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Mechanism, Localization and Cure of Atrial Arrhythmias Occurring After a New Intraoperative Endocardial Radiofrequency Ablation Procedure for Atrial Fibrillation

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OBJECTIVES The purpose of this study was to test a new pattern of radiofrequency ablation for atrial fibrillation (AFib) intended to optimize atrial activation, and to demonstrate the usefulness of catheter techniques for mapping and ablation of postoperative atrial arrhythmias.

BACKGROUND Linear radiofrequency lesions have been used to cure AFib, but the optimal pattern of lesions is unknown and postoperative tachyarrhythmias are common.

METHODS A radial pattern of linear radiofrequency lesions (Star) was made using an endocardial open surgical approach in 25 patients. Postoperative arrhythmias were induced and characterized during electrophysiological studies in 15 patients.

RESULTS The AFib was abolished in most patients (91%), but atrial flutter (AFlut) occurred in 96% of patients postoperatively. At postoperative electrophysiological studies, 37 flutter morphologies were studied in 15 patients (46% spontaneous, cycle length [CL] 223 ± 25 ms). Seven mechanisms (lesions discontinuity, n = 6; focal mechanism, n = 1) of AFlut were characterized in six patients. In these cases, flutter was abolished using further catheter radiofrequency ablation. In the remaining cases, flutter was usually localized to an area involving the interatrial septum, but no critical isthmus was identified for ablation. After 16 ± 10 months, 15 patients (65%) were asymptomatic with (n = 3) or without (n = 12) antiarrhythmic medications. Eight (35%) patients had persistent arrhythmias. Postoperative atrial electrical activation was near physiological.

CONCLUSIONS The AFib may be abolished using a radial pattern of linear endocardial radiofrequency lesions, but postoperative AFlut is common even when lesions are made under optimal conditions. Endocardial mapping techniques can be used to characterize the flutter mechanisms, thus enabling subsequent successful catheter ablation. (J Am Coll Cardiol 2000;35:442–50) © 2000 by the American College of Cardiology

Early attempts to perform minimally invasive catheter radiofrequency ablation procedures based on the original surgical operation of Cox et al. (1,2) have been encouraging, but postprocedural tachyarrhythmias and complications have been a major problem (3–8). Failure of catheter techniques may be due to poor electrode/tissue contact, failure to create continuous and transmural linear lesions and inaccurate placement of lesions. The relative importance of these variables is unknown.

Our long-term goal was to develop a catheter-based ablation technique. However, given the high incidence of atrial flutter (AFlut) after catheter procedures for cure of atrial fibrillation (AFib) we elected first to make the radiofrequency lesions during open-heart surgery. Performing ablation under direct vision ensured accurate placement of lesions, excellent tissue contact and visibly continuous lesions.

The aim of this study was to determine the antiarrhythmic efficacy of an endocardial radiofrequency ablation technique using a new pattern of linear lesions for cure of AFib. We also tested the ability of standard catheter techniques to determine the mechanism and location of residual postoperative atrial arrhythmias with a view to performing further catheter radiofrequency ablation.

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Abbreviations and Acronyms

AFib = atrial fibrillation
AFlut = atrial flutter
CL = cycle length
TA = tricuspid annulus

A different pattern of lesions was used to that described by Cox and colleagues (2). The aims of the new design were to minimize electrical isolation of areas of atrial myocardium and minimize disruption of the normal pattern of atrial activation. During the Cox maze procedure the incision encircling the pulmonary veins electrically isolates that section of the posterior left atrium. We replaced the incision encircling the pulmonary veins with lesions that allow the posterior left atrium to be electrically activated (Fig. 1). Tsui et al. (9) demonstrated that this area represents 36% of the surface area of the left atrial muscle. Preserved contraction of this area may improve the hemodynamic performance of the left atrium and reduce left atrial stasis and the tendency to thromboembolism (10).

To minimize disruption to the normal pattern of atrial activation, the lines of radiofrequency ablation were placed, where possible, parallel to the normal direction of wavefront propagation. We therefore created seven adjacent corridors radiating from the sinus node to the atrioventricular junction. Because of the stellate pattern of lesions we referred to the new operation as the *Star* procedure.

