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Pulsed Doppler Evaluation of Atrial Mechanical Function After Electrical Cardioversion of Atrial Fibrillation

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Full recovery of atrial mechanical activity may not occur immediately after successful electrical cardioversion of atrial fibrillation to sinus rhythm. To examine the time course of recovery of left atrial mechanical function, serial two-dimensional, M-mode and transmitral pulsed Doppler echocardiographic studies were performed in 21 patients after elective direct current cardioversion of atrial fibrillation of 3 weeks' to 24 months' duration (mean 5 months).

Over 3 months of follow-up, there were significant increases in both peak A wave velocity ($p < 0.005$) and percent atrial contribution to total left ventricular filling ($p < 0.005$). Compared with values in a normal control population, peak A wave velocity and percent atrial contribution to total left ventricular filling did not return to normal until 3 weeks after cardioversion in patients who remained in sinus rhythm. Left atrial dimension also decreased over the follow-up period ($p < 0.05$) in patients with persistent sinus rhythm.

These results may have important implications in guiding the appropriate duration of anticoagulant therapy after cardioversion, and in clinically assessing the hemodynamic benefit of restoring sinus rhythm in an individual patient.

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Atrial fibrillation is characterized by a lack of organized electrical and mechanical atrial activity. The loss of unified atrial systolic function results in inadequate atrial emptying and, in some patients, impaired ventricular filling. Stasis of blood in the atria also favors formation of thrombi, which may result in pulmonary and systemic embolism (1,2). Direct current (DC) cardioversion of atrial fibrillation is frequently performed in an effort to restore the atrial contribution to ventricular filling and thereby both improve cardiac function and decrease blood stasis in the atria. Successful cardioversion, however, is associated with a small risk of immediate embolism (3,4), and late embolism has been described several days to weeks after cardioversion in patients who have maintained sinus rhythm (3–6).

Electrocardiographic evidence of restoration of normal sinus rhythm is often assumed to be accompanied by a return of effective atrial contraction. Some invasive and noninvasive studies (5,7–9), however, have suggested that return of normal atrial mechanical function may not occur immediately after successful cardioversion. The delayed recovery of atrial mechanical function may involve one or both atria (5,8).

Pulsed Doppler echocardiography permits serial noninvasive evaluation of flow across the mitral valve. This technique has been used to accurately characterize patterns of transmitral left ventricular filling in normal individuals and in patients with a variety of cardiac abnormalities (10). Given its ability to assess the left atrial systolic contribution to total left ventricular filling, the pulsed Doppler technique is well suited for assessment of changes in left atrial function after successful cardioversion of atrial fibrillation to sinus rhythm. To determine if there is a delay in the return of normal left atrial contraction after cardioversion, imaging and pulsed Doppler echocardiography were used to serially evaluate left atrial size and function in 21 patients undergoing elective cardioversion of atrial fibrillation.

Methods

Study patients. Twenty-one patients undergoing elective DC cardioversion of atrial fibrillation were studied. Excluded were patients who had significant aortic or mitral valve stenosis or regurgitation, a valvular prosthesis or

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echocardiographic evidence of left atrial or left ventricular thrombus. The mean age of the 12 men and 9 women was 71 years (range 49 to 88). The underlying cardiovascular or systemic disorder predisposing to atrial fibrillation was ischemic heart disease in seven, hypertension in six, hyperthyroidism in one, aortic stenosis in three, and mild mitral stenosis in one; there was no evident predisposing condition in three patients. Before cardioversion, patients underwent anticoagulant therapy for ≥2 weeks with doses of oral warfarin sufficient to maintain a prothrombin time of 1.5 to 2 times control. The known duration of atrial fibrillation ranged from 3 weeks to 24 months (mean 5 months). All patients were receiving either digoxin (20 patients), which was withheld for 1 or 2 doses before scheduled cardioversion, or verapamil (1 patient). Treatment with quinidine sulfate, procainamide or encainide was begun the day before scheduled cardioversion in nine patients, two patients and one patient, respectively. Patients were continued on maintenance doses of the same medications throughout the observation period. Precardioversion studies were not performed.

