Left Atrial “Stunning” Following Radiofrequency Catheter Ablation of Chronic Atrial Flutter

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Objectives. This study examined the effect of radiofrequency ablation (RFA) on left atrial (LA) and left atrial appendage (LAA) function in humans with chronic atrial flutter (AFL).

Background. Atrial stunning and the development of spontaneous echocardiographic contrast (SEC) is a consequence of electrical cardioversion of AFL to sinus rhythm. This phenomenon has been termed “stunning” and is associated with thrombus formation and embolic stroke. Radiofrequency ablation is now considered to be definitive treatment for chronic AFL, but whether this procedure is complicated by LA stunning is unknown.

Methods. Fifteen patients with chronic AFL undergoing curative RFA underwent transesophageal echocardiography to evaluate LA and LAA function and SEC before and immediately, 30 minutes and 3 weeks after RFA. To control for possible direct effects of RFA on atrial function, seven patients undergoing RFA for paroxysmal AFL were also studied. In this group, RF energy was delivered in sinus rhythm and echocardiographic parameters were assessed before and immediately and 30 minutes following RFA.

Results. Chronic AFL: Mean arrhythmia duration was 17.2 ± 13.3 months. Twelve patients (80%) developed SEC following RF energy application and reversion to sinus rhythm. LAA velocities decreased significantly from 54.0 ± 14.2 cm/s in AFL to 18.0 ± 7.1 cm/s in sinus rhythm after arrhythmia termination (p < 0.01). These changes persisted for 30 minutes. Following 3 weeks of sustained sinus rhythm, significant improvements in LAA velocities (68.9 ± 23.6 vs. 18.0 ± 7.1 cm/s, p < 0.01) and mitral A-wave velocities (49.8 ± 10.3 vs. 13.4 ± 11.2 cm/s, p < 0.01) were evident and SEC had resolved in all patients. Paroxysmal AFL: Radiofrequency energy delivered in sinus rhythm had no significant effect on any of the above indexes of LA or LAA function and no patient developed SEC following RFA.

Conclusions. Radiofrequency ablation of chronic AFL is associated with significant LA stunning and the development of SEC. Left atrial stunning is not secondary to the RF energy application itself. Sustained sinus rhythm for 3 weeks leads to resolution of these acute phenomena. Left atrial stunning occurs in the absence of direct current shock or antiarrhythmic drugs, suggesting that its mechanism may be a function of the preceding arrhythmia rather than the mode of reversion.

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The cardioversion of atrial fibrillation to sinus rhythm is associated with transient mechanical dysfunction of the left atrium (LA) and left atrial appendage (LAA) and the development of spontaneous echocardiographic contrast (SEC). This phenomenon has been termed “stunning” and is considered an important factor for thromboembolic stroke following reversion of atrial fibrillation to sinus rhythm (1–5). For this reason, it is recommended that patients undergo 3 to 4 weeks of postreversion anticoagulation to reduce the risk of stroke (6).

Atrial stunning may also occur following the electrical cardioversion of atrial flutter (AFL) to sinus rhythm (2,7,8). Although this stunning may be less than that which occurs following reversion of atrial fibrillation, recent evidence demonstrates that the risk of thromboembolic events in patients with AFL and who are cardioverted from AFL is significant (7,9,10).

Curative radiofrequency ablation (RFA) is now considered by many to be the treatment of choice for patients with chronic AFL (11–13). Despite the increasing experience with AFL ablative techniques, whether these procedures are associated with atrial stunning is unknown and uniform recommendations for periprocedural anticoagulation have not been adopted.

The aim of this study was to use transesophageal echocardiography (TEE) to determine whether atrial stunning occurs following reversion of chronic AFL to sinus rhythm when radiofrequency (RF) energy is the mode of reversion. In those patients in whom atrial mechanical dysfunction was observed following reversion to sinus rhythm, we also investigated whether this was reversible in the short and medium term by

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Abbreviations and Acronyms

AFL = atrial flutter  
DC = direct current  
ECG = electrocardiographic, electrocardiography  
FAC = fractional area change  
LA = left atrial  
LAA = left atrial appendage  
RF = radiofrequency  
RFA = radiofrequency ablation  
SEC = spontaneous echocardiographic contrast  
TEE = transesophageal echocardiography

performing repeat transesophageal echocardiographic evaluations 30 minutes and 3 weeks following RFA.

