NONPHARMACOLOGIC TREATMENT OF COMMON ATRIAL FLUTTER GUIDED BY TRANSIENT ENTRAINMENT

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We attempted nonpharmacologic treatment for common atrial flutter in 7 patients, with direct current catheter ablation (CA) in 5 cases and cryoablation in 2 cases under the guidance of transient entrainment. Transient entrainment during common atrial flutter by pacing from the mid or low lateral right atrium (LLRA) revealed a long conduction time between the LLRA and the orifice of the coronary sinus (CSo) (73% to 121% of flutter cycle length). In 2 cases, fragmented electrograms were recorded at the low right atrial septum with durations of 150 msec and 155 msec, respectively. Because these fragmented electrograms were transiently entrained by rapid atrial pacing in the same manner as other atrial electrograms and were recorded in an area of long conduction, we believe that fragmented electrograms represent critical slow conduction. In 5 cases, CA was directed at this area. Three cases were successfully treated with 3 to 4 DC shocks of 100 J (follow up periods of 11.3, 4.5 and 3 months). Two cases which received 1 to 2 DC shocks of 100 J had recurrence of atrial flutter 6 and 4.7 months later, respectively. Two cases which showed atrial septal defect received surgical treatment. They were successfully treated with extended cryoablation in this same area (follow up periods of 26 and 9.2 months). In conclusion, extended ablation of an area of fragmented electrograms using transient entrainment may be an effective treatment for common atrial flutter.

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WHILE there has been a great deal of controversy regarding the mechanism of common atrial flutter, recent progress in the field of clinical electrophysiology using transient entrainment has revealed that it is a macroreentry with an excitable gap.^{1,2} Analysis of electrophysiologic phenomena during transient entrainment of common

atrial flutter has made it possible to demonstrate the existence of slow conduction in the reentry circuit.³ Furthermore, some studies have used transient entrainment to show that the reentry pathway is located in the right atrium, and that the area of slow conduction may exist in the lower right atrial septum.^{4–7}

A wide range of pharmacologic and nonpharmacologic treatments are available for arrhythmia. Rapid atrial pacing is effective in terminating an arrhythmia and anti-

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TABLE I SUBJECTS

case	gender	age	basic heart disease	flutter cycle length
<i>Catheter Ablation</i>				
1	F	55	atrial septal defect II post op	280 msec
2	M	78	no	300 msec
3	M	71	old myocardial infarction post CABG	200 msec
4	M	72	hypertrophic cardiomyopathy	220 msec
5	F	56	atrial septal defect II post catheter ablation	265 msec
<i>(case 1)</i>				
<i>Cryoablation</i>				
6	M	67	atrial septal defect I sick sinus syndrome	240 msec
7	F	67	atrial septal defect II sick sinus syndrome	250 msec

CABG=coronary aorta bypass graft, op=operation.

TABLE II ENDOCARDIAL ATRIAL MAPPING DURING COMMON TYPE OF ATRIAL FLUTTER

case		1	2	3	4	5	6	7
FCL		280 ms	300 ms	200 ms	220 ms	265 ms	240 ms	250 ms
<i>mapping site</i>								
CS	orifice	0	0	0	0	0	0	0
	middle	50	30	30	20	35	30	30
	distal		50		50	70	60	50
HBE		65	70	30	40	80	50	20
HRA	lateral	105	155	70	100		100	110
	posterior	110	130	110	80			
	septal		120		80			
	anterior		130				70	
APP		135		50	90			
MRA	lateral	155	120	120	110		70-150*	120
	anterior		85		50			
	septal	145	70	70	80		80-160*	
LRA	lateral	-15		25, 150#	110, 160#	205	25	140
	anterior				120, 160#			
	septal		70	50	180-70*	245-80*		
<i>mapped %</i>		55%	52%	75%	100%	100%	67%	56%

All values are presented in milliseconds. *=with fragmentation, # = double potentials. CS=coronary sinus, HBE=His bundle electrogram, HRA=high right atrium, APP=appendage, MRA=middle right atrium, LRA=low right atrium.

tachycardia pacemakers have been recommended in some cases⁸⁻¹⁰ Catheter ablation of the atrioventricular node or His bundle is also an effective therapy for eliminating clinical symptoms, but the patient must undergo permanent pacemaker implantation^{11,12} Nonpharmacologic treat-

ment, including surgery or electrical catheter ablation, may be able to eliminate the arrhythmia, but there are some difficulties in detecting the target critical area in the atrium, i.e. the area of slow conduction in the reentry circuit. We used transient entrainment to detect the area of slow conduc-

Fig.1. Endocardial atrial mapping during common atrial flutter.