Direct current cardioversion. After induction of general anesthesia with intravenous methohexital sodium (Brevital), DC cardioversion was performed with use of anteroposterior paddles. The initial echocardiographic studies were performed within 1 h after cardioversion, following a recovery period.

Echocardiographic studies. Two-dimensional and pulsed Doppler echocardiograms were obtained with a Hewlett-Packard (model 77020A) combined imaging Doppler echocardiograph and a 2.5 MHz phased array transducer. Pulsed Doppler transmitral inflow velocities were recorded from the apical four chamber view with the sample volume positioned between the tips of the mitral leaflets. Hard copy recordings were made at paper speeds of 50 or 100 mm/s. All recordings were taken during quiet respiration with the patient in the left lateral position.

Doppler signals were digitized with use of a graphics tablet (Summagraphics) and microcomputer (IBM AT) with custom-written software (Datastat). Digitization consisted of manual tracing of the velocity curves. Peak velocities of the early filling (E) wave and atrial filling (A) wave were determined (Fig. 1). The velocity-time integrals of the E and A waves were then obtained by integrating the flow velocity profile at 4 ms intervals. Percent atrial systolic contribution (percent A wave) to total left ventricular filling was assessed by dividing the area under the A wave by the total area under the diastolic velocity-time curve. Heart rate was <90 beats/min in all patients without mitral stenosis, with clear separation of E and A waves. In the patient with mild mitral stenosis, percent A was calculated by the method of Labovitz et al. (11). For all Doppler measurements, values reported represent the mean of measurements made from five cardiac cycles.

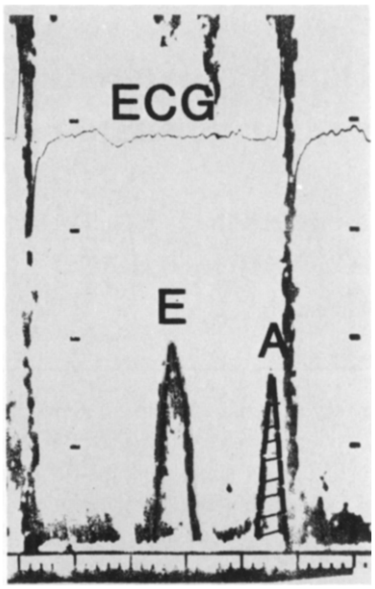


Figure 1. Example of a spectral display of a Doppler transmitral flow velocity profile. The percent A wave filling was calculated as A wave area (**hatched area**) divided by the entire area beneath the diastolic flow velocity profile. A = atrial contraction wave. E = early filling wave; ECG = electrocardiogram.

Left atrial dimension was measured at end-systole in the parasternal long-axis view, with use of the leading edge technique, from two-dimensionally-guided M-mode tracings (12).

Follow-up two-dimensional and pulsed Doppler studies were performed immediately (n = 21), 3 to 6 h (n = 9), 24 h (n = 14), 4 to 7 days (n = 12), 12 to 25 days (n = 12) and 3 months (n = 11) after cardioversion. One patient was lost to follow-up and one patient died during the follow-up period. The follow-up protocol was discontinued if reversion to atrial fibrillation, documented by electrocardiogram (ECG), occurred.

Control group. Pulsed Doppler echocardiograms were also performed in 12 control subjects (9 men and 3 women; mean age 69 yrs, range 62 to 76) without cardiac abnormalities as assessed by history, physical examination or echocardiographic study.

Statistical analysis. All data are expressed as mean ± SD. Statistical significance of serial changes in Doppler variables after cardioversion was assessed with the Student's paired *t* test and multiple-single comparison method. Values for patients and control subjects were compared with the Student's unpaired *t* test. Comparison of immediate and 24 h data was performed by analysis of variance (ANOVA).