The mechanism of atrial stunning following direct current (DC) reversion is incompletely understood. Some evidence suggests that atrial stunning occurs as a result of the preceding atrial arrhythmia and that atrial stunning represents a form of tachycardia-mediated atrial cardiomyopathy (14–16). Other data implicates the DC shock as a significant contributor to the mechanism of stunning (1,3,4,17–20). The present study provided an opportunity to determine whether atrial stunning occurs in the absence of a DC shock or acutely administered antiarrhythmic agents, thereby gaining some insight into the mechanisms of this phenomenon. To control for possible direct effects of RF energy on atrial function, we also studied a group of patients undergoing RFA for paroxysmal AFL. These patients had been in sinus rhythm for at least 4 weeks before the procedure.

Methods

Study patients. The study population comprised 15 male patients undergoing RFA of chronic AFL. Demographic details are presented in Table 1. The patients had documented AFL for 17.2 ± 13.3 months and antiarrhythmic drugs had failed a mean of 2.4 ± 0.6 months before RFA. Structural heart disease was present in six patients and absent in nine patients other than the presence of atrial dilation. All patients gave written informed consent to the study, which was approved by the Board of Medical Research of Royal Melbourne Hospital.

Patient preparation. All antiarrhythmic drugs other than amiodarone were discontinued more than five half-lives before RFA and were not recontinued (Table 1). The single patient taking amiodarone had therapy permanently discontinued four days before RFA. Radiofrequency ablation was performed under general anesthesia to facilitate performance of sequential TEE analyses without patient discomfort. A standardized general anesthetic with endotracheal intubation and mechanical ventilation was administered with propofol induction followed by anesthesia maintained with the volatile agent isoflurane and the muscle relaxant atracurium.

Echocardiographic analysis. Following general anesthesia, TEE was performed using a 5-MHz phased array multiplane probe connected to a Hewlett-Packard Sonos 2500 ultrasound system (Hewlett-Packard, Palo Alto, CA). Images were recorded on 0.5 in. super VHS tape and analyzed off-line using Hewlett-Packard computerized software.

Left atrial and LAA function were assessed at four defined time points: 1) during chronic AFL immediately before ablation; 2) immediately on reversion of AFL to sinus rhythm; 3) 30 minutes following reversion of AFL to sinus rhythm; and 4) three weeks following the RFA procedure.

Left atrial appendage flow velocities were assessed using pulsedwave Doppler imaging, placing the sample volume 1 cm into the mouth of the LAA. A multiplane probe was used and the LAA was scanned in planes from 0° to 130° to establish an angle at which maximal flow velocities could be obtained. This angle was retained for all subsequent analyses. The angles employed in this study ranged from 0° to 95°. All velocity measurements were performed off-line and averaged over 20 consecutive cardiac cycles. Left atrial appendage areas were determined by planimetry and measured at the angle at which LAA flow velocities were obtained. Over 20 consecutive cardiac cycles, consecutive maximal and minimal LAA areas were assessed and LAA fractional area change (FAC) was calculated using the following formula (1): $\text{FAC} = \frac{\text{maximum area} - \text{minimum area}}{\text{maximum area}}$.

Left atrial function was assessed following AFL termination with TEE pulsedwave interrogation of the mitral inflow at the level of the mitral valve leaflet tips (points 2 to 4). Doppler envelopes for the mitral A-wave velocities during AFL were difficult to obtain and were excluded from the data set. Mitral A-wave velocities were analyzed off-line and averaged over 20 cardiac cycles.