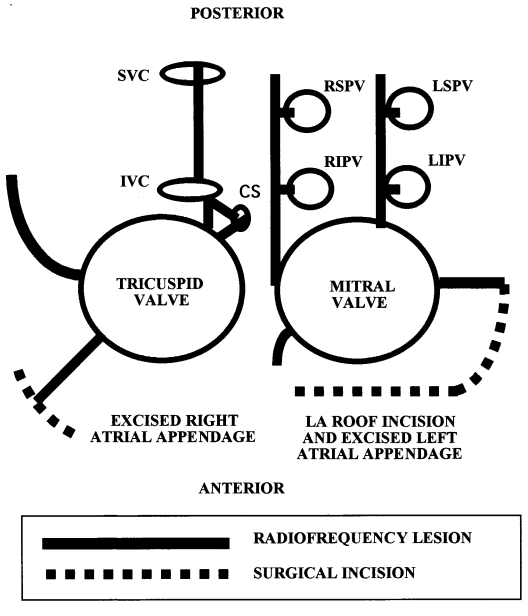


Figure 1. Pattern of endocardial radiofrequency lesions. In this schematic view the atria are opened superiorly and the anterior and posterior parts of the atria laid flat to expose the endocardial surface. SVC = superior vena cava; IVC = inferior vena cava; CS = coronary sinus; RSPV = right superior pulmonary vein; RIPV = right inferior pulmonary vein; LSPV = left superior pulmonary vein; LIPV = left inferior pulmonary vein.

METHODS

Patient population. The new procedure was performed on 25 patients between July 1995 and July 1998. The indications for surgery were symptomatic documented and sustained AFib not responding to drug therapy (n = 13) or AFib in patients requiring cardiac surgery for other lesions (n = 12). More detailed patient characteristics are provided in Table 1. The left atrium was enlarged in 19 patients (76%). The mean left atrial size determined by transthoracic echocardiography was 45 ± 7 mm.

The radiofrequency ablation technique. Radiofrequency lesions were produced using one of two handheld devices (Fig. 2). The first consisted of a straight or J-tipped electrode at the end of a 20-cm handle. The electrode was flat with a length of 12 mm and a width of 2.5 mm. The long handle and shape of the probe allowed the electrode to be positioned firmly against the endocardial surface through an atriotomy under direct vision. Radiofrequency current was delivered between the probe electrode and a large diathermy electrode positioned on the patient's skin. A thermistor in the probe electrode and a closed loop feedback system were used for temperature control. The temperature was set at 80° to 90°C with a settling time (time taken to achieve the set temperature) of approximately 30 s. Each radiofrequency application was 60 s in duration. The large electrode size and accurate temperature control were used to maximize lesion depth. This electrode design was tested in dogs prior to commencement of human studies. In canine ventricular muscle the mean lesion depth was 5.3 ± 0.9 mm. Lesions in canine atria were transmural in both trabeculated and smooth regions. The mean lesion width was 7.7 ± 0.4 mm. Radiofrequency lesions were placed end to end with overlap to make continuous long linear lesions.

A second ablation device had four 6-mm-by-2-mm electrodes with a 3-mm interelectrode distance mounted in sequence on a 33-mm-long flexible tip. This enabled the surgeon to shape the ablation device to conform to the shape of the atrial endocardial surface. Simultaneous, in-phase, unipolar ablation was performed between all four electrodes and the large surface electrode. Using this instrument a 35-mm lesion could be produced using electrode temperatures of 80° to 90°C over a single period of 60 s. This device was tested in canine atria during cardiopulmonary bypass and consistently produced transmural lesions with a duration of ablation of 30 s.

The surgical technique. A midline sternotomy was performed and cardiopulmonary bypass established using bicaval cannulation. The aorta was cross-clamped and cardioplegic solution infused. The atrial appendages were excised to gain access to the endocardial surface. The scheme for the radiofrequency lesions is shown in Figure 1.

Table 1. Preoperative Clinical Characteristics

Patient No.	Age (yrs)	Gender M/F	Duration of AFib (yrs)	Paroxysmal/ Chronic	Number of Failed Drugs	Etiology of Atrial Fibrillation	Concurrent Surgery
1	40	M	9	P	3	Lone fibrillator	Nil
2	58	F	10	C	1	Aortic stenosis	Aortic valve replacement
3	53	M	0.7	C	1	Atrial septal defect	Repair of atrial septal defect
4	49	F	2	P	4	Lone fibrillator	Nil
5	70	F	1	C	2	Mitral stenosis	Mitral valve replacement
6	59	M	6	C	2	Ischemic heart disease	Coronary artery bypass
7	14	F	0.8	P	1	Ebstein's anomaly	Bidirectional glen, right ventricular plication, tricuspid valve repair
8	32	F	1	C	1	Mitral incompetence	Mitral valve repair
9	42	F	3	C	1	Mitral stenosis	Mitral valve replacement
10	60	M	0.4	C	2	Mitral incompetence	Mitral valve repair
11	58	M	12	P	3	Ischemic heart disease	Coronary bypass grafting
12	64	M	7	C	2	Cardiomyopathy	Nil
13	66	M	6	C	4	Lone fibrillator	Nil
14	63	F	3	P	5	Lone fibrillator	Nil
15	56	M	13	C	2	Lone fibrillator	Nil
16	52	M	10	P	3	Mitral incompetence	Mitral valve repair
17	65	M	10	C	3	Lone fibrillator	Nil
18	53	M	10	C	4	Lone fibrillator	Nil
19	48	M	3	C	4	Lone fibrillator	Nil
20	39	M	5	C	0	Tricuspid incompetence	Tricuspid valve repair
21	70	M	10	P	4	Ischemic heart disease	Coronary bypass grafting
22	50	M	2	P	2	Cardiomyopathy	Nil
23	66	M	5	P	2	Idiopathic	Nil
24	50	M	2	P	2	Idiopathic	Nil
25	52	M	5	C	3	Cardiomyopathy	Nil
Mean	52 ± 13		6 ± 4		3 ± 1		

