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Transesophageal Echocardiography Before and During Direct Current Cardioversion of Atrial Fibrillation: Evidence for "Atrial Stunning" as a Mechanism of Thromboembolic Complications

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Objectives. The purpose of this study was to evaluate the usefulness of transesophageal echocardiography before electrical cardioversion in patients with atrial fibrillation and to determine the mechanism of thromboembolism after cardioversion.

Background. Thromboembolic complications after electrical cardioversion of atrial fibrillation have been attributed to the dislodgment of preexistent left atrial thrombus during the resumption of atrial contraction. Transesophageal echocardiography has been proposed as a method of screening patients for left atrial thrombus before cardioversion.

Methods. Seventy transesophageal echocardiographic studies were performed in 66 patients, predominantly with nonvalvular atrial fibrillation, before direct current cardioversion. In addition, transesophageal echocardiography was performed during the cardioversion procedure in 15 patients and immediately after in 1 patient.

Results. Left atrial thrombus was detected in one patient (1.4%), and cardioversion was canceled. Thromboembolic complications occurred in 4 patients, none of whom had evidence of left atrial thrombus before cardioversion. Within 10 s of successful cardioversion, left atrial spontaneous echo contrast appeared in five patients, increased in one patient and was unchanged in nine patients. Patients with new or increased spontaneous echo contrast had more impaired atrial contraction and slower initial heart rates after cardioversion than those without. Left ventricular contraction was also impaired transiently by cardioversion.

Conclusions. Transesophageal echocardiographic detection of left atrial thrombus before direct current cardioversion is important but infrequent in patients with predominantly nonvalvular atrial fibrillation. The occurrence of thromboembolic complications in the absence of demonstrable left atrial thrombus and the new development of spontaneous echo contrast in association with the transient atrial dysfunction ("stunning") caused by cardioversion suggest that cardioversion may promote new thrombus formation, in which case all patients should receive full anticoagulant therapy at the time of cardioversion.

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Atrial fibrillation predisposes to thromboembolism (1,2), but electrical cardioversion of this arrhythmia also may be complicated by thromboembolic events in up to 7% of cases (3–9). Thromboembolism after cardioversion has been attributed to the dislodgment of preformed atrial mural thrombus during the resumption of atrial contraction (1,8,10). Transesophageal echocardiography has been demonstrated to be more sensitive than transthoracic echocardiography for the detection of left atrial thrombus and spontaneous echo contrast, a putative marker of thromboembolic risk (11–15). Consequently, transesophageal echocardiography might be a reliable method of screening patients before electrical cardioversion.

In the present study, we performed transesophageal echocardiography in a consecutive series of patients with atrial fibrillation before and, in some cases, during direct current cardioversion. Our observations suggest that thromboembolism after cardioversion may more often arise as a consequence of the effects of cardioversion on atrial function than from the dislodgment of a preexistent thrombus.

Methods

Study patients. Transesophageal echocardiography was performed in 66 consecutive patients with atrial fibrillation before direct current cardioversion. In our institution, patients are routinely referred for echocardiographic assessment of thromboembolic risk before cardioversion, with the exception of those with atrial fibrillation complicating coronary artery bypass graft surgery who have a relatively low embolic risk. There were 41 men and 25 women, aged 28 to 84 years (mean age 61 years). The duration of atrial fibrilla-

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tion was <24 h to 12 months (mean duration 57 days). Eight patients had mitral valve disease (rheumatic stenosis in 6, valve replacement in 1, valve repair in 1), and 58 patients (88%) had nonvalvular atrial fibrillation. Because 4 patients underwent cardioversion twice, 70 transesophageal echocardiograms were performed. Sixty-four direct current cardioversions were subsequently performed in 60 patients; in others, sinus rhythm was restored in the 24 h before cardioversion, and in 1 patient the cardioversion procedure was canceled when a left atrial thrombus was found on the transesophageal study.

Echocardiography before cardioversion. Transthoracic two-dimensional and Doppler echocardiography were performed before cardioversion with a Hewlett-Packard ultrasonograph (HP 77030A) and a 2.5-MHz transducer. Left atrial size and left ventricular fractional shortening were estimated from M-mode tracings (16). Transesophageal echocardiography was performed with a monoplane (29 patients) or biplane (41 patients) 5-MHz probe (HP 21362A or HP 21363A). Fasting patients received topical pharyngeal anesthesia with 1% lidocaine and sedation with intravenous midazolam hydrochloride (1 to 2 mg). The left atrium and left atrial appendage were inspected for thrombus, spontaneous echo contrast and other potential sources of cardiogenic thromboembolism (11). The severity of spontaneous echo contrast was graded independently by two observers according to the following criteria: 0 (nil) = absence of echogenicity; 1+ (mild) = minimal echogenicity located in the left atrial appendage or sparsely distributed in the main cavity of the left atrium, may be detectable only transiently during the cardiac cycle, imperceptible at operating gain settings for two-dimensional echocardiographic analysis; 2+ (mild to moderate) = more dense swirling pattern than 1+ but with similar distribution, detectable without increased gain settings; 3+ (moderate) = dense swirling pattern in the left atrial appendage, generally associated with somewhat lesser intensity in the main cavity, may fluctuate in intensity but detectable constantly throughout the cardiac cycle; 4+ (severe) = intense echo density and very slow swirling patterns in the left atrial appendage, usually with similar density in the main cavity. Interobserver differences in grading occurred in seven cases but did not exceed one grade and were resolved by a third observer.

