Impact of Electrical Cardioversion for Atrial Fibrillation on Left Atrial Appendage Function and Spontaneous Echo Contrast: Characterization by Simultaneous Transesophageal Echocardiography

RICHARD A. GRIMM, DO, WILLIAM J. STEWART, MD, FACC, JAMES D. MALONEY, MD, FACC, GERALD I. COHEN, MD, GREGORY L. PEARCE, MS, ERNESTO E. SALCEDO, MD, FACC, ALLAN L. KLEIN, MD, FACC

Cleveland, Ohio

Objectives. This study assessed the function of the left atrial appendage in the pericardioversion period to gain insights into mechanisms involved in thromboembolism after cardioversion of atrial fibrillation.

Background. Systemic embolization associated with electrical cardioversion of atrial fibrillation is thought to originate from the left atrium or left atrial appendage, or both. However, the mechanism involved is poorly understood.

Methods. We studied left atrial appendage function with transesophageal echocardiography in 20 patients with atrial fibrillation before and after successful electrical cardioversion. We measured left atrial appendage emptying and filling velocities by pulsed wave Doppler echocardiography, characterized Doppler emptying patterns, measured atrial appendage areas and assessed the presence or absence of spontaneous echo contrast or thrombus.

Results. Organized left atrial appendage function returned in 16 (80%) of 20 patients immediately after cardioversion. Atrial appendage emptying velocities before cardioversion were greater in patients without (0.39 ± 0.02 m/s) than in those with (0.25 ± 0.12 m/s) spontaneous echo contrast (p = 0.045). Furthermore, emptying velocities before cardioversion were significantly greater than late diastolic emptying velocities after cardioversion (0.31 ± 0.15 vs. 0.14 ± 0.12 m/s, p = 0.0001), as well as in both the group with (0.25 ± 0.12 vs. 0.13 ± 0.13 m/s, p = 0.001) and the group without (0.39 ± 0.02 vs. 0.15 ± 0.12 m/s, p = 0.01) spontaneous echo contrast. In addition, left atrial and atrial appendage spontaneous echo contrast developed in 4 of 20 patients and increased in intensity in 3 of 20 patients in the immediate postcardioversion period.

Conclusions. Organized left atrial appendage function returns in most patients immediately after cardioversion of atrial fibrillation. However, its function is impaired compared with that before cardioversion. Furthermore, spontaneous echo contrast increased in 7 (35%) of 20 patients after cardioversion. These observations suggest that stunned left atrial appendage function after cardioversion may predispose the chamber to thrombus formation, which may play a role in the mechanism involved in the occurrence of embolization after cardioversion.

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version. The purpose of this study is to 1) assess the relation between spontaneous echo contrast and left atrial appendage function in the pericardioversion period, and 2) determine what changes occur in appendage emptying velocities and size as a result of electrical cardioversion of atrial fibrillation.

**Methods**

**Study patients.** Transesophageal echocardiography was performed in 20 patients in whom atrial fibrillation had been present for \( \geq 3 \) days (mean 160 days, range 4 days to 2 years) immediately before and after successful cardioversion to sinus rhythm. Our study group consisted of 14 men and 6 women with a mean age of 65 ± 11 years. Informed consent was obtained in all cases and the study was approved by the Institutional Review Board of the Cleveland Clinic Foundation.