Results

In all patients atrial fibrillation was successfully cardioverted to sinus rhythm, with readily discernible atrial depo-

Table 1. Initial and Follow-Up Peak Transmitral A Wave Velocity and Percent A Wave Filling in 21 Patients After Direct Current Cardioversion

	Peak A Wave Velocity (m/s)						Percent A Wave Filling					
Patient	Immed	3 to 4 h	24 h	3 to 5 days	2 to 3 wks	>3 mo	Immed	3 to 6 h	24 h	3 to 5 days	2 to 3 wks	>3 mo
Patients With Reversion to Atrial Fibrillation												
1	0.28		0.21				16.7		14.5			
2	0.83	0.72					30.2	24.2				
3	0.20		0.28	0.26			17.5		21.4	13.9		
4	0.40			0.60	1.03		3.2			9.8	31.0	
5	0.52	0.46	0.56		0.55		4.3	5.6	5.4		18.0	
6	0.47	0.41					11.8	12.4				
7	0.35		0.26				21.4		13.4			
8	0.16	0.18	0.16	0.17			10.8	11.1	8.5	9.4		
Patients Who Maintained Sinus Rhythm During Follow-Up												
9	0.22		0.30		0.39	0.55	29.7		31.6		42.3	52.1
10	0.13		0.26	0.64	1.02	1.18	6.8		11.8	21.5	41.2	43.6
11	0.66			0.74	0.75	0.74	53.3			44.2	49.4	27.7
12	0.09	0.10	0.08	0.08	0.25	0.76	5.9	4.7	3.8	2.3	9.9	28.3
13	0.00	0.25	0.44		0.34	0.35	0.0	18.4	29.3		19.1	21.3
14	0.27			0.51	0.61	0.73	18.5			31.7	34.7	43.7
15	0.28		0.21		0.63	0.69	21.9		14.5		41.8	45.2
16	0.32		0.36	0.38	0.71	0.71	25.8		21.6	18.8	31.9	33.9
17	0.36			0.39	0.54	0.68	22.5			21.6	32.5	40.2
18	0.24	0.39	0.68				11.0	11.8	19.1			
19	0.24		0.30	0.28			17.9		17.8	23.7		
20	0.17	0.21	0.16	0.21		0.60	11.0	13.7	9.5	11.7		40.6
21	0.09	0.23		0.21	0.54	0.50	3.0	6.0		9.0	35.9	31.5

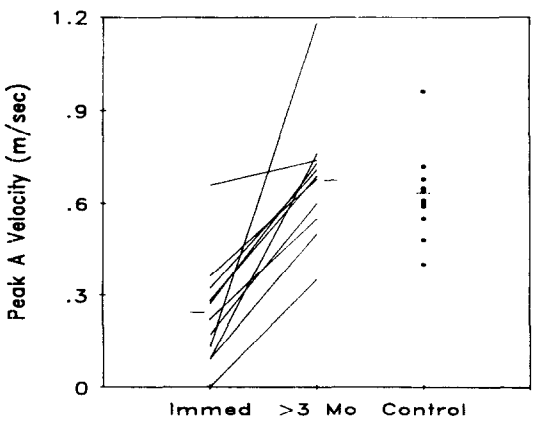
Immed = immediate.

larization waves on ECG. No patient had clinically evident pulmonary or systemic embolism after cardioversion. Initial and follow-up data for individual patients are shown in Table 1.

Pulsed Doppler transmitral recordings. Identifiable atrial filling waves were seen in 20 of the 21 patients on the pulsed Doppler transmitral recordings performed within 1 h of cardioversion. The initial value of mean peak A wave velocity, however, was significantly lower than that value at 3 months ($p < 0.005$) or that in the healthy control subjects ($p < 0.005$) (Fig. 2). At 24 h after successful cardioversion, mean peak A wave velocity of the entire study group had not significantly improved compared with the immediate result (Fig. 3), and remained less than that of the control subjects ($p < 0.005$). In the group with persistent sinus rhythm, mean peak A wave velocity significantly improved over the 3 months of follow-up (Fig. 4). Figure 5 shows serial pulsed Doppler signals obtained from a single individual over a 3 month follow-up period. There were continued increases in peak A wave velocity and percent A wave filling over the observation period. Neither initial nor 24 h peak A velocity was predictive of the maintenance of sinus rhythm. Further, there was no significant correlation between the duration of atrial fibrillation and initial peak A wave velocity after cardioversion.