Examples of atrial mapping from case 4 (left) and case 5 (right) are shown. In case 4, fragmented electrograms with a duration of 150 msec were recorded in the low septal area (LSRA1 and LSRA2). In case 5, fragmented electrograms were recorded at the same area as in case 4, but the duration of the fragmented electrograms was 155 msec. These catheters were located close to the low atrial septum near the orifice of the coronary sinus (Fig. 2). II and VI=surface ECG leads II and VI, HRA=high right atrium, LLRA=low lateral right atrium, LSRA=low septal right atrium, HBE=atrioventricular junction area, CSo=orifice of the coronary sinus, CSm=middle of the coronary sinus, CSd=distal coronary sinus.

tion in the right atrium and attempted non-pharmacologic treatment for common atrial flutter. In this article, we report the methods and results of our nonpharmacologic approach to this arrhythmia.

SUBJECTS AND METHODS

Subjects: (Table I) The study population consisted of 7 patients, 4 males and 3 females, from 55 to 78 years old. Each patient had drug refractory common atrial flutter and symptoms such as palpitation and dyspnea. They were administered class Ia antiarrhythmic drugs (procainamide, disopyramide and quinidine), but these drugs were ineffective in eliminating the arrhythmias. Case 4 also had an atrial fibrillation attack before catheter ablation. He was also administered class Ia antiarrhythmic drugs, but they were ineffective against common atrial flutter. Quinidine was effective only against atrial fibrillation. Each patient, ex-

cept case 2, had additional heart disease. Two patients with atrial septal defect underwent cryoablation simultaneously with a correction of the anomaly. Catheter ablation was attempted in the remaining 5 cases. Cases 1 and 5 are the same patient who underwent repeated catheter ablation due to recurrence of common atrial flutter.

Electrophysiologic study: After receiving written informed consent from the subjects, an electrophysiologic study was performed in the fasting state in our cardiac catheterization laboratory. A catheter with hexapolar electrodes, and a 2 mm interelectrode spacing and 2 cm between electrode pairs (Mansfield, USA), was inserted into the coronary sinus through the left subclavian vein. The proximal pair of electrodes was positioned at the orifice of the coronary sinus. A catheter with quadripolar electrodes and a 5 mm interelectrode spacing, was positioned at the atrioventricular junc-

Fig.2. Catheter position in case 4

Posterior-anterior view of a chest X-ray is shown. Fragmented electrograms were recorded with the distal electrode pairs of the 2 catheters (LSRA1 and 2). The electrode spacing of the catheter was 5 mm and the recording area of fragmented electrograms was small. Abbreviations are the same as in Fig. 1.

tion for recording His bundle electrograms. One or 2 quadripolar-electrode catheters were positioned at the high lateral right atrium or in the right atrium for endocardial atrial mapping during atrial flutter through the femoral veins. Surface electrograms and intracardiac electrograms were recorded with a NIHON KOHDEN RMC-2000 polygraph system. During common atrial flutter, atrial mapping was performed at 7 to 15 sites in the right atrium and coronary sinus. After confirming counterclockwise rotation of excitation^{3,5} the entrainment study was performed by pacing from the high lateral right atrium, middle lateral right atrium or low lateral right atrium. The conduction time during transient entrainment from the pacing site to the orifice of the coronary sinus was measured, especially from the middle or low lateral right atrium, to confirm manifest transient entrainment to the orifice of the coronary sinus.^{5,7} The area of slow conduction during common atrial flutter was believed to be located between 2 sites where transient entrainment showed a long conduction time despite the short distance between the sites.^{5,7,13-16} In 2 cases (cases 4 and 5), we recorded fragmented electrograms in this

area during atrial flutter, which may represent critical slow conduction in the reentry circuit!¹⁷

