**Effects of Cavotricuspid Isthmus Ablation on Atrioventricular Node Electrophysiology in Patients With Typical Atrial Flutter**

Ching-Tai Tai, MD; Chin-Feng Tsai, MD; Ming-Hsiung Hsieh, MD; Wei-Shiang Lin, MD; Yung-Kuo Lin, MD; Shih-Huang Lee, MD; Wen-Chung Yu, MD; Yu-An Ding, MD, PhD; Mau-Song Chang, MD; Shih-Ann Chen, MD

**Background**—The atrial musculature in the cavotricuspid isthmus is a part of posterior inputs to the AV node. In patients with typical atrial flutter, effects of radiofrequency ablation of this isthmus on AV node conduction are still unknown.

**Methods and Results**—This study included 16 patients with clinically documented typical atrial flutter. Group 1 had 8 patients without and group 2 had 8 patients with dual AV nodal pathway physiology. Electrical pacing from the interatrial septum and low right atrium was performed to evaluate antegrade AV node function before and after ablation of the cavotricuspid isthmus. In group 1, the AV node conduction properties were similar before and after ablation. In group 2, the AV node Wenckebach cycle length and maximal AH interval during low right atrium (356±58 versus 399±49 ms, P=0.008; 303±57 versus 376±50 ms, P=0.008) and interatrial septum (365±62 versus 393±59 ms, P=0.008; 324±52 versus 390±60 ms, P=0.008) pacing were significantly longer after ablation. Elimination of the slow pathway after ablation was noted in 2 patients, including 1 with AV nodal reentrant echo beats.

**Conclusions**—Radiofrequency ablation of the cavotricuspid isthmus was effective in eliminating typical atrial flutter without injury of antegrade fast AV node conduction. The atrial musculature in the cavotricuspid isthmus significantly contributed to the slow AV node conduction. (Circulation. 2001;104:1501-1505.)

**Key Words:** atrial flutter ■ atrioventricular node ■ catheter ablation ■ conduction

**Methods**

**Patients**

The study population consisted of 16 patients with clinically documented typical atrial flutter. All patients were referred to receive electrophysiological study and radiofrequency catheter ablation in this institution. Group 1 included 8 patients (age, 61±21 years; 4 men and 4 women) with a continuous AV node conduction curve; 2 patients had hypertensive cardiovascular disease, and 1 patient had coronary artery disease. Group 2 included 8 patients (age, 60±21 years; 5 men and 3 women) with a discontinuous AV node conduction curve; 1 patient had mitral valve prolapse with mild mitral regurgitation, and 1 patient had coronary artery disease.

**Baseline Electrophysiological Study**

As described previously, each patient underwent a baseline electrophysiological study in the fasting, unsedated state ≥5 half-lives after discontinuation of antiarrhythmic drugs. A signed consent form for the study and ablation was obtained from each patient. One deflectable, 20-pole “halo” catheter (Cordis-Webster) was placed around the tricuspid annulus to record the activation of the lateral wall and low atrial isthmus simultaneously (Figure 1). Three multipolar, closely spaced (2-mm interelectrode spacing) electrode catheters (Boston Scientific Inc, Mansfield Division) were introduced from the right and left femoral veins and placed in the interatrial septum (IAS), low right atrium (LRA) near the crista terminalis, and...
His bundle area for programmed electrical stimulation and recording (Figure 1). One decapolar electrode catheter (Daig Corp) was introduced from the right internal jugular vein and placed in the coronary sinus with the proximal electrode pair just at the ostium. Intracardiac electrograms were displayed simultaneously with ECG leads I, II, and V1 on a multichannel oscilloscopic recorder (Prucka Engineering) and were recorded on paper at a speed of 100 to 200 mm/s. The filter was set from 30 to 500 Hz. A programmed digital stimulator (DTU-210 or 215, Bloom Associates Ltd) was used to deliver 2.0-ms-long electrical impulses at approximately twice the diastolic threshold.