The percent atrial contribution to total left ventricular filling (percent A wave) was also significantly lower immediately after cardioversion than in control subjects ($p < 0.005$) (Fig. 6). By 12 to 22 days after cardioversion, percent

Figure 2. Comparison of individual peak transmitral A wave velocities immediately (Immed) and at 3 months after direct current cardioversion in 11 patients with persistent sinus rhythm. Values for 12 normal control subjects are also plotted. Mean values for each group are indicated by the **horizontal bars**.



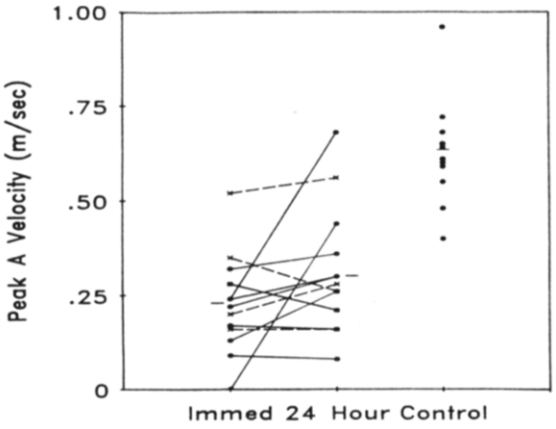


Figure 3. Comparison of individual peak transmitral A wave velocities immediately and at 24 h after cardioversion in 14 patients. The **dashed lines** (x – – x) indicate five patients subsequently reverting to atrial fibrillation and the **solid lines** (● – – – – – ●), nine patients who maintained sinus rhythm. Values for 12 control subjects are also plotted. Mean values for each group are indicated by the **horizontal bars.**

A wave filling had significantly increased and was not different from that in the control group.

Follow-up: reversion to atrial fibrillation. Over the 3 months of follow-up, spontaneous reversion to atrial fibrillation occurred in eight patients (in six within 2 weeks and in two between 2 weeks and 2 months of cardioversion). Immediately after cardioversion, peak A wave velocity was significantly higher (0.40 ± 0.21 m/s) in patients with ultimate reversion to atrial fibrillation than in those who remained in sinus rhythm (0.24 ± 0.16 m/s, $p < 0.05$). In patients in whom sinus rhythm was maintained, however, peak A wave velocity increased over the observation period,

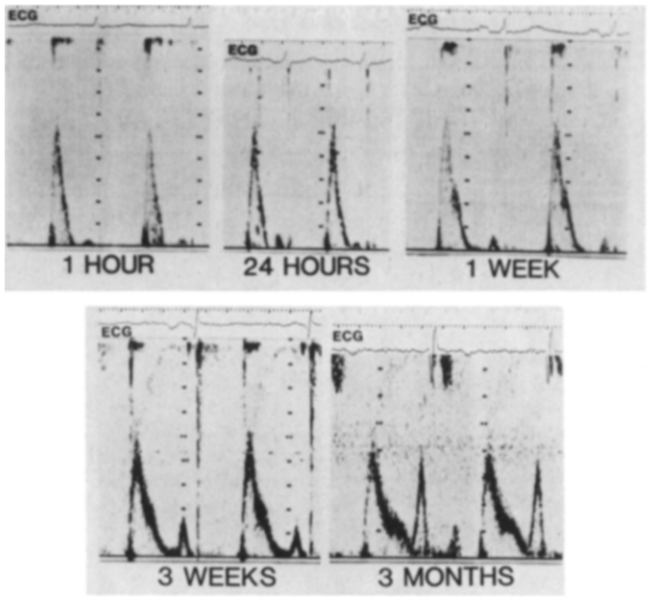


Figure 5. Serial transmitral pulsed Doppler inflow velocity profiles from an individual patient. Note the progressive increase in peak *A* wave velocity and percent A wave filling over the observation period. For each spectrum, the vertical calibration markings indicate 0.2 m/s flow velocity; horizontal distances between calibration markings indicate 1 s.

whereas in six of eight patients with reversion to atrial fibrillation, peak A wave velocity declined or failed to increase during the observation period. Repeated ANOVA of the immediate and 24 A wave velocities did not demonstrate a statistical difference between these two groups.