Direct current catheter ablation: We used direct current discharge in catheter ablation, because we believed that direct current energy could ablate atrial myocardium more deeply and more widely than radiofrequency!¹⁸ In 5 cases, 1 to 4 sequential DC shocks of 100 joules were discharged at the area of slow conduction which had been identified in the transient entrainment study. In cases 4 and 5, DC shock was discharged at the area of fragmented electrograms. The cumulative energies ranged from 100 to 400 joules per session. Catheter ablation was performed with the above-mentioned 6 F quadripolar-electrode catheters and a direct current cardioverter (Life Pack 9, Physio-Control, USA). A distal electrode tip or a second electrode tip was used as the cathode and a large patient back plate was used as the anode under intravenous thiamylal anesthesia.

Cryoablation: In the 2 cases with atrial septal defect, the area of slow conduction

Fig.3. An example of transient entrainment in case 5.

Flutter cycle length was 260 msec and the pacing cycle length was 240 msec. Last pacing stimulus and last entrained electrograms are indicated by asterisks. Atrial flutter and fragmented electrograms were transiently entrained by rapid atrial pacing from the mid lateral right atrium. During transient entrainment, the relationship between fragmented electrograms and the other electrograms was constant.

II and VI=surface ECG leads II and VI, MLRA=mid lateral right atrium, LSRA=low septal right atrium, HBE=atrioventricular junction area, CSo=orifice of the coronary sinus, CSm=middle of the coronary sinus, CSd=distal coronary sinus.

was ablated during surgery by a cryoprobe with a diameter of 1 cm (Cryobar, Japan). In case 7, during a partial normothermic cardiopulmonary bypass, endocardial atrial mapping near the defect and a transient entrainment study were performed. The area of slow conduction, as identified in the preoperative entrainment study and the electrophysiologic study during surgery, was ablated at a temperature of -170°C at 2 or 3 areas.

RESULTS

Endocardial atrial mapping: The results of endocardial atrial mapping during common atrial flutter are shown in Table II. In case 5, endocardial atrial mapping was abbreviated. The electrogram of the orifice of the coronary sinus was defined as the reference point (0 msec) and the mapping

sequence of each site in the atria was shown in milliseconds. Mapped % represents mapped duration of 1 flutter cycle length. Counterclockwise excitation rotation was observed in all cases and 52 to 100% of 1 flutter cycle could be mapped. Fragmented electrograms or double potentials were obtained in 3 cases. In cases 4 and 5, we recorded fragmented electrograms at the low septal right atrium near the orifice of the coronary sinus using a catheter with an interelectrode distance of 5 mm. These fragmented electrograms were located between the low lateral right atrium electrogram and the electrogram of the coronary sinus orifice, and reached into the next cycle. As shown in Fig. 1, the duration of the fragmented electrograms was 150 msec in case 4 (left) and 155 msec in case 5 (right). Fig. 2 (cases 4) shows 2 ablation catheters positioned at the low septal right atrium where the frag-

Fig.4. An example of transient entrainment in case 5

Pacing cycle length was 210 msec. The fragmented electrograms were transiently entrained by rapid atrial pacing. Compared with Fig. 3, constant fusion and progressive fusion of intraatrial electrograms are observed. In addition, the stimulus-HBE conduction time during transient entrainment changed from 30 msec to 260 msec. Abbreviations are the same as in Fig. 3.

mented electrograms were recorded. In the other 5 cases, 25% to 48% of the flutter cycle could not be recorded in the mapping sequence between the low lateral right atrium and the orifice of the coronary sinus during counterclockwise excitation rotation.

