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Radiofrequency Catheter Ablation of Common Atrial Flutter: Comparison of Electrophysiologically Guided Focal Ablation Technique and Linear Ablation Technique

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Objectives. **The purpose of this study was to study electrophysiologic characteristics and compare the electrophysiologically guided focal ablation technique and linear ablation technique in patients with common atrial flutter in a prospective randomized fashion.**

Background. **Catheter ablation of the common atrial flutter circuit can be performed with different techniques. To date, these two techniques have not been compared prospectively in a randomized study.**

Methods. **Sixty patients with drug-refractory common atrial flutter were randomly assigned to undergo radiofrequency catheter ablation performed with the electrophysiologically guided focal ablation (Group I) or linear ablation technique (Group II). In Group I, radiofrequency energy was delivered to the site characterized by concealed entrainment with a short stimulus–P wave interval (<40 ms) and a postpacing interval equal to the atrial flutter cycle length. In Group II, continuous migratory application of radiofrequency energy was used to create two linear lesions in or around the inferior vena cava–tricuspid ring isthmus. Serial 24-h ambulatory electrocardiographic (Holter) and follow-up electrophysiologic studies were performed to assess recurrence of tachycardia and possible atrial arrhythmogenic effects.**

Results. **Successful elimination of the flutter circuit was achieved in 28 of 30 patients in Group I and 29 of 30 patients in Group II. More atrial premature beats and episodes of short run atrial tachyarrhythmias in the early period (within 2 weeks) after ablation were found in Group II. Recurrence rate (2 of 28 vs. 3 of 29) and incidence of new sustained atrial tachyarrhythmias (3 of 28 vs. 3 of 29) was similar in the two groups. Occurrence of recurrent atrial flutter and new sustained atrial tachyarrhythmias was related to associated cardiovascular disease and atrial enlargement in both groups. However, in Group II, the procedure time (104 ± 17 vs. 181 ± 29 min, $p < 0.01$) and radiation time (22 ± 8 vs. 42 ± 13 min, $p < 0.01$) were significantly shorter than those in Group I.**

Conclusions. **Radiofrequency ablation of the common atrial flutter circuit was safe and effective with either the electrophysiologically guided focal ablation or linear ablation technique. However, the linear ablation technique was time-saving.**

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The optimal target site for successful ablation of common atrial flutter remains controversial (1–6). Previous reports (1–7) have indicated that radiofrequency energy delivered to the low right atrium, including the areas of fragmented electrograms, double potentials, presumed entry site (near the inferior vena cava–tricuspid ring isthmus) and exit site (near the coronary sinus orifice–tricuspid ring isthmus) of the slow conduction area could achieve a high immediate success rate. However, a high recurrence rate with new types of atrial tachyarrhythmias was found after successful ablation. Two major methods (according to electrophysiologic characteristics or anatomic basis) used to ablate the atrial tissue critical for maintenance of atrial flutter have been described (1–6). Because no previous studies have compared these two techniques, it is unclear whether one approach is preferable to the other. Furthermore, the factors related to recurrent tachycardia and new atrial tachyarrhythmias have not been determined.

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Methods

Patient characteristics (Table 1). Sixty consecutive patients with a documented history of paroxysmal common atrial flutter were included. We excluded patients with clinically documented episodes of paroxysmal atrial fibrillation or uncommon atrial flutter, or both, and patients with sustained uncommon atrial flutter or atrial fibrillation, or both, induced (>1 min) during the baseline electrophysiologic study. All patients had been unable to tolerate or had not responded to

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Table 1. Comparisons of Clinical and Electrophysiologic Characteristics and Effects of Radiofrequency Ablation Performed With Two Different Methods in Patients With Common Atrial Flutter

	Group I (n = 30)	Group II (n = 30)	p Value
Age (yr)	52 ± 7	55 ± 6	NS
Men/women	27 of 3	25 of 5	NS
Associated CV disease	9 of 30	11 of 30	NS
Atrial enlargement	7 of 30	9 of 30	NS
Atrial flutter CL (ms)	217 ± 18	223 ± 16	NS
Radiofrequency pulse (no.)	3 ± 1	4 ± 1	NS
Procedure time (min)	181 ± 29	104 ± 17	< 0.01
Radiation time (min)	42 ± 13	22 ± 8	< 0.01
Success rate	28 of 30	29 of 30	NS
Recurrence rate	2 of 28	3 of 29	NS
Complication rate	0	0	—
Follow-up period (mo)	21 ± 11	19 ± 11	NS
New sustained atrial tachyarrhythmias	3 of 28	3 of 29	NS

Data are presented as mean value ± SD or number of patients. CL = cycle length; CV = cardiovascular.

a mean of 3 ± 1 (range 2 to 5) antiarrhythmic drugs before referral. Twenty patients had associated cardiovascular disease, including rheumatic heart disease in 1 patient, hypertension in 10, coronary heart disease in 3, mitral valve prolapse with moderate mitral regurgitation in 2, chronic pericarditis in 1 and cardiomyopathy in 3. Echocardiographic examinations revealed left atrial enlargement in 5 patients and biatrial enlargement in 11.