In a number of cases the pattern of lesions was altered in the posterior left atrium. The purpose of these changes was to reduce the risk of postoperative atrial flutter evident in the initial patients. The changes were based on the results of postoperative electrophysiological studies (performed on the preceding patients), the details of which are documented below. None of the alterations proved to be superior to the original design.

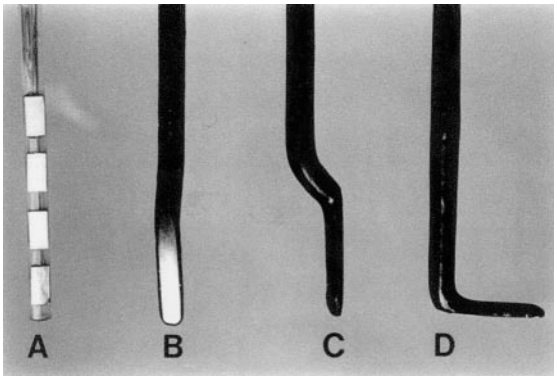


Figure 2. Devices used for radiofrequency ablation. The multielectrode probe **(A)** and three unipolar probes **(B–D).**

Electrophysiological studies. Electrophysiological studies were performed on 21 patients preoperatively. These studies were used to exclude causes of AFib treatable by standard catheter methods, to assess sinus node function and to measure intra-atrial conduction times. Postoperative electrophysiological studies were performed 6 to 12 months postoperatively or earlier if electrophysiological assessment was required for postoperative arrhythmias. To date, 15 patients have had late postoperative electrophysiological studies after a mean of 9 ± 7 months.

Catheter electrodes were placed in the high right atrium (quadpolar), adjacent to the bundle of His (quad- or

octapolar), in the coronary sinus (decapolar), around the tricuspid annulus (TA) (20 polar) and in the left atrium either by a transeptal or retrograde aortic/mitral approach. The study protocol included measurement of the P-wave duration, atrial and regional conduction times and determination of sinus node recovery times at six different pacing cycle lengths (CLs) (300 to 600 ms). Regional conduction times were determined by measuring the time from the earliest endocardial atrial activation to standard sites in the right atrium, septum and coronary sinus during sinus rhythm. The total activation time was determined in sinus rhythm by measuring the time from the earliest to the last endocardial activation in any of the standard catheter positions.

Vigorous attempts were made to induce AFib in all patients to determine whether the procedure truly prevented induction of sustained AFib. First, rapid burst pacing was delivered using an interval of 280 ms and decrementing by 10- to 20-ms intervals in subsequent bursts until 1:1 atrial capture was lost. Then very rapid atrial pacing was performed using 10-s bursts at CLs of 20 to 100 ms with broad-pulse widths (2 to 10 ms) and high current (20 mA) stimuli.

Tests of lesion integrity were performed by pacing in each atrial corridor close to the atrioventricular junction and recording the activation sequence in the adjacent corridor. Pacing caused a caudocranial activation sequence in the paced corridor, but where lesions were intact the activation sequence in the adjacent corridors was craniocaudal.

In cases where arrhythmias were induced the activation sequence and response to pacing were studied for localization and to establish the mechanism. Regularity of each arrhythmia was determined by calculating the standard deviation of 10 consecutive CLs. The morphology of the intracardiac electrograms, CL and surface electrocardiograms (ECGs) were used to classify the induced arrhythmia as AFib or AFlut. Arrhythmias with constant CL (SD $<$ 10 ms), uniform intracardiac electrogram morphology and distinct flutter waves on the surface ECG were classified as AFlut. Activation sequences were studied to identify possible macroreentrant circuits. Large areas of the atria could be excluded from involvement in the tachycardia mechanism because of varying degrees of inter- and intra-atrial conduction block. Pacing was used to identify concealed or manifest entrainment. At the end of a train of burst pacing the return cycles were studied to help identify areas that were part of a macroreentrant circuit.