The delay between these echocardiographic studies and the scheduled cardioversion was <24 h for 37 procedures (58%), 24 to 72 h for 7 procedures, 72 h to 1 week for 10 procedures and >1 week for 10 procedures. In the latter group, the duration of atrial fibrillation ranged from 3 weeks to 6 months, and no changes in the hemodynamic or anticoagulation status were observed in the interval between transesophageal echocardiography and cardioversion.

Echocardiography during cardioversion. In a subgroup of 16 patients, detailed echocardiographic assessment of left atrial and ventricular function was made before, during and after cardioversion. Transthoracic echocardiography performed before the procedure included measurement of orthogonal left atrial diameters in three planes (anteroposterior in the parasternal long-axis view [D1], mediolateral in the parasternal short-axis [D2] and superoinferior in the apical four-chamber views [D3]); left atrial area was determined by planimetry in the apical four-chamber view. In 15 of the 16 patients, transesophageal echocardiography was performed with a monoplane probe immediately before cardioversion, within 5 to 10 min after the induction of anesthesia. In the five patients who had recent transesophageal echocardiograms before study entry, hemodynamic variables were compared before and after anesthesia. During delivery of the cardioversion shock, the transesophageal probe was left in situ but was transiently disconnected from the ultrasonograph to prevent grounding of the electrical discharge by way of the probe. The following variables were recorded before and at 0.5, 1, 3, 5 and 10 min after successful cardioversion: grade of spontaneous echo contrast, heart rate, left atrial and left ventricular fractional shortening and, in the last 10 patients, Doppler velocity profiles of the mitral inlet and the orifice of the left atrial appendage. Left atrial fractional shortening was derived from transesophageal two-dimensional echocardiographic images in the four-chamber view using atrial diameters measured orthogonally from the atrial septum near its junction with the mitral annulus at atrial end-diastole (defined as the onset of the electrocardiographic [ECG] P wave) and at atrial end-systole (defined as the minimal diameter observed after the P wave), with the following formula: (End-diastolic diameter − End-systolic diameter)/End-diastolic diameter (Fig. 1, A and B). An index of atrial stasis was calculated for each time interval as the product of left atrial fractional shortening and heart rate. Left ventricular fractional shortening was derived from transverse ventricular diameters in the four-chamber view, measured at a position just distal to the tips of the open mitral leaflets (analogous to standard transthoracic measurements (Fig. 1, C and D). In 1 of the 16 patients, transesophageal echocardiography was performed before and at 10 min after cardioversion, without additional anesthesia or endotracheal intubation.

Follow-up transthoracic echocardiography was performed 24 h and 72 h after cardioversion. Transesophageal echocardiography was performed 72 h after cardioversion. Patients with recurrent atrial fibrillation at these time intervals were not restudied.

For comparison of indexes of atrial and ventricular function with normal values, a cohort of 16 age- and gender-matched control subjects with no cardiac structural abnormalities was selected from patients in our department who underwent transesophageal echocardiography for exclusion of a cardiogenic source of thromboembolism or infective endocarditis.

Direct current cardioversion. Fasting patients underwent sedation with intravenous midazolam (1 to 4 mg) or propofol (1 to 3 mg/kg body weight). Incremental direct current discharges were delivered through cutaneous electrode patches. A procedure was defined as successful if sinus

