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Left Atrial Appendage "Stunning" After Electrical Cardioversion of Atrial Flutter: An Attenuated Response Compared With Atrial Fibrillation as the Mechanism for Lower Susceptibility to Thromboembolic Events

RICHARD A. GRIMM, DO, FACC, WILLIAM J. STEWART, MD, FACC, KRISTOPHER L. ARHEART, EDD, JAMES D. THOMAS, MD, FACC, ALLAN L. KLEIN, MD, FACC

Cleveland, Ohio

Objectives. **This study sought to determine whether left atrial appendage stunning occurs in patients with atrial flutter and to compare left atrial appendage function in the pericardioversion period with that in patients with atrial fibrillation.**

Background. **Left atrial appendage stunning has recently been proposed as a key mechanistic phenomenon in the etiology of postcardioversion thromboembolic events in atrial fibrillation. Atrial flutter is thought to be associated with a negligible risk of thromboembolic events; therefore, anticoagulation is commonly withheld before and after cardioversion in these patients.**

Methods. **Sixty-three patients with atrial flutter (n = 19) or atrial fibrillation (n = 44) underwent transesophageal echocardiography immediately before and after electrical cardioversion. In addition to assessing the presence of thrombus and spontaneous echo contrast, we measured left atrial appendage emptying velocity and calculated shear rates by pulsed wave Doppler and two-dimensional echocardiography.**

Results. **Patients with atrial flutter exhibited greater left atrial appendage flow velocities before cardioversion than those with atrial fibrillation (42 ± 19 vs. 28 ± 15 cm/s [mean ± SD], $p < 0.001$). Left atrial appendage shear rates were also higher in patients with atrial flutter (103 ± 82 vs. 59 ± 37 s^{-1}, $p < 0.001$). After cardioversion, left atrial appendage flow velocities decreased compared with precardioversion values in patients with atrial fibrillation (28 ± 15 before to 15 ± 14 cm/s after cardioversion, $p < 0.001$) and atrial flutter (42 ± 19 to 27 ± 18 cm/s, respectively, $p < 0.001$). Shear rates decreased from 59 ± 37 before cardioversion to 30 ± 31 s^{-1} after cardioversion in atrial fibrillation ($p < 0.001$), and from 103 ± 82 s to 65 ± 52 s^{-1}, respectively ($p < 0.001$), in atrial flutter. This decrease in flow velocity from before to after cardioversion occurred in 36 (82%) of 44 patients with atrial fibrillation and 14 (74%) of 19 with atrial flutter. The impaired left atrial appendage function after cardioversion was less pronounced in the group with atrial flutter (27 ± 18 cm/s for atrial flutter vs. 15 ± 14 cm/s for atrial fibrillation, $p < 0.001$). New or increased spontaneous echo contrast occurred in 22 (50%) of 44 patients with atrial fibrillation versus 4 (21%) of 19 with atrial flutter ($p < 0.05$).**

Conclusions. **Left atrial appendage stunning also occurs in patients with atrial flutter, although to a lesser degree than in those with atrial fibrillation. These data suggest that patients with atrial flutter are at risk for thromboembolic events after cardioversion, although this risk is most likely lower than that in patients with atrial fibrillation because of better preserved left atrial appendage function.**

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It is widely believed that patients with atrial flutter are less susceptible to embolic complications of cardioversion than those with atrial fibrillation. However, although some investigators (1) have suggested that this is the result of a more synchronous atrial activity during atrial flutter, the mechanism of this reduced embolic risk is currently undefined. Left atrial appendage stunning after electrical cardioversion of atrial fibrillation has recently been proposed as a mechanism for thromboembolism in the immediate postcardioversion period (2). This stunning phenomenon has been demonstrated by Doppler echocardiography to consist of impaired left atrial appendage function and increased atrial spontaneous echo contrast. The theoretic mechanism of postcardioversion thrombogenesis has acquired support from a recent publication (3) that reported on embolic events in 17 patients with negative results on a precardioversion transesophageal echocardiographic examination for thrombus. Because a similar mechanism for thromboembolic events in patients with atrial flutter seems likely and, if established, could alter prevailing

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Address for correspondence: Dr. Richard A. Grimm, The Cleveland Clinic Foundation, 9500 Euclid Avenue, Department of Cardiology, Desk F-15, Cleveland, Ohio 44195-5064. E-mail: grimmr@cesmtp.ccf.org.

