Left ventricular diastolic function after electrical cardioversion of atrial fibrillation

H J Muntinga, F van den Berg, M G Niemeyer, P K Blanksma, E E van der Wall, H J G M Crijns

In chronic atrial fibrillation (AF) left ventricular function may gradually diminish as a result of an intrinsic tachycardiomyopathy. This tachycardia induced cardiomyopathy is characterised by decreased systolic function, ventricular dilation, and raised ventricular filling pressures. In such patients left ventricular systolic function may gradually improve after electrical cardioversion. Little is known about left ventricular diastolic function in AF. In animal models with chronic rapid pacing induced cardiomyopathy, systolic dysfunction is accompanied by an increased diastolic wall stress and impaired myocardial relaxation. In addition, recovery from supraventricular tachycardia induced cardiomyopathy in animal models is associated with hypertrophy, reduced myocardial blood flow, and diastolic dysfunction. Whether the same changes in diastolic left ventricular function hold true for humans with AF remains to be investigated. The present study was performed to investigate diastolic filling in patients with AF after successful cardioversion compared to a control group.

METHODS
Seventeen patients who had successfully undergone direct current electrical cardioversion for persistent lone AF or atrial flutter were studied. Lone AF was diagnosed when the absence of hypertension, significant valvar disease, coronary artery disease, hyperthyroidism or other cardiovascular disease could be established. Informed consent was given by all patients. Oral anticoagulation was instituted. Rate controlling drug treatment and antiarrhythmic drug therapy was instituted singly or in combination. Direct current electrical cardioversion was performed. Six hours after successful cardioversion left ventricular radionuclide angiography was performed. Results were compared with a control group of 17 patients with normal findings in coronary arteriography and left ventriculography.

Left ventricular function was evaluated by equilibrium blood pool scintigraphy using a gamma camera (Siemens Orbiter) with an all purpose parallel hole collimator interfaced with a computer. The radionuclide imaging procedure was performed in the supine position on a gamma camera equipped with a high resolution parallel hole collimator interfaced with a computer. 

Table 1 Characteristics of patients and radionuclide angiography derived left ventricular filling parameters of 17 patients with lone atrial fibrillation (AF) direct post cardioversion compared to 17 normal controls (mean (SD))

<table>
<thead>
<tr>
<th></th>
<th>Control (n=17)</th>
<th>Lone AF (n=17)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>57 (11)</td>
<td>65 (13)*</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>7/10</td>
<td>11/6</td>
</tr>
<tr>
<td>Atrial fibrillation duration [median, range, days]</td>
<td>–</td>
<td>135 (60–1215)</td>
</tr>
<tr>
<td>Echocardiography (mm)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDD</td>
<td>48 (5)</td>
<td>45 (5)</td>
</tr>
<tr>
<td>LVESD</td>
<td>29 (4)</td>
<td>27 (2)</td>
</tr>
<tr>
<td>FS (% of LVEDD)</td>
<td>39 (6)</td>
<td>39 (5)</td>
</tr>
<tr>
<td>LA (LA)</td>
<td>35 (6)</td>
<td>42 (5)*</td>
</tr>
<tr>
<td>LA (AP)</td>
<td>51 (2)</td>
<td>61 (7)**</td>
</tr>
<tr>
<td>Heart rate (per min)†</td>
<td>66 (8)</td>
<td>67 (11)</td>
</tr>
<tr>
<td>Electrocardiography</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PR interval [ms]†</td>
<td>165 (19)</td>
<td>215 (22)**</td>
</tr>
<tr>
<td>QRS width [ms]†</td>
<td>91 (9)</td>
<td>90 (21)</td>
</tr>
<tr>
<td>Systolic blood pressure [mm Hg]†</td>
<td>137 (14)</td>
<td>120 (13)</td>
</tr>
<tr>
<td>Diastolic blood pressure [mm Hg]†</td>
<td>84 (7)</td>
<td>66 (7)**</td>
</tr>
<tr>
<td>Radionuclide angiography</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEF [% of EDV]†</td>
<td>61 (5)</td>
<td>58 (5)</td>
</tr>
<tr>
<td>PFR [FV/s]</td>
<td>3.28 (0.64)</td>
<td>3.81 (0.67)*</td>
</tr>
<tr>
<td>TPFR [ms]</td>
<td>199 (42)</td>
<td>125 (44)**</td>
</tr>
<tr>
<td>EDF [%FV]</td>
<td>67 (7)</td>
<td>69 (6)</td>
</tr>
<tr>
<td>DF [%FV]</td>
<td>14 (7)</td>
<td>18 (5)</td>
</tr>
<tr>
<td>AFF [%FV]</td>
<td>31 (5)</td>
<td>24 (6)**</td>
</tr>
</tbody>
</table>

*p<0.05 v control; **p<0.01 v control; ***p<0.001 v control.
†For AF patients: after cardioversion.