Statistics. Paired or unpaired Student *t* tests were used for comparisons within or between groups. A significance level of 5% was used throughout. Continuous variables are expressed as means ± SD. Cycle lengths, activation times and echocardiographic parameters are expressed as means ± SD.

The study was approved by the Western Sydney Area Health Service Human Ethics Committee and conducted in a manner conforming to the Ethical and Scientific Principles set out by the National Health and Medical Research Council of Australia. Informed consent was obtained before inclusion of subjects in the study, and institutional guidelines were observed.

RESULTS

Operative details. The concurrent surgical procedures are provided in Table 1. The mean duration of bypass was 158 ± 37 min and the mean cross clamp time was 117 ± 29 min. There were two postoperative deaths (patients 7 and 16) attributed primarily to the coexistent pathology and concurrent surgical procedures.

Postoperative cardiac arrhythmias. During a mean follow-up period of 16 ± 10 months clinical AFib and AFlut were documented in two patients (9%) and AFlut only was documented in a further 20 patients (87%). Seventeen of these patients (74%) experienced atrial tachyarrhythmias prior to discharge from hospital. In the 72 h after surgery, eight patients (38%) had periods of sinus arrest or bradycardia requiring temporary pacing. One required long-term pacing for sinus node dysfunction. At the most recent follow-up, 12 patients (52%) were asymptomatic (no symptoms or documented arrhythmias for at least three months) without medications. Another three (13%) were asymptomatic on medication (sotalol [n = 2], amiodarone [n = 1]). Eight patients (35%) continued to experience tachycardia. Four from this group underwent His bundle ablation and insertion of a permanent pacemaker. Six patients (26%) were taking antiarrhythmic medications.

Characterization of postoperative arrhythmias. In the 15 patients studied electrophysiologically in the postoperative period, 37 different AFlut morphologies occurred either spontaneously (51%) or were induced by programmed stimulation. In three patients, AFib was also either present spontaneously (n = 1) or inducible (n = 2). The CL of these arrhythmias varied widely from 123 ms (AFib) to 439 ms (stable AFlut). The mean CL of the 37 AFluts was 223 ± 25 ms.

Entrainment was possible in 65% of the inducible arrhythmias. The mean CL of the entrainable arrhythmias was longer than that of the nonentrainable arrhythmias, but this difference did not achieve statistical significance (248 ± 35 ms vs. 202 ± 34 ms, respectively).

Detailed studies showed clear deficiencies in lines of radiofrequency ablation in six patients. Flutter was made possible in one patient by a discontinuity in the tricuspid annulus-inferior vena cava isthmus lesion close to the tricuspid valve annulus. The macroreentrant circuit passed up the right atrial free wall and down the septum in a clockwise direction (as seen from the left anterior oblique view). The lesion was completed by catheter radiofrequency ablation. Subsequently, the clinical arrhythmia was no longer inducible and has not recurred.

JACC Vol. 35, No. 2, 2000
February 2000:442–50

In the other five cases the deficiencies were sited at the point where the posterior left atrial lesions met the atrioventricular junction. Flutter circuits in these patients used these residual connections to circulate around the lines of ablation. Radiofrequency ablation using standard transvascular catheter techniques was also used successfully to interrupt the flutter circuit in all five cases. The site of successful ablation was always inside the coronary sinus at a point corresponding to the position of the original endocardial linear lesion in the posterior left atrium. An example of AFlut due to discontinuity in a line of ablation is illustrated in Figure 3.

Figure 4 shows the endocardial recordings and site of successful radiofrequency ablation in the patient likely to have had a focal tachycardia mechanism (microreentrant or automatic). Although this tachycardia may have been due to increased automaticity, the delay of at least 70 ms in activation between the distal and proximal electrode pair (approximately 10 mm) did indicate profoundly delayed local conduction that might have supported a microreentrant circuit. The tachycardia was successfully ablated by application of radiofrequency energy within the right superior pulmonary vein.

In nine cases it was not possible to define a discrete deficiency in a line of ablation or a critical isthmus suitable for radiofrequency ablation. Activation sequence mapping and entrainment studies in these cases suggested circuits or foci involving the interatrial septum or adjacent areas. The mean CL of the clinical AFlut in these patients was 206 ± 38 ms compared with 281 ± 83 ms for those in whom AFlut was successfully ablated ($p = 0.07$). There was no significant difference in the regularity or the presence of an excitable gap in the two groups of AFlut.

