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Impaired Left Atrial Mechanical Function After Cardioversion: Relation to the Duration of Atrial Fibrillation

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Objectives. We hypothesized that the time course of the recovery of atrial systolic function may be related to the duration of atrial fibrillation before cardioversion and sought to study noninvasively the recovery of left atrial mechanical function utilizing serial transthoracic Doppler studies.

Background. Recovery of atrial mechanical function may be delayed for several weeks after successful cardioversion of atrial fibrillation to sinus rhythm.

Methods. After successful cardioversion, 60 patients with atrial fibrillation of brief (≤2 week, 17 patients), moderate (>2 to 6 weeks, 22 patients) or prolonged (>6 weeks, 21 patients) duration were followed up with serial transmitral pulsed Doppler echocardiography immediately (60 patients) and at 24 h (45 patients), 1 week (41 patients), 1 month (31 patients) and >3 months (30 patients) after cardioversion.

Results. Atrial mechanical function is greater immediately and at 24 h and 1 week after cardioversion in patients with "brief" compared with "prolonged" atrial fibrillation. In all groups, atrial mechanical function increases over time, ultimately achieving similar levels. Full recovery of atrial mechanical function, however, is achieved within 24 h in patients with brief atrial fibrillation, within 1 week in patients with moderate-duration atrial fibrillation and within 1 month in patients with prolonged atrial fibrillation.

Conclusions. Recovery of left atrial mechanical function is related to the duration of atrial fibrillation before cardioversion. These findings have important implications for assessing the early hemodynamic benefit of successful cardioversion.

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Atrial fibrillation is a common arrhythmia, occurring in 0.4% of the general population and in up to 4% of people >60 years of age (1,2). Clinically atrial fibrillation is characterized by symptoms of palpitations, and physiologically by a lack of organized atrial mechanical activity with a concomitant decrease in stroke volume and cardiac output (3). This loss of a unified atrial contraction results in blood stasis, a condition that favors the formation of thrombi, which may subsequently embolize. Chemical or electrical cardioversion of atrial fibrillation is generally performed in an effort to improve cardiac function, relieve symptoms and decrease the incidence of thrombus formation (4). Successful cardioversion, however, is sometimes associated with a thromboembolic event. Although they often occur immediately after cardioversion, such events have been described several days to weeks after cardioversion (5–7) in patients who have apparently maintained sinus rhythm.

Pulsed Doppler echocardiography offers the opportunity to evaluate noninvasively the left atrial systolic contribution to total left ventricular filling by measuring flow across the mitral orifice during atrial systole. We have previously reported the marked delay in return of left atrial mechanical function in a small group of patients with atrial fibrillation of several months' duration (8) and hypothesized that the delay in the return of atrial mechanical function is related to the duration of atrial fibrillation before cardioversion. We now report our findings in a group of 60 patients with atrial fibrillation of 0.3 to 36 weeks' duration who underwent serial echocardiographic study after successful cardioversion.

Methods

Study patients. We studied 60 adult patients who underwent successful cardioversion from atrial fibrillation to sinus rhythm at the Beth Israel Hospital (49 patients, including 19 previously studied [8]) and the University of Connecticut Health Center (11 patients) (28 men, 32 women; mean [±SD]

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age 73 ± 12 years, range 30 to 90), with an estimated duration of atrial fibrillation of 6.0 ± 7.4 weeks (0.3 to 2 weeks, 17 patients [28%]; >2 to 6 weeks, 22 patients [37%]; >6 weeks, 21 patients [35%]). No patient had severe aortic or mitral stenosis or a prosthetic mitral valve. The underlying cardiovascular or systemic disorder predisposing to atrial fibrillation included one or more of the following: hypertension (29 patients), ischemic heart disease (13 patients), pneumonia (3 patients), mild mitral stenosis (1 patient) and alcohol intake (1 patient). In 18 patients, no underlying predisposing condition was identified. All patients were receiving either oral digoxin (45 patients), a calcium-channel blocking agent (verapamil, 4 patients; diltiazem, 5 patients) or a beta-adrenergic blocking drug (11 patients) for rate control before cardioversion. Patients continued to receive maintenance doses of the same medications throughout the observation period.