Electrical pacing from the IAS and LRA was performed to evaluate antegrade AV node function, including the Wenckebach cycle length, effective refractory period (ERP), and maximal atrio-His (AH) interval. The methods of atrial flutter induction included (1) baseline pacing (8-beat drive, twice the diastolic threshold) with single and double premature stimuli at 3 different cycle lengths (600, 500, and 400 ms) and (2) burst pacing (>20 beats, twice the diastolic threshold or up to 10 mA) at progressively shorter cycle lengths until 2:1 atrial capture from the LRA and coronary sinus ostium.

Radiofrequency Catheter Ablation

As described previously, radiofrequency energy was delivered with a thermistor ablation catheter with a 4-mm distal electrode (7F, EP Technologies, Inc.) to achieve a tip-tissue interface temperature of 70° during sinus rhythm with pacing from the coronary sinus ostium. The preset duration of each radiofrequency pulse was 120 seconds. Continuous application of radiofrequency energy during pullback of the ablation catheter from the right ventricle toward the atrial septum was used to create linear lesions of the cavitricuspid isthmus. Successful ablation was defined as achievement of bidirectional isthmus conduction block without induction of typical atrial flutter.

After successful ablation of atrial flutter, all patients were observed in the electrophysiological laboratory for >20 minutes, and electrophysiological study was repeated to ensure complete bidirectional isthmus conduction block. Electrical pacing from the IAS and LRA was repeated to evaluate antegrade AV node function.

Follow-Up

After hospital discharge, all patients were followed up closely and came back to the outpatient clinic in the second week, first month, and second month after ablation, and then every 3 months. Long-term efficacy was assessed clinically on the basis of the resting surface ECG, 24-hour Holter monitoring, and clinical symptoms.

Definitions

Antegrade AV node conduction curves were drawn from the results of programmed atrial extrastimulus testing. Dual-pathway physiology was characterized by a jump (>50 ms) in H1-H2 or H2-H3 at a critical range of A2-A3 or A3-H3 coupling intervals (10-ms decrease) that resulted in a discontinuity between the portion of the curve to the right of the jump in H1-H2 or H2-H3 (fast pathway conduction) and the portion of the curve to the left of the jump (slow pathway conduction). Comparable discontinuous A1-A2 versus A2-H3 curves were also demonstrated. Continuous AV node conduction curves were defined as those without a jump of H1-H2 or A1-H2 at all ranges of A2-A3 coupling intervals. The maximal AH interval was defined as the longest AH interval measured during extrastimulus testing.

Typical atrial flutter was defined as atrial flutter exhibiting either a counterclockwise or a clockwise activation around the cavitricuspid annulus in the right atrium. Counterclockwise atrial flutter was defined as atrial flutter with craniocaudal activation of the anterior and lateral walls of the right atrium and caudocranial activation of the atrial septum, inverted P waves in the inferior leads, and positive P wave in lead V1. Clockwise atrial flutter was defined as atrial flutter with a similar flutter cycle length and reverse activation sequence of the counterclockwise flutter. Complete isthmus conduction block was defined by the observation of a purely descending wave front at the lateral wall down to the low right atrial isthmus when pacing from the coronary sinus ostium at 600-ms cycle length and observation of a purely descending wave front at the septal wall to reach the coronary sinus ostium, resulting in activation of the His bundle region preceding the coronary sinus ostium when pacing from the LRA at 600-ms cycle length.

Statistical Analysis

All data are expressed as mean±SD. The Wilcoxon signed-rank test was used to compare the continuous data before and after ablation of the cavitricuspid isthmus. A value of P<0.05 was considered to be statistically significant.

Results

Radiofrequency Ablation and Follow-Up

Two patients had clockwise atrial flutter, and the other 14 patients had counterclockwise atrial flutter. All 16 patients underwent successful ablation of typical atrial flutter with creation of complete bidirectional isthmus conduction block. There was no significant difference in the number of energy application between group 1 and group 2 patients (2.3±1.4 versus 2.8±1.8, P>0.05). During the follow-up period of 7.2±1.7 months, 15 patients maintained sinus rhythm, and 1 patient had occurrence of atrial fibrillation.