**Precardioversion echocardiographic examination.** Transesophageal echocardiography was performed using commercially available equipment (Hewlett-Packard Sonos 1000 or Acuson 128 XP/10) equipped with a 5-MHz phased array monoplane or biplane transducer. Patients were examined in a fully equipped electrophysiology laboratory. Topical cetracaine spray and viscous lidocaine solution were used to anesthetize the oropharynx. Patients were positioned in the left lateral decubitus position and then sedated with a combination of meperidine hydrochloride (25 to 50 mg) and midazolam hydrochloride (1 to 5 mg intravenously) before the probe insertion. Vital signs were monitored using continuous electrocardiographic (ECG) monitoring and an automated sphygmomanometer. Oxygen saturation was monitored utilizing a pulse oximeter. A complete transesophageal echocardiographic examination was performed, with attention given to imaging the left atrium and left atrial appendage to assess for the presence or absence of spontaneous echo contrast and thrombus. The left atrial appendage was imaged in the basal transverse plane at the level of the aortic valve and in the longitudinal plane in the two-chamber left ventricular inflow view. Pulsed wave Doppler evaluation of the left atrial appendage and mitral inflows was performed. The sample volume was placed 1 to 2 cm into the mouth of the atrial appendage and at the mitral leaflet tips for measurement of respective Doppler flows. All patients underwent transthoracic echocardiography for measurement of left atrial appendage and at the mitral leaflet tips for measurement of respective Doppler flows. All patients underwent transthoracic echocardiography for measurement of left atrial appendage and mitral inflows was performed. The pulsatile inflow view. Pulsed wave Doppler echocardiography (as well as the precordial Doppler flows) as well as the precordial Doppler flows was scanned, looking for the most consistent patterns of atrial appendage function before and after cardioversion. Left atrial appendage peak emptying and filling fibrillatory velocities before cardioversion and peak emptying and filling late diastolic velocities after cardioversion were identified and digitized using a computer system (Dextra Inc.). Peak flow velocities were averaged over six cardiac cycles for atrial fibrillation and three cardiac cycles for sinus rhythm. Because filling velocities were generally equal in magnitude to the emptying velocities that preceded them, data were reported mainly in terms of emptying velocities.

The maximal and minimal left atrial appendage areas were measured off-line by planimetry. Precardioversion maximal and minimal areas were measured by visual estimation of the largest and smallest size during one cardiac cycle regardless of timing in the cardiac cycle. Postcardioversion left atrial appendage maximal areas were measured in atrial diastole and minimal areas in atrial systole. An emptying fraction (EF) of the left atrial appendage (LAA) was calculated using the following equation: 

\[ EF = \frac{LAA_{max} - LAA_{min}}{LAA_{max}} \]

Note that even though "emptying fractions" were calculated before cardioversion during atrial fibrillation, these are not necessarily a measure of ejection but rather a measure of the change in atrial appendage size, which may be mostly passive. Electrical mechanical intervals to correlate early and late diastolic atrial appendage flows with early and late mitral flows were measured at a paper speed of 50 to 100 mm/s using standard calipers.

Mitral regurgitation was assessed qualitatively, using Doppler color flow mapping (mild = 1+, moderate = 2+, moderately severe = 3+, severe = 4+), by visually assess-
ing the maximal jet area (15). Left ventricular function was determined by qualitative grading (mild, moderate, moderately severe or severe), using the transthoracic short axis of the left ventricle. Left atrial diameter was measured by transthoracic echocardiography in the parasternal long-axis view.

**Determination of intra- and interobserver variation.** The detection of spontaneous echo contrast was reviewed before and after cardioversion, on-line and off-line, by two separate experienced observers who were blinded to their own readings as well as to each other's. The timing of the observation was blinded only for the off-line analysis because blinding of the pre- versus the postcardioversion observation during the on-line analysis was, of course, not possible. The determination of an increase in the intensity or the development of new spontaneous echo contrast after cardioversion was made by a consensus reached by the two observers.

**Statistical methods.** Analysis of variance (ANOVA) techniques were used to evaluate the relation between spontaneous echo contrast and continuous echocardiographic variables. An F test with a resulting probability < 0.05 was considered significant. Changes over time (pre- to postcardioversion) were made using repeated measures ANOVA. Again, p values < 0.05 were considered significant. Results are reported as mean ± SD.

**Results**

**Clinical characteristics.** Etiologies of atrial fibrillation included hypertension (n = 5), valvular heart disease (n = 4), hypertrophic obstructive cardiomyopathy (n = 3), idiopathic (n = 4), and coronary artery disease (n = 2). Two patients had undergone open heart surgery. Only 3 (15%) of 20 patients exhibited mitral regurgitation of moderate severity or greater and 3 other patients were found to have at least moderate left ventricular dysfunction. 11 but 2 of the 20 patients were receiving anticoagulant therapy at the time of cardioversion, whereas 18 were on an antiarrhythmic regimen that included at least one agent for ventricular rate control, as well as a second agent for maintenance of sinus rhythm (Table 1). Conversion to sinus rhythm was accomplished in all 20 patients as demonstrated by 12-lead ECG with an average of 1.3 shocks (range 1 to 2) and 235 ± 75 J.