Baseline electrophysiologic study, endocardial mapping and entrainment pacing study. Each patient gave informed, written consent. Research protocols were approved by the Human Research Committee at this institution. As described previously (8,9), all antiarrhythmic drugs were discontinued for at least 5 half-lives before the study. The orifices of the inferior vena cava and coronary sinus were identified by venograms. All patients had sustained common atrial flutter reproducibly induced by programmed stimulation. The methods of induction included 1) baseline pacing (8-beat drive, twice diastolic threshold) with single and double premature stimuli at three cycle lengths (600, 500 and 400 ms), and 2) burst pacing from the high right atrium (>20 beats, twice diastolic threshold or up to 10 mA) at progressively shorter cycle lengths until 2:1 capture was noted.

Only 30 patients assigned to the electrophysiologic approach underwent very detailed endocardial mapping and an entrainment pacing study. In the first six of these patients, detailed endocardial mapping of the right atrium (anteromedial, anterior, anterolateral, lateral, posterolateral, posterior, posteromedial and septal positions at the high, middle and low levels) was performed by a multipolar, closely spaced (2-mm) electrode catheter. Among the remaining 24 patients, a deflectable "halo" catheter (Webster) was adjusted at an appropriate position to record the electrograms of the lateral aspect of the right atrium, inferior vena cava–tricuspid ring isthmus, coronary sinus orifice and the septal area simultaneously. A multipolar, closely spaced (2-mm) electrode catheter with a deflectable tip was used to map and perform entrainment pacing in this low right atrium (the area near the inferior vena cava–tricuspid ring isthmus, coronary sinus orifice–tricuspid ring isthmus and the area between the two isthmuses).

Entrainment pacing was started at a pacing cycle length 20 ms shorter than the atrial flutter cycle length until 2:1 atrial capture was achieved. During entrainment pacing, 12-lead surface electrocardiograms (ECGs) were simultaneously recorded and the tracings compared with the baseline atrial flutter. *A normal discrete bipolar atrial electrogram* was defined as a rapidly increasing, discrete potential with an amplitude >1 mV and a duration <50 ms. *An abnormal bipolar atrial electrogram* included 1) a fragmented electrogram (a low amplitude potential [<1 mV] either with a slow rate of rise or with multiple peaks and a duration >50 ms, or 2) double potentials (two discrete potentials separated by an isoelectric baseline [10,11]). *The stimulus–P wave interval (SP interval), postpacing interval and manifest and concealed entrainment* of atrial flutter were defined according to classic criteria (1,12). *The presumed exit site of the slow conduction area* was identified by the presence of concealed entrainment pace-mapping with a relatively short SP interval (<40 ms) and a postpacing interval equal to the atrial flutter cycle length.

Radiofrequency ablation technique. All the patients were randomly assigned to Group I or II. *In Group I patients,* radiofrequency energy was delivered to the site characterized by concealed entrainment with a short SP interval (<40 ms) and a postpacing interval equal to the atrial flutter cycle length. The SP interval and postpacing interval were measured from the slowest stimulus trains that entrained the atrial flutter to avoid the possible decremental property of the slow conduction area. Energy (Radionic-3C, Radionics) was delivered at a power setting of 30 to 40 W, duration 20 to 30 s and was terminated immediately in the event of impedance rise or catheter dislocation. If atrial flutter was terminated during application of energy, a full 60-s application was performed at that site. If an ablation attempt failed to terminate atrial flutter, entrainment pace-mapping was performed again to identify an area consistent with the same criteria by repositioning the ablation catheter (Fig. 1 to 4). *In Group II patients,* radiofrequency energy was delivered primarily according to anatomic landmarks. Continuous migratory application of radiofrequency energy was used to create two linear lesions: the beginning and ending points of the linear lesion were identified and recorded in the cine films as guides for ablation. Initially, the ablation catheter was introduced into the right ventricle (ventricular electrogram only, without atrial electrogram) and then progressively pulled back to the atrial margin of the tricuspid annulus with a very small atrial electrogram and very large ventricular electrogram; the tip of the ablation catheter was positioned inferiorly at the 6 o'clock to 7 o'clock position (60° left anterior oblique view) on the tricuspid ring. In the course of migratory movement, the atrial electrogram became progressively larger, then disappeared again when the

