Atrial Mechanical Performance After Internal and External Cardioversion of Atrial Fibrillation*
An Echocardiographic Study

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Objectives: To compare the time course of resumption of mechanical performance of the left and right atrium after the novel method of internal low-energy cardioversion (CV) and conventional external CV of atrial fibrillation (AF).

Background: Right atrial performance has been shown to normalize before the left atrium after external CV. However, no data on atrial function after internal CV are available.

Patients and interventions: Sixty-three patients with chronic AF were randomized to participate in either external or internal CV.

Measurements: Echocardiographic examinations were carried out before as well as immediately after CV (day 0), and at days 1, 7, and 28 thereafter for the determination of cardiac dimensions, volumes, and transvalvular flow patterns.

Results: After randomized internal CV or external CV, stable sinus rhythm was restored in 59 patients. Irrespective of the mode of CV, the right atrium resumed its mechanical function immediately after CV, whereas the left atrium was stunned beyond day 7. The mode of CV, internal or external, had no influence on the recovery of atrial mechanical function.

Conclusions: The right atrium resumes its normal function immediately after internal as well as external CV, whereas left atrium function is delayed. In contrast to the assumption that low-energy internal CV would impact less on atrial mechanical recovery, the type of method of CV used has no effect on such recovery.

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Key words: atrial fibrillation; atrial performance; echocardiographic parameters; external cardioversion; internal cardioversion

Abbreviations: A = active atrial contraction; AF = atrial fibrillation; CV = cardioversion; E = passive ventricular filling; E/A ratio = ratio of the amplitude of the waves created by early diastolic filling and atrial contraction

Compared to conventional external cardioversion (CV) with respect to both primary success rate and long-term success, the novel method of internal CV of atrial fibrillation (AF) has been shown to be advantageous. Following external CV, the right atrium resumes its normal mechanical activity earlier than the left atrium, which has been attributed to the fact that the left atrium is more affected by the underlying disease than the right atrium. Since the high energy applied with external CV is assumed to impact on recovery of mechanical atrial function, and since internal and external CV have different energy requirements, it was the purpose of the present randomized study to evaluate both electrical CV methods with respect to the time course of resumption of left and right atrial mechanical activity following either internal or external CV of chronic AF. It was also of special interest to determine
whether the time course of normalization of atrial performance would be related to atrial dimensions or volumes prior to electrical CV and whether the maintenance of sinus rhythm would be predicted by echocardiographic parameters following CV.

**Materials and Methods**

**Patients**

Sixty-three consecutive patients with chronic AF (mean duration ± SD, 10.9 ± 27.6 months, range 0.5 to 180 months) were studied. Patients were excluded from the study if there was ineffective anticoagulation with warfarin (international normalized ratio of 2.0 to 3.0 for 3 weeks) or evidence of digitalis toxicity, abnormal electrolyte levels, or hyperthyroidism, in addition to patients with a history of long QT syndrome, an acute myocardial infarction within the past 6 weeks, a history of embolism, or either an atrioventricular valve disease greater than moderate in degree or a mechanical atrioventricular valve prosthesis. Clinical examinations were performed, and medical histories were obtained. Routine 12-lead ECG; routine laboratory testing comprising of blood cell count, liver enzymes, serum electrolytes, serum creatinine, urea, blood coagulation parameters, and analysis of thyroid parameters; and M-mode and Doppler echocardiography were performed in all patients. For exclusion of thromboembolic risks (ie, presence of left atrial thrombi), transesophageal echocardiography was performed in all patients before CV. Creatine kinase and potassium values were determined before and at 6 h after each CV attempt. Antiarrhythmic treatment, other than either sotalol or bisoprolol, was withheld for at least five half-lives before CV. Patients were then randomized to undergo either external or internal CV.

**Informed Consent and Documentation**

The benefits and risks of the study were explained to the patients. Written informed consent was obtained from all patients based on the protocol of the study as approved by the Ethics Committee of the Klinikum rechts der Isar.

**Interventions**

**External CV:** External CV was performed under the supervision of an anesthetist. Up to a maximum of three R wave-triggered shocks of increasing energy (200 J, 360 J, and again 360 J) were applied by an external defibrillator as described elsewhere. Up to a maximum of five R wave-triggered shocks of increasing energy (3, 6, 9, 12, and 15 J) were applied using biphasic shocks.