### Table 1. Clinical and Echocardiographic Characteristics of the Patient Population Undergoing Radiofrequency Ablation of Chronic Atrial Flutter

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>15</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>57.6 ± 14.0</td>
</tr>
<tr>
<td>Men</td>
<td>15</td>
</tr>
<tr>
<td>Duration of atrial flutter (mo)</td>
<td>17.2 ± 13.3 (range 2–24)</td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>3</td>
</tr>
<tr>
<td>ASD repair</td>
<td>1</td>
</tr>
<tr>
<td>Mitral valve replacement</td>
<td>1</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>1</td>
</tr>
<tr>
<td>LA size (cm)</td>
<td>4.8 ± 0.4</td>
</tr>
<tr>
<td>LVEDD (cm)</td>
<td>5.3 ± 0.4</td>
</tr>
<tr>
<td>LVESD (cm)</td>
<td>3.2 ± 0.5</td>
</tr>
<tr>
<td>LVFS (%)</td>
<td>33.1 ± 7.6</td>
</tr>
</tbody>
</table>

ASD = atrial septal defect; LA = left atrial; LVEDD = left ventricular end-diastolic diameter; LVESD = left ventricular end-systolic diameter; LVFS = left ventricular fractional shortening.
Spontaneous echocardiographic contrast was defined by the appearance of swirling clouds of echogenicity in the left atrium and LAA distinct from white noise artifact. Gain settings were reduced sequentially to distinguish SEC from noise and maintained for the study duration. SEC was assessed independently by two observers at the four time points and graded as absent, mild or marked (21). Spontaneous echocardiographic contrast analysis was performed off-line and observers were unaware of patient identification, AFL type (chronic vs. paroxysmal), the timing of TEE in relation to RFA and the other's interpretations. Changes in SEC grading were resolved by consensus. Left atrial appendage thrombus was defined as a mass adherent to the wall of the LAA with either independent motion or different echogenicity. Attention was directed at differentiating pectinate muscles from potential thrombus (8).

All recordings at the time of the RFA (points 1 to 3) were performed during periods of apnea to minimize potential velocity variations caused by mechanical ventilation. Transesophageal echocardiography at 3 weeks following the RFA was performed with intravenous benzodiazepine sedation; velocity measurements during apnea were not possible.

Atrial flutter definition. Typical AFL was defined by its characteristic 12-lead surface electrocardiogram. This was confirmed at electrophysiologic study on the basis of established criteria (11–13). These include: 1) counterclockwise activation sequence in the right atrium in the frontal plane, and 2) manifest entrainment from the high right atrium and concealed entrainment from within the subeustachian isthmus. Chronicity was defined as the presence of AFL for at least 14 days. AFL duration was defined as the time from initial 12-lead ECG diagnosis to the time of the RFA procedure.

Electrophysiological evaluation and ablation. Intracardiac catheters were positioned following the initial TEE evaluation to avoid physical termination of AFL before collection of baseline echocardiographic data. Catheters were placed in the coronary sinus, His bundle and tricuspid annulus positions as previously described in detail (11). An 8F ablation catheter was positioned in the subeustachian isthmus and was used for entrainment mapping and delivery of RF energy as previously described (11). In all patients RFA was initially performed during AFL. After electrophysiologic confirmation of the flutter mechanism, an anatomic approach was used to create a line of conduction block between the inferior tricuspid annulus and the eustachian ridge (11,12). The end point for successful ablation was the demonstration of bi-directional isthmus block using well known techniques (11–13). Atrial flutter reinduction employing rapid atrial pacing following isthmus block was not attempted. At no time during the procedure were DC shocks or intravenous antiarrhythmic drugs administered.

All antiarrhythmic drugs discontinued before the ablation procedure were not recommended following successful RFA. All patients received anticoagulation with warfarin until follow up at 3 weeks. An international normalized ratio of 2.0 to 2.5 was considered satisfactory.

Control Group

Study patients. To assess the direct effect of RF energy application on atrial mechanical function, seven male patients (age 62.4 ± 14.9 years) with paroxysmal AFL were also evaluated. These patients were included with the sole purpose of determining if the procedure itself and the application of RF energy depressed atrial function. Paroxysmal AFL was defined by a history of 3 or more episodes of typical AFL confirmed on a 12-lead electrocardiogram as described previously. No patient described symptoms attributable to paroxysmal AFL in the four weeks before RFA. All patients in the control group were in sinus rhythm before ablation and underwent the same preoperative preparation as described previously for the group with chronic AFL.