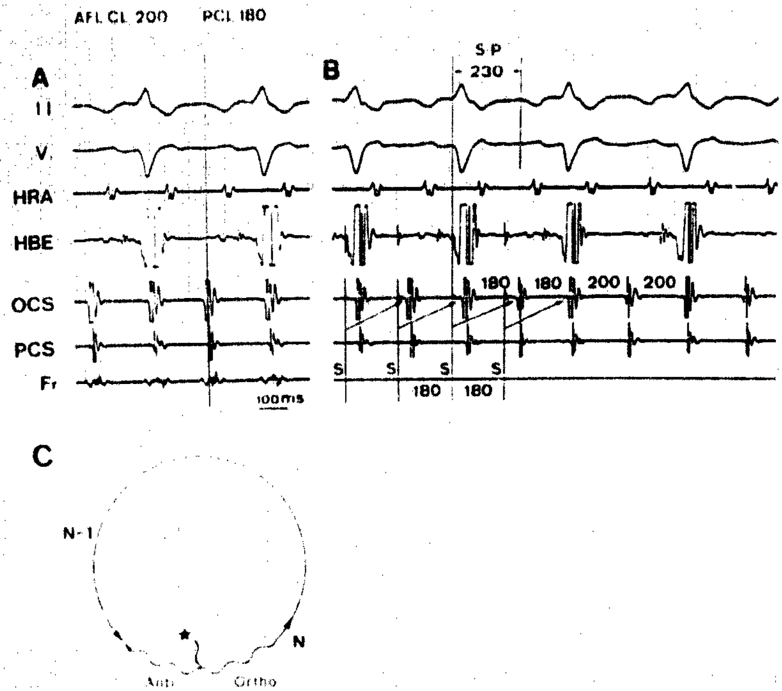

Figure 1. A, Fragmented electrograms (Fr) recorded from the inferior vena cava-tricuspid ring isthmus lasted for 95 ms. **B,** Entrainment pacing with cycle length 180 ms was performed from the area with fragmented electrograms. The SP interval (S-P) prolonged to 230 ms. The P wave configuration was similar between entrainment pacing and atrial flutter. **C,** Concealed entrainment with a long SP interval also suggested that the pacing site was near the entry site of slow conduction or in the "dead end" of the slow conduction area. The orthodromic wave front (N) from the pacing site (**star**) conducted through the "dead end" pathway to the exit site of the slow conduction area, resulting in a long SP interval. The antidromic wave front would either block in the slow conduction area or collide with the orthodromic wave front of the preceding paced beat (N-1) in the slow conduction area. AFL CL = atrial flutter cycle length; Anti = antidromic; HBE = His bundle electrogram; HRA = high right atrium; OCS = coronary sinus orifice; ortho = orthodromic; PCL = pacing cycle length; PCS = proximal coronary sinus; S = stimuli; II = lead II; V_1 = lead V_1.

ablation catheter was in the inferior vena cava (Fig. 5). The slow migratory movement lasted 120 s from the beginning to the ending point. Application of energy (30 W) was continued until the tip electrode of the ablation catheter was clearly in the inferior vena cava. When the ablation catheter was close to the inferior vena cava, the power of radiofrequency current was decreased a little if the patient felt pain. The second ablation line was located in the more lateral position at the 8 o'clock position close to the junction between the inferior vena cava–tricuspid ring isthmus and the lateral wall of the right atrium; the second lesion was created after the first linear lesion had been created, whether or not atrial flutter had been terminated. If atrial flutter had been terminated after the two linear lesions, radiofrequency energy was again delivered to the first and second linear lesions to ensure success. If atrial flutter had not been terminated, application of energy to the first and second linear lesions was repeated for a maximum of four times. Approximately 30 min after successful ablation of atrial flutter, induction of atrial flutter (including burst pacing using electrical current up to 10 mA) was repeated.

Postablation follow-up. All patients underwent 24-h ambulating electrocardiographic (Holter) monitoring 1 day before, immediately after and at 1 and 2 weeks and 1 and 3 months after ablation (13). All patients received a hand-held wrist recorder for 3 months on the day immediately after ablation. When the patients experienced symptoms suggestive of tachycardia, 24-h Holter monitoring with use of a hand-held wrist recorder and a follow-up electrophysiologic study were performed to define the cause of clinical symptoms.

Statistical analysis. Quantitative values are expressed as mean value ± SD and were compared by *t* test. The chi-square test and Fisher exact test were used for analysis of categoric data. Absence of recurrent tachycardia and new sustained atrial tachyarrhythmias was assessed by the Kaplan-Meier curve analysis; comparisons between groups were assessed by log-rank test. A p value <0.05 was considered statistically significant.

Results

Group I patients. *1) Endocardial mapping and entrainment pacing from presumed exit sites.* Endocardial activation of the right atrium proceeded "down" the free wall and "up" the septum during atrial flutter in all patients. Entrainment pacing at locations with fragmented electrograms in the three areas (inferior vena cava–tricuspid ring isthmus, area between the two isthmuses and coronary sinus orifice–tricuspid ring isthmus) revealed concealed entrainment with SP intervals of 252 ± 62, 187 ± 41 and 110 ± 33 ms ($p < 0.01$), respectively (Fig. 1). Pacing at locations with discrete electrograms in the