**Internal CV:** Internal CV was performed in the cardiac catheterization laboratory. A single-lead atrial CV catheter (EP Med Systems; Mount Arlington, NJ) was inserted via either the right femoral or brachial venous access as previously reported. Up to a maximum of three R wave-triggered shocks of increasing energy (3, 6, 9, 12, and 15 J) were applied using biphasic shocks.

**Follow-up Evaluation**

All patients received follow-up evaluations in the outpatient department. A 12-lead ECG was obtained, and M-mode and Doppler echocardiography were performed immediately before CV and at days 1, 7, and 28 after CV. Patients were randomly treated with either sotalol (n = 25; mean daily dose, 178 ± 37 mg; range, 120 to 240 mg) or bisoprolol (n = 28; mean daily dose, 6 ± 3 mg; range, 2.5 to 20 mg) after effective CV to sinus rhythm. Treatments with angiotensin-converting enzyme inhibitors, diuretics, and digoxis were continued based on the clinical status of the patients. Anticoagulation was continued for 4 weeks after successful CV.

**Echocardiographic Evaluation**

End-diastolic and end-systolic left and right ventricular diameters as well as left and right atrial dimensions were measured from a parasternal long-axis view and apical four-chamber view according to American Society of Echocardiography criteria using the respective longest and shortest longitudinal and cross-

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**Table 1—Baseline Characteristics of Patients With AF Who Received Successful Elective CV***

<table>
<thead>
<tr>
<th>Parameters</th>
<th>External CV</th>
<th>Internal CV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients, No.</td>
<td>31</td>
<td>28</td>
</tr>
<tr>
<td>Age, yr</td>
<td>62 ± 12</td>
<td>63 ± 12</td>
</tr>
<tr>
<td>Height, cm</td>
<td>177 ± 9</td>
<td>174 ± 9</td>
</tr>
<tr>
<td>Body weight, kg</td>
<td>84 ± 13</td>
<td>84 ± 14</td>
</tr>
<tr>
<td>Male/female ratio</td>
<td>26:5</td>
<td>22:6</td>
</tr>
<tr>
<td>Minimum left atrial diameters (determined at</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ventricular diastole), cm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Longitudinal</td>
<td>53.6 ± 5.2</td>
<td>56.3 ± 6.0</td>
</tr>
<tr>
<td>Cross-sectional</td>
<td>39.4 ± 6.2</td>
<td>41.0 ± 5.9</td>
</tr>
<tr>
<td>Minimum right atrial diameters (determined at</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ventricular diastole), cm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Longitudinal</td>
<td>51.3 ± 6.6</td>
<td>53.2 ± 5.0</td>
</tr>
<tr>
<td>Cross-sectional</td>
<td>40.9 ± 7.8</td>
<td>38.4 ± 4.2</td>
</tr>
<tr>
<td>Underlying heart disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>6 (19)</td>
<td>8 (29)</td>
</tr>
<tr>
<td>Arterial hypertension</td>
<td>14 (45)</td>
<td>12 (43)</td>
</tr>
<tr>
<td>Valvular heart disease</td>
<td>2 (6)</td>
<td>1 (4)</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>3 (10)</td>
<td>4 (14)</td>
</tr>
<tr>
<td>Lone AF</td>
<td>6 (19)</td>
<td>3 (11)</td>
</tr>
<tr>
<td>Duration of current episode of AF, mo</td>
<td>10.3 ± 31</td>
<td>11.2 ± 25</td>
</tr>
<tr>
<td>Range, mo</td>
<td>(0.5–180)</td>
<td>(0.5–120)</td>
</tr>
<tr>
<td>Prior unsuccessful attempts of electrical CV of AF, No.</td>
<td>2 (6)</td>
<td>5 (18)</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD or No. (%) unless otherwise indicated.
sectional diameters (Sonos 5500; Hewlett Packard; Palo Alto, CA). Mitral and tricuspid flow velocities were measured with pulsed Doppler echocardiography to obtain peak early (passive ventricular filling [E]) and late (active atrial contraction [A]) flow velocities. Flow velocity across the left ventricular outflow tract was assessed by pulsed Doppler echocardiography. Volumetric quantification was performed using automated calculation of volumes based on the rotational ellipsoid.

**Statistical Analyses**

Statistical analyses were performed according to mode of treatment. Continuous variables are expressed as mean ± SD. Categorical variables were analyzed using the χ² test. Student’s t test for paired or unpaired samples was applied as appropriate. Comparisons of more than two groups of data were performed with a repeated-measures analysis of variance, subjected to multiple comparison according to the Friedman test. Statistical univariate and multivariate analyses were performed with the Statistical Package for Social Sciences (SPSS; Chicago, IL) for clinical and echocardiographic parameters, outcome, and follow-up. Statistical significance was assumed at p < 0.05.