Paroxysmal AFL had been present for 14.8 ± 7.1 months and a mean of 2.8 ± 0.8 antiarrhythmic drugs could not be used before RFA. Two patients had ischemic heart disease and one patient had had a Ross procedure for aortic regurgitation. Structural heart disease other than LA dilation was absent in four patients. The mean LA dimension was 4.5 ± 0.4 cm and the mean left ventricular end-diastolic dimension was 5.1 ± 0.5 cm. Before RFA, three patients were taking amiodarone and four patients were taking sotalol; these drugs were discontinued 1 week before RFA.

Echocardiographic analysis. Following general anesthesia and before intracardiac catheter placement, TEE was performed as described above. Left atrial and LAA function at three defined time points were evaluated (1–3): 1) in sinus rhythm before ablation; 2) in sinus rhythm, immediately following isthmus block after successful application of RF energy in the low right atrial isthmus and 3) 30 minutes following application of RF energy. Transesophageal echocardiography at three weeks was not performed.

Electrophysiologic evaluation and ablation. Intracardiac catheter placement in the control group was the same as that described previously for patients with chronic AFL. Pacing from the coronary sinus ostium at a cycle length of 600 ms was used during RFA and success was defined as previously. These conventional techniques have been described elsewhere (11–13).

Statistical analysis. All hemodynamic variables are reported as mean ± SD. A repeated measures analysis of variance was used to compare continuous variables. Schefle’s F test was used for multiple comparisons. Statistical significance was established at p < 0.05. Seven patients were included in the paroxysmal AFL control group. This part of the study was prospectively designed to detect a mean fall in velocity of 14 ± 13 cm/s with 80% power (2.20,22).

Results

Chronic Atrial Flutter Group

Radiofrequency ablation. The mean AFL cycle length of the study population was 255 ± 38 ms. In all 15 patients, RFA was successful in terminating AFL and bi-directional isthmus
block was achieved in all patients. The mean fluoroscopy time for the procedure was 28.7 ± 11.7 min. The mean number of RF energy applications was 18 ± 11.

**Left atrial appendage function (Figs. 1 and 2).** The mean LAA velocity before RFA was 54.0 ± 14.2 cm/s. Immediately following termination of AFL by RF energy, LAA velocity decreased significantly to 18.0 ± 7.2 cm/s during sinus rhythm (p < 0.01). Re-evaluation 30 minutes following termination of AFL disclosed no further significant change in LAA velocity (17.4 ± 8.3 vs 18.0 ± 7.2 cm/s).

All patients were re-evaluated 3 weeks following RFA. All patients manifested sinus rhythm as evidenced by discrete P waves on the 12-lead surface electrocardiogram. The mean LAA velocity at 3 weeks was 68.9 ± 23.6 cm/s, which was significantly greater than that in sinus rhythm following AFL termination (18.0 ± 7.1 cm/s, p < 0.01), and also significantly greater than that during AFL before RFA (54.0 ± 14.2 cm/s, p < 0.05). Recovery of LAA mechanical function was seen in all patients by 3 weeks (Fig. 1).

**Left atrial appendage fractional area change (Fig. 3).** The mean preablation LAA FAC was 47.0 ± 23.8%. Fractional area change immediately following termination of AFL was significantly reduced to 24.5 ± 18.9% (p < 0.01). There were no significant changes in FAC in the subsequent 30 minutes following AFL termination (24.5 ± 18.9% vs. 26.8 ± 17.8%). Left atrial appendage FAC increased significantly from 24.5 ± 18.9% immediately after RFA to 68.1 ± 14.5% in sinus rhythm at 3 weeks (p < 0.01). Fractional area change at 3 weeks was also significantly greater than that during AFL before RFA (68.1 ± 14.5% vs. 47.0 ± 23.8%, p < 0.05).