Echocardiographic studies. Two-dimensional imaging and two-dimensional guided pulsed Doppler transthoracic echocardiographic studies were obtained with Hewlett-Packard model 77020A, Sonos 500, Sonos 1000 or Sonos 1500 echocardiograph with a 2.0- or 2.5-MHz transducer or Acuson 128XP/10 echocardiograph with 2.0- or 2.5-MHz transducer. M-mode left atrial dimension was measured at end-systole in the parasternal long-axis view using standard techniques (9). Transmitral Doppler inflow velocities were recorded from the apical four-chamber view with the sample volume positioned between the tips of the mitral leaflets. All measurements were made during quiet respirations with the patient in the left lateral position. Hard-copy recordings were made at paper speeds of 50 or 100 mm/s.

Doppler data from three to five consecutive spectra were digitized utilizing an off-line workstation (Cardiology Workstation, Freeland Systems) by an observer unaware of the clinical history. Peak velocities of the early filling (E) wave and atrial filling (A) wave were determined, as were their velocity time integrals, percent E filling and percent A filling, respectively.

Echocardiographic studies were performed immediately (<2 h, 60 patients), and at 24 h (45 patients), 1 week (41 patients), 1 month (31 patients) and >3 months (30 patients) after cardioversion. One patient was lost to follow-up, and two refused to return for their >3-month studies because of physical disabilities or transportation difficulties. Both of the latter two patients were in sinus rhythm at clinical follow-up with their primary physicians >3 months after study entry. The follow-up protocol was discontinued if the patient's rhythm reverted to atrial fibrillation, as documented by electrocardiography.

Cardioversion. Cardioversion from atrial fibrillation was accomplished chemically in 21 patients (quinidine, 14 patients; procainamide, 4 patients; disopyramide, 1 patient; flecainide, 2 patients) and by direct current cardioversion in 39 patients with anteroposterior paddles, after pharmacologic conversion attempts had failed.

Statistical analysis. All data are expressed as mean values ± 1 SD. Analysis of serial changes in peak A velocity and percent A-wave filling among groups immediately after cardioversion and at 24 h and 1 week was performed by analysis of variance for repeated measures. Statistical significance of serial changes in Doppler variables after cardioversion in individual groups was assessed with the Student paired t test and multiple single-comparison method. Changes in left atrial size between patients who remained in sinus rhythm and those who reverted to atrial fibrillation were compared by use of the Mann-Whitney U test for unpaired samples, and comparisons within groups of study entry and 3-month data were made using the Student paired t test. A p value ≤ 0.05 was considered significant, and in all instances a two-tailed test was performed.

The protocol was approved by the Investigational Review Board of both hospitals, and informed consent was obtained from all participants.

Results

After cardioversion, all patients had readily discernible atrial depolarizations on their electrocardiogram, and no patient had clinical evidence of an immediate or delayed thromboembolic event.

Pulsed Doppler transmitral recordings. Both the immediate peak A velocity and percent A-wave velocities were significantly lower (Fig. 1) in the groups with atrial fibrillation of moderate (>2 to 6 weeks) and prolonged (>6 weeks) duration compared with patients with atrial fibrillation of only brief (≤2 weeks) duration (both $p < 0.05$). This depression in peak A velocity was also present at the 1-week study ($p < 0.05$). In addition, at 1 week after cardioversion, both peak A velocity and percent A-wave filling were significantly depressed in the group with atrial fibrillation of prolonged duration compared with the group with atrial fibrillation of moderate duration (Fig. 1) (both $p < 0.05$).

Serial evaluation of the group with atrial fibrillation of brief duration (≤2 weeks) demonstrated a significantly lower peak A velocity immediately after cardioversion compared with 1-week, 1-month and >3-month studies. No significant depression was present at 24 h compared with 1- or 3-month studies (Fig. 2). Analysis of percent A-wave filling failed to demonstrate a significant difference of this index on any study, with the immediate postcardioversion data being similar to >3-month data.

The study group with atrial fibrillation of more moderate duration (2 to 6 weeks) demonstrated a significantly depressed peak A velocity (Fig. 3) and percent A-wave filling immediately after cardioversion and at 24 h compared with 1-week and >3-month studies. Although there was continued improvement in the group mean of both indexes after the 1-week study, this did not reach statistical significance.