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attitudes toward the susceptibility to thromboembolic complications of cardioversion (and in turn the need for anticoagulation), we elected to study left atrial appendage function before and after cardioversion in a group of patients with atrial flutter and compared them with patients with atrial fibrillation, using transesophageal Doppler echocardiography. The purpose of this study was therefore threefold: 1) to determine whether left atrial appendage stunning occurred in atrial flutter; 2) to compare the magnitude of left atrial appendage stunning in patients with atrial flutter with that in patients with atrial fibrillation; and 3) to gain insight into the mechanism for the reduced thromboembolic risk in atrial flutter versus fibrillation by analyzing left atrial appendage function.

Methods

Patients. We performed transesophageal Doppler echocardiography before and after successful electrical cardioversion in 63 patients with atrial fibrillation (n = 44) and atrial flutter (n = 19) of at least 2 days in duration in whom a transesophageal echocardiogram was requested to rule out the presence of left atrial thrombi. All patients were enrolled and studied before the initiation of the Assessment of Cardioversion Utilizing Transesophageal Echocardiography (ACUTE) trial (4), and therefore patient selection was not influenced by this study recruitment. Informed consent was obtained before the procedure, and the study was approved by the Institutional Review Board of the Cleveland Clinic Foundation. Twenty of these patients were part of the original series describing left atrial appendage stunning in atrial fibrillation and have been reported on previously (2). The presenting rhythm was confirmed by 12-lead electrocardiography before transesophageal echocardiography. Patients found to have atrial thrombi detected at the time of transesophageal echocardiography as well as those with unsuccessful cardioversion were excluded from the study. Transesophageal echocardiography was performed using commercially available equipment (Hewlett-Packard Sonos 1500 or 1000 or Acuson 128 XP/10) equipped with a 5-MHz phased array biplane transducer. All patients were examined in a fully equipped electrophysiology laboratory. After obtaining consent, 1% lidocaine viscous plus 1% cetocaine topical spray was used to anesthetize the oropharynx. Patients were subsequently sedated using Versed (1 to 4 mg) and Demerol (12.5 to 50 mg) intravenously as needed, after which the transesophageal echocardiographic probe was inserted into the esophagus. After a complete transesophageal echocardiographic examination, with special attention given to left atrial and atrial appendage anatomy and function, patients were anesthetized with brevitol (methohexital, 0.5 to 1.0 mg/kg body weight), with the total duration of anesthesia lasting ~5 to 10 min. After ensuring adequate anesthesia, electrical cardioversion was performed (5) using a Zoll device and 200 J of energy as the initial charge for patients with atrial fibrillation and 100 J of energy for patients with atrial flutter. Once successful cardioversion was achieved and cardiopulmonary stability assumed, the transesophageal echocardiographic probe was reinserted, and reexamination of the left atrial cavity and appendage was performed. On completion of the echocardiographic examination, each patient was monitored until recovery from sedation and anesthesia was confirmed, and patients were observed in the hospital until the following morning.

Echocardiographic analysis. Left atrial cavity and appendage anatomy and function were evaluated both before and after electrical cardioversion, with special attention given to identifying spontaneous echo contrast or thrombi, or both. Left atrial appendage function was assessed before and after cardioversion using pulsed Doppler echocardiography by placing the sample volume 1.5 cm into the mouth of the atrial appendage in the basal transverse plane at the level of the aortic valve. Peak flow velocities at end-diastole were measured and averaged over six cardiac cycles for patients in atrial fibrillation and flutter before cardioversion and over three cardiac cycles for all patients in sinus rhythm after cardioversion. Left atrial appendage areas were measured by planimetry, and atrial appendage diameters were measured off-line in the basal transverse plane. Diameters were obtained at the level of the sample volume (1.5 cm into the mouth of the atrial appendage) so as to obtain a radius measurement for the calculation of shear rate.

Left atrial appendage shear rates were calculated using peak velocity data (V_m) and left atrial appendage diameters (LAA_d) using a formula derived from Poiseuilles' law. Assuming a parabolic flow profile across the diameter of the left atrial appendage (unpublished data), we estimated that shear rates would be greatest along the periphery of the cavity. Therefore, shear rates (SR) were calculated using the equation

$$SR = \frac{2 \times V_m}{LAA_d/2}.$$

Left atrial cavity function was assessed after cardioversion by pulsed Doppler interrogation of mitral inflow (with the sample volume placed at the leaflet tips) and measurement of the A wave peak velocity. The mitral A wave velocities were then averaged over three cardiac cycles.