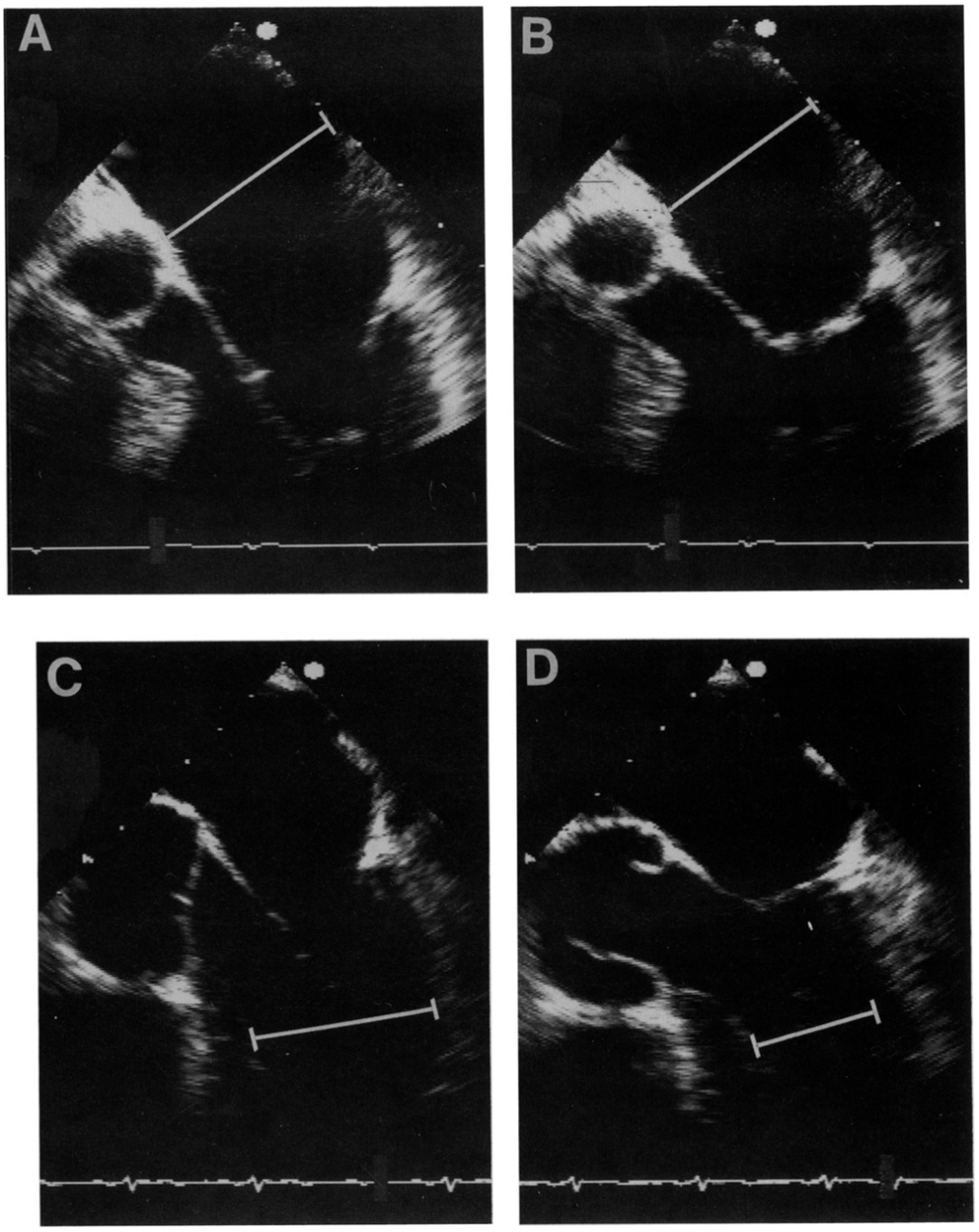

Figure 1. Left atrial and left ventricular fractional shortening were derived from transesophageal two-dimensional echocardiographic images in the four-chamber view using measurements (see bars) of transverse atrial diameters at atrial end-diastole (A) and atrial end-systole (B) and transverse ventricular diameters at end-diastole (C) and end-systole (D).

rhythm was achieved. In the subgroup of patients who underwent transesophageal echocardiography during the cardioversion procedure, additional informed, written consent and approval from the institutional Research and Ethics Committee were obtained. These patients received intravenous thiopentone sodium (3 to 5 mg/kg, nine patients) or propofol (1 to 3 mg/kg, six patients), and endotracheal intubation was performed.

Anticoagulation. Anticoagulation regimens varied widely among the various referring physicians in our institution. Fifty-two patients received anticoagulant therapy before cardioversion: long-term coumadin therapy (11 patients) or short-term anticoagulation (1 to 12 days) with intravenous heparin (30 patients), subcutaneous heparin (8 patients) or coumadin (3 patients). On the day of cardioversion, anticoagulation levels were therapeutic (activated partial thromboplastin time >55 s, or international normalized ratio >2.0) in 38 patients. After cardioversion, 18 patients continued receiving intravenous (14 patients) or subcutaneous (4 patients) heparin for 12 to 48 h. Twenty-five patients were given long-term therapy with coumadin. Aspirin was received by 10 patients before and 14 patients after cardioversion. Two patients before and six patients after cardioversion had no antithrombotic therapy.

Statistical analysis. Mean differences in hemodynamic variables for groups of patients were obtained from the mean value of the differences in individual patients. The presence of significant changes in hemodynamic variables over time was ascertained by repeated measures analysis of variance. The significance of changes within groups was determined

Table 1. Clinical and Echocardiographic Characteristics of Four Patients With a Thromboembolic Event After Electrical Cardioversion

	Patient 1	Patient 2	Patient 3	Patient 4*
Age (yr)/gender	57/M	77/M	65/F	84/F
Etiology of AF	Lone	Postoperative	Hypertension	Lone
Pattern of AF	Paroxysmal	1st episode	1st episode	Paroxysmal
Duration of current episode (days)	28	14	14	1) 42; 2) 11
LA-M (mm)	43	58	43	33
LV FS <28%	No	No	No	No
LA thrombus	No	No	No	1) No; 2) N/A
LA SEC†				
Before CV	0	1+	0	0
During CV	N/A	N/A	2+	1+
Time from TEE to CV	24 h	3 wk	6 days	1) Immediate; 2) N/A
Time from CV to event	48 h	7 days	6 days	1) N/A; 2) 24 h
Event	TIA	Stroke	Stroke	TIA
Rhythm at event	Sinus	Sinus	Sinus	Sinus
AC therapy	Aspirin	S/C heparin	IV heparin/coumadin	1) IV heparin; 2) S/C heparin
AC status				
At CV	N/A	Subtherapeutic	Therapeutic	1) Therapeutic; 2) therapeutic
At event	N/A	Subtherapeutic	Subtherapeutic	1) N/A; 2) subtherapeutic

*Two cardioversions performed. Recurrence of atrial fibrillation (AF) after 5 days. †See text for explanation of 0, 1+ and 2+. AC = anticoagulant; CV = cardioversion; F = female; IV = intravenous; LA = left atrial; LA-M = left atrial diameter (by M-mode echocardiography); LV FS = left ventricular fractional shortening; M = male; N/A = not applicable; S/C = subcutaneous; SEC = spontaneous echo contrast; TEE = transesophageal echocardiography; TIA = transient ischemic attack.

with the paired Student *t* test and between groups with the Mann-Whitney *U* test. The relation between improvements in the grade of spontaneous echo contrast after cardioversion and atrial function was determined by using changes in the atrial stasis index, which was normalized to final values to facilitate comparisons between individual patients. A p value < 0.05 was considered significant.

