Assessment of Atrioventricular Nodal Electrophysiological Characteristics After Radiofrequency Catheter Ablation of the Slow Pathway in Atrioventricular Nodal Reentrant Tachycardia — 3-Month Follow Up —

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Radiofrequency catheter ablation of the slow pathway is commonly used to treat atrioventricular (AV) nodal reentrant tachycardia. However, there has been little study of the follow-up assessment of AV nodal physiology. We compared AV nodal electrophysiological characteristics before, immediately after, and again 3 months after successful catheter ablation in 17 patients (mean age 50 ± 16 years). Sinus cycle length, Wenckebach cycle length, A-H interval at a paced cycle length of 600 ms, effective refractory period and functional refractory period of the fast pathway were significantly changed immediately after catheter ablation, but had recovered 3 months after the procedure. There were no significant differences between the electrophysiological parameters immediately after catheter ablation and those 3 months after the procedure under the intravenous injection of atropine sulfate.

We conclude that, due to changes in autonomic nervous tone, AV nodal electrophysiological characteristics are influenced immediately after catheter ablation of the slow pathway in AV nodal reentrant tachycardia.

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ATRIOVENTRICULAR (AV) nodal reentrant tachycardia is a common cause of paroxysmal supraventricular tachycardia.^{1,2} Radiofrequency catheter ablation of the slow pathway with AV nodal reentry, without disturbing AV nodal conduction, is often performed in patients who are refractory to antiarrhythmic drug therapy.^{3–7} We conducted a follow-up electrophysiological study to assess the AV nodal electrophysiological characteristics influenced by radiofrequency catheter ablation of the slow pathway.

Key words:

AV nodal reentrant tachycardia
Slow pathway
Radiofrequency catheter ablation

SUBJECTS AND METHODS

Patients

The patient characteristics are summarized in Table I. We studied 17 patients who had been diagnosed as having a common form of AV nodal reentrant tachycardia and who underwent successful radiofrequency catheter ablation of the slow pathway between August 1992 and December 1994. There were 3 males and 14 females (mean age 50 ± 16 years, range 27 to 73 years). None had structural heart disease. All were symptomatic and were either refractory to, or had experienced adverse effects from antiarrhythmic drug therapy. The duration

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TABLE I PATIENTS' CHARACTERISTICS

No.	Age	Sex	Duration (y)	Antiarrhythmic drugs	RF application time	Total energy (J)
1	49	F	3	V	4	2000
2	47	F	3	D	8	3585
3	66	F	9	V,D	2	1000
4	32	F	2	V	3	1300
5	41	F	10	D,A,C	3	1500
6	56	M	10	V	2	1000
7	66	M	18	V	3	1500
8	27	F	16	V,P	2	1000
9	38	F	17	V,C	4	1850
10	61	F	12	V	4	2000
11	39	F	3	D	4	1875
12	48	F	6	V,D	5	1925
13	57	F	20	D	9	4180
14	62	F	3	V	2	800
15	35	M	7	V	5	2250
16	73	F	25	V	6	2270
17	59	F	9	V	2	1000

V: verapamil D: disopyramide A: aprindine C: cibenzoline P: propranolol

TABLE II COMPARISON OF ELECTROPHYSIOLOGICAL PARAMETERS AMONG BEFORE, IMMEDIATELY AFTER AND 3 MONTHS AFTER SUCCESSFUL RADIOFREQUENCY CATHETER ABLATION OF SLOW PATHWAY WITH AV NODAL REENTRY