Spontaneous echo contrast was defined as dynamic intracavitary echoes with a characteristic swirling pattern distinct from white noise artifact. Gain settings were decreased in a stepwise manner to exclude white noise artifact due to excessive gain. These settings were adjusted for optimal visualization of spontaneous echo contrast and maintained for the postcardioversion study. The degree of spontaneous echo contrast was categorized independently by two different observers (R.G., A.K.) as absent, mild or severe based on the system described by Daniel et al. (6) and Beppu et al. (7). *Mild spontaneous echo contrast* was defined as being present if dynamic intracavitary microechoes were seen only with high gain, whereas *severe spontaneous echo contrast* was present if spontaneous contrast was noted with low gain. The detection and grading of spontaneous echo contrast was reviewed before and after cardioversion, on-line by two separate experienced echocardiographers (R.G., A.K.) who had no knowledge of the other

interpretations. The determination of increased intensity or the development of new smoke after cardioversion was made if new spontaneous contrast was observed or if an increase from mild to severe spontaneous contrast was agreed on by consensus.

Determination of intraobserver and interobserver variation in spontaneous contrast. The timing of the observation was blinded only for the off-line analysis because blinding of the precardioversion versus postcardioversion observation during the on-line analysis was not possible. The results for the interobserver and intraobserver variation for the detection of left atrial spontaneous echo contrast were published previously in a smaller group of patients and found to be 91% and 84%, respectively (2).

Statistical methods. Dichotomous, ordinal and continuous variables comprised the data set that was analyzed. The Fisher exact test compared groups for dichotomous variables; the Wilcoxon test analyzed ordinal variables; and the *t* test was used for continuous variables. A repeated measures analysis of variance with planned comparisons was used to analyze preconversion with postconversion velocities and shear rates. Logistic regression was performed to model the occurrence of left atrial appendage stunning and the development of increased spontaneous echo contrast. Analyses with p values ≤ 0.05 were considered significant.

Table 1. Clinical and Echocardiographic Characteristics of Patients With Atrial Fibrillation or Atrial Flutter Undergoing Electrical Cardioversion

	Atrial Fibrillation (n = 44)	Atrial Flutter (n = 19)	p Value
Age (yr)	65 ± 12	66 ± 8	0.546
Male/female	14/30	9/10	0.250
AF duration (mo)	6.8 ± 8.5	4.3 ± 4.9	0.143
≥ moderate MR	4	1	1.000
LVEF (%)	53 ± 16	47 ± 17	0.141
LVEDD (mm)	54 ± 11	56 ± 11	0.405
LVESD (mm)	37 ± 13	41 ± 13	0.22
LA size (mm)	50.1 ± 6.9	48.5 ± 4.9	0.37
LAA area (cm^2)	6.9 ± 2.8	5.9 ± 1.7	0.105
Heart rate (beats/min)	95 ± 21	98 ± 27	0.640
Hypertension	13 (30%)	5 (26%)	0.792
Valvular disease	13 (30%)	3 (16%)	0.204
ASHD	4 (9%)	6 (32%)	0.051
HOCM	6 (14%)	0 (0%)	0.008
Idiopathic	5 (11%)	2 (11%)	0.922
DCM	3 (7%)	3 (16%)	0.329

Data presented are mean value ± SD or number (%) of patients. AF = atrial fibrillation; ASHD = atherosclerotic heart disease; DCM = dilated cardiomyopathy; HOCM = hypertrophic obstructive cardiomyopathy; LA = left atrial; LAA = left atrial appendage; LVEDD = left ventricular end-diastolic dimension; LVEF = left ventricular ejection fraction; LVESD = left ventricular end-systolic dimension; MR = mitral regurgitation.

Results

Study group. We studied 63 consecutive patients with atrial fibrillation (n = 44) and atrial flutter (n = 19) of at least 2 days in duration undergoing transesophageal echocardiography before scheduled cardioversion. All patients had successful cardioversions as defined by the maintenance of sinus rhythm for at least 24 h. Patients with atrial fibrillation and atrial flutter were similar in mean age of 65 ± 12 and 66 ± 8 years, respectively; however, duration of the arrhythmia was longer for those with atrial fibrillation, although this difference was not statistically significant (6.8 ± 8.5 vs. 4.3 ± 4.9 months for atrial flutter, p = 0.143). Suspected underlying etiologies for the atrial arrhythmias included hypertension, valvular heart disease, hypertrophic obstructive cardiomyopathy, dilated cardiomyopathy, idiopathic and coronary artery disease. There was a greater proportion of patients with valvular heart disease and hypertrophic cardiomyopathy in the atrial fibrillation group than in the atrial flutter group, whereas the atrial flutter group had a greater proportion of patients with coronary artery disease and dilated cardiomyopathy (Table 1). Left ventricular function was similar in both groups: mean ejection fraction 53 ± 16% (range 20% to 78%) versus 47 ± 17% (range 15% to 72%) for atrial fibrillation versus atrial flutter (p = 0.141). In reference to the cardioversion procedure itself, patients with atrial fibrillation received higher energy levels (328 ± 211 vs. 185 ± 64 J, $p < 0.001$) as well as a greater number of total shocks (1.5 ± 0.6 vs. 1.1 ± 0.3, p = 0.017). At the completion of a 4-week follow-up period, no thromboembolic events were reported, although 42 (95%) of 44 patients with atrial fibrillation and 13 (68%) of 19 with atrial flutter were receiving anticoagulation at the time of the cardioversion. In the atrial fibrillation group, heparin was used in 11 of 44 patients, warfarin in 31 of 44 and both heparin plus warfarin in 4 of 44 before and immediately after cardioversion. In the atrial flutter group, heparin was used in 5 of 19 patients, warfarin in 8 of 19 and both heparin and warfarin in 1. Additionally, 11 patients with atrial fibrillation had a previous cerebrovascular event, whereas no patient with atrial flutter had such an event.