**Spontaneous echo contrast.** Precardioversion spontaneous echo contrast was detected in the left atrium in 12 (60%) of the 20 patients and in the left atrial appendage in 10 of those 12 (50% of all 20 patients). One patient in this series was scheduled to undergo cardioversion 8 weeks earlier; however, when transesophageal echocardiography was performed according to this protocol, he was found to have a thrombus in the left atrial appendage with subsequent postponement of cardioversion. The patient was treated with coumadin and 8 weeks later repeat transesophageal echocardiography revealed resolution of the clot with residual severe spontaneous echo contrast in the left atrium and left

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<th>Table 1. Clinical Characteristics of Patients With Atrial Fibrillation Undergoing Electrical Cardioversion Related to the Presence of Spontaneous Echo Contrast</th>
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<tr>
<td>All Patients (n = 20)</td>
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<td>Smo. (n = 12)</td>
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<td>No Smoke (n = 8)</td>
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<td>Male</td>
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<td>Status post OHS</td>
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<td>HCM</td>
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<td>Idiopathic</td>
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<td>Values are presented as mean value ± SD or number of patients. AF = atrial fibrillation; ASHD = atherosclerotic heart disease; HCM = hypertrophic cardiomyopathy; LA = left atrial; LV = left ventricular; MR = mitral regurgitation; post OHS = after open heart surgery.</td>
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Figure 1. Left atrial appendage (LAA) before (A) and after (B) cardioversion, showing a very mild degree of smoke in the left atrium (LA) and left atrial appendage before cardioversion with an increase in the intensity of smoke in the left atrial appendage after cardioversion. LV = left ventricle.
patients found to have either an increase in or the development of smoke after cardioversion as determined by consensus of the observers. Thirteen patients demonstrated no new smoke after cardioversion. Intraobserver concordance for the detection of spontaneous echo contrast was 84%. Interobserver concordance was 91%.

**Left atrial appendage function and size before cardioversion.** During atrial fibrillation, the left atrial appendage demonstrated a fibrillatory pattern in 18 (90%) of the 20 patients. This was defined as a rapid repetitive emptying and filling flow pattern by pulsed wave Doppler echocardiography (Fig. 2). Peak emptying velocities before cardioversion averaged $0.32 \pm 0.15$ m/s in the 18 patients, whereas peak filling velocities averaged $0.33 \pm 0.16$ m/s. The mechanical cycle length of these fibrillatory waves ranged from 140 to 210 ms (average $174 \pm 29$). The remaining two patients exhibited a passive pattern, defined as prolonged low velocity flow without discernible systolic or diastolic signals. Left atrial appendage maximal and minimal area values and emptying fractions are listed in Table 2.

**Left atrial appendage function after cardioversion.** After conversion to sinus rhythm, four distinct pulsed wave Doppler velocity patterns were noted in the left atrial appendage. Three (15%) of the 20 patients exhibited a “sinus” pattern (Fig. 3), characterized primarily by a late diastolic forward flow followed by a reversed flow that temporally corresponded to the ECG P wave.

Thirteen patients (65%) demonstrated a “sinuslike” pattern (Fig. 4), characterized by both early and late diastolic emptying and filling flow. The late diastolic emptying flow has been termed the left atrial appendage $a$ wave, signifying its relation to atrial depolarization. The mean left atrial appendage $a$ wave flow velocity was $0.17 \pm 0.11$ m/s and occurred within $90.3 \pm 36.9$ ms ($n = 15$; 1 patient had an uninterpretable p wave) of the ECG P wave. The P wave to mitral A wave interval was $94.2 \pm 27.0$ ms. The early diastolic emptying wave was termed the left atrial appendage $e$ wave, signifying its temporal relation to the mitral E wave. The peak left atrial appendage $e$ wave occurred an average of $532.9 \pm 42$ ms after the QRS complex, whereas the mitral E wave occurred an average of $524.4 \pm 79$ ms after the QRS complex. The mean emptying flow velocity of the left atrial appendage $e$ wave was $0.15 \pm 0.08$ m/s.