Transient entrainment during common atrial flutter Fig. 3 and 4 show examples of transient entrainment by pacing from the mid lateral right atrium (case 5). Flutter cycle length was 260 msec. Pacing cycle length was 240 msec (Fig. 3) and 210 msec (Fig. 4). Last pacing and last entrained electrograms are indicated by asterisks. In both figures, during rapid atrial pacing, each of the electrograms was entrained to the pacing cycle length (constant fusion). The relative locations of the electrograms in intracardiac recording during rapid atrial pacing were different at pacing cycle lengths of 240 msec and 210 msec (progressive fusion). The conduction time from the pacing impulse to the electrogram of the His bundle area was 30 msec at a pacing cycle length of 240 msec (Fig. 3) and 260 msec at a pacing cycle

length of 210 msec (Fig. 4), i.e., 2 different conduction times were observed at 2 different pacing cycle lengths, which represents the fourth criterion of transient entrainment.¹⁶ Fragmented electrograms and the electrograms of the coronary sinus orifice were accelerated to each pacing cycle length, which implies manifest entrainment. During rapid atrial pacing, these criteria of transient entrainment were fulfilled.

Another case is shown in Fig. 5. We did not perform catheter ablation in this case, or include this case in the study. Flutter cycle length in this case was 330 msec and entrainment pacing cycle length was 280 msec (left) and 250 msec (right). Last pacing and last entrainment electrograms are indicated by asterisks. Fragmented electrograms and double potentials (LSRA2 and LSRA3) were recorded at the low septal right atrium, but were actually entrained by rapid atrial pacing from the high lateral right atrium (left panel). At a pacing cycle length of 250 msec, atrial flutter was interrupted by conduction block of fragmented electrograms

Fig.5 An example of transient entrainment and interruption of atrial flutter in relation to fragmented electrograms.

Flutter cycle length was 330 msec. Last pacing stimulus and last entrained electrograms are indicated by asterisks. Fragmented electrograms (shown by brackets) were recorded at the low septal right atrium. Pacing from the high lateral right atrium (pacing cycle length of 280 msec) transiently entrained these electrograms. At a pacing cycle length of 250 msec, atrial flutter was interrupted with conduction of fragmented electrograms (shown by double bars). Abbreviations are the same as in Fig.3.

TABLE III LONG CONDUCTION TIME DURING TRANSIENT ENTRAINMENT OF COMMON TYPE OF ATRIAL FLUTTER BY PACING FROM MID OR LOW LATERAL RIGHT ATRIUM

case	FCL	PCL	conduction time from St to CSo	conduction time %FCL
<i>Catheter Ablation</i>				
1	280 ms	200 ms	300 ms	107%
2	300 ms	240 ms	240 ms	80%
3	200 ms	180 ms	205 ms	103%
4	240 ms	210 ms	250 ms	119%
5	265 ms	210 ms	190 ms	73%
<i>Cryoablation</i>				
6	240 ms	200 ms	220 ms	92%
7	240 ms	200 ms	290 ms	121%

FCL=flutter cycle length, PCL=pacing cycle length, St=stimulus, CSo=orifice of coronary sinus.

(double bars). These results strongly suggest that fragmented electrograms at the low septal area are a critical portion of the reentry circuit and represent the slow conduction of atrial flutter.

In Fig. 3 and 4, the conduction time from the pacing site (mid lateral right atrium) to the orifice of the coronary sinus was 190

msec at a pacing cycle length of 240 msec or 210 msec, respectively. Fragmented electrograms were located in this long conduction area. A long conduction time was recognized in all cases during transient entrainment from the mid or low lateral right atrium (Table III). Conduction time is presented as a percentage of the flutter cycle

TABLE IV DIRECT CURRENT CATHETER ABLATION

case	ablation site	fragmentation	energy	induction immediately after ablation
1	coronary sinus orifice	(-)	100 J	not inducible
	low septal right atrium	(-)	100 J	
2	low septal right atrium	(-)	100 J	not performed
3	low septal right atrium	(-)	100 J	not performed
	coronary sinus orifice	(-)	100 J×2	
	(2 sites)			
4	low septal right atrium	(+)	100 J×4	non sustained
	(4 sites)			uncommon atrial flutter
5	low septal right atrium	(+)	100 J×4	not inducible
	(4 sites)			

TABLE V CRYOABLATION

case	ablation site	fragmentation	temperature	induction post operation
6	mid lateral atrium near defect	(+)	-170°C×1.5 min	not inducible
	mid septum (2 sites)	(+)	-170°C×1.5 min×2	
	coronary sinus orifice	(-)	-170°C× 3 min	
7	coronary sinus orifice	(-)	-170°C× 3 min×2	not inducible
	(2 sites)

Fig.6. Schematic representation of the cryoablation sites in cases 6 and 7.