Results

A successful immediate outcome was obtained in 56 direct current cardioversions (88%). Thromboembolic complications occurred in four patients (Table 1).

Transesophageal echocardiography before cardioversion. A thrombus was found, with a monoplane probe, at the orifice of the left atrial appendage in one patient before cardioversion (1.4%). None of the patients with embolic events after cardioversion had evidence of left atrial thrombus before cardioversion (Table 1). The precardioversion clinical and echocardiographic characteristics of those patients with and without embolic complications after cardioversion are shown in Table 2. Although the number of patients with embolic complications was too small to permit meaningful statistical comparisons between the two groups, it is notable that those with embolic complications did not have a longer duration of atrial fibrillation, a larger left atrium or a greater prevalence of spontaneous echo contrast, mitral valve disease or left ventricular dysfunction when compared with those without embolic complications.

Transesophageal echocardiography during cardioversion. *Effect of anesthesia.* After the induction of general anesthesia, the precardioversion grade of left atrial spontaneous echo contrast was 0 in nine patients, 1+ in three patients and 2+ in three patients. The grade of spontaneous echo contrast was unchanged by anesthesia when comparisons with previous transesophageal echocardiograms were made in five patients. There was a mean reduction of 14.7% in left ventricular fractional shortening after anesthesia ($p = 0.02$). The difference between the 11.2% decrease in left ventricular fractional shortening in the nine patients who received thiopentone as an induction agent and the 21% mean reduc-

Table 2. Clinical and Echocardiographic Characteristics Before Electrical Cardioversion in Patients With and Without Thromboembolic Events After Cardioversion

	Thromboembolic Events	
	No (n = 60)	Yes (n = 4)
Male/female	37/19	2/2
Age (yr)	60 ± 13	71 ± 12
Duration of AF (days)	59 ± 75	25 ± 14
Mitral valve disease	21 (35)	1 (25)
MS ± MR	6	0
MVR	2	0
MR only	13	1
LA diameter (mm)	45 ± 6	46 ± 8
LV FS <28%	20 (33)	0 (0)
LA thrombus	0 (0)	0 (0)
LA SEC	23 (38)	1 (25)
Patent foramen ovale	3 (5)	1 (25)
ASD/ASA	0 (0)	0 (0)
Atrial tumor	0 (0)	0 (0)

Values presented are mean value ± SD or number (%) of patients. ASD/ASA = atrial septal defect or aneurysm; MR = mitral regurgitation; MS = mitral stenosis; MVR = mitral valve replacement or repair; other abbreviations as in Table 1.