Despite clear sinus rhythm on the ECG after cardioversion, the left atrial appendage flow in three patients exhibited

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<th>Table 2. Left Atrial Appendage Areas and Emptying Fractions Before and After Cardioversion in Patients With Versus Those Without the Presence of Smoke</th>
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<td>Emptying Fraction (%)</td>
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Values presented are mean ± SD. Post = after cardioversion; Pre = before cardioversion.
an akinetic pattern, with no discernible emptying or filling flow. One patient demonstrated a fibrillatory pattern but did have an emptying wave consistently associated with the P wave.

**Left atrial function and size after cardioversion.** Left atrial function returned in 15 (75%) of 20 patients immediately after cardioversion. In three of the five patients, the lack of evidence for atrial function (absence of mitral A wave) was associated with an absence of atrial appendage function. The two remaining patients demonstrated dissociation between the return of atrial and atrial appendage function. The mean left atrial size as measured by transthoracic echocardiography before cardioversion was 48.9 ± 5.9 mm. There was no significant difference in left atrial size between patients with (49.3 ± 6.8 mm) and patients without (48.3 ± 4.6 mm) spontaneous echo contrast.

**Relation between precardioverson smoke and left atrial appendage size and function.** In the 12 patients in whom smoke was found before cardioversion, the left atrial appendage peak fibrillatory flow velocities measured before cardioversion were lower than in the 8 patients without smoke (0.25 ± 0.12 vs. 0.39 ± 0.02 m/s, respectively, F = 4.62, p = 0.045) (Fig. 5). In addition, a significantly lower precardioversion left atrial appendage emptying fraction was observed in those with smoke compared with those without smoke (12.0 ± 9.0% vs. 28.0 ± 16.8%, respectively, F = 7.76, p = 0.012) (Table 2). In patients with smoke before cardioversion, the left atrial appendage initial diastolic emptying velocity was 8.54 ± 3.64 cm²/s, with a left atrial appendage minimal area of 7.58 ± 3.41 cm². In the eight patients without smoke, maximal and minimal areas (6.08 ± 3.03 and 4.34 ± 2.42 cm², respectively) were lower compared with values in the group with smoke; however, this difference was not significant.

In the 12 patients with smoke before electrical cardioversion, the postcardioversion left atrial appendage emptying fraction (14.8 ± 15.6%) was significantly lower than the emptying fraction in patients without smoke before cardioversion (32.8 ± 17.0%, F = 5.91, p = 0.026) (Table 2). However, the left atrial appendage a wave velocities were not significantly different between those with and without smoke (0.13 ± 0.13 vs. 0.15 ± 0.12 m/s). In patients with smoke before cardioversion (n = 12), the postcardioversion left atrial appendage maximal and minimal areas were 8.81 ± 3.93 and 7.64 ± 3.89 cm², respectively. In the eight patients without smoke before cardioversion, maximal and minimal areas (6.53 ± 2.83 and 4.36 ± 2.35 cm², respectively) were lower as compared with maximal and minimal areas in the group with smoke; however, this difference was not significant.

**Reduction in left atrial appendage emptying velocities after cardioversion.** For the entire group of 20 patients, the peak left atrial appendage fibrillatory emptying velocities before cardioversion were significantly greater than the peak left atrial appendage a wave emptying velocities after cardioversion (0.31 ± 0.15 vs. 0.14 ± 0.12 m/s, F = 24.59, p = 0.0001). Velocity-time integrals also demonstrated a significant change from the pre- to the postcardioversion period (0.028 ± 0.033 vs. 0.012 ± 0.010 m, respectively, p = 0.01). If the two patients without fibrillatory emptying flows before cardioversion are excluded and only those patients with evidence of atrial appendage function before cardioversion are included for comparison, a similar difference from pre- to postcardioversion also exists (0.32 ± 0.15 vs. 0.15 ± 0.13 m/s, F = 27.26, p = 0.0001).

Furthermore, the magnitude of the reduction in left atrial appendage emptying velocity did not differ significantly between groups, decreasing from 0.25 ± 0.12 to 0.13 ± 0.13 m/s in patients with smoke before cardioversion and from 0.39 ± 0.02 to 0.15 ± 0.12 m/s in patients without
tying and filling wave as well as an early diastolic emptying trum. The sinuslike pattern includes the late diastolic emptying wave and manifesting either new smoke or no change in smoke after cardioversion.

None of these observations have been previously described. This may represent a transitional finding of this study deserves comment. The most significant difference before compared with after cardioversion (96 ± 27 vs. 75 ± 14 beats/min, respectively), there was no association between heart rate and flow velocity.