Before cardioversion. At the time of presentation to the electrophysiology laboratory, patients with atrial flutter exhibited greater left atrial appendage flow velocities than those with atrial fibrillation (42 ± 19 vs. 28 ± 15 cm/s, $p < 0.001$). Left atrial appendage shear rates were also higher in patients with atrial flutter (103 ± 82 vs. 59 ± 37 s^{-1}, $p < 0.001$). Spontaneous echo contrast was more prevalent in patients with atrial fibrillation (80% vs. 42% in atrial flutter, $p < 0.001$), and left atrial diameter was not significantly different between groups. Although left atrial appendage area was larger in the atrial fibrillation group, this difference was not significantly different (6.9 ± 2.8 vs. 5.9 ± 1.7 cm^2, p = 0.105).

After cardioversion. After cardioversion, left atrial appendage flow velocities (Fig. 1) and shear rates (Fig. 2) decreased compared with precardioversion values in patients with atrial fibrillation as well as atrial flutter. In atrial fibrillation, the average flow velocities decreased from 28 ± 15 before to 15 ± 14 cm/s after cardioversion ($p < 0.001$), whereas shear rates decreased from 59 ± 37 to 30 ± 31 s^{-1}, respectively ($p < 0.001$). Left atrial appendage stunning, which was defined as a

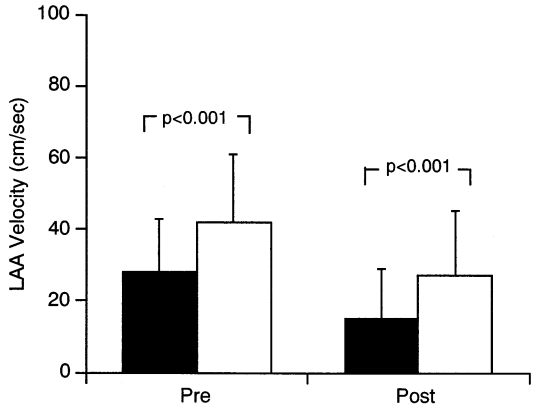

Figure 1. Left atrial appendage (LAA) flow velocity changes between atrial fibrillation **(solid bars)** and atrial **(open bars)** flutter before and after cardioversion. Left atrial appendage velocity decreased from before to after cardioversion in patients with atrial fibrillation and atrial flutter. Significant differences ($p < 0.001$) in left atrial appendage flow velocities are also observed between atrial fibrillation and atrial flutter, both before as well as after cardioversion.

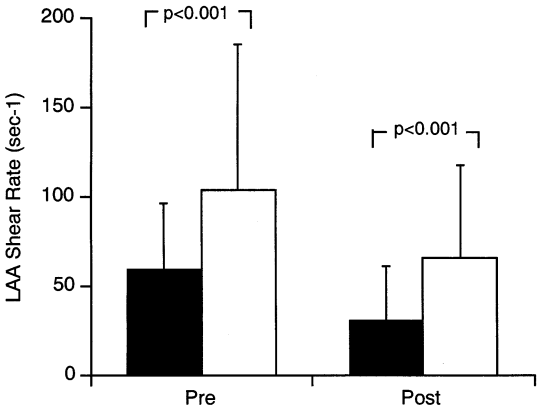

Figure 2. Effect of electrical cardioversion on left atrial appendage (LAA) shear forces between atrial fibrillation **(solid bars)** and atrial flutter **(open bars)** before and after cardioversion. Left atrial appendage shear rate decreased from before to after cardioversion in patients with atrial fibrillation and atrial flutter. Significant differences ($p < 0.001$) in left atrial appendage shear rates are also observed between atrial fibrillation and atrial flutter, both before as well as after cardioversion.