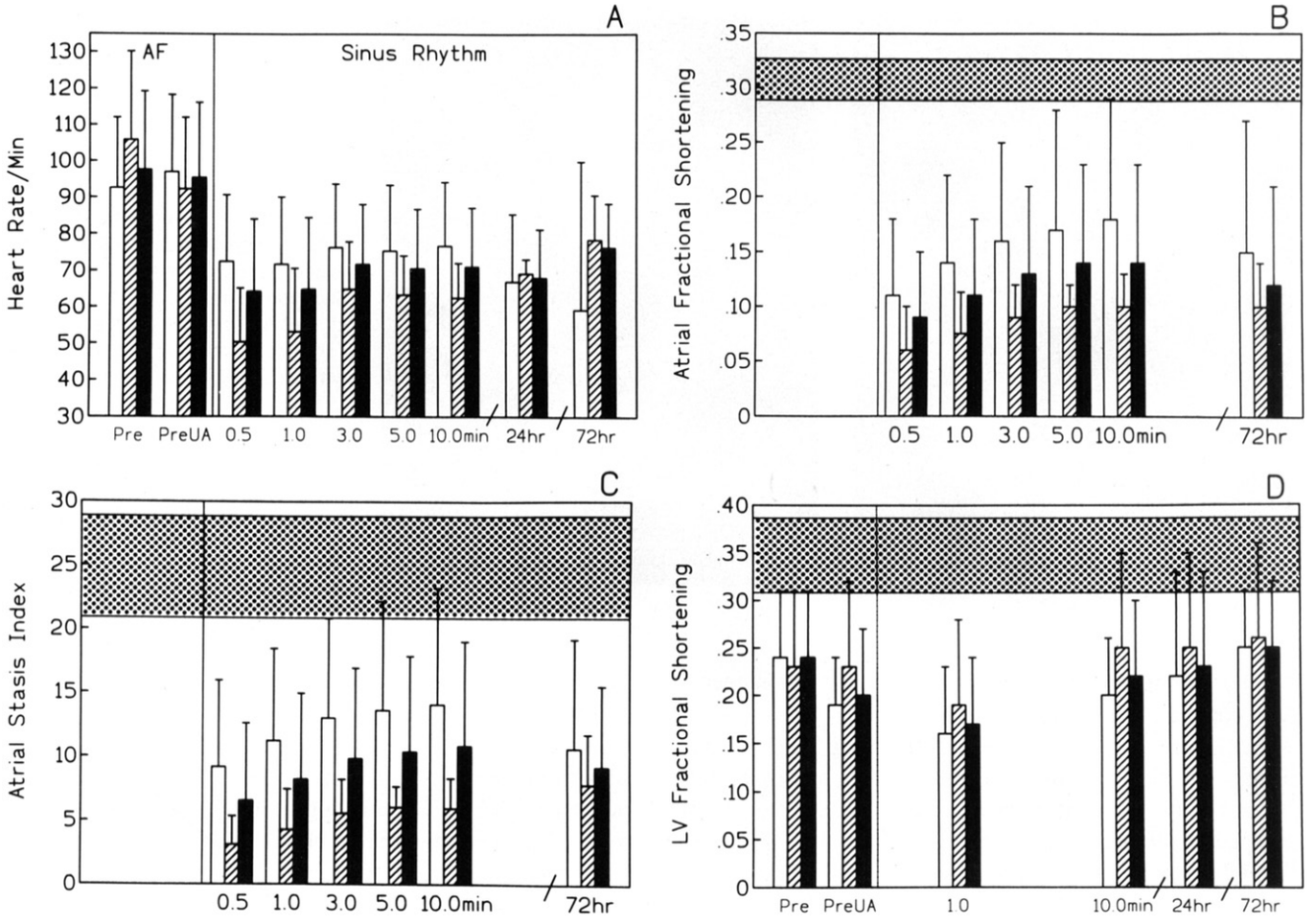

Figure 2. Heart rate (A), left atrial fractional shortening (B), left atrial stasis index (C) and left ventricular (LV) fractional shortening (D) in patients with and without new or worse spontaneous echo contrast after successful cardioversion. Pre = before cardioversion; PreUA = before cardioversion and after the induction of general anesthesia; **vertical line** = time of cardioversion; **open bars** = spontaneous echo contrast unchanged; **hatched bars** = new or increased spontaneous echo contrast; **solid bars** = all patients; **shaded areas** = normal range of variables in age- and gender-matched control subjects. Data are expressed as mean value ± SD.

tion in the six patients who received propofol did not achieve statistical significance. No differences were found in the effect of general anesthesia on either heart rate or left ventricular fractional shortening between those patients who subsequently developed new or increased spontaneous echo contrast and those who did not (Fig. 2, A and D).

Effects of cardioversion. A successful immediate outcome was obtained in 15 of the 16 patients who underwent transesophageal echocardiography during and immediately after cardioversion.

Spontaneous echo contrast. Within 10 s of the restoration of sinus rhythm, new spontaneous echo contrast appeared in the left atrium in five patients (Fig. 3). The grade of spontaneous echo contrast was maximal within 1 min: 1+ in three patients, 2+ in one patient and 3+ in one patient. One additional patient with 1+ spontaneous echo contrast before cardioversion, had a transient increase to 3+ after each of two unsuccessful shocks, and 3+ spontaneous echo contrast developed after a third shock that was successful. In nine patients, no changes in the grade of spontaneous echo contrast were observed after successful cardioversion: 0 in four patients, 1+ in two patients and 2+ in three patients.

In the patients with new or increased spontaneous echo contrast, the spontaneous echo contrast had disappeared in two patients, decreased in grade in one patient and was unchanged in three patients by 10 min after cardioversion. By 72 h after cardioversion, spontaneous echo contrast had disappeared in one additional patient, decreased in grade in one patient, was unchanged in one patient and could not be restudied in one patient. In the patients with spontaneous echo contrast unchanged by the cardioversion procedure, no changes were observed by 10 min, but by 72 h the spontaneous echo contrast had disappeared in one patient, decreased in grade in one patient, was unchanged in two patients and could not be restudied in five patients.

In the patients with new or worse left atrial spontaneous echo contrast, there was a longer duration of atrial fibrillation (51 ± 32 vs. 8 ± 9 days, $p < 0.01$), a higher mean number of shocks (2.7 ± 0.8 vs. 1.4 ± 0.7, $p = 0.02$) and higher mean energy delivered (516 ± 204 vs. 272 ± 319 J,

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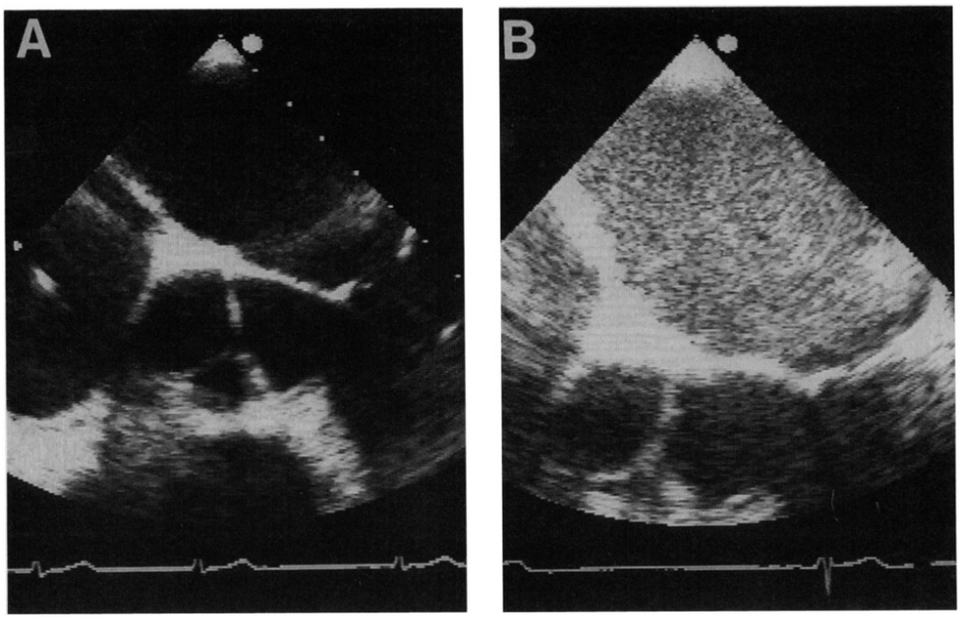

Figure 3. New spontaneous echo contrast appeared in the left atrium, accompanied by sinus bradycardia and poor atrial contraction, within 10 s of successful cardioversion in a 73-year old man. A, Before cardioversion. B, After cardioversion.