Patients with atrial fibrillation of prolonged duration (>6 weeks) demonstrated an even more pronounced delay in the return of atrial function. Both peak A-wave velocity (Fig. 4)

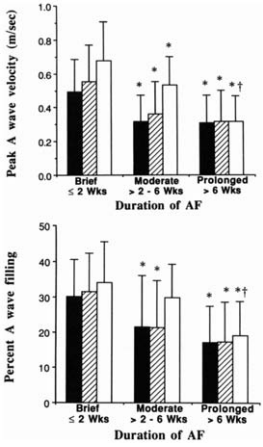


Figure 1. Comparison of (top) peak A-wave velocity and (bottom) percent A-wave filling in patients with brief, moderate and prolonged duration of atrial fibrillation immediately after (solid bars) and at 24 h (hatched bars) and 1 week (open bars) after cardioversion. *p < 0.05 versus brief duration. †p < 0.05 versus moderate duration.

and percent A-wave filling did not change over the first week after cardioversion. At 1 month after cardioversion, peak A-wave velocity had markedly increased compared with baseline, 24-h and 1-week data. No further improvement was noted at >3 months. Percent A-wave filling demonstrated a significant improvement at both the 1- and 3-month studies ($p < 0.05$).

Follow-up: reversion to atrial fibrillation. Twenty-seven (45%) patients reverted to atrial fibrillation during follow-up, as documented by electrocardiography. The duration of atrial fibrillation before cardioversion was longer in the group that reverted to atrial fibrillation compared with the group with sustained sinus rhythm (10.2 ± 10.1 vs. 5.3 ± 5.9 weeks, p = 0.04). There was no difference in left atrial dimension (4.5 ± 0.5 vs. 4.4 ± 0.6 cm, p = NS), patient age (72 ± 10 vs. 73 ± 13 years, p = NS) or mode of cardioversion between these two groups, with 33% of those converting with pharmacologic therapy reverting to atrial fibrillation during follow-up (vs. 44% of those converted electrically, p = NS). Analysis of immediate, 24-h and 1-week data from patients who remained in sinus rhythm throughout the follow-up period compared with those who reverted to atrial fibrillation did not reveal a statistical difference in either peak A-wave velocity or percent A-wave filling (Fig. 5).

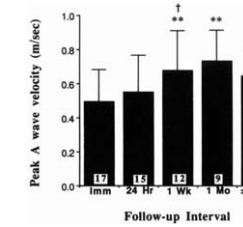


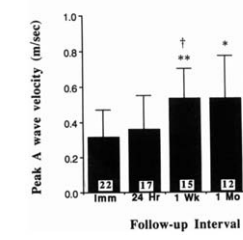
Figure 2. Serial peak A-wave velocity in the group with atrial fibrillation of brief duration. *p < 0.05 and **p < 0.02 versus immediately (Imm) after cardioversion. †p < 0.03 versus 24 h after cardioversion. Numbers within bars are numbers of patients in sinus rhythm studied at each interval.

Left atrial dimension. Assessment of left atrial size demonstrated no difference among the three groups in initial postcardioversion left atrial dimension data. Follow-up changes in left atrial dimension demonstrated no significant change between the immediate postcardioversion and >3-month studies for the group with brief atrial fibrillation (4.4 ± 0.5 vs. 4.3 ± 0.5 cm, p = NS). There was a trend toward a decrease in left atrial dimension in the group with moderate-duration atrial fibrillation (4.5 ± 0.6 vs. 4.1 ± 0.9 cm, p = 0.076). Analysis of those with prolonged atrial fibrillation demonstrated a significant decrease in left atrial dimension during the 3-month follow-up period (4.5 ± 0.6 vs. 4.2 ± 0.6 cm, p = 0.023).

Discussion

In this prospective study, we demonstrated the time-dependent recovery of left atrial mechanical function and its

Figure 3. Serial peak A-wave velocity in the group with atrial fibrillation of moderate duration. *p < 0.05 and **p < 0.01 versus immediately (Imm) after cardioversion. †p < 0.05 versus 24 h after cardioversion. Numbers within bars are numbers of patients in sinus rhythm studied at each interval.