Group 1 Patients

The sinus cycle length (670±80 versus 679±120 ms), AH interval (72±17 versus 70±14 ms) during sinus rhythm, AH interval during atrial pacing at 500-ms cycle length (83±21 versus 82±19 ms and 83±19 versus 80±19 ms during LRA and IAS pacing, respectively), AV node Wenckebach cycle length (311±51 versus 311±44 ms and 321±50 versus 311±47 ms during LRA and IAS pacing, respectively), and maximal AH interval (171±28 versus 171±26 ms and 156±19 versus 156±22 ms during LRA and IAS pacing, respectively) were similar before and after radiofrequency ablation of the cavitricuspid isthmus (Figures 2 and 3 and the Table).

Group 2 Patients

One patient had an AV nodal reentrant echo beat during extrastimulation testing before ablation. The sinus cycle length (729±161 versus 739±148 ms), AH interval (90±18 versus 93±13 ms) during sinus rhythm, AH interval during
atrial pacing at 500-ms cycle length (112±34 versus 114±35 ms and 111±33 versus 109±32 ms during LRA and IAS pacing, respectively), antegrade fast pathway ERP (343±76 versus 342±62 ms and 351±76 versus 339±66 ms during LRA and IAS pacing, respectively), and antegrade slow pathway ERP (297±61 versus 316±66 ms and 308±70 versus 311±68 ms during LRA and IAS pacing, respectively) were similar before and after radiofrequency ablation of the cavotricuspid isthmus (Figure 4 and the Table). However, the AV node Wenckebach cycle length (356±58 versus 399±49 ms and 365±62 versus 393±59 ms during LRA and IAS pacing, respectively) and maximal AH interval (303±57 versus 376±50 ms and 324±52 versus 390±60 ms during LRA and IAS pacing, respectively) were significantly longer after ablation of the cavotricuspid isthmus (Figures 5 and 6 and the Table). Elimination of the slow pathway after ablation was noted in 2 patients, including 1 with AV nodal reentrant echo beats.

**Discussion**

**Major Findings**

In the present study, we first demonstrated that ablation of the cavotricuspid isthmus eliminated the slow AV nodal pathway or significantly prolonged the AV node Wenckebach cycle length and maximal AH interval during atrial pacing at 500-ms cycle length; AVNWCL, AV node Wenckebach cycle length; FP, fast pathway; and SP, slow pathway. All values are expressed in milliseconds and are mean±SD.

**Relationship Between Atrial Inputs and Dual AV Nodal Pathways**

In the present study, ablation of the cavotricuspid isthmus eliminated only the slow AV nodal pathway or significantly prolonged the AV node Wenckebach cycle length and maximal AH interval in patients with dual AV nodal pathway physiology, but it did not influence the AV node conduction and refractoriness in patients with a continuous AV node function curve. This finding suggested that the atrial musculature in the cavotricuspid isthmus plays an important role in the slow pathway conduction.

**Figure 2.** AV node Wenckebach cycle length (AVNWCL; A) and maximal AH interval (AHmax; B) during atrial pacing at LRA and IAS before and after ablation of cavotricuspid isthmus in group 1 patients.

**Figure 3.** ERP of fast pathway (FP) before and after ablation of cavotricuspid isthmus in group 1 patients.
coronary sinus before entering the posterior aspect of the AV node. Clinical and experimental evidence also suggests that the dual AV nodal pathways have different sites of atrial input: The fast pathway appears to have a more anterior septal input, whereas the slow pathway appears to have a posterior atrial input. Furthermore, high-resolution mapping of the perinodal region in humans with AV nodal reentrant tachycardia demonstrated higher conduction velocities in the anterior interatrial septum than in the posteroseptal region.