$p = 0.07$) compared with those with unchanged spontaneous echo contrast.

Heart rate. After cardioversion, the mean heart rate was significantly slower in the patients with new or worse spontaneous echo contrast compared with patients with unchanged spontaneous echo contrast ($p = 0.001$) (Fig. 2A). The differences between the groups were most marked in the immediate postcardioversion period.

Atrial fractional shortening. After cardioversion to sinus rhythm, atrial fractional shortening was depressed in all patients compared with age- and gender-matched control subjects (Fig. 2B). A greater degree of impairment of atrial fractional shortening was observed in the subgroup of patients who developed new or increased spontaneous echo contrast ($p = 0.001$). The differences between the groups were maintained for all time periods up to 72 h. In both groups, similar trends of improvement in atrial fractional shortening over time were noted. Concurrent trends of improvement in left atrial appendage peak outflow and mitral inflow "a" wave velocities were found (Fig. 4).

Atrial stasis index. In individual patients, the new onset or worsening of left atrial spontaneous echo contrast was accompanied by a variable degree of bradycardia or impairment of atrial contraction, or both (Fig. 2C). For this reason, the product of heart rate and fractional shortening was determined as an index of atrial stasis that incorporated both factors. The atrial stasis index was depressed in all patients compared with age- and gender-matched control subjects. A greater degree of atrial stasis was observed in the patients with new or worse spontaneous echo contrast compared with those with spontaneous echo contrast that was unchanged ($p < 0.001$). A reduction in the grade of spontaneous echo contrast was accompanied by an improvement in the atrial stasis index (Fig. 5).

Left ventricular systolic function. A highly significant impairment of left ventricular fractional shortening was

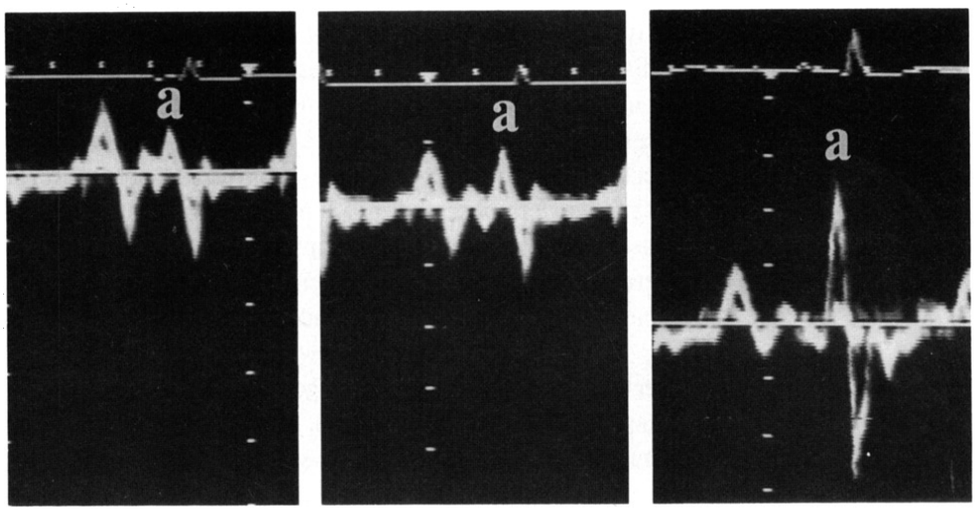

Figure 4. Left atrial appendage Doppler velocity profiles at 3 min (left), 10 min (middle) and 72 h (right) after cardioversion in a 67-year old man. An increase in the peak velocity of the left atrial appendage "a" wave, corresponding to increased left atrial appendage contraction, was noted after cardioversion.

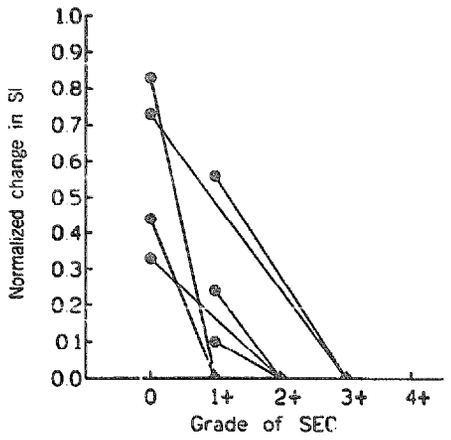

Figure 5. Reductions in the grade of spontaneous echo contrast (SEC) in seven patients after successful cardioversion were accompanied by improvements in the atrial stasis index (SI).

induced by the cardioversion procedure (mean decrease 27.4%, $p < 0.001$) (Fig. 2D). In all patients, left ventricular contraction had returned to precardioversion levels by 24 h, and improvement to greater than baseline levels occurred in six patients. The extent of left ventricular systolic dysfunction induced by cardioversion was variable in individual patients, with no statistically significant differences between the subgroups of patients with and without new or worse spontaneous echo contrast.

Left atrial size. Although some reductions were found in individual patients, mean left atrial size, indexed by left atrial diameter (M-mode tracing) or planimetered left atrial area, at 24 h and 72 h after cardioversion was not significantly different from precardioversion values in either the total population or in subgroups with and without new or worse spontaneous echo contrast. By 24 h, however, a marked reduction in the D2 left atrial diameter was noted in seven patients, with no changes in either the D1 or D3 diameters, indicating an early change in left atrial shape in some patients. The mean D2 diameter at 24 h was significantly smaller in the patients who had unchanged spontaneous echo contrast than in those with new or worse spontaneous echo contrast ($p = 0.03$). By 72 h, however, the differences between the groups were not significant.