**Left atrial cavity function (Fig. 4).** Mean mitral inflow A-wave velocity in sinus rhythm following AFL termination was 13.4 ± 11.2 cm/s. There was no significant difference between the velocity measured immediately following AFL and that of 30 minutes later (12.7 ± 11.3 cm/s). Left atrial cavity function exhibited improvements in contractility at 3 weeks. The mean mitral inflow A-wave velocity at 3 weeks was significantly greater than that immediately following AFL termination (49.8 ± 10.3 cm/s vs. 13.4 ± 11.2 cm/s, p < 0.01).

**Left atrial spontaneous echocardiographic contrast and thrombus.** Spontaneous echocardiographic contrast and LAA thrombus were absent in all patients before RFA and AFL termination. Immediately following AFL termination, 12 of 15 patients (80%) developed new LA SEC. Four patients developed marked SEC and eight developed mild SEC. These changes persisted at re-evaluation 30 minutes following AFL termination. No patient developed LA or LAA thrombus in the interval between AFL termination and reassessment at 30 minutes. The mean LAA velocity immediately following reversion in the 12 patients who developed LA SEC was 16.1 ± 7.1 cm/s. This velocity was not significantly different from that in the 3 patients who did not develop LA SEC (19.6 ± 8.7 cm/s).

Spontaneous echocardiographic contrast had resolved by the 3-week reevaluation in all 12 patients in whom SEC had
developed following RFA. No embolic events occurred during the 3-week follow-up period.

**Atrial cycle length.** There was no significant difference in atrial cycle length immediately following compared with 30 minutes following AFL termination (987 ± 220 ms vs. 997 ± 202 ms). The mean sinus rhythm rate demonstrated no significant difference from 60 ± 16 beats/min at AFL termination to 62 ± 10 beats/min at 3 weeks.

**Control Group**

**Radiofrequency ablation.** Radiofrequency ablation of paroxysmal AFL was successful in all patients. Mean fluoroscopy time for the procedure was 29.2 ± 12.1 min and the mean number of RF energy applications was 19 ± 9. Atrial flutter was not induced and DC shocks and antiarrhythmic drugs were not administered during the procedure.

**Left atrial appendage function (Fig. 5).** Before RFA, the mean LAA velocity in sinus rhythm was 55.0 ± 10.5 cm/s. Mean LAA velocities in sinus rhythm immediately (58.8 ± 12.2 cm/s) and 30 minutes following RFA (57.5 ± 10.5 cm/s) were not significantly different from baseline.

**Left atrial appendage fractional area change.** Mean LAA FAC before RFA was 60.0 ± 10.5%. Immediately and 30 minutes following RFA, FAC was 58.0 ± 10.8% and 55.8 ± 11.4%, respectively. These values were not significantly different from baseline.

**Spontaneous echocardiographic contrast.** No patient demonstrated SEC or thrombus before, immediately following, or 30 minutes following RFA.

**Sinus rate.** There were no significant differences in heart rate between baseline (65 ± 10 beats/min), immediately after (65 ± 8 beats/min) and 30 minutes after RFA (68 ± 10 beats/min) values.

**Discussion**

**New findings.** This study presents important new information regarding the development of atrial mechanical stunning following termination of chronic AFL.

First, significant atrial stunning occurs following RFA of chronic AFL. Both LAA and LA velocities became markedly depressed and a significant number of patients developed SEC. Second, this phenomenon occurred in the absence of a DC shock or the administration of antiarrhythmic agents and was not observed in patients undergoing RFA of paroxysmal AFL. These observations strongly implicate the preceding atrial arrhythmia as an important causative factor in the develop-
ment of atrial stunning. Finally, LA and LAA mechanical function recover toward normal values within 3 weeks following RFA of chronic AFL. These findings have important implications both for the clinical management of patients undergoing RFA of chronic AFL and for the mechanism of development of atrial stunning.

Comparison with previous studies. Left atrial stunning following reversion of atrial flutter to sinus rhythm. Atrial stunning immediately following DC cardioversion of atrial fibrillation to sinus rhythm has been demonstrated by several studies (1–5,16,20,22,23). Few studies, however, have examined atrial function following the reversion of AFL to sinus rhythm (2,7,23).