Left atrial dimension. At study entry there was no significant difference in left atrial dimension between the group that remained in sinus rhythm (4.2 ± 1.0 cm) and the group

Figure 4. Group mean ± SD peak transmitral A wave velocities for the 3 months after cardioversion in 11 patients who maintained sinus rhythm throughout the follow-up period. * = $p < 0.05$ versus immediate study; ** = $p < 0.005$ versus immediate study; # = $p < 0.005$ versus 24 h study; & = $p < 0.05$ versus 4 to 7 day study. D = days; H = hours; Imm = immediate study; M = months.

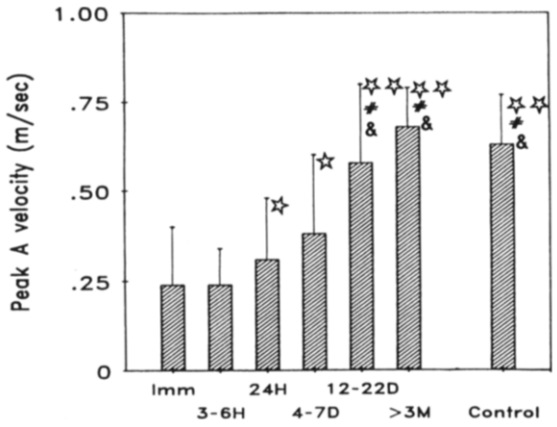
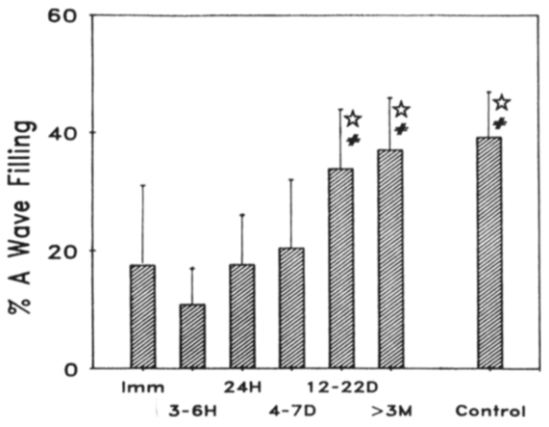


Figure 6. Group mean ± SD percent A wave contribution to total left ventricular filling in 11 patients who maintained sinus rhythm throughout the follow-up period. * = $p < 0.005$ versus immediate study, # = $p < 0.05$ versus 24 h study and versus 4 to 7 day study. Abbreviations as in Figure 5.



with reversion to atrial fibrillation (4.3 ± 0.7 cm). Over 3 months of follow-up, left atrial dimension at end-systole decreased slightly in patients with persistent sinus rhythm (to 3.8 ± 1.0 cm, $p < 0.05$). Mean heart rate in this same group did not significantly change over the observation period.

Discussion

Atrial contribution to ventricular filling. A forceful, appropriately timed atrial contraction augments ventricular filling. Harvey (13) first recognized the importance of the atrial contribution to ventricular filling in 1628. Subsequent experimental and clinical studies have confirmed its importance. Atrial contribution to ventricular filling, even at rest, is increased in many disease states associated with impaired left ventricular diastolic function, including hypertrophic and some dilated cardiomyopathies, secondary hypertrophy and left ventricular ischemia (14–18). In addition, impaired diastolic function has been associated with the normal aging process (19), making healthy older subjects more dependent than younger individuals on the atrial contribution to ventricular filling.