Discussion

Thromboembolism after cardioversion has been attributed to the dislodgment of preformed thrombus from the left atrium with the resumption of sinus rhythm and atrial contraction (1,8,10). Several investigators have reported a reduction in the prevalence of thromboembolic complications in patients receiving anticoagulant therapy (6–8). In the largest series, Bjerkelund and Orning (6) found a 5.3% prevalence of embolic events in patients without anticoagulant therapy and only a 0.8% prevalence in patients with long-term anticoagulant therapy. Although not formally evaluated in clinical trials, an empiric regime of anticoagulation for 2 to 4 weeks before the cardioversion procedure has been recommended (17).

Transesophageal echocardiography before cardioversion. In an attempt to further reduce the prevalence of thromboembolic complications after electrical cardioversion, transesophageal echocardiography has been proposed as a method of screening patients for left atrial thrombus before the procedure (18,19). Transesophageal echocardiography is more sensitive than transthoracic echocardiography for the detection of thrombi in the left atrium, particularly in the left atrial appendage (11–13). In two small series of patients with rheumatic mitral valve disease undergoing valve replacement surgery, a sensitivity of 83% to 100% and specificity of 97% to 100% were reported for the detection of left atrial thrombus by transesophageal echocardiography when compared with the operative findings as the reference standard (12,13). Left atrial thrombus is not frequently observed by transesophageal echocardiography before cardioversion. Similar to our own findings, Grimm et al. (18) detected only one thrombus (2.5%) in 40 patients with atrial fibrillation or flutter. Manning et al. (19) reported 14 thrombi in 12 (13%) of 94 patients. However, because patients receiving long-term anticoagulant therapy were excluded from that study, a higher prevalence of thrombus might be expected. In our institution, cardioversion is not performed in patients with a long duration of atrial fibrillation or a large left atrium, because these patients are unlikely to maintain sinus rhythm after the procedure and are also more likely to be at increased risk for thrombus. Therefore, although a 7% to 16% prevalence rate of thrombus has been reported in groups with predominantly nonvalvular atrial fibrillation (20,21), a lower rate may be found in patients selected for cardioversion. The single thrombus in this series was identified with a monoplane probe, and no thrombi were detected in any of the 41 patients examined with a biplane probe. Although it is possible that the use of a monoplane probe in 29 patients in this study may have led to an underestimation of thrombus prevalence, in the absence of prospective data comparing monoplane with biplane or multiplane techniques, it remains to be determined whether these additional planes increase the sensitivity for thrombus detection.

Thromboembolic complications after cardioversion. A major finding of this study is that thromboembolic complications may occur after cardioversion despite negative results on precardioversion transesophageal echocardiography. Thromboembolic complications were observed after cardioversion in four patients, none of whom had evidence of left atrial thrombus before cardioversion. One possible explanation for these observations may be that some thrombi may be large enough to lodge in small cerebral vessels and cause ischemic events yet still be too small to be detected by transesophageal echocardiography. In this case, screening transesophageal echocardiography before cardioversion would not be sufficient to prevent such events.

An alternative explanation is that thrombus formation

may occur after the cardioversion procedure. Our results support a concept of acute atrial stunning induced by electrical cardioversion that may predispose to blood stasis and thrombus formation after the procedure. Several factors may contribute to atrial dysfunction after cardioversion. The electrical energy delivered during cardioversion may impair cardiac function, with both laboratory and clinical evidence of myocardial damage after direct current electrical discharge (4,5,22). The extent of injury has been related to the amount of energy delivered (22), consistent with our own observations. Patients who developed new or increased spontaneous echo contrast received a greater number of shocks and a higher mean energy shock level than did patients who had no change in spontaneous echo contrast. In addition, short-acting anesthetic agents commonly used during electrical cardioversion, such as thiopentone and propofol, may depress myocardial contraction transiently and reduce cardiac output. The effects of these agents on heart rate may vary, depending on levels of sympathetic activation before anesthesia, baroreceptor responsiveness and concomitant drug therapy (23–25).

Despite the restoration of P waves on the ECG after successful cardioversion, the previously fibrillating left atrium may not return immediately to normal function. Doppler echocardiographic studies examining serial changes in mitral inflow velocity profiles have demonstrated that restitution of normal atrial contraction after cardioversion is variable and may take up to 3 months (26–28). The time course of atrial mechanical restitution may be related to the duration of atrial fibrillation before cardioversion, left atrial size, left ventricular function, underlying cardiac disease and the myocardial depressant effects of prophylactic antiarrhythmic therapy. It is possible that the higher cardioversion energy required in patients with new or increased spontaneous echo contrast may reflect a greater difficulty in achieving cardioversion in those more prone to delayed mechanical restitution. However, Manning et al. (28) recently reported that patients undergoing electrical cardioversion of atrial fibrillation may have greater and more prolonged depression of atrial function than that in patients whose arrhythmia is converted pharmacologically. A combination of electrically and chemically induced myocardial depression and delayed restitution of atrial contraction may predispose to blood stasis and potential thromboembolism for a variable period after cardioversion.