Grimm et al. (2) compared atrial function before and immediately following DC cardioversion of atrial fibrillation in 44 patients and AFL in 19 patients using TEE. Significant LA stunning developed in both groups but was less marked in those patients with AFL. New or increased SEC developed in 21% of the patients with AFL.

Atrial mechanical dysfunction following DC cardioversion of AFL has been demonstrated in two studies using trans-thoracic echocardiography to evaluate transmitral flow. Irani et al. (7) studied 40 patients with AFL for a mean duration of 4 weeks. Mitral A-wave velocities were absent in 11 of the 40 patients (28%) following cardioversion. Jordaens et al. (23) used transthoracic echocardiography to assess mitral A-wave velocities following DC cardioversion in 16 patients and overdrive pacing in six patients with AFL. All patients received disopyramide before and after reversion; atrial stunning was observed in both groups. These studies are limited by the fact that the mitral A wave cannot be accurately assessed during AFL and because LAA function and SEC require TEE for determination.

Although the above studies have evaluated the effect of DC reversion of AFL on atrial function, we used TEE to demonstrate that chronic AFL leads to significant atrial stunning when RF energy is the mode of arrhythmia termination. This is important because curative RFA is now considered by many to be the treatment of choice for patients with chronic AFL (11–13). In the present study, LAA velocities were depressed to a level that is considered to represent high risk for thrombus formation; new SEC was observed in 80% of patients (1–4). The greater degree of atrial stunning and higher incidence of SEC compared with that observed by Grimm et al. (2) may reflect the longer duration of AFL in patients included in the present study.

Unlike larger studies that have demonstrated predisposing factors to the development of atrial stunning following reversion of atrial fibrillation (3), small patient numbers in the present study precluded a determination of risk factors for SEC following RFA of chronic AFL. This should be an important focus of future research in larger patient cohorts with AFL.

Recovery of left atrial mechanical function over time. Several studies have examined the recovery of LA function following cardioversion of atrial fibrillation to sinus rhythm (3,5,24–26). Manning et al. (3) employed serial transthoracic echocardiography to identify arrhythmia chronicity as a major determinant of the recovery of atrial function following DC cardioversion of atrial fibrillation. Serial TEE was used to demonstrate the return of LA and LAA function 7 days following the endocardial defibrillation of atrial fibrillation by Omran et al. (5).

Recovery of LA mechanical function over time following reversion of AFL to sinus rhythm has not been studied extensively. Jordaens et al. (23) used transthoracic echocardiography to evaluate recovery of LA function following DC cardioversion of AFL. Following cardioversion of AFL to sinus rhythm, late diastolic filling peak A-wave velocities increased from 28 cm/s after 1 h to 39 cm/s after 24 h and reached 54 cm/s after 6 weeks.

Transesophageal echocardiography has been used previously to evaluate LA mechanical function immediately following electrical and spontaneous cardioversion of AFL to sinus rhythm (2,16). To our knowledge, long-term recovery of LA and LAA function and the evolution of SEC following reversion of AFL to sinus rhythm has not been studied previously using TEE. The present study has shown that atrial stunning resolves and LAA function returns to clinically normal values following 3 weeks of sustained sinus rhythm. In addition, SEC that develops following reversion of AFL resolves after 3 weeks of sustained sinus rhythm. These findings suggest that: 1) sustained sinus rhythm following curative ablation of chronic AFL may lead to normalization of LA and LAA function and 2) the tachycardia-mediated atrial cardiomyopathy that may be responsible for atrial stunning in AFL is reversible (14).

Mechanism of atrial stunning. Both low energy internal and higher energy external defibrillation of atrial fibrillation may be associated with atrial stunning (1–5,20). Whether DC energy causes direct damage to atrial myocardium and hence, is a major determinant of stunning is unclear. Fatkin et al. (4) demonstrated a relationship between the number and energy level of DC shocks delivered in atrial fibrillation and the development of SEC on reversion to sinus rhythm. Manning et al. (24) suggested that electrical cardioversion of atrial fibrillation was associated with more significant atrial stunning than that occurring during pharmacologic cardioversion. In addition, other studies have provided direct biochemical and ultrastructural evidence of myocardial damage following delivery of DC shocks for ventricular arrhythmias (27,28). In contrast, other investigators have shown no significant differences in atrial function following cardioversion achieved through either DC shock or pharmacologic agents (29), that external DC shocks delivered during ventricular tachycardia or sinus rhythm are not associated with atrial stunning (30,31) and that DC cardioversion of atrial fibrillation is not associated with an elevation of specific cardiac enzymes such as cardiac troponin I (32).