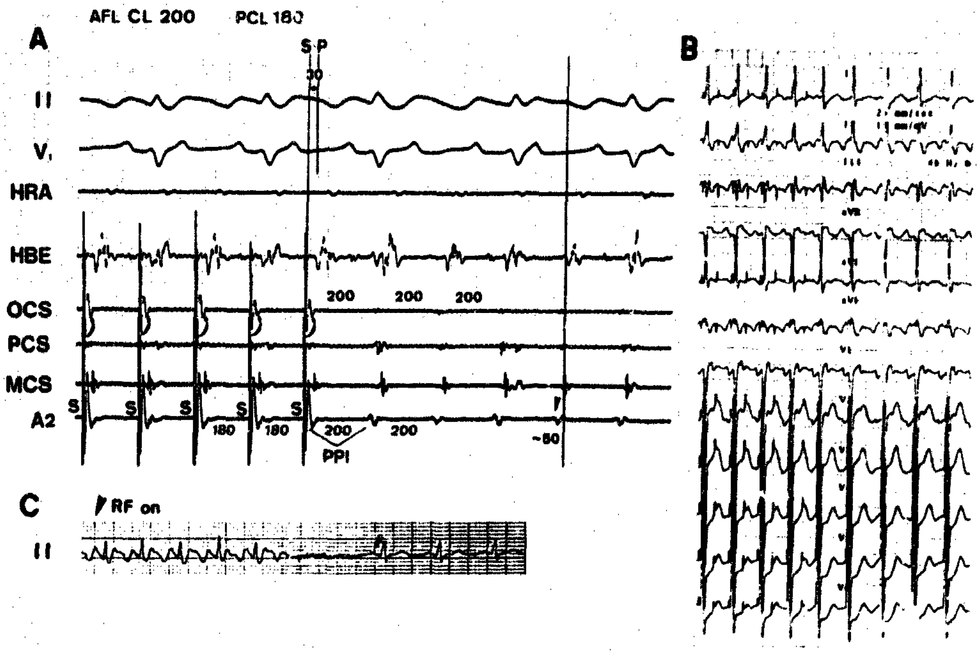

Figure 2. A, The local activation time of the discrete electrogram **(arrowhead)** was 50 ms before the onset of the P wave; the SP interval was 30 ms, the postpacing interval (PPI) was equal to the atrial flutter cycle length, and concealed entrainment was also demonstrated in the 12-lead surface electrocardiogram (**B**), suggesting that this stimulation site was at the exit site of the slow conduction area. P wave configurations were similar during entrainment pacing and atrial flutter (**B**). C, There is abrupt termination of atrial flutter with the appearance of sinus rhythm **(small arrowhead)** after application of radiofrequency energy (RF). A_2 = 2 cm away from coronary sinus orifice; MCS = mid-coronary sinus; other abbreviations as in Figure 1.

three areas also revealed concealed entrainment with shorter SP intervals of 110 ± 19, 83 ± 17 and 50 ± 14 ms ($p < 0.01$), respectively (Fig. 2). In the three areas with discrete electrograms, the earliest local activation time before the onset of the inverted P wave was in the inferior vena cava–tricuspid ring isthmus (−101 ± 18, −80 ± 14 and −52 ± 9 ms, respectively, $p < 0.01$). The extent of SP interval prolongation during concealed entrainment pacing with decreasing pacing cycle lengths in the three areas was 15 ± 4, 11 ± 3 and 6 ± 4 ms, respectively ($p < 0.01$), and postpacing interval prolongation was 16 ± 5, 10 ± 4 and 6 ± 4 ms, respectively ($p < 0.01$) (Fig 3). Entrainment pacing from the locations with double potentials in the three areas all showed manifest entrainment (Fig. 4).

2) Radiofrequency ablation over the presumed exit site of slow conduction area. Atrial flutter was terminated in 28 (93.3%) of 30 patients (8 patients needed only one pulse) (Fig. 2). After successful ablation, nonsustained uncommon atrial flutter or atrial fibrillation, or both, was induced in 11 of the 28. The successful ablation sites were posterior to (n = 14), inferior to (n = 9) or in (n = 5) the coronary sinus orifice, and they had discrete electrograms with a mean local activation time of 43 ± 8 ms (range 30 to 58) before the onset of the P wave. The mean SP interval was 32 ± 7 ms (range 15 to 40) and the postpacing interval almostly equaled the atrial flutter cycle length (mean difference 5 ± 3 ms [range 0 to 10]). Of the two patients without successful ablation, one continued to receive medication and the other received atrioventricular node ablation with implantation of VVIR pacemaker.

3) Follow-up. Two patients had recurrence in the 2nd and the 3rd month, respectively, and both had a successful second ablation by the same method used in the first ablation with no late recurrence. Three patients had new sustained atrial tachyarrhythmias (uncommon atrial flutter in one patient and atrial fibrillation in two patients). Late follow-up electrophysiologic studies (92 ± 7 days later) were performed in 20 patients; sustained atrial tachyarrhythmias were induced only in the three patients with clinically documented tachyarrhythmias. These arrhythmias were treated effectively by oral amiodarone. The incidence of recurrence and new sustained atrial tachyarrhythmias was significantly related to associated cardiovascular disease and atrial enlargement, but not to patient age (Fig. 6 to 8).

Group II patients. *1) Radiofrequency ablation.* Atrial flutter was eliminated in 11 patients with a single application of radiofrequency energy to the first (n = 6) or second (n = 5) linear lesion. In the other 18 patients, atrial flutter was