Invasive evaluation of atrial systolic function after cardioversion of atrial fibrillation. The ability to pharmacologically or electrically convert atrial fibrillation was associated with the expectation that atrial systolic function would return concomitantly with atrial electrical activity. Invasive evaluation of atrial function after cardioversion has included the assessment of the presence and height of atrial pressure waves in right atrial and left atrial (or pulmonary capillary wedge) pressure tracings and of changes in stroke volume or cardiac output. Scott and Patterson (20) serially assessed cardiac output over 3 days after cardioversion, using a dye-dilution technique. They found no significant change in rest cardiac output immediately after cardioversion or at 3 h. Increases in stroke volume and cardiac output, however, were evident 3 days after restoration of sinus rhythm. Reznekov (21) also found no immediate change in cardiac output at rest, assessed by the Fick technique, but did observe a significantly greater increase in cardiac output with exercise after restoration of normal sinus rhythm. Duchelle (7) evaluated the height of the atrial pressure wave in right atrial tracings immediately after cardioversion, and at 24 h. An increase in atrial wave pressure was noted only at 24 h. Rowlands et al. (9) measured right and left atrial pressures in patients immediately after cardioversion. Twelve of 13 patients with successful cardioversion were noted to have right atrial pressure waves. Only five of eight patients undergoing simultaneous left atrial measurements developed discernible left atrial contraction waves. Two patients who developed right atrial contraction waves had no evident return of left atrial function.

Noninvasive evaluation of atrial systolic function after cardioversion of atrial fibrillation. Before the development of Doppler echocardiographic techniques, the noninvasive evaluation of left atrial function focused primarily on findings from physical examination, apex kinetocardiography and M-mode echocardiography. Ikram et al. (5) recorded displacement curves at the left ventricular apex in 12 patients undergoing cardioversion from atrial fibrillation. Five of the patients manifested an a wave in the left ventricular displacement curve immediately after cardioversion. Six patients first developed evidence of left atrial activity 3 to 6 days after cardioversion and one patient had no evident return of atrial function throughout the 3 week observation period. In contrast, return of right atrial function, assessed by presence of a waves in the jugular venous pulse tracing, generally appeared immediately after cardioversion. Also using kinetocardiography, Mahlich et al. (22) detected atrial mechanical function in almost all patients both immediately and at 24 h after cardioversion. In half of the patients, the amplitude of the atrial waves was normal. Furthermore, patients maintaining sinus rhythm chronically after cardioversion were noted to have higher a waves immediately and at 24 hours after cardioversion than those patients with recurrent fibrillation. These findings conflict with our data, which do not support the hypothesis that the strength of atrial contraction has prognostic significance for persistence of sinus rhythm in patients undergoing cardioversion for atrial fibrillation. Further, our data failed to show a relation between the duration of atrial fibrillation and initial peak A wave velocity after cardioversion.

More recently, Dethy et al. (23), also using Doppler mitral inflow velocities, suggested that early restoration of atrial mechanical activity does identify a group of patients more likely to remain in sinus rhythm. Our disparate findings may, in part, be due to different patient characteristics, including differences in age, etiology of atrial fibrillation, left ventricular function and exclusion criteria. None of our patients had significant mitral regurgitation or severe mitral stenosis. In addition, our patient sample is small. A larger study may be necessary to determine whether the strength of atrial activity immediately after conversion has any predictive role relative to maintenance of sinus rhythm. Mahlich et al. (22) noted that a decrease in the size of the left atrial displacement wave over the first 24 h was often observed in patients with reversion to atrial fibrillation. We also observed a similar trend in our patients with reversion to atrial fibrillation.

Duration of atrial fibrillation may also play a role in the return of left atrial systolic activity. Ikram et al. (5) studied patients who had successful cardioversion of atrial fibrillation of ≥5 years' duration. Left atrial mechanical activity, as assessed by the presence of an a wave on the left ventricular apex displacement curve, was apparent in only 5 of the 12 patients immediately after cardioversion. Interestingly, six of the seven patients who had no apparent left atrial activity

did have a waves in jugular venous tracings, suggesting return of right atrial mechanical activity. Left atrial a waves were detected in three more patients several days after cardioversion. Delay in return of a detectable left atrial a wave was more common in patients with underlying rheumatic heart disease. Although not statistically significant, mean duration of atrial fibrillation was longer (6.8 ± 12 months) for our group of patients with reversion to atrial fibrillation than in the group with persistent sinus rhythm (4.2 ± 4 months).