**RESULTS**

In 59 of 63 patients, sinus rhythm could be restored successfully, the clinical and echocardiographic data are given in Table 1. Upon examination by transesophageal echocardiography or echo contrast, none of the patients with chronic AF, all of whom needed to exhibit a mandatory effective anticoagulation with international normalized ratio values of >2 for at least 3 weeks before undergoing CV, revealed left atrial thrombus dense enough to withhold or postpone CV. Of five patients with an AF duration of <4 weeks, four patients had a history of recurrent AF for which they were receiving warfarin at CV as well. The fifth patient received heparin IV to bridge the time necessary for achievement of an international normalized ratio of >2 after initiation of oral anticoagulation.

As expected, energies required for external CV were considerably higher than for internal CV (256 ± 83 J vs 8.2 ± 4.1 J; p < 0.001). Earlier findings of a positive correlation between duration of AF before CV and internal CV defibrillation energy requirements were confirmed (p < 0.01). No such association could be demonstrated for patients undergoing external CV. Creatine kinase values after CV were 118.4 ± 131.4 U/L (external) vs 37.5 ± 27.3 U/L (internal) [p < 0.01].

When comparing both internal and external electrical CV modalities, we found that there were no differences regarding maintenance of sinus rhythm at 28 days or with atrial and ventricular volumes, diameters, or ejection fractions at all points in time. External (Fig 1, top) and internal CV (Fig 1, bottom) did not differ regarding peak E, peak A, or the ratio of the amplitude of the waves created by early diastolic filling and atrial contraction (E/A ratio) at any point in time. Thus, in contrast to the assumption that low-energy internal CV would have less impact on atrial mechanical recovery, the method of CV had no effect. Accordingly, the data of patients who received external and internal CV were pooled for the remaining analyses.

**Resumption of Mechanical Atrial Function After Electrical CV**

In the left atrium, E remained relatively constant throughout the entire lapse of the 28 days. There was a gradual increase in A and a parallel decline in the E/A ratio over time, both of which were statistically significant after day 7. Until this point in time, the E/A ratio did not return to its normal range, which assumes E/A ratio values in normal subjects between 1 and 2 for both atrioventricular valves.
Because no basal E/A ratio was available for any patient, the values measured on day 28 after CV were taken as baseline characteristic of the individual patients. Because there are many variables with the potential to influence the E/A ratio, a subgroup analysis of patients in sinus rhythm was performed on day 28. Accordingly, patients were dichotomized with respect to their E/A ratio (< 1 or ≥ 1) on day 28. Subgroups did not differ with respect to clinical parameters or underlying heart diseases. There was a constancy of E over time in both subgroups, a constant rise of A, and a subsequent decline in E/A, which was more rapid in the subgroup of patients with a final E/A ratio < 1, reflecting a more rapid gain in atrial contractility (Fig 2).

In the right atrium, there was also a constancy of E values during the entire observation episode. There was a smooth increase in A values and a parallel decline in the E/A ratio. However, as opposed to the left atrium, right atrial E/A assumed values equivalent to those at day 28 almost immediately after CV, which was equivalent to a more rapid return of mechanical function after electrical CV.

**Time Course of Resumption of Atrial Performance in Relation to Preprocedural Dimensions**

On the basis of the assumption that A and the E/A ratio mirror mechanical atrial function, regression analyses were performed to evaluate the relation between atrial dimensions or volumes before CV and time course of normalization of atrial performance, reflected by the differences of A and E/A between days 0 and 28. Neither the diameters of the atria and ventricles nor the volumes of the left atrium and ventricle reliably predicted the resumption of mechanical atrial performance over the 28 days after electrical CV in a statistically significant manner. Of note, left atrial volumes were always larger and left atrial ejection fraction was always smaller than their right atrial counterparts at all points in time and irrespective of rhythmic outcome on day 28.