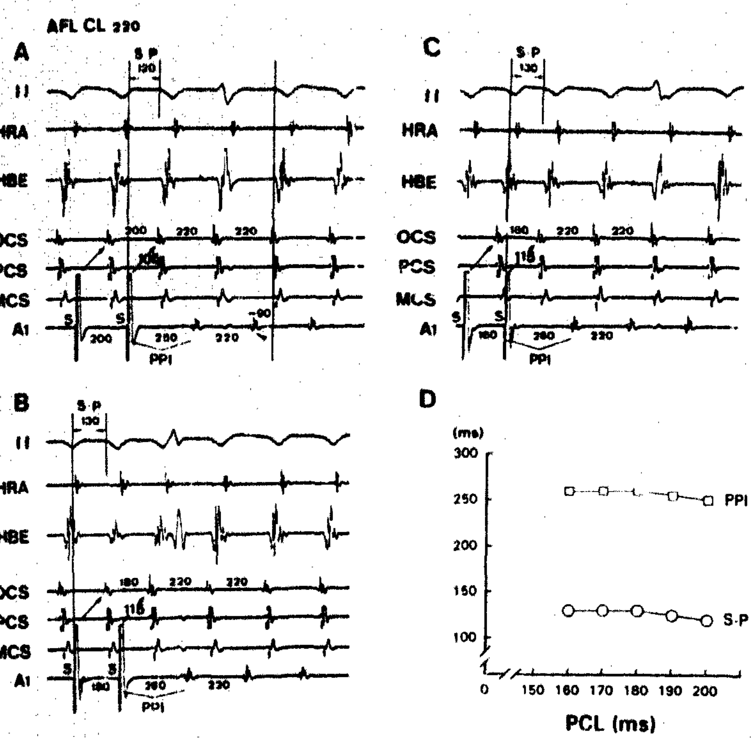

Figure 3. Entrainment pacing from the area between two isthmuses. The SP interval and postpacing interval were prolonged slightly when the entrainment pacing cycle length was decreased progressively (**A to D**). The interval between the pacing site and the coronary sinus orifice (**oblique arrows**) was the source of pacing-induced conduction delays. A_1 = 1 cm away from coronary sinus orifice; other abbreviations as in Figures 1 and 2.

eliminated when more than one radiofrequency pulse was delivered to the first or second linear lesion. One patient without successful ablation underwent AV junction modification to control the ventricular rate. Nonsustained uncommon atrial flutter or atrial fibrillation, or both, was induced in eight patients after successful ablation.

2) Follow-up. Three patients had a recurrence (in the 1st, 4th and 6th month, respectively) and had successful ablation in the second ablation session using the same method used in the first session without recurrence. One patient had uncommon atrial flutter and two patients had atrial fibrillation after successful ablation. Late follow-up electrophysiologic studies (89 ± 6 days after ablation) were performed in 22 patients; a sustained form of atrial tachyarrhythmia was induced in four patients, and only three of them had clinical tachyarrhythmia. Their arrhythmia was treated successfully by administration of oral amiodarone. As in Group I, the incidence of recurrence and new sustained atrial tachyarrhythmias was significantly related to associated cardiovascular disease and atrial enlargement but not to patient age (Fig. 6 to 8).

Comparisons between Group I and Group II patients. The major differences between patient groups were the significantly longer procedure time and radiation exposure time in Group I. Although Group II patients had more atrial arrhythmias in the early follow-up period (within 2 weeks), the number of these atrial arrhythmias was similar in the two groups in the late period (Fig. 6).

Discussion

Major findings. This study demonstrated that both the electrophysiologically guided focal ablation technique and the linear ablation technique could achieve a high success rate, low recurrence rate and low incidence of atrial arrhythmogenesis. However, procedure time and radiation exposure time were longer in the ablation sessions using the electrophysiologically guided focal ablation technique.

Efficacy of different ablation techniques. There is general agreement that the mechanism of typical atrial flutter is a macroreentrant circus movement localized within the right atrium (14–16). Detailed endocardial mapping and entrainment pacing study showed presence of slow conduction area in the low right atrium (1,16). The tricuspid ring–inferior vena cava isthmus is a zone of relatively slow conduction. However, the area of Koch's triangle delineated by the coronary sinus orifice, the tendon of Todaro and the tricuspid ring may also be slowly conducting, but it may or may not be involved in the atrial flutter circuit and is certainly distal to the tricuspid valve–inferior vena cava isthmus and the exits from the isthmus. Feld et al. (1) delivered radiofrequency energy to the presumed exit site of slow conduction area with the shortest stimulus–P wave interval during concealed entrainment. Ten of their 12 patients had successful ablation and only 1 patient (10%) had recurrence during the follow-up period (mean 16 ± 9 weeks [range 2 to 31]). Cosio et al. (2) assumed that the