Atrial activation times and sinus node function. No significant postoperative delay was observed in the total atrial activation time (103 ± 10 ms, preoperatively, 108 ± 14 ms, postoperatively), the P-wave duration (147 ± 10 ms, preoperatively, 142 ± 16 ms, postoperatively) or the regional activation times. One patient not tested preoperatively (because of refractory AFib) required a permanent pacemaker postoperatively after experiencing symptomatic bradycardia. The remaining patients had normal sinus node recovery times postoperatively.

Atrial contractility. Echocardiograms were performed on all patients preoperatively and had been performed on 83% of patients postoperatively. However, most studies were performed during arrhythmias. Echocardiograms in sinus rhythm were available in seven patients preoperatively (28%) and nine patients postoperatively (39%). The postoperative studies were performed 122 ± 80 days after surgery. There was a reduction in the A/E ratio of the mitral valve flows caused by an increase in the E-wave velocity and a downward trend in A-wave velocity; details are shown in Table 2. The left atrial filling fraction was 50 ± 13% preoperatively and 25 ± 2% postoperatively ($p = 0.02$). There was no significant reduction in the right atrial filling fraction.

DISCUSSION

Even under direct vision at open heart surgery using optimal techniques for radiofrequency ablation we found that endocardial radiofrequency ablation tends to leave discontinuities in lines of ablation, especially in the regions of the posterior left atrioventricular groove and interatrial septum. These discontinuities are proarrhythmic and cause both clinical and inducible AFlut.

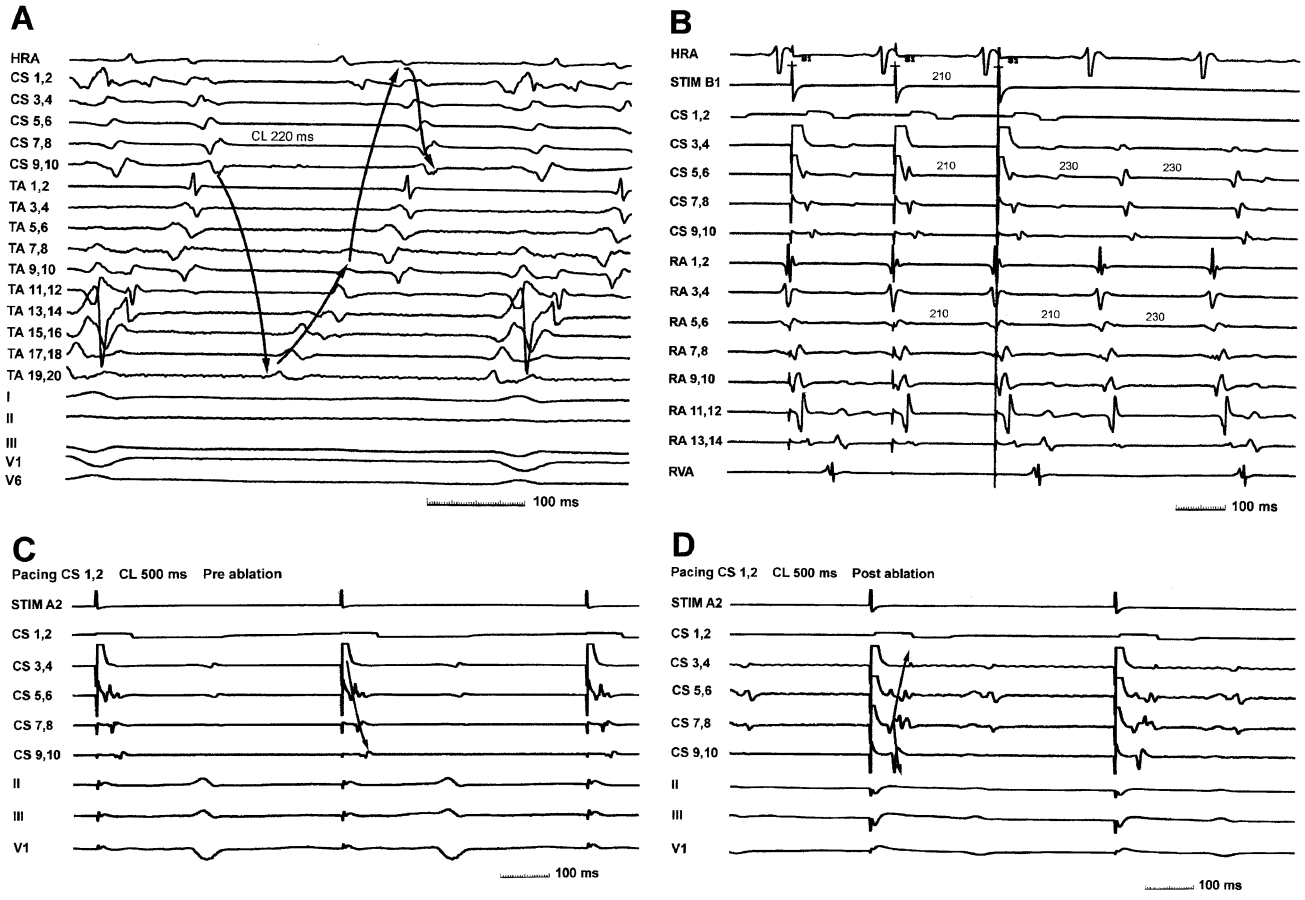
Mechanism for residual AFlut. In six cases we demonstrated that postoperative AFlut was due to a discontinuity in a line of ablation at the left posterior atrioventricular junction or the inferior vena cava–TA isthmus. A discontinuity in a linear radiofrequency lesion leaves an isthmus of surviving myocardium that permits reentry around the line of ablated tissue. In these patients the mechanism of reentry was clearly established by activation mapping and entrainment studies. Abolition of flutter by radiofrequency ablation at the site of discontinuity provided convincing confirmation of this mechanism. After successful radiofrequency ablation at the site of discontinuity it was possible to demonstrate conduction block at the site of the completed radiofrequency lesion. This form of reentry involves two or more adjacent corridors. In these cases the procedure itself created favorable conditions for AFlut to occur. Endocardial lines of ablation, therefore, have the potential to be proarrhythmic if radiofrequency lesions are not transmural or continuous.