In case 6, with atrial septum defect type I, the atrial septum was incised from the defect to the low septum. Fragmented electrograms were recorded at 3 sites and cryoablation was directed at these areas and at the low septum near the orifice of the coronary sinus. In case 7, with atrial septum defect type II, fragmented electrograms were not recorded and cryoablation was directed at the low septum near the orifice of the coronary sinus. SVC=superior vena cava, IVC=inferior vena cava, D=atrial septal defect, Ap=appendage, TA=tricuspid annulus, CS=coronary sinus, RV=right ventricle, LA=left atrium.

length. Conduction time ranged from 73% to 121% of flutter cycle length. In the first 3 cases, catheter ablation was performed at the low septal area near the coronary sinus orifice, and in the latter 2 cases, catheter

ablation was performed at the area of fragmented electrograms.

Direct current catheter ablation: Ablation sites and ablation energies are summarized

Fig. 7. Endocardial atrial mapping near the defect during surgery in case 7.

Mapping was performed during normothermic cardiopulmonary bypass. Flutter cycle length was 240 msec. The mapping reference point was the epicardial side of the orifice of the coronary sinus (0 msec). Sequential atrial activation, from cranial to caudal, was recorded on the lateral side of the defect, but not on the medial side. At 8 o'clock from the defect, a fragmented electrogram was recorded, and at 2 o'clock, a 2:1 electrogram was recorded. Long conduction time was observed between the low lateral right atrium and the orifice of the coronary sinus during transient entrainment. CS os = orifice of the coronary sinus, MLRA = mid lateral right atrium.

in Table IV. In cases 4 and 5, catheter ablation was performed at the sites with fragmented electrograms through a distal electrode and a second electrode of each mapping catheter. In the other 3 cases, catheter ablation was performed near the orifice of the coronary sinus without confirming fragmented electrograms.

Multiple sequential shocks were discharged through sequential electrodes of a catheter. Cumulative energies ranged from 100 joules to 400 joules per session. No complications occurred during or after the catheter ablation procedure.

Cryoablation: Cryoablation sites are summarized in Table V and Fig. 6. In case 6, fragmented electrograms were recorded at the middle and low septal area below the defect during the preoperative electrophysiologic study. After the atrial septum was in-

cised from the defect to the lower septum, cryoablation was directed at areas of fragmented electrograms and the lower atrial septum just outside of the orifice of the coronary sinus, i.e., a total of 4 sites. In Case 7 (Fig. 7), during a normothermic cardiopulmonary bypass, mapping near the defect showed a cranial-to-caudal excitation sequence on the lateral side of the defect. However, on the medial side of the defect, no sequential excitation was seen. Fragmented electrograms were recorded at the low lateral side of the defect. Transient entrainment by pacing from the mid lateral right atrium showed a long conduction time from the pacing site to the orifice of the coronary sinus, which suggests slow conduction between these areas. Cryoablation was directed at an area just lateral to the orifice of the coronary sinus and below the defect, i.e., a total of 2 sites (Fig. 6). The atrial septal defect was simultaneously corrected with a patch closure in both cases.

Electrophysiologic study immediately after ablation: We tried to induced common type of atrial flutter by an atrial extrastimulation method which included up to 3 extrastimuli, and by burst pacing immediately after catheter ablation in 3 cases or after cryoablation in 2 cases. In each case, common atrial flutter was not induced. However, a nonsustained uncommon atrial flutter was induced in case 4.