	Before	Immediately after	3 months after	3 months after (atropine)
Sinus cycle length (ms)	788±134	714±115	910±155	
	└─ P<0.05 ─┘	└─ P<0.01 ─┘		
Wenckebach cycle length of fast pathway (ms)	156±36	179±33	141±35	
	└─ P<0.05 ─┘	└─ P<0.05 ─┘		
A-H interval (BCL 600 ms) (ms)	110±19	97±18*	125±41	96±19*
	└─ P<0.01 ─┘	└─ P<0.01 ─┘	└─ P<0.01 ─┘	
ERP of fast pathway (ms)	357±61	267±54*	382±104	255±44*
	└─ P<0.01 ─┘	└─ P<0.01 ─┘	└─ P<0.01 ─┘	
FRP of fast pathway (ms)	428±59	378±48*	473±99	367±54*
	└─ P<0.01 ─┘	└─ P<0.01 ─┘	└─ P<0.01 ─┘	

ERP: Effective refractory period FRP: Functional refractory period *: NS

of the symptoms due to AV nodal reentry ranged from 2 to 25 years.

Electrophysiological Study

Written and verbal informed consent was obtained from each patient. An electrophysiological study was performed in each patient without sedation just before, immediately after, and 3 months after catheter ablation. All antiarrhythmic drugs were discontinued for at least 5 half-lives before each electrophysiological study. A 5F quadripolar electrode catheter (Vygon, Aachen, Germany) was introduced percutaneously into

the femoral vein and advanced to the high right atrium, a 6F hexapolar electrode catheter (Vygon, Aachen, Germany) was advanced to the His bundle region to record an intracardiac electrogram, and a 5F bipolar electrode catheter (Baxter, Irvine, CA, USA) was advanced to the right ventricular apex to stimulate the right ventricle. Another 5F quadripolar electrode catheter (Vygon, Aachen, Germany) was introduced into the right antecubital vein and positioned in the coronary sinus. A surface electrocardiogram (leads I, aVF, V1) and intracardiac electrograms were displayed on a multichan-

Fig 1. Intracardiac electrogram at the optimal site for radiofrequency catheter ablation of the slow pathway in patients with AV nodal reentrant tachycardia. A wide split atrial wave and slow pathway potential were recorded from the ablation catheter. Asterisks show a slow pathway potential. Abbreviations: Ab., intracardiac electrogram recorded from ablation catheter; CS, intracardiac electrogram recorded at the coronary sinus; HBE, His bundle electrogram;

Fig 2. Radiograms showing the position of the catheter for slow pathway ablation. Left panel: Right anterior oblique view. Right panel: Left anterior oblique view. The ablation catheter (Ab) was positioned posteroinferior to the site from which the His bundle electrogram (HBE) was recorded, and near the ostium of the coronary sinus.

nel oscilloscope and recorded on a thermal recorder (RF-80 Fukuda Denshi Inc, Tokyo, Japan). Electrical stimulation was provided by an electrical stimulator (SEC-3102 Nihon Kodens Inc, Tokyo, Japan). The electrical stimulation protocols were atrial and ventricular extrastimuli following an 8 beat drive train, and atrial and ventricular burst pacing.

Ablation Procedure

A deflectable 7F quadripolar electrode

catheter with a 4 mm distal large tip (Webster, Boston Scientific, Watertown, MA, USA) was introduced via the right femoral vein and positioned near the ostium of the coronary sinus, where the slow pathway potential and a wide or split atrial wave were recorded (Fig 1). The catheter was positioned posterior to and lower than the site from which the His bundle electrogram was recorded (Fig 2). Radiofrequency energy with a 500 kHz continuous unmodulated

current (NL-50-I, Central Kogyo, Chiba, Japan) was delivered at a setting of 20 to 25 watts for 20 s between the distal large-tip electrode and an adhesive electrosurgical dispersive pad on the left posterior chest wall. The delivery of radiofrequency energy was repeated until AV nodal reentrant tachycardia could no longer be induced. Criteria for successful ablation were 1) no AV nodal reentrant tachycardia induced by electrical stimuli. (1 echo beat was permitted) and 2) disappearance of the jump-up phenomenon in the AV conduction curve before radiofrequency ablation. Intravenous heparin was administered as an initial dose of 2000 U at the onset of the procedure, and then infused at 1000 U/h for the duration of the study.