Focal mechanisms. We demonstrated a focal mechanism in one patient with a symptomatic postoperative tachycardia. Tachycardias similar to this have previously been described during attempts to cure AFib using catheter techniques (3,11).

It was not clear in our case whether the focal mechanism had been present preoperatively or created by the surgery. These foci are hard to identify during sustained AFib. Clearly, if a focal tachycardia is present it would be an advantage to attempt focal ablation prior to performing the more extensive Maze-type procedure. More studies are required to characterize those patients who might be suitable for focal radiofrequency ablation of AFib.

Postprocedural AFlut was more common than reported after the Cox Maze procedure. This is presumably because surgical incisions are less likely to contain discontinuities, and the common site for focal tachycardias (11), the posterior left atrium, is isolated by the Cox maze procedure.

Atrial contractility. The pattern of lesions used in this study was designed to minimize delays in atrial electrical depolarization and optimize postoperative atrial mechanical function. We demonstrated a reduction in the mitral Doppler A/E ratio, suggesting left atrial function was impaired after radiofrequency ablation. Impaired atrial mechanical



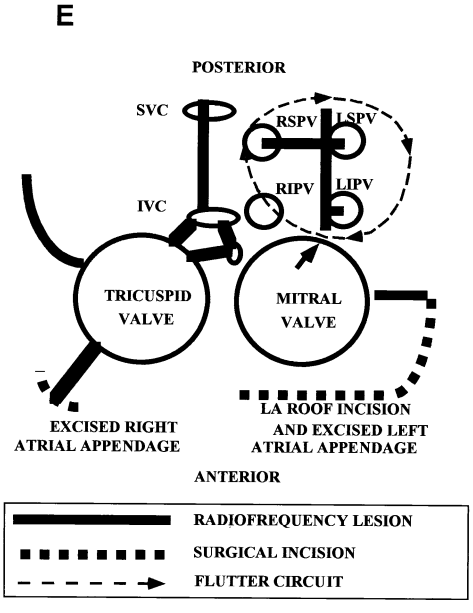


Figure 3. Postoperative AFlut due to reentry around a lesion produced by radiofrequency ablation. **(A)** The pattern of endocardial activation during AFlut. Note that activation of the coronary sinus was from distal to proximal and activation of the septum was from low to high as recorded in the proximal electrode pairs of the tricuspid annulus (TA) catheter (proximal to distal). The TA catheter is in a proximal position with electrodes 16–20 close to the septum. The delay in conduction at TA 15,16 is due to the inferior vena cava to superior vena cava line of ablation, which diverts activation anteriorly around the superior vena cava. The larger delay between TA 11,12 and the distal TA electrodes is due to activation being diverted around the excised atrial appendage to the area lateral to the crista. There is, unexpectedly, minimal delay in conduction along the coronary sinus where a posterior left atrial line of ablation was placed intraoperatively. **(B)** Concealed entrainment of atrial electrograms during pacing of the distal coronary sinus during AFlut. Atrial activation in the two most distal coronary sinus electrograms is not visible owing to stimulus artifact. There is intact conduction along the coronary sinus from distal to proximal through the line of ablation. The right atrial decapole catheter has been moved from the TA position to the posterior right atrium. **(C)** and **(D)** The difference in coronary sinus activation during distal coronary sinus pacing before and after radiofrequency ablation. The 5-mm decapolar coronary sinus catheter is positioned with electrode 10 (the most proximal) at the coronary sinus os. Ablation was performed from within the coronary sinus at a site corresponding with electrode 3. This induced a reversal of the activation sequence in the proximal coronary sinus. Following this application of radiofrequency energy it was no longer possible to induce AFlut with burst pacing. **(E)** The likely circuit of this particular AFlut and the successful site of ablation **(arrow).**