This study has shown that significant atrial stunning occurs following the termination of chronic AFL in the absence of DC shocks or the administration of antiarrhythmic drugs. Furthermore, the observed effect was not mediated by RF energy because atrial stunning was not observed in patients with sinus
rhythm undergoing the same procedure for paroxysmal AFL. Although the etiology of atrial stunning may be multifactorial (4,14–20,24,33) results from the present study suggest that atrial stunning following RFA of chronic AFL may result from a tachycardia-mediated atrial cardiomyopathy rather than the mode of cardioversion (14).

Clinical implications. The development of reversible LA mechanical stunning and SEC following RFA of chronic AFL suggests that a potential for thromboembolic stroke exists for which prophylactic anticoagulation should be considered. These observations suggest that a three-week period of prophylactic anticoagulation may be reasonable following RFA of chronic AFL.

Emerging evidence demonstrates that chronic AFL is not a benign condition. Several studies have demonstrated a significant risk of thromboembolic events in patients with chronic AFL (9,10). In addition, a high prevalence of SEC and/or atrial thrombus was found in patients with sustained AFL by Irani et al. (7). The present study demonstrates that not only is reversion of chronic AFL to sinus rhythm associated with atrial stunning but that this reverses over a period of 3 weeks. Indeed, despite a mean AFL duration of 17 months, LAA velocities returned to levels approaching that of the normal population after 3 weeks of sinus rhythm. These data suggest that the risk of thromboembolic events in patients with chronic AFL might be effectively reduced by curative RFA. This would extend the indications for this procedure to include not only symptomatic patients but all patients with chronic AFL.

The demonstration of significant atrial stunning following reversion of AFL to sinus rhythm in the absence of DC shock suggests that atrial stunning is likely to occur irrespective of the mode of termination. Thus, spontaneous-, pharmacologic- and pace-terminated reversion of chronic AFL may be potentially associated with similar degrees of atrial stunning. Prophylactic anticoagulation should also be considered in these circumstances.

Study limitations. To determine whether atrial stunning following RFA of chronic AFL is reversible, TEE was performed immediately, 30 minutes and 3 weeks following RFA. A 30-minute period was selected to establish whether atrial stunning persisted in the short term. A 3-week interval separating the ablation procedure from the follow-up TEE was selected because it approximated the time anticoagulation is recommended following DC cardioversion of atrial fibrillation (6). It was not feasible to perform more frequent TEE examinations in the intervening period; therefore, the exact time course of recovery of mechanical function could not be determined.

Patients with paroxysmal AFL were included only to determine if the application of RF energy during sinus rhythm had an effect on LA function. A direct comparison with the group with chronic AFL would have required a much larger group of patients with paroxysmal AFL.

Conclusion. Transesophageal echocardiography before and following RFA of chronic AFL has provided insight into the mechanical changes manifested in the left atrium on reversion to sinus rhythm without cardioverting DC shock. After RFA of chronic AFL, significant atrial mechanical dysfunction occurs with the development of SEC. Sinus rhythm for 3 weeks following RFA allows recovery of atrial function with the resolution of SEC. Because atrial stunning and the development of SEC are strongly associated with embolic stroke, prophylactic anticoagulation for 3 weeks should be considered following RFA of chronic AFL. As atrial stunning was observed in the absence of DC shock or antiarrhythmic agents, this study suggests that atrial stunning results from properties of the preceding atrial arrhythmia rather than the mode of reversion.

References
occurs after electrical cardioversion, but not after chemical cardioversion [abstract]. J Am Coll Cardiol 1997;29:292A.