decrease in flow velocity from before to after cardioversion of at least 20%, occurred in 36 (82%) of 44 patients with atrial fibrillation (Fig. 3). In atrial flutter, left atrial appendage function also exhibited a decrease in flow velocity from before to after cardioversion (Fig. 4). During atrial flutter, flow velocities averaged 42 ± 19 cm/s; after conversion to sinus rhythm, flow velocities decreased to 27 ± 18 cm/s ($p < 0.001$). Shear rates also decreased in patients with atrial flutter from 103 ± 82 s^{-1} before to 65 ± 52 s^{-1} after conversion ($p < 0.001$). Overall, this stunning phenomenon was observed in 14 (74%) of 19 patients with pure atrial flutter (Fig. 3) and 5 of 7 patients with atrial flutter with no history of previous atrial fibrillation. Moreover, absolute left atrial appendage function after cardioversion was less depressed in the group with atrial flutter because left atrial appendage velocities were significantly greater in the atrial flutter group after cardioversion at 27 ± 18 cm/s than after cardioversion in the atrial fibrillation group with a mean velocity of 15 ± 14 cm/s ($p < 0.001$). The higher left atrial appendage velocities after cardioversion of atrial flutter were observed despite the finding of similar absolute differences in flow velocities from before to after cardioversion (14 ± 13 cm/s for atrial fibrillation, 17 ± 15 cm/s for atrial flutter, $p = 0.757$). This observation can therefore best be explained by the higher left atrial appendage velocities in patients with atrial flutter before cardioversion than in those with atrial fibrillation (Fig. 1), as opposed to a more significant stunning effect in patients with atrial fibrillation. Furthermore, left atrial cavity function after cardioversion exhibited better contractility during flutter than atrial fibrillation because mitral inflow A wave velocities after cardioversion were significantly higher in the group with atrial flutter (36 ± 24 cm/s) than in the group previously in atrial fibrillation (22 ± 17 cm/s, $p = 0.029$).

Left atrial spontaneous echo contrast. Before undergoing cardioversion, 35 (80%) of 44 patients with atrial fibrillation were found to have spontaneous echo contrast compared with only 8 (42%) of 19 with atrial flutter ($p < 0.002$). After electrical cardioversion, new or increased spontaneous contrast was detected in 22 (50%) of the 44 patients with atrial fibrillation versus only 4 (21%) of 19 with atrial flutter ($p = 0.032$) (Fig. 5).

Predictors of left atrial appendage stunning and increased spontaneous contrast. Stepwise logistic regression analysis was performed in an attempt to identify potential clinical or echocardiographic predictors of new spontaneous echo contrast or left atrial appendage stunning. Precardioversion clinical and echocardiographic variables significant at the 0.2 level by univariate analysis were included, and all models included age, gender, rhythm and left atrial size as confounders. The only significant predictor for new spontaneous echo contrast after cardioversion was atrial fibrillation because patients with atrial fibrillation were 4.7 times more likely to have increased spontaneous echo contrast than those with atrial flutter (95% confidence interval 1.3 to 17.6, $p = 0.021$). The logistic regression model using precardioversion variables did not identify any significant variables for predicting left atrial appendage stunning.

Discussion

Previous investigation from our laboratory (2) demonstrated the phenomenon of left atrial appendage stunning as a mechanism by which de novo thrombogenesis and subsequent thromboembolism could result after the successful electrical cardioversion of patients with atrial fibrillation. The present study confirms this finding in a larger group of patients in addition to elucidating similar but importantly different mechanistic information regarding patients with atrial flutter. In the present study, patients with atrial flutter also exhibited left atrial appendage stunning after cardioversion, although the