Characteristics of patients with new smoke. There was no significant difference in the mean values or magnitude of change of left atrial appendage flow and emptying fractions from before to after cardioversion among the seven patients who developed new smoke after cardioversion compared with the other 11 patients in whom smoke was never present or did not demonstrate increased intensity (Fig. 6).

Discussion

This study demonstrated three primary findings. First, the chaotic fibrillatory contractile pattern of the left atrial appendage in atrial fibrillation is usually converted to a more organized contraction pattern immediately after cardioversion (this observation corresponds to our understanding of the timing of the return of atrial activity after cardioversion). Second, left atrial appendage emptying velocities were lower after cardioversion than those generated before cardioversion, implying reduced mechanical activity of the atrial appendage. Third, spontaneous echo contrast (a phenomenon thought to be related to low shear rates and circulatory stasis) can be generated or intensified in the left atrium or atrial appendage in 35% of patients after cardioversion. None of these observations have been previously reported.

Left atrial appendage Doppler flow patterns. Some additional findings of this study deserve comment. The most common pattern of left atrial appendage mechanical activity after cardioversion, which we termed a sinuslike pattern, has not been previously described. This may represent a transitional pattern of contraction between the fibrillatory and sinus patterns, which represent opposite ends of the spectrum. The sinuslike pattern includes the late diastolic emptying and filling wave as well as an early diastolic emptying wave and filling wave of comparable flow velocities. The uniqueness of the sinuslike pattern is the presence of the early diastolic emptying (left atrial appendage e wave) and filling wave (Fig. 4).

Although the cause of the left atrial appendage e wave is not clear, the most likely explanation is that it represents a passive event that occurs in early diastole. Mitral valve opening could cause a precipitous decrease in left atrial pressure, causing the appendage to empty in early diastole. Alternatively, the left atrial appendage e wave may result from compression of the medial wall of the left atrial appendage by ascent of the ventricle during early rapid filling. Both explanations are supported by our finding of a close temporal relation of the left atrial appendage e wave with the mitral E wave. Previous reports (1,18) of left atrial appendage function in sinus rhythm demonstrated a single emptying and filling wave in late diastole associated with the P wave, without mention of an early diastolic event. However, some of these studies were likely performed using low velocity wall filters selected too high, resulting in obliteration of the relevant signals.

An alternative explanation for the etiology of the left atrial appendage e wave involves an active process, assuming electrical mechanical dissociation of atrial appendage contraction from atrial electrical activity. Support for an active process can be extrapolated from studies involving intraoperative electrophysiologic atrial mapping of atrial flutter and fibrillation because reentrant foci have been documented in the left atrial appendage (19).

Left atrial appendage mechanical activity and smoke. Patients who had spontaneous echo contrast in the left atrial appendage before cardioversion had lower fibrillatory flow velocities and lower emptying fractions than did the group without spontaneous echo contrast. These data are consistent with those from previous reports (1,18). Additionally, although left atrial appendage a wave velocities after cardioversion did not differ between the groups with and without spontaneous echo contrast, the emptying fraction after cardioversion was greater in the latter group.

Emptying fraction is quite different from the more familiar concept of ejection fraction pertaining to active ventricular contraction. With regard to the left atrial appendage in atrial fibrillation, emptying fraction is not necessarily a measure of ejection but rather a measure of change in cavity size, which may sometimes be passive. Avoidance of thrombus formation may depend on this washing phenomenon, whether the appendage empties and fills actively or passively. However, in the period after electrical cardioversion, the emptying fraction is a semiquantitative measure of active appendage contraction.

Left atrial appendage function before versus after cardioversion. Comparing left atrial appendage activity before versus after electrical cardioversion, we found that left atrial appendage fibrillatory flow velocities were consistently greater than left atrial appendage a wave velocities. This finding is discordant with the expectation that there would be
less effective left atrial appendage mechanical activity in atrial fibrillation than in sinus rhythm. The observation that the left atrial appendage generates significant emptying velocities in atrial fibrillation (as opposed to the left atrium, which does not generate detectable mechanical activity during atrial fibrillation as demonstrated by Doppler assessment of mitral inflows) can be explained by the relative size of the left atrial and left atrial appendage cavities as it relates to the constant contractile force. Being smaller in size, the atrial appendage can generate greater flows than the larger left atrial cavity with the same contractile force.