Transesophageal echocardiography during cardioversion. *Spontaneous echo contrast and atrial stunning.* Spontaneous echo contrast is an echogenic swirling pattern of blood flow that has been attributed to the aggregation of cellular components of blood, most commonly in conditions of low velocity blood flow (29). The new appearance of left atrial spontaneous echo contrast after cardioversion may thus be evidence of increased atrial blood stasis induced by the procedure. In the present series, patients with new or increased spontaneous echo contrast had a slower initial mean heart rate and a lower atrial fractional shortening than those of patients with no change in spontaneous echo contrast. When these two variables were multiplied as an index of atrial stasis, marked differences between the subgroups of patients with and without new or increased spontaneous echo contrast were noted.

The duration of spontaneous echo contrast after cardioversion was variable. Spontaneous echo contrast induced or increased by cardioversion had either disappeared or decreased in grade by 72 h in the majority of patients. In one patient, however, new spontaneous echo contrast persisted unchanged at 72 h. In the patients with preexisting spontaneous echo contrast that was unchanged immediately after cardioversion, the spontaneous echo contrast had either disappeared or decreased in grade in two of the four patients studied at 72 h. Reductions in the grade of spontaneous echo contrast at 10 min and at 72 h corresponded with improvements in the atrial stasis index.

Left ventricular dysfunction. Significant transient depression of left ventricular contraction was observed as a result of the cardioversion procedure but appeared to be poorly related to the development of left atrial spontaneous echo contrast. In addition to the amount of energy delivered, anesthetic agents and concomitant antiarrhythmic drug therapy may contribute to the variable degree of left ventricular dysfunction observed.

Implications for anticoagulant therapy during cardioversion. It has been suggested that transesophageal echocardiography before cardioversion may obviate the need for several weeks of prophylactic anticoagulant therapy before cardioversion (9,19). However, because of the low prevalence of embolism after cardioversion, large scale controlled clinical studies would be required to fully evaluate the role of transesophageal echocardiography before cardioversion.

The major clinical implication of this study is that all patients may require full anticoagulant therapy at the time of and for some time after electrical cardioversion. Anticoagulant therapy at the time of cardioversion might not prevent thrombi too small to be seen by transesophageal echocardiography from embolizing but could be expected to reduce the risk of new thrombus formation. None of the four patients in this study who experienced thromboembolic complications after cardioversion received full anticoagulant therapy during the interval between the procedure and the embolic event: Patient 1 received aspirin alone, Patient 2 was a 71-kg man who received only 10,000 U of subcutaneous heparin twice daily (30) and subtherapeutic levels of anticoagulation were demonstrated in Patients 3 and 4. Manning et al. (19) recently found no thromboembolic complications in a series of 31 patients who received short-term anticoagulant therapy with intravenous heparin for a mean 2.1 ± 1.2 days before cardioversion and had adequate anticoagulation at the time of the procedure. The optimal duration of anticoagulant therapy after cardioversion is not known, but theoretic considerations suggest continuation until restitution of atrial contraction sufficient to prevent blood stasis.

Indexes of atrial contraction. Although calculation of fractional shortening or a stasis index can be used to estimate atrial function after cardioversion, the degree and duration of spontaneous echo contrast within the left atrium may provide the most direct evidence of the persistence of blood stasis. Because the left atrial appendage is frequently the site of thrombus formation, the peak "a" wave outflow velocity, an index of contractile function of this structure (31), may be useful in sequential assessment of thromboembolic risk. Mitral valve Doppler profiles used in previous echocardiographic studies after cardioversion (26–28) may be difficult to interpret because of concurrent effects of the cardioversion procedure on both atrial and ventricular function.

Study limitations. The number of patients examined in this study was sufficient to demonstrate both the induction of new spontaneous echo contrast after cardioversion in a substantial proportion of patients and the relation of spontaneous echo contrast to atrial stasis. However, the relatively small number of patients in the study group, the low prevalence of thromboembolic events and the high recurrence rate of atrial fibrillation after cardioversion limit the conclusions that can be drawn from our data. The apparent discrepancy between the longer duration of atrial fibrillation in patients who developed new or increased spontaneous echo contrast after cardioversion and the short duration of atrial fibrillation in the four patients with embolic events may largely be due to the small patient sample.

The anesthetic agents thiopentone and propofol probably contributed to the left atrial and ventricular dysfunction observed in this study. However, transesophageal echocardiographic measurements were made well after the anticipated peak depressant drug effect, the depression of contractile function observed in some patients after induction of anesthesia further increased immediately after cardioversion and successive shocks produced incremental contractile dysfunction in some patients. The study protocol did require more prolonged anesthesia than is usually used for routine cardioversion owing to the inclusion of a 10-min monitoring period after each successful shock.

Conclusions. Precardioversion transesophageal echocardiography is useful to detect preexisting left atrial thrombus, but this is an infrequent finding in patients with predominantly nonvalvular atrial fibrillation. The occurrence of thromboembolic complications in the absence of demonstrable thrombus before cardioversion suggests that either transesophageal echocardiography misses some small thrombi or that thrombus formation may occur after the procedure. Transesophageal echocardiography during cardioversion demonstrated new or increased spontaneous echo contrast in a considerable proportion of patients. In these patients, a slower initial heart rate and more impaired atrial fractional shortening were present compared with findings in patients with no change in spontaneous echo contrast. These observations suggest a phenomenon of transient atrial "stunning" that may promote thromboembolism after successful cardioversion in the absence of preexisting left atrial thrombus. Our findings suggest that full prophylactic anticoagulant therapy may be advisable in all patients immediately before electrical cardioversion of atrial fibrillation and for a variable period thereafter, determined by the duration of atrial contractile dysfunction.

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