function has been well documented after the maze procedure or its modifications relying predominantly on atrial incisions to achieve electrical compartmentalization (12–19). Left atrial contraction was absent in 27% to 39% of patients after the maze procedure. In those in whom atrial contractility was present, the A/E ratio and atrial filling

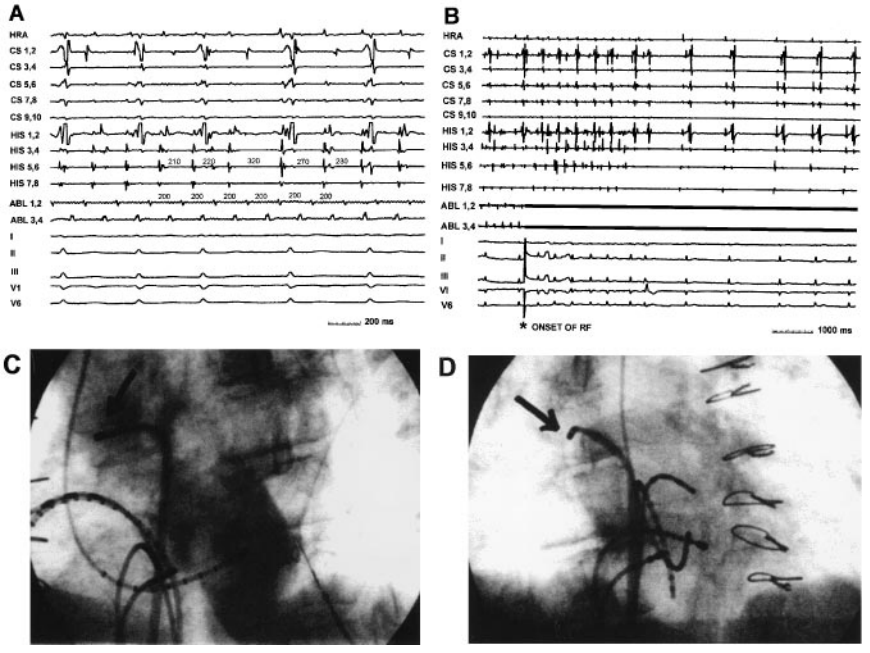


Figure 4. Postoperative tachycardia with focal mechanism. **(A)** Electrograms recorded from a number of endocardial sites before successful focal ablation can be seen. This arrhythmia was rapid (CL 200 ms) and regular at the site of successful ablation (right superior pulmonary vein) in contrast to the irregular but well-formed atrial electrograms at other atrial sites. This was consistent with second-degree conduction block between the focus and more distant atrial sites. **(B)** Termination of the tachycardia 3 s after the onset of the successful radiofrequency application. **(C)** and **(D)** X-ray images showing the position of the successful radiofrequency energy application **(arrows).**

fractions were reduced compared to normal values. A reduction in atrial contractility has not previously been reported after radiofrequency ablation for AFib. The mechanism for this reduction in contractility is unknown, but we have shown that this is not due to delays in electrical activation of the atria. One possible mechanism is loss of myocardial mass. Mitchell et al. (20) showed that in a dog model a pattern of linear lesions to cure AFib may cover 20% to 22% of the endocardial surface. Preliminary trials performed in animals demonstrated that the ablation technique used in the present study produced lesions 7.7 ± 0.4 mm wide that covered a similar proportion of the endocardial surface. Another possible mechanism for the reduction in atrial contractility is splinting of the atria by the linear scars.