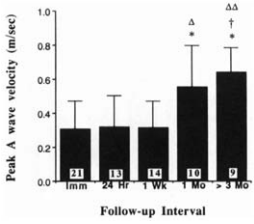


Figure 4. Serial peak A-wave velocity in the group with atrial fibrillation of prolonged duration. *$p < 0.03$ versus immediately (Imm) after cardioversion. †$p < 0.05$ versus 24 h after cardioversion. Δ$p < 0.03$ and ΔΔ$p < 0.01$ versus 1 week after cardioversion. **Numbers within bars** are numbers of patients in sinus rhythm studied at each interval.

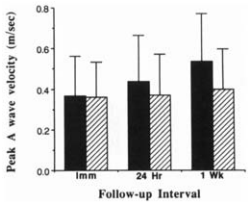


Figure 5. Serial peak A-wave velocity in 32 patients who remained in sinus rhythm (**solid bars**) and in 27 patients who reverted to atrial fibrillation (**hatched bars**) immediately (Imm) and at 24 h and 1 week after cardioversion.

relation to the duration of atrial fibrillation before cardioversion. Patients with atrial fibrillation of only brief (≤2 weeks) duration displayed a rapid return of atrial mechanical function on restoration of sinus rhythm, whereas those with atrial fibrillation of prolonged (>6 weeks) duration had a prominent delay in return of atrial systolic function.

Noninvasive evaluation of atrial mechanical function after cardioversion. Previous investigators have utilized a variety of techniques to examine the return of atrial mechanical function after cardioversion in patients with prolonged atrial fibrillation. Ikram et al. (10) studied patients with atrial fibrillation of ≥5 years' duration using apex kinetocardiography and found left atrial mechanical activity present in <50% of patients immediately after cardioversion. More than 40% of those without evident left atrial mechanical activity developed evidence of activity over the ensuing several days. DeMaria et al. (11) examined M-mode recordings of mitral valve excursion in a group of patients with atrial fibrillation for a mean duration of 14 months and found the A-wave amplitude to be depressed after cardioversion. In 1989 we reported the serial transmitral Doppler evaluation of a group of patients with a mean duration of atrial fibrillation of 5 months before cardioversion (9). Patients with atrial fibrillation of such prolonged duration displayed a depression of both peak A velocity and percent A-wave filling that persisted for several weeks after successful cardioversion. Transesophageal echocardiography, with its superior ability for visualizing the left atrium and left atrial appendage, has been used to assess atrial function after cardioversion. The new appearance of spontaneous echo contrast after electrical cardioversion has recently been described by Grimm et al. (12) and suggests stunning of the atrium associated with cardioversion. These investigators have also described depressed left atrial appendage function immediately after cardioversion as seen on pulsed Doppler spectra (12).

Investigations of patients with short- and moderate-duration atrial fibrillation. Few data are available on atrial mechanical function in patients with atrial fibrillation of very brief duration (<2 days), possibly because of the assumption that atrial fibrillation of such transient length would not be associated with a depression of atrial systolic function. This assumption was confirmed by Shapiro et al. (13) who reported transmitral Doppler data in 18 patients with "acute atrial fibrillation" (mean duration 1.8 ± 0.4 days) after cardioversion to sinus rhythm. These investigators found no depression in peak A-wave velocity or percent A-wave filling when patients were studied within 24 h of cardioversion.

Studies of atrial mechanical function in patients with atrial fibrillation of an intermediate (3 days to 4 weeks) duration have been limited by the relative scarcity of such patients. This is most likely because of the recommendation that patients with atrial fibrillation of >2-day duration receive anticoagulation for 3 to 4 weeks before cardioversion to decrease the incidence of embolic complications associated with cardioversion (14). Cardioversion in patients who would be assigned to this intermediate group at presentation is therefore postponed, placing them in the prolonged group with its greater delay in the return of atrial mechanical function.

