve might in fact consist of 2 distinct components, which may be linked to one or the other pathway, respectively. Atrial burst pacing and isoproterenol were required for the initiation of AVNRT in 3 of these patients. One other patient required A1 A2 A3 to initiate AVNRT. The slow pathway conduction could be achieved in these circumstances to set up for reentry to occur. However, it was difficult to draw any conclusions on the electrophysiological characteristics of the AVN in such a small number of patients.

The anterograde AVN ERP, AVNWCL, and A3H3max remained unchanged in group 1. A3H3max was the only electrophysiological parameter that shortened significantly after successful ablation. To facilitate the ablation procedure, it may be much easier to use A1A2A3 as defined in the report to establish an acceptable therapeutic end point, ie, A3H3max may be a better indicator than A1H3max in determining and confirming the success of the ablation.

Lessons Learned From Ablation

This study further supports the hypothesis that use of double atrial extrastimulation could reveal distinct discontinuity in the A1A2/A2H2 curves in most patients with smooth A1A2/A2H2 curves. Of note, loss of the terminal portion of the A2A3/A3H3 curves (to the left of the discontinuity) representing slow pathway conduction occurred after the elimination of the AVNRT, leaving a continuous A2A3/A3H3 curve. At times, the “slow pathway” zone was only modified, leaving a discontinuous A2A3/A3H3 curve after elimination of the inducibility of AVNRT. Nonetheless, A3H3max always shortened in a significant magnitude. In those who still failed to show discontinuous A2A3/A3H3 curves during A1A2A3, successful ablation always resulted in the loss or marked diminution of the terminal portion of the curve on the left, which may be linked to the slow pathway component.

A3H3max decreased in a parallel fashion after successful ablation in both groups. Both A3H3max and A1H3max were shorter in those with smooth A1A2/A2H2 curves, implying a “less decremental” slow pathway, as suggested by previous studies.2,7 The data suggest that the use of the A1 could increase the difference in conduction time of the fast and slow pathways, although the A1A2/A2H2 curves remained continuous in some patients.
Limitations
In this study, we did not use autonomic blockade to control for autonomic tone. However, autonomic blockade has been shown not to affect the observed changes in postablation refractoriness.\textsuperscript{19} In our A\textsubscript{1} A\textsubscript{2} A\textsubscript{3} study, it might be true that neither “A\textsubscript{2} usually at 50 to 100 ms greater than the ERP of AVN or fast pathway” nor “A\textsubscript{2} H\textsubscript{2} \textless 180 ms” is an indicator of fast pathway conduction. Although we always checked the AH during induction of single AVN echo, induction and maintenance of the AVNRT to determine that the predefined A\textsubscript{2} and thus A\textsubscript{2} H\textsubscript{2} did not fall into the slow pathway range. In fact, the predefined A\textsubscript{2} H\textsubscript{2} was most often \textless 150 ms. However, this would not guarantee that slow pathway–coupled A\textsubscript{2} did not occur.

Another potential limitation is the choice of the A\textsubscript{2}, which may be arbitrary in such a way that A\textsubscript{2} H\textsubscript{max} may not really be the longest attainable interval during A\textsubscript{1} A\textsubscript{2} A\textsubscript{3}. It is also likely that the longest attainable AH interval, AHmax, could be obtained during atrial burst pacing instead of atrial extrastimulation.

We did not routinely use a third drive cycle length to expose discontinuity of the A\textsubscript{1} A\textsubscript{2} H\textsubscript{2} and/or A\textsubscript{2} A\textsubscript{3} H\textsubscript{3} curves. It is possible that use of various atrial sites,\textsuperscript{16, 3} or more extrastimuli, or pharmacological intervention could have revealed discontinuity in some of the patients with smooth AVN curves.

Clinical Implications
It has been well established that total elimination or modification of the slow pathway, to the extent that repetitive AVN reentry cannot be induced, is an acceptable therapeutic end point that portends a good prognosis.\textsuperscript{6, 13, 21–24} However, the end point is less distinct when the AVN conduction curves do not demonstrate a classic jump at the transition from the fast to slow pathway conduction. After successful ablation, A\textsubscript{2} H\textsubscript{max} might shorten significantly in group I patients, as demonstrated by Tai et al\textsuperscript{7} and Sheahan et al.\textsuperscript{2} In this study, however, the postablation A\textsubscript{2} H\textsubscript{max} did reveal a trend to decrease, although the difference was noted to be statistically insignificant. The anterograde AVN ERP and AVNWCL also remained unchanged after ablation in this group. Because...
A\textsubscript{H}max is the only electrophysiological parameter that shortened in a greater magnitude, it may be shown that A\textsubscript{H}max is a good (if not better) indicator compared with A\textsubscript{H}max in portending a successful outcome for ablation. It is hoped that this approach will facilitate the ablation procedure in patients with smooth A\textsubscript{A}/A\textsubscript{H}\textsubscript{2} curves who otherwise have typical AVNRT. Finally, the study suggests that a decrease of \( \geq 100 \) to 150 ms in A\textsubscript{H}max may be indicative of clinical success. This merits further study in a large patient population.

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