Table 2. Mitral and Tricuspid Valve Flow Patterns

	Preoperative	Postoperative	p Value
Mitral Valve			
E wave	0.70 ± 0.12	1.18 ± 0.29	0.01
A wave	0.71 ± 0.14	0.49 ± 0.12	0.08
A/E	0.98 ± 0.22	0.56 ± 0.10	0.04
Tricuspid Valve			
E wave	0.67 ± 0.30	0.69 ± 0.13	0.8
A wave	0.58 ± 0.30	0.49 ± 0.12	0.7
A/E	0.86 ± 0.08	0.81 ± 0.09	0.54

Implications for the development of catheter ablation procedures for cure of AFib. Even when radiofrequency current is applied endocardially under direct vision at open heart surgery with a bloodless field, and a relatively large electrode and electrode temperature accurately controlled at 80° to 90°C, it is still common to have residual discontinuities in these lesions. We found that the problem areas include the atrioventricular groove in the posterior left atrium, the posterior septal space and probably also the interatrial septum. When discontinuities occur they are highly proarrhythmic, causing AFlut that is often resistant to antiarrhythmic therapy. Because discontinuities occur in the comparatively ideal conditions of intraoperative radiofrequency ablation under direct vision, we expect they will continue to be a major problem with current catheter ablation techniques for AFib.

Failure of lesions at the posterior left atrial atrioventricular junction to be transmural is likely to be due to deep bundles of myocardium located in the fat of the atrioventricular groove or in the wall of the coronary sinus. Conduction along the coronary sinus between the right atrium and the posterior left atrial wall was recently studied by Antz et al. (21). The anatomy of this region is illustrated by the pathology specimen shown in Figure 5. Ablation from the left atrial endocardial surface should interrupt fibers in the posterior wall of the left atrium and usually interrupt fibers in the anterior wall of the coronary sinus. However, the deeper fibers in the posterior wall of the coronary sinus or

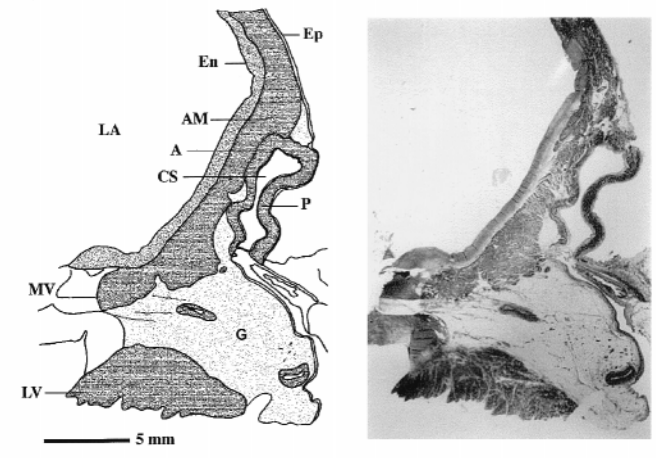


Figure 5. The human atrioventricular groove at the posterior left atrium. A = anterior wall of coronary sinus; AM = atrial myocardium; CS = coronary sinus; LV = left ventricle; En = endocardial surface of the left atrium; Ep = epicardial surface of the left atrium; G = fat of the atrioventricular groove; LA = left atrium; MV = mitral valve annulus; P = posterior wall of coronary sinus.

small muscle bundles coursing through the atrioventricular fat may be spared.

A number of other problems unique to the catheter technique will need to be overcome before high success rates can be achieved. It is difficult to attain reliably good contact between the ablating electrode and the endocardial surface during catheter-based procedures, especially with the drag technique. This can directly cause discontinuities in lesions. It is more likely that the lesions will not be overlapped accurately during catheter ablation due to the limitations of current imaging and guidance techniques. Lesions placed end to end or using a drag technique can become discontinuous because of minor changes in the catheter position during lesion formation.

In this study we introduced a new pattern of lesions designed to minimize delays in electrical activation of the atria. This pattern of lesions usually provides normal or near-normal atrial contraction and may be easier to reproduce using catheters than the original patterns described by Cox.

Conclusions. A radial pattern of radiofrequency linear lesions is associated with normal or near-normal electrical activation of the atrial myocardium but may be associated with loss of atrial contractility. This study demonstrated that in most cases AFib may be abolished, at least in the medium term, by this pattern of endocardial radiofrequency ablation. However, even when optimal techniques are used to ensure that deep linear radiofrequency lesions are delivered accurately to the atrial myocardium, AFlut commonly occurs postoperatively and often results in significant symptoms.

At least two mechanisms were responsible. Most commonly AFlut was due to a residual discontinuity in a line of ablation. In the remainder, reentry within a corridor, multiple discontinuities in a line of ablation, or focal mechanisms were the major possibilities. These mechanisms are likely to be important after catheter radiofrequency ablation procedures and will lead to a high rate of residual AFlut unless current techniques are modified to solve the problems we have described. Additional catheter radiofrequency ablation can be used successfully to ablate residual AFlut after initial attempts at endocardial radiofrequency ablation for AFib.

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