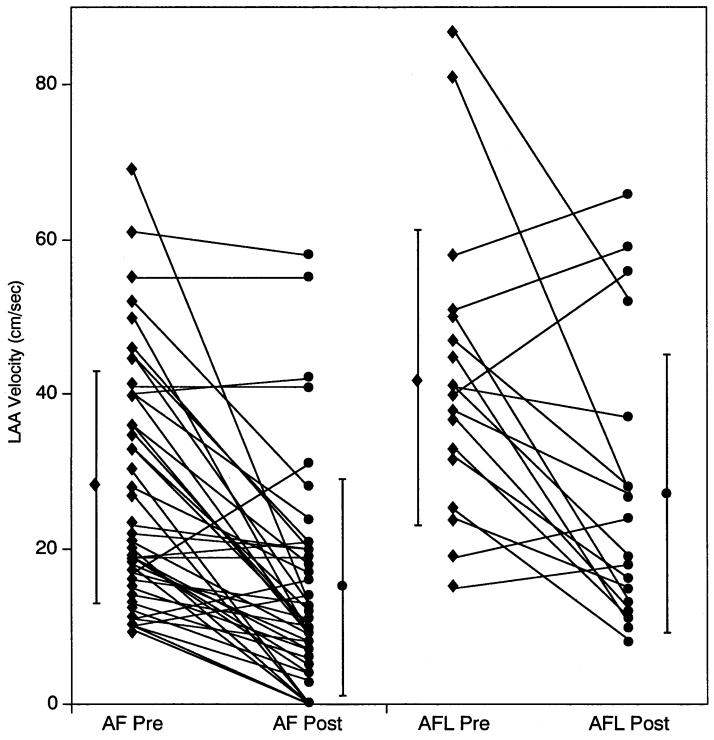

Figure 3. Effect of electrical cardioversion on left atrial appendage (LAA) flow velocity in patients with atrial fibrillation (AF) **(left)** and atrial flutter (AFL) **(right)** before (Pre) and after (Post) cardioversion. **Vertical bars** = mean value ± SD.

stunning phenomenon was significantly less pronounced than in those with atrial fibrillation. Additionally, postcardioversion left atrial appendage function is better preserved, in terms of flow velocities, in atrial flutter than in atrial fibrillation. Finally, some patients with atrial flutter also demonstrated increased spontaneous echo contrast immediately after cardioversion. However, this finding of increased spontaneous echo contrast was observed at a markedly reduced frequency in patients with

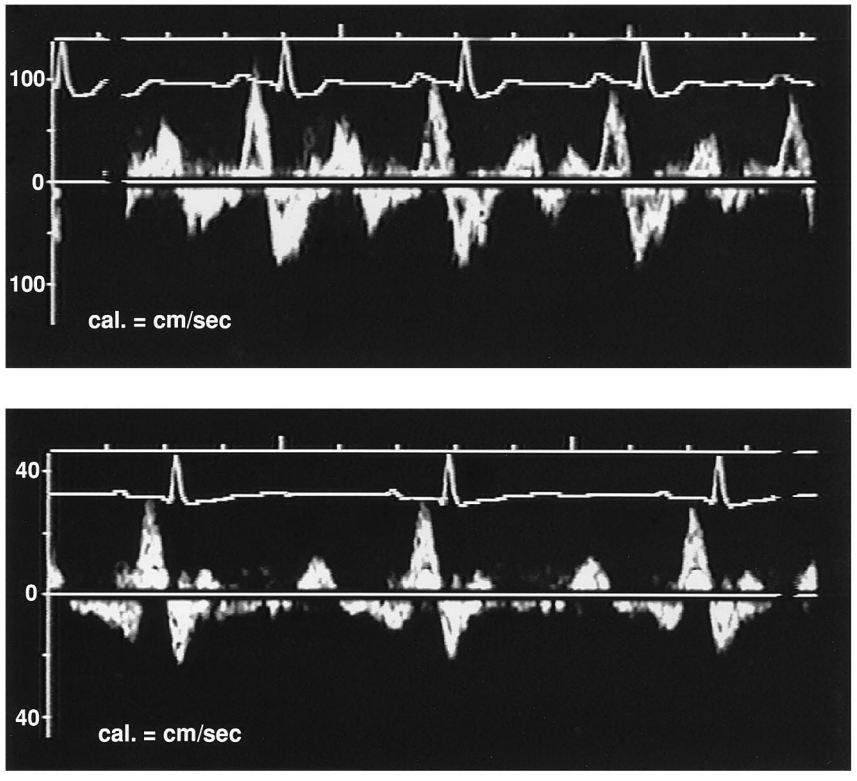

Figure 4. Representative left atrial appendage pulsed Doppler flows illustrating the "stunning" phenomenon after electrical cardioversion in a patient with atrial flutter. Left atrial appendage flow in atrial flutter before cardioversion **(top)** with "flutter waves" on the electrocardiographic tracing preceding each forward and reverse Doppler flow and left atrial appendage flow in sinus rhythm after cardioversion **(bottom)** with a p wave preceding the late diastolic forward and reverse flow. paper speed = 50 mm/s.

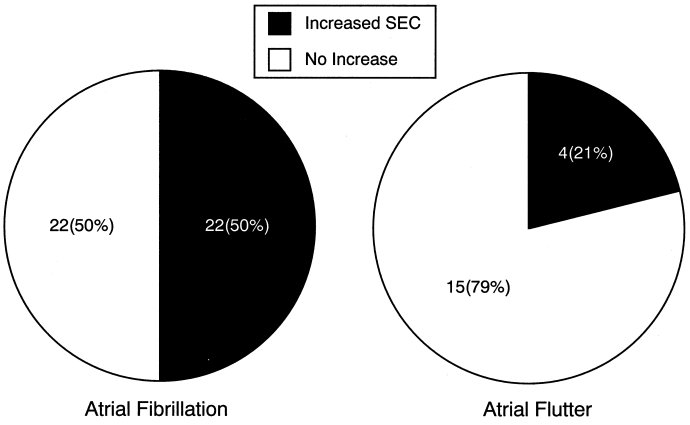

Figure 5. Frequency of new or increased spontaneous echo contrast (SEC), or both, after cardioversion in patients with atrial fibrillation **(left)** and atrial flutter **(right).** New or increased spontaneous contrast was detected in 22 (50%) of the 44 patients with atrial fibrillation versus only 4 (21%) of 19 with atrial flutter ($p = 0.032$).