Abbreviations: AF, atrial fibrillation; AFF, additional filling fraction; DF, diastasis filling fraction; EDF, early diastolic filling fraction; EDV, end diastolic volume; FS, fractional shortening; FV, filling volume; LA (AP), left atrium apical view; LA (LA), left atrium long axis; LVEDD, left ventricular end diastolic diameter; LVESD, left ventricular end systolic diameter; LVEF, left ventricular ejection fraction; PFR, peak filling rate; TPFR, time to peak filling rate.
with a Pinnacle computer (Medasys Inc, Ann Arbor, Michigan, USA). Only a 5% cycle length window with forward gating was accepted. Acquisition was completed after 150 000 counts per frame of 20 ms. Temporal smoothing was performed by 5 Fourier harmonics. From the left ventricular time-activity curve we directly measured left ventricular ejection fraction (EF). For interindividual comparison of volume fractions, measured filling counts were converted to percentages of the filling volume (FV, measured filling counts = 100% FV). All patients showed separation of the diastolic portion of the curve into early filling phase and additional filling phase. In seven patients a long pronounced diastasis period was observed. This allowed volume measurement of early diastolic filling fraction (EDF), diastasis filling fraction (DF), and additional filling fraction (AF) relative to the left ventricular filling volume. The first derivative of the time versus relative filling volume curve expresses instantaneous filling rates measured in filling volume/second. With this normalisation the measurement does not vary with the residual volume and the EF. This curve was used to calculate peak filling rate (PFR) and time to peak filling rate (TPFR). PFR is the rapid instantaneous filling rate during early diastolic filling. Timing was performed in relation to the beginning of filling. The data are expressed as mean (SD). For comparison of the data between the control group and the lone AF group statistical analysis was performed with Student's t test for independent samples assuming equal variances. All p values were two tailed. A value of p < 0.05 was considered significant.

RESULTS
Patient characteristics of AF patients are compared to control subjects in table 1. All patients had clear atrial depolarisation waves on the ECG after cardioversion with a relatively long PR relationship. Mean resting heart rate decreased from 84 (18)/min before cardioversion to 67 (11)/min after cardioversion (p < 0.01). Mean diastolic blood pressure decreased from 82 (12) mm Hg before cardioversion to 66 (7) mm Hg after cardioversion (p < 0.05). None of the patients had clinical signs of cerebral or peripheral embolism. After cardioversion mean peak filling rate was higher and mean time to peak filling rate was shorter in AF patients. Additional left ventricular filling fraction was less in AF patients compared to normal controls. In all AF patients additional filling was detectable early after cardioversion.

DISCUSSION
The present study shows that left ventricular filling is disturbed in patients with normal left ventricular systolic function early after electrical cardioversion for chronic lone AF. If anything, increased PFR and decreased TPFR in the present study suggest the presence of atrial remodelling—that is, decreased atrial compliance early after cardioversion for chronic AF. The atrioventricular pressure difference is not only influenced by left ventricular relaxation, but also by left atrial pressure. Increased left atrial pressure may produce increased atrioventricular pressure gradient and concomitant increased early diastolic filling. This may either be produced by increased atrial loading and volume or decreased atrial compliance. In chronic AF atrial enlargement is commonly described. Also longstanding AF may produce interstitial fibrosis and degeneration of atrial myocytes. In AF increased atrial pressure can be measured. We therefore consider decreased atrial compliance (producing an increased v wave) a probable explanation for the increased early diastolic filling rate measured in the present study.

It is unlikely that in the present study increased ventricular relaxation velocity explains the observed filling pattern. Studies of animal models with rapid pacing induced heart failure showed diastolic dysfunction consisting of decreased relaxation, and increased myocardial stiffness and end diastolic wall stress early after pacing. If anything, the difference in age between the control subjects and the patients with lone AF in the present study would produce higher PFR and lower TPFR instead of the reverse in the control subjects. The higher blood pressure in control subjects compared to patients with lone AF could have produced a higher afterload to the left ventricle with a lower relaxation velocity, explaining a lower PFR in the controls. Although previous investigators reported a significant decrease of left ventricular relaxation because of increased afterload, early left ventricular filling responds variably to blood pressure elevations in subjects with normal left ventricular systolic function without mitral regurgitation.

The finding of decreased additional filling fraction compared to control subjects in the present study is probably explained by decreased atrial contraction. Factors with potential influence on atrial mechanical function include duration of AF and the properties of the left ventricle—that is, left ventricular relaxation and compliance.

These data show abnormal filling of the left ventricle early after electrical cardioversion in patients with lone AF, and are consistent with decreased atrial compliance and contractility caused by atrial remodelling.

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