Our group has been conducting a trial investigating the use of transesophageal echocardiography to assess the atria for thrombi and thereby facilitate early cardioversion with only short-term anticoagulation in patients in whom thrombi are not seen (15). This concurrent transesophageal study has allowed us to recruit patients with a duration of atrial fibrillation of 2 days to 4 weeks before cardioversion and to study the return of atrial mechanical function in patients with atrial fibrillation of an intermediate duration. All patients in this report with a duration of atrial fibrillation <4 weeks either were receiving long-term warfarin therapy or were participants in the transesophageal echocardiographic study.

The finding that a rapid return of atrial mechanical function is found in the group with atrial fibrillation of brief (≤2 weeks) duration and a more rapid return is found in

patients with atrial fibrillation of moderate duration (compared with prolonged duration) is particularly important because a significant number of patients admitted to the hospital with new atrial fibrillation have an estimated duration of atrial fibrillation within this intermediate time period. Almost two-thirds of patients admitted to our hospital during the past 2 years have a clinically estimated duration of atrial fibrillation <1 month. Although our study does not specifically address differences in clinical outcome in relation to impaired atrial mechanical function, it is logical to assume that patients with a significant atrial contribution would have improved cardiac performance and that a goal of treatment should be a more rapid return of atrial mechanical function.

Potential clinical implications. These results also have potential clinical implications for guiding the duration of anticoagulation after cardioversion. Warfarin anticoagulation is commonly administered for several weeks after cardioversion for prophylaxis against thrombus formation both during return of atrial mechanical function and in case of reversion to atrial fibrillation. The current study demonstrates that patients with a duration of atrial fibrillation of <6 weeks before cardioversion have nearly complete recovery of atrial mechanical function within 1 week. These data suggest that these patients may be at lower risk for new thrombus formation after this initial period. Studies by others have also suggested that maintenance of sinus rhythm is related to the duration of atrial fibrillation before cardioversion (16). Before short-term anticoagulation can be recommended in this group, however, more detailed studies examining the relation between maintenance of sinus rhythm and duration of atrial fibrillation will need to be done.

We did not find an association between the return of atrial mechanical function and maintenance of sinus rhythm during the follow-up period. This may be because a patient who develops atrial fibrillation in response to myocardial ischemia is likely to revert to atrial fibrillation when ischemia recurs, regardless of peak A velocity when in sinus rhythm. Other investigators (17) have also failed to find a relation between peak A-wave velocity after cardioversion and maintenance of sinus rhythm but have cited a lower percent increase in A-wave velocity at 24 h (<10%) as having predictive value for the recurrence of atrial fibrillation.

Changes in left atrial dimension with atrial fibrillation. We found no significant change in left atrial dimension during the follow-up period for the groups with atrial fibrillation of brief and moderate duration. These data are consistent with those recently reported by Petersen et al. (18), who studied the change in left atrial dimension in patients with atrial fibrillation <3 months and >1 year in duration. The greatest increase was found in the latter group, with only a minimal increase in the former. Their data suggest that the increase in left atrial dimension is related to the duration of atrial fibrillation. Our data suggest that the decline in left atrial size associated with successful cardioversion occurs primarily in those patients with prolonged atrial fibrillation. This may be because this same group has experienced the largest growth in left atrial size during the period of atrial fibrillation.

Study limitations. A limitation of our study is that peak A-wave velocity and percent A-wave filling provide only indirect assessment of atrial mechanical function. These indexes have been shown to be dependent on heart rate, age, preload, afterload, ventricular compliance and Doppler sample position. Heart rate did not significantly change during the follow-up period for each group, and care was made to maintain consistent pulsed Doppler sample position with each study by having the same technician perform serial studies on individual patients. In addition, different antiarrhythmic agents, atrioventricular node blocking agents and cardioversion regimens were used, and the affects of these on atrial mechanical function is unknown. Each patient, however, served as his or her own control for follow-up data. Further studies will need to be performed to address these issues.

Conclusions. Recovery of atrial mechanical function is dependent on the duration of atrial fibrillation before cardioversion. Patients with atrial fibrillation of only brief duration demonstrate recovery of atrial mechanical function very quickly (within 24 h), whereas atrial function of those with moderate-duration atrial fibrillation recovers within 1 week. Recovery of atrial function among those patients with atrial fibrillation of prolonged duration may extend several weeks. These data have important implications for assessing the early hemodynamic benefit of successful cardioversion and potentially for guiding the duration of anticoagulant therapy after cardioversion. Because warfarin is also advocated for prophylaxis should the patient revert to atrial fibrillation, further studies need to be performed before short-term postcardioversion anticoagulation can be advocated for those with brief atrial fibrillation.