Emptying fraction was not found to be significantly different before and after cardioversion, a finding that does not correspond to the Doppler data. However, because comparable left atrial appendage views were not always obtainable, a comparison of atrial appendage areas before and after cardioversion does not seem valid. Therefore, using pulsed Doppler flow velocities as a measure of left atrial appendage function before and after cardioversion is significantly more representative of contractile function.

The etiology for the left atrial appendage stunning that occurs in the early postcardioversion period has not been elucidated by the current study. However, one possibility is that this phenomenon results from the administration of the electrical energy. The effect of electrical versus chemical cardioversion on left atrial appendage smoke and function is unknown but might serve to differentiate whether the phenomenon is a result of the type of cardioversion applied or the conversion to sinus rhythm itself.

Potential mechanisms for thromboembolic events. Many clinicians presume that a thromboembolic event occurring after electrical cardioversion of atrial fibrillation is a result of motion of indwelling left atrial thrombus present before cardioversion that is propelled into the systemic circulation by the return of atrial contraction (20-22). Although appealing, this theory is inconsistent with studies (8-10) that have observed a lower stroke rate in those with paroxysmal compared with chronic atrial fibrillation. Petersen and Gottfredsen (8) reported that the presence of thrombus in the atrial appendage significantly raises the risk of embolic events. The observation that patients with paroxysmal atrial fibrillation and large left atrial appendage size correlated with the occurrence of thromboembolic events that occur within hours or days after electrical cardioversion (6,26,27), the strong likelihood that it is freshly formed thrombus that breaks off and embolizes and the occurrence of events in patients with atrial fibrillation of relatively short duration (26).

Clinical implications. Transesophageal echocardiography has been proposed by some investigators (28) as a potentially useful diagnostic tool in the screening of patients before cardioversion to preclude the need for anticoagulation if thrombus can be excluded. Its high sensitivity for detecting both left atrial and left atrial appendage thrombus (29,30) is the basis for this proposal. Reducing patients’ exposure to anticoagulant agents may have advantages such as decreased bleeding complications, convenience and reduced hospitalization costs. However, the present study suggests that a thrombogenic milieu may develop as a result of electrical cardioversion and it provides further support for anticoagulant therapy for patients with atrial fibrillation undergoing cardioversion. Therefore, the absence of thrombus before cardioversion would not be sufficient grounds for eliminating anticoagulation before or after cardioversion.

An alternative modification to the conventional anticoagulant regimen (31) using transesophageal echocardiography as a guide to therapy would be to ensure therapeutic anticoagulation at the time of cardioversion and thereafter for a week if thrombus can be excluded. If thrombus is identified, several weeks of anticoagulant therapy before cardioversion would be initiated. A recent study by Manning et al. (32) examined a similar approach for early cardioversion without the emphasis on postcardioversion anticoagulation in the presence of a negative transesophageal echocardiographic study for thrombus (33).
Study limitations. The absence of thromboembolic events in this series of patients in the postelectrical cardioversion phase and the small number of patients studied preclude any definitive correlation between the noted observations and the mechanism involved in thromboembolic complications related to cardioversion. In addition, the relatively small sample size may have prevented the identification of subgroups of patients at particularly high risk for the development of a thrombogenic potential. Finally, the gradation of severity of spontaneous echo contrast is qualitative. To minimize this, we utilized a grading system that we believed had the least potential for interobserver error.

Conclusions. Organized left atrial appendage function returns in most patients with atrial fibrillation immediately after electrical cardioversion. However, its function is impaired after as compared with before cardioversion. In addition, left atrial and atrial appendage spontaneous echo contrast can develop or increase in severity in the immediate postcardioversion period. These findings suggest that stunned left atrial appendage function after cardioversion may produce a thrombogenic milieu that could be a mechanism for systemic embolization after cardioversion. Further investigation is required into the mechanisms involved in cardioembolic events after cardioversion of atrial arrhythmias so as to provide a rational basis for our treatment. In addition, prospective studies are needed to define the role of transesophageal echocardiography in patients undergoing cardioversion, as well as to determine the safest and most efficacious mode of anticoagulation.

References