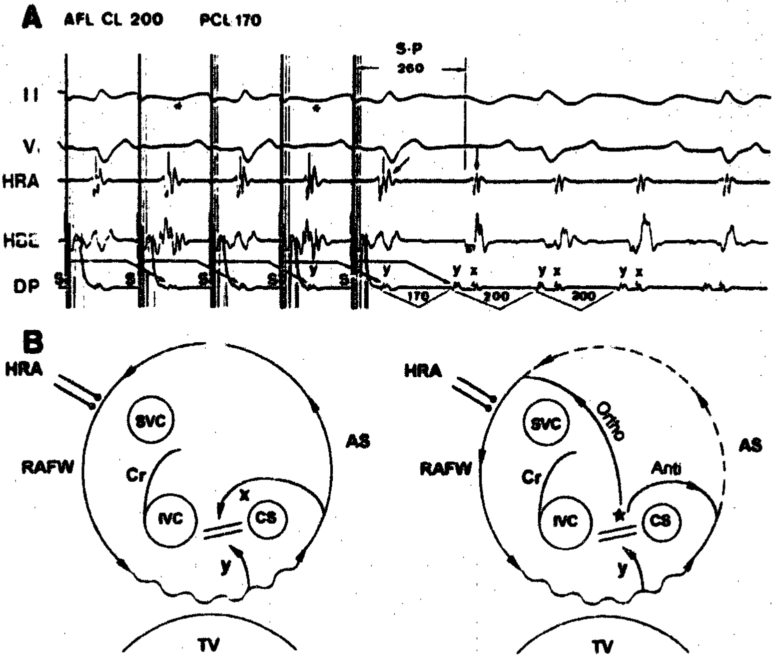

Figure 4. **A,** Entrainment pacing and recording from the area with double potentials (DP). Manifest entrainment was demonstrated: 1) constant fusion of P waves in the surface electrocardiogram (lead II, *), 2) a long SP interval (260 ms) in the surface electrocardiogram, and 3) the local electrograms of the high right atrium recorded during pacing (**oblique arrow**) were different from those recorded during atrial flutter (**vertical arrow**). **B,** Diagram showing the proposed circuit of atrial flutter (**left**) and the mechanism of entrainment pacing (**right**) in **A.** The x potential was defined as that occurred shortly after the onset of the P wave in lead II, and the y potential then came late just prior to P wave onset. The pacing site was probably at or above the line of block demarcating a lateral border of the tricuspid ring–inferior vena cava isthmus, accounting for the early capture of the x potential antidromically (Anti) and the y potential orthodromically (Ortho) through the area of slow conduction with a long stimulus–y interval. Pacing from this area (★) also demonstrated manifest entrainment with fusion of the P wave and high right atrial electrogram. AS = atrial septum; Cr = crista terminalis; Cs = coronary sinus; IVC = inferior vena cava; RAFW = free wall of right atrium; SVC = superior vena cava; TV = tricuspid valve; other abbreviations as in Figure 1.

inferior vena cava–tricuspid ring isthmus was a critical path for circus movement of typical atrial flutter. The isthmus was not ablated completely, and four (44%) of nine patients had a recurrence during the short-term follow-up period. Kirkorian et al. (4) delivered radiofrequency energy to the whole isthmus in 22 patients. A high immediate success rate (86%) with a relatively low recurrence rate (10%) was demonstrated (mean follow-up interval 13 months). Wolfe et al. (17) delivered radiofrequency energy to the isthmus between the tricuspid anulus, coronary sinus orifice and inferior vena cava to perform linear ablation in 85 patients. A high immediate success rate (94%) with a relatively low recurrence rate (11%) was demonstrated (mean follow-up interval 10 months). Lesh et al. (3) delivered radiofrequency energy to the entry site, middle portion or the exit site of slow conduction area using an anatomic approach in 18 patients (the linear ablation method was not used). The results showed a high immediate success rate (94%) with a recurrence rate up to 29% (mean follow-up interval 10 months). Fischer et al. (6) used both anatomic landmarks and electrophysiologic variables (not based on entrainment study) as guides for ablation; the immediate success rate was 90% and the recurrence rate 19% (mean follow-up interval 20 months). They suggested that ablation of the atrial flutter circuit was more feasible in the inferior vena cava–tricuspid ring isthmus than in other areas. In our series, an ablation technique using either a primarily anatomic approach or an electrophysiologic approach was capable of terminating typical atrial flutter.

Anatomic approach. Fischer et al. (6) demonstrated that radiofrequency energy was most effective for ablation when delivered to the area between the tricuspid ring and inferior vena cava. Because the activation wave of atrial flutter proceeded either anteriorly or posteriorly to the ostium of the coronary sinus, the other areas were less effective. Cosio et al. (2) and Kirkorian et al. (4) also showed that the isthmus between the inferior vena cava and the tricuspid ring was a critical site for ablation. This structure was located more laterally than the target area selected by Feld et al. (1). The

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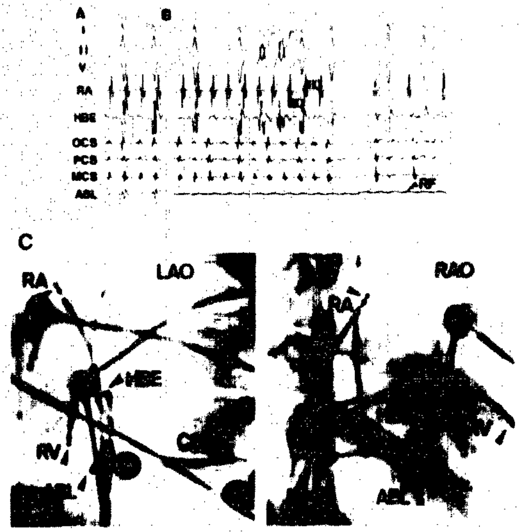

Figure 5. With the ablation (ABL) catheter pulled back to the atrial margin of the tricuspid annulus, there is a very small atrial electrogram and a very large ventricular electrogram. The tip of the ablation catheter was positioned at the 6 o'clock position (60% left anterior oblique [LAO] view) on the tricuspid ring (panels A and C). **B,** Termination of atrial flutter after delivery of radiofrequency energy (RF) in this inferior vena cava–tricuspid ring isthmus. I = lead I; RA = right atrium; RAO = right anterior oblique view; RV = right ventricle; other abbreviations as in Figures 1, 2 and 4.