Electrophysiological Follow-up

The following electrophysiological parameters were measured before, immediately after (10 to 15 min), and 3 months after successful radiofrequency catheter ablation: 1) sinus cycle length, 2) Wenckebach cycle length of the fast pathway, 3) A-H interval at a basic paced cycle length of 600 ms, 4) the effective refractory period of the antegrade fast pathway and 5) the functional refractory period of the fast pathway. The A-H interval, the effective refractory period of the fast pathway and the functional refractory period of the fast pathway were measured again after administration of atropine sulfate, 1–2 mg intravenously, 3 months after radiofrequency catheter ablation. The effective refractory period of the fast pathway was defined as the period with the longest A1-A2 coupling interval during which it was not possible to conduct in an antegrade manner through the fast pathway to the ventricle. The functional refractory period was defined as the shortest H1-H2 interval which conducted the fast pathway in an antegrade manner.

Statistical Analysis

Data are presented as the mean $\pm$ SD. Data within a group over time were first compared by ANOVA for repeated measures and then by the t-test with the Bonferroni correction. A p value of less than 0.05 was considered statistically significant.

RESULTS

Ablation Procedure

Slow pathway potential and a wide or split atrial wave were recorded in all 17 patients before ablation. The total number of applications of radiofrequency energy ranged from 2 to 9 (mean, 4 ± 2), and the total energy applied ranged from 800 to 4180 joules (mean, 1826 ± 914). No AV nodal reentrant tachycardia could be induced by any electrical stimulation immediately after the procedure. None of the patients experienced paroxysmal supraventricular tachycardia during the follow-up period of 3 months despite the absence of antiarrhythmic drug therapy. AV nodal reentrant tachycardia was not induced by programmed electrical stimulation in any of patients in the 3 months period after successful catheter ablation.

Electrophysiological Parameters

Sinus Cycle Length (Fig 3)

Sinus cycle length immediately after ablation was significantly shortened from 788 ± 134 to 714 ± 115 ms ($p < 0.05$). However, it was prolonged to 910 ± 155 ms at 3 months after ablation, which was significantly longer than those before and immediately after the procedure ($p < 0.01$).

Wenckebach Cycle Length (Fig 4)

The Wenckebach cycle length of the fast pathway during atrial pacing immediately after ablation was significantly longer than those before and 3 months after the procedure ($p < 0.05$).

A-H Interval (Fig 5)

The A-H interval was significantly shortened from 110 ± 19 ms to 97 ± 18 ms ($p < 0.01$) immediately after catheter ablation, and was prolonged to 125 ± 41 ms 3 months after the procedure ($p < 0.01$). There was no significant difference in the A-H interval between pre-ablation and 3 months post-ablation. In addition, there was no significant difference in the A-H interval between immediately after catheter ablation and with atropine sulfate at 3 months (97 ± 18 vs 96 ± 19).

Effective Refractory Period of the Fast Pathway (Fig 6)

The effective refractory period of the

Fig 3. Comparison of the sinus cycle length before, immediately after and 3 months after successful catheter ablation of the slow pathway. The sinus cycle length immediately after ablation was significantly shorter than that before ablation.

Fig 4. Comparison of the Wenckebach cycle length of the fast pathway in each period. The Wenckebach cycle length immediately after ablation was significantly higher than that before the procedure. It had recovered at 3 months after ablation.

antegrade fast pathway, 357 ± 61 ms in the control state, was shortened to 267 ± 54 ms immediately after catheter ablation ($p < 0.01$), but had recovered to 382 ± 104 ms at 3 months after ablation ($p < 0.01$). This was shortened to 255 ± 44 ms after intravenous administration of atropine sulfate 3 months after catheter ablation. There was no sig-

Fig 5. Comparison of the A-H interval at a paced cycle length of 600 ms in each period. The A-H interval was significantly shortened immediately after the slow pathway was eliminated, but had recovered at 3 months after the procedure. There was no significant difference between the A-H interval immediately after ablation and that after the administration of atropine sulfate at 3 months.

nificant difference between the effective refractory period immediately after catheter ablation and that at 3 months after the procedure with atropine sulfate. The effective refractory periods of the retrograde fast pathway could not be compared at each follow-up period, because it was equal to or shorter than that of the ventricular muscle in 15 of the 17 patients.