Clinical follow up: All patients, except case 4, were initially followed up without any drug administration. Clinical recurrence of common atrial flutter was observed in 2 cases who had been treated with catheter ablation after 6 months (case 1) and 4.7 months (case 2). These 2 patients were administered disopyramide 300 mg daily after recurrence. Case 2 did not have any recurrences after drug administration. Since disopyramide and quinidine were ineffective in case 1, we tried re-catheter ablation (case 5). After the second catheter ablation, she had no recurrence during 3 months of follow up without any drug administration. Because case 4 had suffered from atrial fibrillation before catheter ablation, he was administered oral quinidine after catheter ablation. Although quinidine had been in-

TABLE VI CLINICAL FOLLOW UP

<i>case</i>	<i>follow up (months)</i>	<i>initial follow up drug</i>	<i>clinical outcome</i>	<i>other arrhythmia</i>	<i>additional therapy</i>
<i>catheter ablation</i>					
1	12 m	no	recurrence (6 m)	no	disopyramide re-catheter ablation (case 5)
2	8.4 m	no	recurrence (4.7 m)	no	disopyramide
3	11.3 m	no	no recurrence	no	no
4	4.5 m	quinidine	no recurrence	atrial fibrillation	quinidine
5	3 m	no	no recurrence	no	no
<i>cryoablation</i>					
6	26 m	no	no recurrence	slow atrial tachycardia	disopyramide AAI pacemaker
7	9.2 m	no	no recurrence	uncommon atrial flutter	DDD pacemaker

effective against common type of atrial flutter before catheter ablation, he has had no recurrence of atrial flutter after catheter ablation. Atrial fibrillation was still present after quinidine administration, but the frequency of attacks was reduced. Case 6 suffered from sick sinus syndrome and an AAI antitachycardia pacemaker was implanted in case of recurrence of common type of atrial flutter. Common type of atrial flutter did not recur, but once slow atrial tachycardia (120 bpm) was clinically observed. Disopyramide was effective against this atrial tachycardia and antitachycardia pacing was not in use. In case 7, a DDD pacemaker was implanted because of the previous existence of sick sinus syndrome. He suffered from uncommon type of atrial flutter once immediately after cryoablation, but no recurrence was clinically observed during the follow up period. In summary, 5 cases, 3 with catheter ablation and 2 with cryoablation, had no clinical recurrence of common type of atrial flutter.

DISCUSSION

Transient entrainment of a tachycardia indicates that the mechanism of a tachycardia is macroreentry with an excitable gap^{1,2}. During transient entrainment, an orthodromic wave front propagates into an area of slow conduction and appears at the exit of this area after a long conduction time.¹³⁻¹⁶ The reentry circuit of common atrial flutter

can also be transiently entrained by rapid atrial pacing, which suggests that it also has a mechanism of macroreentry with an excitable gap^{2,3}. Some studies using transient entrainment have determined that slow conduction of common type of atrial flutter is located in the low septal right atrium and that the orifice of the coronary sinus is an exit from the slow conducting area⁵⁻⁷. We used this phenomenon to identify the area of slow conduction in the reentry circuit of atrial flutter. Slow conduction was observed in the lower septal right atrium, and ablation of this area was eliminated common atrial flutter in 5 cases out of 7.

In our cases, transient entrainment showed a long conduction time from the pacing site to the orifice of the coronary sinus. Fragmented electrograms were recorded at the low septal right atrium between the low lateral right atrium and the orifice of the coronary sinus in counterclockwise excitation rotation. As shown in the figures, these fragmented electrograms were actually transiently entrained by rapid atrial pacing, and in 1 case common type of atrial flutter was interrupted with a conduction block in these electrograms. These phenomena strongly suggest that fragmented electrograms are a critical part of the reentry circuit of common type of atrial flutter, and represent the slow conduction of atrial flutter.

There is still some disagreement over whether double potentials or fragmented electrograms represent critical slow conduc-

tion^{7,17} In our study, because fragmented electrograms with a long duration were recorded in a small area and transiently entrained, we believed that they represented critical slow conduction. Another method which could be used to show that fragmented electrograms represent slow conduction is to confirm concealed entrainment by pacing from the fragmented electrogram area.¹⁹ However, it is difficult to identify fusion flutter electrograms in a surface ECG during transient entrainment. In experimental study, double potentials and fragmented electrograms may represent the conduction block in the center of the reentry circuit, and not the reentry circuit itself or slow conduction.²⁰ Further electrophysiologic studies in humans will be necessary to clarify this problem.