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References

1. Ostrander LC Jr, Brandt RL, Kjelsberg MO, Epstein FH. Electrocardiographic findings among the adult population of a total natural community, Tecumseh, Michigan. Circulation 1965;31:888–98.
2. Kannel WB, Wolf PA. Epidemiology of atrial fibrillation. In: Falk RH, Podrid P, editors. Atrial Fibrillation: Mechanisms and Management. New York: Raven Press, 1992:81–92.
3. Morris JJ, Entman M, North WC, Kong Y, McIntosh H. The changes in cardiac output with reversion of atrial fibrillation to sinus rhythm. Circulation 1965;31:670–8.
4. Pritchett ELC. Management of atrial fibrillation. N Engl J Med 1992;326: 1264–71.
5. Resnekov L, McDonald L. Complications in 220 with cardiac dysrhythmias treated by phased direct current shock and indications for electroconversion. Br Heart J 1967;29:926–35.
6. Bjerkelund CH, Orning OM. The efficacy of anticoagulant therapy in preventing embolism related to DC electrical conversion of atrial fibrillation. Am J Cardiol 1969;23:208–15.
7. Arnold AZ, Mick MJ, Mazurek RP, Loop FD, Trohman RG. Role of

prophylactic anticoagulation for direct current cardioversion in patients with atrial fibrillation or atrial flutter. J Am Coll Cardiol 1992;19:851–5.

8. Manning WJ, Leeman DE, Gotch PJ, Come PC. Pulsed Doppler evaluation of atrial mechanical function after electrical cardioversion of atrial fibrillation. J Am Coll Cardiol 1989;13:617–23.
9. Sahn DJ, DeMaria A, Kisslo J, Weyman A. Recommendations regarding quantification in M-mode echocardiography: results of a survey of echocardiographic measurements. Circulation 1978;58:1072–83.
10. Ikram H, Nixon PGF, Arcan T. Left atrial function after electrical *conversion to sinus rhythm. Br Heart J 1968;30:80–3.*
11. DeMaria AN, Lies JE, King JF, Miller RR, Amsterdam EA, Mason DT. Echographic assessment of atrial transport, mitral movement and ventricular performance following electroversion of supraventricular arrhythmias. Circulation 1975;51:273–82.
12. Grimm RA, Stewart WJ, Maloney JD, et al. Impact of electrical cardioversion for atrial fibrillation on left atrial appendage function and spontaneous echo contrast: characterization by simultaneous transesophageal echocardiography. J Am Coll Cardiol 1993;22:1359–66.
13. Shapiro EP, Effron MB, Lima S, Ouyang P, Siu CO, Bush D. Transient atrial dysfunction after conversion of chronic atrial fibrillation to sinus rhythm. Am J Cardiol 1988;62:1202–7.
14. Dunn M, Alexander J, de Silva R, Hildner F. Antithrombotic therapy in atrial fibrillation. Chest 1989;95 Suppl:118s–27s.
15. Manning WJ, Silverman DI, Gordon SPF, Krumholz HM, Douglas PS. Cardioversion from atrial fibrillation without prolonged anticoagulation using transesophageal echo to exclude atrial thrombi. N Engl J Med 1993;328:750–5.
16. *Dittrich HC, Erickson JS, Schneiderman T, Blacky AF, Savides T, Nicod* PH. Echocardiographic and clinical predictors for outcome of elective cardioversion of atrial fibrillation. Am J Cardiol 1989;63:193–7.
17. Dethy M, Chassai C, Roy D, Mercier LA. Doppler echocardiographic predictors of recurrence of atrial fibrillation after cardioversion. Am J Cardiol 1988;62:723–6.
18. Petersen P, Kastrup J, Brinch K, Godtfredsen J, Boysen G. Relation between left atrial dimension and duration of atrial fibrillation. Am J Cardiol 1987;60:382–4.