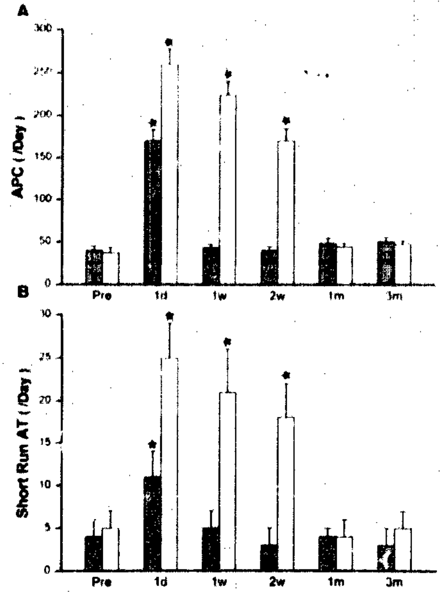

Figure 6. Results of serial 24-h ambulatory electrocardiographic (Holter) monitoring in patients in Group I (**hatched bars**) and Group II (**white bars**). Panels A and B show numbers of atrial premature contractions (APC) and episodes of short-run atrial tachyarrhythmias (AT), respectively (*$p < 0.01$). d = day; m = month; w = week.

inability of radiofrequency energy applications to terminate atrial flutter did not absolutely indicate that the ablation site was away from the reentrant circuit. If the critical area for maintaining the tachycardia involved broad sheets of atrial tissue in the low right atrium with different exits, a relatively small radiofrequency lesion was unlikely to interrupt it. Because the lesion size required for successful ablation is unknown, it remains uncertain whether continuous linear ablation or multiple applications are necessary. Significantly higher recurrence rates were found in the study of Cosio et al. (2), who did not ablate the isthmus completely, and in the study of Lesh et al. (3), who delivered energy only to the point site. Lesh et al. (3) and Feld et al. (18) also showed that a large (10-mm or 8-mm) tip ablation catheter could improve results with fewer lesions. Their data indicated that portions of the tricuspid ring–inferior vena cava isthmus suitable for ablation might be larger than the lesion created with a 4-mm tip ablation catheter and that a larger electrode may more easily ablate larger isthmus with fewer lesions.

Electrophysiologic characteristics in the low right atrium. *Fragmented electrogram.* Lesh et al. (3) reported that electrograms at sites of successful radiofrequency ablation were fragmented in 12 of 15 patients. However, the present study and others (1,5) showed that application of radiofrequency energy over the areas with fragmented electrograms failed to terminate atrial flutter. The present study also demonstrated that entrainment pacing from the areas with fragmented electrograms could show concealed entrainment with a long SP interval; most of the pacing sites with this phenomenon were presumably near the entrance site of the tricuspid ring–inferior vena cava isthmus, where descending pectinate muscle produced significant anisotropy and fragmented conduction. Although the long SP interval could be explained by the decremental property of slow conduction area responding to shorter pacing cycle lengths, this finding also suggested the possibility of "dead end" or "bystander" areas of slow conduction, which have been described in patients with atrial flutter and ventricular tachycardia (1,19). These areas might be adjacent to or outside the reentrant circuit and were not critical for the maintenance of tachycardia.

Double potentials. An experimental model of atrial flutter (20,21) demonstrated that double potentials could be recorded only at the functional center of the reentrant circuit during atrial flutter and represented sequential activation on either side of block. In human studies, Cosio et al. (11,16) suggested that double potentials could be explained either by a line of complete block or by slow conduction. Olshansky et al. (10,22) demonstrated that double potentials probably reflected activation on either side of an area of block. The present study demonstrated that entrainment pacing from the areas with double potentials revealed manifest entrainment. The preced-

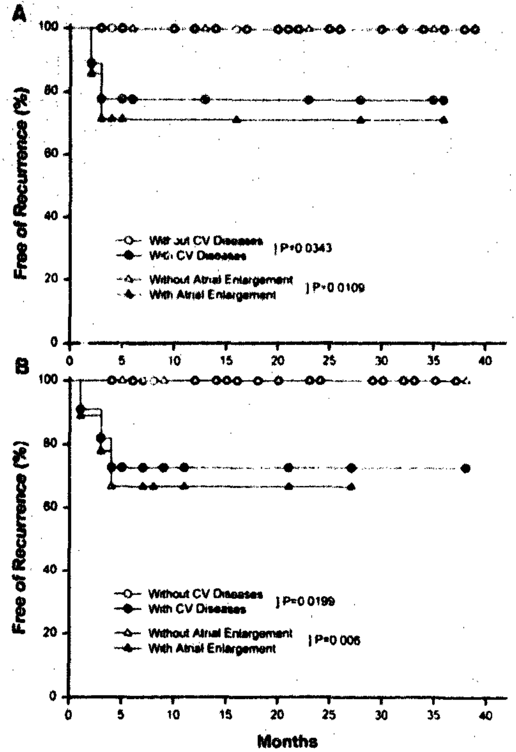

Figure 7. Proportion of patients in Group I (A) and Group II (B) without recurrent atrial flutter, according to the presence or absence of associated cardiovascular (CV) diseases or atrial enlargement.