In the first 3 cases, catheter ablation was performed at the low septal area, because we believed that flutter excitation showed counterclockwise rotation, and the coronary sinus orifice was believed to be the exit from the area of slow conduction, based on the entrainment study. In the latter 2 cases, we actually recorded fragmented electrograms and ablated these areas. Chauvin and Brechenmacher⁴ and Saoudi et al⁶ reported the results of endocardial fulguration for common type of atrial flutter. The lesion they targeted for fulguration in the right atrium was an area which showed fragmented electrograms during atrial flutter. Using an entrainment study, they ensured that these fragmented electrograms were critical to slow conduction, and not merely slow, uninvolved, pathways. We also found significance in the fact that a long conduction time was observed between the 2 sites, despite their close proximity. We searched for fragmented electrograms around these sites and ablated these areas. We believe that our ablation area corresponds to those of Chauvin and Saoudi.

We used direct current ablation through an electrode catheter with an energy of 100 joules for up to 4 times. In cases 3, 4 and 5, we ablated 3 or 4 sites in the low septal area per session and, especially in cases 4 and 5, ablation was directed only at the area of fragmented electrograms. In the cases which showed recurrence (cases 1 and 2), the total amount of energy delivered was 200 J and

100 J to 2 and 1 sites, respectively. This energy may have been too low to ablate the area of slow conduction. We believe that slow conduction observed during atrial flutter extends across in a large 2-dimensional plane on atrial myocardium.^{3,21} In those cases which did not show recurrence, cryoablation was performed at 2 or 4 sites, and the total ablated area was evidently sufficiently large to produce satisfactory results. Cryoablation can penetrate deeply into atrial myocardium. Direct current power can also ablate myocardium deeply and widely due to its barotrauma and electrical power, even with soft catheter contact to atrial myocardium.^{22,23} Recently, radiofrequency power has been used in catheter ablation of accessory pathways.²⁴ Radiofrequency can produce a clearly delineated lesion, but the ablated area is small and shallow. Since we believe that successful treatment requires ablation of an extended area, and since a radiofrequency source was not available, we did not use radiofrequency power in the present study. However, radiofrequency energy appears to be worthy of future study in the ablation of common atrial flutter.

Clinical limitation: Patients with common atrial flutter often have other types of atrial arrhythmias, i.e. uncommon type of atrial flutter, atrial fibrillation or sick sinus syndrome. In the present study, case 4 suffered from atrial fibrillation before catheter ablation, and cases 7 and 8 suffered from sick sinus syndrome. The basic heart disease in case 4 was hypertrophic cardiomyopathy. He has not had a recurrence of common type of atrial flutter since catheter ablation, but still suffers from atrial fibrillation. In cases of atrial myocardial degeneration (cardiomyopathy, longstanding valvular heart disease), there is a problem of clinical indication and clinical application of catheter ablation. After successful ablation of common type of atrial flutter, patients require drug administration or pace maker implantation. However, in a patient with many types of atrial tachyarrhythmias, catheter ablation could reduce the risk of at least one of these unpleasant atrial tachyarrhythmias. Because catheter ablation is still being studied, care is required regarding its indication and applica-

tion. In 3 cases, 2 with catheter ablation and 1 with cryoablation, atrial fibrillation, atrial tachycardia and uncommon type of atrial flutter were clinically observed after ablation. These arrhythmias may actually be the result of catheter ablation or cryoablation.¹⁸ Ablation itself may have predisposed these arrhythmias by producing myocardial damage. This implies that some problems may exist after successful ablation for common atrial flutter. Therefore, medical treatment may be required after successful ablation, which may reduce the benefit of catheter ablation. However, the patients in the present study had less severe symptoms in these arrhythmias after ablation than before ablation.

Conclusions: We believe that catheter ablation or cryoablation under the guidance of transient entrainment and fragmented electrograms is a useful treatment for common type of atrial flutter. However, because of catheter ablation is still being studied, care should be taken regarding its application, and further study with a long term follow up in a larger number of patients is necessary.

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