Functional Refractory Period (Fig 7)

The functional refractory period of the fast pathway was significantly shortened from 428 ± 59 ms before ablation to 378 ± 48 ms immediately after the procedure. It then recovered to 473 ± 99 ms 3 months after the procedure. There was no significant difference between the functional refractory period immediately after ablation and that at 3 months under the intravenous injection of atropine sulfate.

Each of these electrophysiological parameters changed in the same manner in all of the patients despite differences in the total applied radiofrequency energy and the number of applications.

Fig 6. Comparison of the effective refractory period of the fast pathway in each period. A shortening of refractoriness of the antegrade fast pathway was observed immediately after radiofrequency catheter ablation, but refractoriness had recovered at 3 months after elimination of the slow pathway. Refractoriness of the fast pathway after administration of atropine sulfate did not differ from that immediately after radiofrequency catheter ablation.

Fig 7. Comparison of the functional refractory period of the fast pathway in each period. The functional refractory period of the fast pathway was significantly shortened immediately after ablation, but had recovered at 3 months after the procedure. There was no significant difference between the functional refractory period immediately after and 3 months after the procedure with the intravenous injection of atropine sulfate.

DISCUSSION

Radiofrequency catheter ablation of the slow pathway in patients with AV nodal reentrant tachycardia has become a common mode of therapy in those refractory to antiarrhythmic drugs.³⁻⁷ Jazayeri et al⁶ reported that the slow pathway is ablated by the application of anatomically guided radiofrequency energy to the region of the coronary sinus ostium without recording discrete potentials. In this anatomical approach, the ablation catheter is positioned to the atrial margin of the A-V annulus at 4-6 o'clock on the tricuspid ring, and radiofrequency current is then applied toward the ostium of the coronary sinus. On the other hand, when the ablation site is determined by the electrophysiological approach, radiofrequency energy is delivered at the site where the slow pathway potential or a wide and split atrial wave was recorded. The ablation site of the slow pathway is beneath and close to the AV node in both approaches. Although the reentrant circuit of AV nodal reentrant tachycardia is reportedly extranodal⁸⁻¹¹ and the slow pathway has a retrograde exit located posterior and inferior to that of the fast pathway,¹² the physiology of the AV node after catheter ablation of the slow pathway remains controversial. Kay et al¹³ reported that the effective refractory period of the fast pathway was shortened after radiofrequency catheter ablation of the slow pathway in AV nodal reentrant tachycardia. In contrast, Wathen et al⁸ and Takahashi et al¹⁴ reported no significant change after elimination of the slow pathway. In addition, Jazayeri et al⁶ and Wathen et al⁸ reported that the A-H interval was unchanged after the procedure. These contradictory reports only discussed the characteristics of AV nodal physiology that occurred immediately after the procedure. No assessment of AV nodal physiology was made during the long-term follow-up. This is the first report to compare AV nodal physiology immediately after to that 3 months after radiofrequency catheter ablation. Our study demonstrated that the sinus cycle length, the Wenckebach cycle length of the fast pathway, the A-H interval, the antegrade effective refractory period of the fast pathway and the functional refractory period of

the fast pathway were shortened immediately after radiofrequency catheter ablation, but had recovered at 3 months after the procedure. These findings show that A-V nodal physiology is affected by radiofrequency catheter ablation of the slow pathway in AV nodal reentrant tachycardia, but recovers within 3 months. Although we could not determine exactly when AV nodal physiology had recovered, ablation of the slow pathway in AV nodal reentrant tachycardia can be safely performed without disturbing AV nodal physiology.