Effects of Cavotricuspid Isthmus Ablation on AV Nodal Conduction
In the present study, ablation of the cavotricuspid isthmus significantly prolonged the AV node Wenckebach cycle length and maximal AH interval in patients with dual AV nodal pathways but not in patients without dual AV nodal pathways. Alteration of the slow AV nodal pathway conduction was the underlying mechanism. Spach and Josephson demonstrated that the transitional zone of the AV node has marked nonuniform anisotropic properties and therefore could provide a mechanism for the slow and fast pathway characteristics of the AV junction. Hocini et al also showed anisotropic conduction in the triangle of Koch of mammalian hearts. Stimulation from the anterior and posterior sites of the tricuspid valve annulus and from the high right atrium resulted in rapid conduction parallel to the alignment of fibers; stimulation from the oval fossa and from sites near the orifice of the coronary sinus led to narrow zones of slow conduction in the posterior part of Koch’s triangle. On the basis of a recent human study by Sanchez-Quintana et al, the atrial musculature in the cavotricuspid isthmus was a main part of the posteroinferior fibers toward the AV node. It is possible that ablation of this isthmus changes the direction of wave-front propagation relative to the orientation of muscle fibers.
fibers in the transition zone and increases conduction time in the slow AV nodal pathway. Furthermore, several investigators have demonstrated that the relative timing of activation at the major input sites of the AV node is critical to patterns of AV nodal conduction. Thus, alteration of the activation pattern in the atrial inputs after ablation of the cavotricuspid isthmus resulted in inhomogeneous conduction in the AV node, which contributed to conduction delay or block in the slow pathway.

**Study Limitations**

Because this study included only patients without AV nodal reentrant tachycardia, effects of the cavotricuspid isthmus ablation on dual AV nodal pathway physiology in patients with AV nodal reentrant tachycardia were unknown. Although the mean AV node Wenckebach cycle length and maximal AH interval did not change significantly after ablation of the cavotricuspid isthmus in group 1 patients, the individual result as shown in Figure 2 was not consistent. This finding might be partly explained by the alteration of the autonomic tone after ablation of the cavotricuspid isthmus. The epicardial fat pad located between the coronary sinus ostium and inferior vena cava contains the parasympathetic ganglia and nerve fibers; these postsynaptic fibers selectively innervate the AV node. Electric stimulation of this area has been demonstrated to induce negative dromotropic effects in humans and animals. Thus, the radiofrequency energy applied in the cavotricuspid isthmus might cause variable thermal effects on the autonomic nervous activity and lead to the variable change in the AV node conduction. However, the consistent changes in the AV node Wenckebach cycle length and maximal AH interval in group 2 patients (Figure 6) suggested that the influence of the autonomic change induced by ablation on the AV node conduction might be slight.

**Conclusions**

Radiofrequency catheter ablation of the cavotricuspid isthmus was effective in eliminating typical atrial flutter without injury of antegrade fast AV node conduction. Prolongation of the AV node Wenckebach cycle length and maximal AH interval in patients with dual AV nodal pathway physiology provided evidence that the atrial musculature in the cavotricuspid isthmus significantly contributed to the slow AV node conduction.

**Acknowledgments**

This work was supported in part by grants from the National Science Council (NSC 88-2314-B-075-090, 88-2314-B-010-093, 88-2314-B-010-094) and Tsou’s Foundation (VGHYM-S4-30, VGHYM-S4-31), Taipei, Taiwan, ROC.

**References**

Effects of Cavotricuspid Isthmus Ablation on Atrioventricular Node Electrophysiology in Patients With Typical Atrial Flutter
Ching-Tai Tai, Chin-Feng Tsai, Ming-Hsiung Hsieh, Wei-Shiang Lin, Yung-Kuo Lin, Shih-Huang Lee, Wen-Chung Yu, Yu-An Ding, Mau-Song Chang and Shih-Ann Chen

_Circulation._ 2001;104:1501-1505
doi: 10.1161/hc3801.078813

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2001 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/104/13/1501

**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/