**Discussion**

The first major finding of the present study is that the resumption of mechanical atrial function as assessed by Doppler echocardiographic flow across the mitral and tricuspid valves is independent of the mode of CV. It is known that AF is associated with electrical and mechanical remodeling of the atria.15 These alterations take place as early as within a few days after onset of constant AF, leading to atrial dilatation, myocyte hypertrophy, atrial apoptosis, fibrosis, and consecutive decline in atrial natriuretic peptide production. There is also myofiber disarray and redistribution of connexins and, finally, dispersion of the atrial refractory period.5,15,18–23 All of these findings develop in proportion to the duration of the arrhythmia and are more likely to be reversible when AF is shorter. These findings may also explain results of studies18,23,24 that show long-standing AF requires higher energies at both internal and external CV. CV energy is determined by the duration of AF,19,25 and the long-term outcome for maintenance of sinus rhythm is not dependent on the method chosen for CV; organic and structural alterations seem to determine restoration and maintenance of sinus rhythm after electrical CVs.

The higher total energy application with external CV is reflected by higher creatine kinase values (118 U/L vs 37 U/L), especially in patients who required three or more shocks. According to one study,26 elevations of creatine kinase are found in > 50% of patients undergoing external CV, but without concomitant elevation of the creatine kinase-MB fraction or cardiac troponin I. Indeed, there was only one patient in whom the MB fraction was found elevated (reaching 13 U/L). Therefore, the creatine kinase increase with external CV primarily results from muscular contractions that are not found with low-energy internal CV.

The second important finding of the present randomized study is that the resumption of mechanical atrial activity occurs earlier in the right atrium, when compared with the left atrium. There is a constancy in E velocity mirroring the passive ventricular filling across both atrioventricular valves over time; however, normalization of A velocity, reflecting the active mechanical atrial contraction,27 and return of E/A ratio occur almost immediately in the right

**Figure 2.** E/A ratios over time for patients with a final (i.e., at 28 days after CV) E/A ratio < 1 (open circles) and patients with a final E/A ratio > 1 (solid circles). ***p < 0.001 between patient subgroups.
atrium. In contrast, no change in these parameters was observed in the left atrium within 7 days after CV. This phenomenon had been observed earlier after exclusively external CV. In these nonrandomized studies, patients were included for electrical CV after failure of pharmacologic attempts. Because both underlying heart disease and AF duration have the potential to lead to structural alterations of both atria, which, in turn, predominate the left atrium, the atria of patients requiring electrical CV were the more seriously affected, rendering non-electrical measurements as well as low-energy electrical CV less effective. These findings are also in keeping with a report on the rapidity of atrial recovery in association with the duration of AF before CV. Accordingly, it is not the procedure, but rather the extent of the organic affection of the atria, the reversibility, and perhaps, the intrinsic differences in electrophysiology between both atria that determine the time-course of mechanical atrial recovery after CV. It is not only atherosclerosis, pressure volume overload, or the duration of AF that impact on atrial electrophysiology, but also inborn structural differences between both atria that render the left atrium more vulnerable to arrhythmias than the right atrium. The coexistence of sinus rhythm in the right atrium and AF emanating from the left have been reported. Consequently, the delayed recovery of mechanical and electrical left atrial function after either CV mode forms another basis to continue oral anticoagulation for up to 4 weeks, despite the presence of sinus rhythm.

There is, however, one study inferring an identical and rapid time course of mechanical recovery of both atria, normalizing even within 24 h after chemical CV. Methodologic reasons may explain its conclusions. First, only 31 of 50 patients converted within 24 h of receiving amiodarone and formed the patient group. Because chemical CV is known to be less effective than electrical CV, we can assume that the 31 patients had relatively little atrial organic alteration. This can be deduced from their AF duration (approximately 4 weeks, on average) and their preserved left ventricular function, which is known to deteriorate substantially in proportion to AF duration. Second, mean values of atrial ejection forces were different from that in control subjects, almost twice as high in the left atrium at 24 h and remaining higher on day 7 after CV; however, because of the small patient number, they failed to reach statistical significance. Finally, one half of the patients were receiving various antiarrhythmic treatments with the potential of affecting electrophysiology and contractility, and medical treatment was unknown for 12 patients. Thus, mechanical recovery of both atria also must be regarded as different when treated with chemical CV of AF of a relatively short duration.

In summary, after both internal and external CV of AF with modes and energies as described, there was no evidence of damage to the atrial myocardium. The present study adds to the growing evidence that the atrial stunning process is the result of the reversion to sinus rhythm itself, regardless of the method used. Normalization of left atrial performance takes significantly longer than normalization of the right atrium. Electrical CV mode exerts no influence on the time course of restoration of atrial performance. However, because of the delay in normalization of mechanical function of the left atrium, there is a rationale for continuation of anticoagulation in patients after CV even with stable sinus rhythm.

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