Kay et al¹³ reported that a shortening of the effective refractory period of the fast pathway after radiofrequency catheter ablation of the slow pathway is caused by an increase in sympathetic nervous tone, or by the selective destruction of parasympathetic innervation. Kocovic et al¹⁵ reported that the increase in the sinus cycle length after radiofrequency catheter ablation of the slow pathway is caused by a decrease in parasympathetic nerve tone. Innervation of the human AV node is less understood than that of animals. In contrast, parasympathetic innervation in dogs is well understood. Kay et al¹³ and Lazzara et al¹⁶ reported that the parasympathetic fibers that innervate the AV node of the dog are clustered near the crux of the heart and traverse the triangle of Koch. Parasympathetic fibers penetrate to the AV node and extend across the right atrium and sulcus terminalis to innervate the sinus node.¹⁶ Therefore, it is possible that they are selectively destroyed by application of radiofrequency energy for ablation of the slow pathway, which results in parasympathetic denervation of the AV node and sinus node. Natale et al¹⁷ suggested that the cause of this shortening of the effective refractory period was heat injury. On the other hand, an electrical interaction between the fast and slow pathways may produce these changes. Natale et al¹⁷ reported that the durations of myocardial refractoriness and of the action potential depend on the pattern and timing of activation. The sequence of excitations and different speeds of propagation of the impulse would affect local recovery properties through electrotonic interactions during the excitation process. Electrotonic effects reflect the existence of anisotropy and nonuniform activation in the

myocardium. In the case of AV node tissue and the surrounding myocardium, when the entire region has been excited, an impulse from the slow pathway may retard repolarization of areas excited earlier in the activation sequence of the fast pathway. Loss of this interaction after ablation may shorten the refractoriness of the fast pathway.¹⁷ If interaction between the fast and slow pathways is important, the changes in the electrophysiological characteristics of the AV node should last more than 3 months. Although our findings, including shortening of the sinus cycle length, the A-H interval, the antegrade effective refractory period of the fast pathway and the functional refractory period immediately after the radiofrequency catheter ablation of the slow pathway, are the same as those in previous reports, shortening of the A-H interval and of the effective refractory period of the fast pathway observed immediately after radiofrequency catheter ablation were not observed 3 months after ablation, even though the slow pathway had been perfectly ablated. The effective and functional refractory periods of the fast pathway immediately after radiofrequency catheter ablation did not differ significantly from those after injection of atropine sulfate at 3 months post-ablation. These results indicate that such changes may be caused by a decrease in parasympathetic nerve tone. The shortening of the sinus cycle length and the A-H interval immediately after the procedure also suggests a decrease in parasympathetic nerve tone.

Although the electrophysiological parameters had recovered at 3 months after the ablation, we did not determine exactly when these abnormalities recovered to the control state. In animal models, parasympathetic reinnervation requires 6 weeks.¹⁸ If parasympathetic nerve denervation was the cause of these abnormalities, and parasympathetic innervation in humans is similar to that in animals, these changes would be expected to recover gradually with a time-course consistent with reinnervation.

Limitations:

We could not exclude the possibility that an increase in sympathetic nerve tone contributed to the electrophysiological changes

after radiofrequency catheter ablation. We did not examine either the serum or urinary catecholamine concentration and did not inject β -blocking agents immediately after catheter ablation. Therefore, to examine the possible role of sympathetic nervous tone, future studies should use β -blocking agents and examine catecholamine metabolism.

CONCLUSION

Although the changes in the characteristics of the AV node are attributed to a decrease in parasympathetic nerve tone that may be caused by electrical heat injury or some other mechanism, the true cause is not known. AV nodal physiology was affected immediately after radiofrequency catheter ablation of the slow pathway in patients with AV nodal reentrant tachycardia, and this effect was not observed at 3 months. Therefore, this procedure appears to be safe for treating AV nodal reentrant tachycardia in patients who are refractory to, or who are intolerant of, antiarrhythmic agents.

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