atrial flutter than in those with atrial fibrillation. These results provide the first mechanistic support for historical data that suggest that patients with atrial flutter are less likely than those with atrial fibrillation (yet still prone) to experience thromboembolic complications after cardioversion.

Published reports to date on the cardioversion of atrial flutter strongly suggest that these patients are at a very low risk of thromboembolic events before and after cardioversion; hence the rationale of many physicians for avoiding the use of anticoagulation in patients with atrial flutter as well as the somewhat arbitrary tone of the current anticoagulation guidelines that advocate anticoagulation for atrial flutter solely in the presence of a history of fibrillation. However, review of the published reports reveals that relatively few patients with atrial flutter have been carefully studied. In 1965, Jensen et al. (8) studied 50 patients undergoing electrical cardioversion, 7 of whom were in atrial flutter, with no embolic events detected in either group. Two years later, Wikland et al. (9) reported on 74 patients undergoing electrical cardioversion, with 8 of the patients in atrial flutter. One mesenteric embolic event was reported; however, the underlying rhythm in this patient was not revealed. Fritz and Aberg (10) reported on 29 patients with atrial flutter undergoing electrical cardioversion in 1970, none of whom sustained an embolic event. Roy et al. (11) reported embolic events in 2 of 30 patients with atrial flutter, both of whom were not receiving anticoagulation at the time of cardioversion. Arnold et al. (12), from our institution, retrospectively evaluated 122 patients with atrial flutter of 454 undergoing electrical cardioversion. No embolic events were detected regardless of anticoagulation status. More recently, Pagadala et al. (13) reported a 9% incidence of thromboembolic events among 85 patients with atrial flutter, many of whom had a history of atrial fibrillation. Other investigators have combined patients with atrial flutter with those in atrial fibrillation in studies on outcomes after cardioversion; however, details regarding the exact numbers of patients in atrial flutter and a breakdown of event rates in flutter versus fibrillation are often omitted in these reports (14–17).

Similar to the paucity of data on thromboembolism after cardioversion of atrial flutter, few data exist on the prevalence of intraatrial thrombus and the risk for thromboembolism in atrial flutter. However, the prevailing notion has been to assume that intraatrial thrombus is a rare phenomenon. A recent study by Bikkina et al. (18) challenges traditional thinking and provides some interesting insight into this area of study. These investigators demonstrated a surprisingly high incidence of atrial thrombus in a group of 24 consecutive patients with atrial flutter admitted to the hospital. Intraatrial thrombus was detected by transesophageal echocardiography in 5 (21%) of 24 patients with atrial flutter versus 6 (3%) of 184 in a control group. Predictors of the presence of thrombus in this cohort included male gender, ejection fraction <40% and atrial flutter. Therefore, it would appear from this isolated report that the potential for thrombogenesis in atrial flutter may have been underestimated in the past.

Support for left atrial appendage stunning as a mechanism for thromboembolic events. Clinical evidence supporting left atrial appendage stunning and postcardioversion thrombogenesis was provided by Black et al. (3) who described 17 patients with atrial fibrillation and sustained thromboembolic events after cardioversion despite a transesophageal echocardiogram that demonstrated no evidence for thrombus. In a smaller series of patients with atrial flutter studied by Baruch et al. (19), two embolic events were observed after cardioversion, again despite a transesophageal echocardiogram that was negative for the presence of thrombus. Contrary to the traditionally described etiologic mechanism for thromboembolic events after cardioversion, as first proposed by Goldman et al. (20), that suggested that preexisting thrombus was the sole etiology of thromboembolic events, these more recent data (3,19) support postcardioversion thrombogenesis as a more important pathophysiologic mechanism than previously recognized. Furthermore, the physiologic data presented in our study suggest that left atrial appendage stunning is a likely mechanism for the observed thromboembolic episodes after cardioversion, as described in patients with atrial fibrillation as well as in those with atrial flutter. One might also argue that the high incidence of atrial appendage stunning and increase in spontaneous echo contrast intensity in both groups could be an argument against atrial stunning as a mechanism of

thromboembolic events given the rather low event rate of cardioversion-related embolic events of ~1.6%. However, these observations simply highlight circumstances (i.e., atrial appendage stunning) that appear to provide a mileu for thrombogenesis because frank thrombus was never identified, and anticoagulation was present in 95% and 68% of patients with atrial fibrillation and atrial flutter, respectively.