Echocardiography would appear to be a more sensitive technique for detecting atrial systolic activity than left ventricular apex displacement and recording of a waves from the jugular venous pulse. DeMaria et al. (24) used M-mode echocardiography to evaluate diastolic movement of the mitral valve before and 1 h after successful elective cardioversion of atrial fibrillation. Thirty-three of 35 patients were noted to have A waves on the M-mode echocardiogram 1 h after cardioversion. The mean value for A wave amplitude (7.5 mm) in the postcardioversion patients, however, was less than the mean amplitude of 13 mm observed in normal subjects, suggesting that atrial contraction, although present soon after cardioversion, was reduced. The two patients with atrial electromechanical dissociation had reversion to atrial fibrillation within 1 week, whereas of those with evident A waves 1 h after cardioversion, 84% remained in normal sinus rhythm at 3 weeks and 54% remained in sinus rhythm at 6 months. Orlando et al. (25) also used M-mode echocardiography to evaluate atrial function after cardioversion. Eleven (73%) of their 15 patients had a detectable A wave on the mitral valve echogram 5 h after cardioversion. Sixty-seven percent of their patients remained in normal sinus rhythm at follow-up studies ranging from 1 week to 9 months. In their study, as in ours, neither the presence of an A wave nor its amplitude correlated with long-term maintenance of sinus rhythm.

Role of peak A wave velocities on Doppler echocardiography. The present study employed another echocardiographic technique, pulsed wave Doppler echocardiography, to evaluate flow velocities and integrals of flow velocity across the mitral valve. This Doppler technique has been shown to provide accurate information about the time course of ventricular filling and the relative contribution of atrial systole to total ventricular filling (10). Our studies corroborate findings from previous studies, using displacement curves and M-mode echocardiography, that there is early return of some atrial transport function in the majority of patients undergoing electrocardioversion of atrial fibrillation. The strength of atrial contraction, however, appears reduced in the early postcardioversion period. DeMaria et al. (24) did not extend their observations beyond 1 h after cardioversion.

Our results suggest that the strength of atrial contraction progressively increases over several weeks to several months after cardioversion. By 3 weeks, the contribution of atrial systole to total left ventricular filling did not differ significantly from that seen in control subjects. Our control subjects represented a group of healthy individuals, similar in age to the patient population. The elderly patients undergoing cardioversion included patients with concomitant left ventricular dysfunction due to ischemia, cardiomyopathy and left ventricular hypertrophy, conditions that have been associated with increased dependence on atrial systole for adequate ventricular filling (15–18). It is possible that the full peak A wave velocities in our patients might be higher than those observed in the normal elderly control population. Because patients were not studied in sinus rhythm before the onset of fibrillation and because the follow-up period was limited to 3 months after cardioversion, the possibility that further increases in A wave amplitude and flow velocity integral would have occurred cannot be excluded. Three of our patients have had repeat studies 12 months after cardioversion. There was no further change from the 3 month data in peak A wave velocity or percent A wave filling in this small group.

Potential clinical implications. The finding that atrial contraction may be weak immediately after cardioversion, with progressive increases in the strength of atrial activity over the next 3 weeks, would appear to have important clinical implications. If atrial contraction is weak, patients may remain at risk for formation of atrial thrombus, which may then result in late embolism after return of more effective atrial contraction. Consideration should be given to continuing anticoagulant therapy for at least several weeks after cardioversion, or until there is documented return of a strong atrial contraction. Second, the full hemodynamic benefit resulting from restoration of atrial mechanical function will likely not be evident for several weeks after successful cardioversion. Premature assessment of hemodynamic status after cardioversion may thus cause underestimation of the benefit of maintaining sinus rhythm.

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