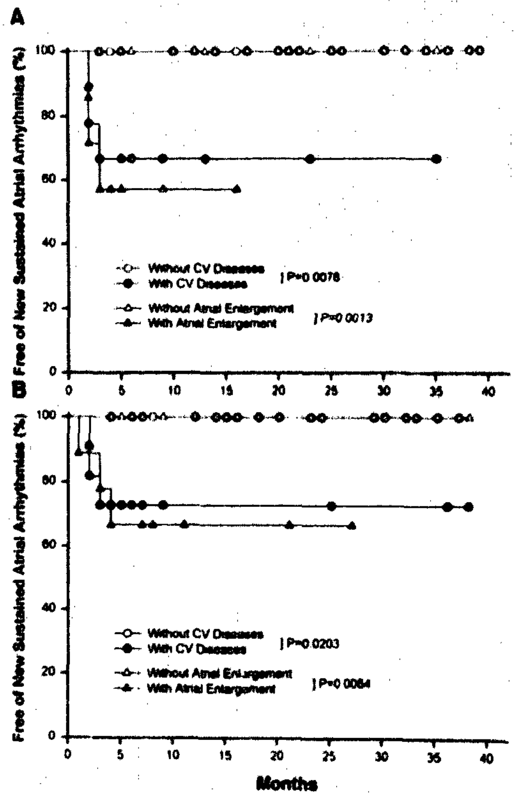

Figure 8. Proportion of patients in Group I (A) and Group II (B) without new sustained atrial tachyarrhythmias, according to the presence or absence of associated cardiovascular (CV) diseases or atrial enlargement.

ing findings demonstrated that double potentials might be away from the exit site of the slow conduction area. Shimizu et al. (21) also suggested that ablation at the area with double potentials should be avoided, as it might simply change a region of functional block in the center of the reentrant circuit into a region of anatomic block. Furthermore, the line (between the inferior vena cava and coronary sinus orifice) of block demarcating the border of the tricuspid annulus–inferior vena cava isthmus, opposite the tricuspid annulus, is probably the eustachian valve (20,23).

Early local activation time stimulus–P wave interval and postpacing interval. Entrainment pacing from the inferior vena cava–tricuspid ring isthmus consistently revealed concealed entrainment with a long SP interval (>40 ms), and the postpacing interval was always longer than the atrial flutter cycle length. The area characterized by concealed entrainment with a short SP interval (<40 ms) and a postpacing interval equal to the flutter cycle length was supposed to be near the exit site of slow conduction area (near the coronary sinus orifice) and optimal for radiofrequency ablation. In this specific area, discrete electrograms with local activation 30 to 60 ms before the onset of the flutter wave were consistently observed. Morady et al. (24) and Stevenson et al. (19) found that entrainment with concealed fusion alone was a poor predictor of successful catheter ablation of ventricular tachycardia, suggesting the possible role of bystander areas of slow conduction. Stevenson et al. (19) demonstrated that analysis of the postpacing interval could provide further information. The postpacing interval represents the conduction time from the pacing site through the reentrant circuit and back to the pacing site; thus it is always longer than the tachycardia cycle length when the pacing site is outside the reentrant circuit. For sites in the reentrant circuit, the postpacing interval is equal to the tachycardia cycle length if pacing does not alter the conduction velocity of propagation in the circuit. However, the postpacing interval is longer than the tachycardia cycle length when conduction velocity slows in response to premature stimuli or rapid stimulus trains, even when the pacing site is in the reentrant circuit. Furthermore, this study showed that the SP interval and the postpacing interval prolonged slightly when the entrainment pacing cycle lengths were decreased.

Atrial arrhythmogenic effects and recurrent tachycardia. Spontaneous or electrical stimulation-induced sustained atrial fibrillation and atypical atrial flutter were not found before or during the procedures; thus, new sustained atrial arrhythmias were considered to result from the natural course of cardiovascular disease–related atrial myopathy or arrhythmogenesis from a radiofrequency ablation lesion. However, the absence of documented atrial fibrillation before ablation does not exclude the possibility of previous latent paroxysmal episodes, as can be detected by Holter monitoring. It is not clear whether a larger ablation lesion will be more arrhythmogenic. In the present study, the ablation lesion created by linear ablation was larger than that obtained with the electrophysiologic approach. However, the incidence of new atrial arrhythmias in the late period after ablation was similar in the two groups. A previous study (13) had demonstrated that arrhythmogenic effects of radiofrequency ablation disappear ~1 month after ablation, and the present study showed that these new sustained atrial tachyarrhythmias are related to associated cardiovascular diseases and atrial enlargement. Philippon et al. (25) also showed that new sustained atrial fibrillation was related to structural heart disease. These findings suggest that new sustained atrial tachyarrhythmias may not be related to the lesion produced by radiofrequency ablation. However, the role of structural heart disease in the genesis of newly occurring atrial tachyarrhythmias does not exclude a role for ablation, as the risk of their occurrence with ablation may be potentiated by a preexisting cardiac process. The present study demonstrated that the recurrence rates are similar with the two techniques. Previous studies (1,4) also showed that the recurrence rate was significantly lower in patients who underwent ablation of the exit site of slow conduction (guided by electrophysiologic characteristics) and in patients who underwent complete ablation of the inferior vena cava–tricuspid ring isthmus. Wolfe et al. (17) reported that the presence of structural heart disease did not affect the recurrence rate. In the present study, the actuarial freedom from recurrent tachycardia was affected by the presence of associated cardiovascular disease and atrial enlargement. These differences in findings might result from the use of different ablation techniques.

Conclusions. Both linear ablation and electrophysiologically guided focal ablation achieved a high rate of success with a low recurrence rate in radiofrequency ablation of a typical flutter circuit. Possible arrhythmogenic effects of radiofrequency ablation were not found in the late follow-up period. The anatomic approach was time-saving, with respect to the procedure time and radiation exposure time.

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