Because all patients included in the present study underwent electrical cardioversion, the question regarding the potential role of the electrical energy in the development of atrial appendage stunning requires discussion. Previous investigations implicated the electrical energy as the cause for atrial stunning (2,21); however, recent evidence (22) suggests that left atrial appendage stunning can be seen independent of the application of electrical energy. In the present study, two patients undergoing transesophageal echocardiography converted to sinus rhythm spontaneously during the transesophageal examination and demonstrated left atrial appendage stunning despite the absence of antiarrhythmic drugs or an electric shock. One of the patients was in atrial fibrillation and the other in atrial flutter. Furthermore, others investigators (23) have demonstrated that patients examined by transesophageal echocardiography before and after failed attempts at cardioversion of atrial fibrillation did not exhibit diminished fibrillatory flow velocities after the electric shock. Although it is possible that the electrical energy contributes to the atrial appendage stunning, these recent studies would suggest that it is unlikely to be the etiologic mechanism for this phenomenon.

Clinical implications. Current recommendations by the American College of Chest Physicians (1) with regard to anticoagulation for patients with atrial flutter in the pericardioversion period, suggest that anticoagulation is not required for patients with atrial flutter unless the patient has had a previous history of atrial fibrillation. The data presented in the present study demonstrate that the left atrial appendage stunning phenomenon occurred in atrial flutter, independent of a previous history of atrial fibrillation, suggesting that the possibility of postcardioversion thrombogenesis exists regardless of previous history of fibrillation. Although the existence of left atrial appendage stunning implies that all patients with atrial flutter are at a potential risk for thromboembolic events and therefore should be undergo anticoagulation (similar to those with atrial fibrillation), the duration of anticoagulation required after the procedure is likely to be significantly shorter than that required for atrial fibrillation. The basis for this hypothesis is that the return of left atrial appendage function after conversion to normal values is likely to be more rapid in atrial flutter than in atrial fibrillation. Return of left atrial appendage function beyond the immediate postcardioversion period was not examined in the present study, and therefore the time course for return to normal function remains unknown, hence a limitation of the current study. Manning et al. (24), studied the return of atrial cavity function in patients with acute and chronic atrial fibrillation by transthoracic echocardiography and demonstrated that mitral A wave inflow velocities return to near normal values earlier in patients with acute than in those with chronic atrial fibrillation. They therefore suggested that the required duration of anticoagulation after cardioversion is shorter in patients with acute versus chronic atrial fibrillation. For example, patients with acute atrial fibrillation and otherwise normal hearts may require anticoagulation for only 1 week after cardioversion, whereas those with chronic atrial fibrillation may need a full 4 weeks of anticoagulation because atrial function may not fully return to normal until the fourth week. Other factors, in addition to chronicity, are likely to affect the degree and duration of left atrial appendage stunning, such as the underlying pathology, left atrial size and left ventricular function. Unfortunately, the limited sample size in the study by Manning et al. (24) and the lack of long-term follow-up in our study preclude such risk stratification at the present time. Clearly, further investigation regarding return of left atrial appendage function after cardioversion is imperative to provide data to support tailoring the duration of anticoagulation therapy after cardioversion in patients with atrial fibrillation and atrial flutter.

Role of echocardiography in patients with atrial flutter undergoing cardioversion. Similar to its role in patients with atrial fibrillation (25), the utility of transesophageal echocardiography in patients with atrial flutter is dependent on the need to rule in or rule out the presence of thrombus. Because of the existence of atrial appendage stunning in atrial flutter, the exclusion of a thrombus before cardioversion does not obviate the need for anticoagulation after cardioversion (2,3,25), although it should lower the risk of cardioversion as one of the two potential mechanisms for thromboembolic events after cardioversion is excluded. Additionally, utilization of transesophageal echocardiography may allow earlier cardioversion and the avoidance of 3 to 4 weeks of anticoagulation before cardioversion (25,26).

Conclusions. The phenomenon of left atrial appendage stunning and the development of new or increased left atrial spontaneous echo contrast previously reported for patients with atrial fibrillation undergoing cardioversion also occur in patients with atrial flutter. These data suggest that the potential for the development of a thrombogenic milieu does exist in patients with atrial flutter as a result of left atrial appendage stunning, and therefore these patients should not be considered risk free for thromboembolic events after cardioversion. However, the likelihood of this embolic risk is probably significantly lower than that in patients with atrial fibrillation because of the attenuated degree of left atrial appendage stunning in atrial flutter compared with that in atrial fibrillation.

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