Hemodynamics and Echocardiograms Before and After Cardioversion of Atrial Fibrillation to Normal Sinus Rhythm*

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Hemodynamic and echocardiographic measurements were obtained in 15 patients prior to cardioversion and immediately, one, three, and five hours after cardioversion of atrial fibrillation to normal sinus rhythm. At five hours after cardioversion, there was improvement in cardiac index (P < 0.005), in stroke index (P < 0.005), and in stroke work index (P < 0.025), but no significant change in heart rate, mean pulmonary capillary wedge pressure, left atrial dimensions (LAD), left ventricular end-diastolic dimensions (LVEDD), or left ventricular end-systolic dimension (LVESD). At follow-up one week to nine months after cardioversion, 10 of the 15 patients (67 percent) remained in normal sinus rhythm. Neither the size of the left atrium, changes in hemodynamics, LAD, LVEDD, or LVESD, nor the presence or amplitude of the A-wave on the mitral valve echogram correlated with the persistence of normal sinus rhythm.

Direct-current cardioversion of atrial fibrillation is frequently performed to improve cardiac function by returning the atrial contribution to ventricular filling. Electrocardiographic evidence of restoration of normal sinus rhythm is often assumed to be accompanied by effective atrial contraction. However, numerous hemodynamic studies performed before and after cardioversion of atrial fibrillation to normal sinus rhythm reported conflicting results regarding improvement in hemodynamic function.1-8 DeMaria and associates8 assessed by echocardiography the effect of direct-current cardioversion of atrial arrhythmias (atrial fibrillation in 31 of 35 patients) to normal sinus rhythm. In their study, the majority of patients showed effective atrial contraction and decreased left atrial size within one hour of direct-current cardioversion. Measurements of left ventricular function performed in 12 patients also showed improvement.

We performed a prospective study in 20 patients with atrial fibrillation not due to significant primary valve disease, and undergoing direct current cardioversion for clinical reasons. Our study was undertaken to evaluate the effect of direct-current cardioversion on hemodynamic measurements, and to determine whether any hemodynamic changes would be accompanied by corresponding changes in left atrial dimension (LAD), left ventricular end-systolic dimension (LVESD), and left ventricular end-diastolic dimension (LVEDD) measured by M-mode echocardiography. We also correlated changes in left atrial and left ventricular dimensions and in hemodynamic measurements, and the presence and amplitude of the A-wave on the mitral valve echogram with maintenance of normal sinus rhythm at follow-up. The data from this study are reported here.

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Methods

Fifteen men between the ages of 44 and 74, mean age 57.4 ± 6.6 years, underwent successful cardioversion from atrial fibrillation to normal sinus rhythm. The 15 patients had atrial fibrillation for three weeks to ten months. Six patients had idiopathic atrial fibrillation, three had coronary heart disease, two had hypertensive heart disease, two had idiopathic cardiomyopathy, one had treated hyperthyroidism, and one patient had prolapse of the mitral valve by echocardiography. One patient had mild mitral insufficiency due to a prolapse of the mitral valve, and another had mild mitral insufficiency secondary to left ventricular dysfunction. Nine of these 15 patients (60 percent) had an enlarged LAD by echocardiography. A measurement of LAD at systole of greater than 4.0 cm was considered an enlarged LAD.10 These 15 patients underwent successful direct-current car-
dioversion from atrial fibrillation to normal sinus rhythm for clinical reasons in a prospective study. Only patients in whom technically satisfactory echocardiograms could be recorded rapidly were included.

Five additional patients between the ages of 58 and 74, mean age 63 ± 7.3 years, remained in atrial fibrillation after an unsuccessful attempt at cardioversion (four patients) or reverted to atrial fibrillation within two hours after cardioversion (one patient). These patients had atrial fibrillation for one to two years, and their clinical, hemodynamic, and echocardiographic characteristics were similar to those of the 15 patients who underwent successful direct-current cardioversion from atrial fibrillation to normal sinus rhythm. All 20 patients signed informed consent forms and the study was approved by the institutional committees on human research.

All 20 patients received intravenous diazepam prior to cardioversion. Digoxin was stopped 48 hours prior to cardioversion. In the 15 patients who were cardioverted successfully to normal sinus rhythm, digoxin was restarted on the day following cardioversion. Maintenance doses of quinidine were instituted in six of these 15 patients within two hours prior to cardioversion, and in the other nine patients after cardioversion. Four of the five patients who had atrial fibrillation at two hours after cardioversion received quinidine sulfate, 300 mg, within two hours prior to cardioversion.

On the morning of cardioversion, right-heart catheterization was performed using a number 7 triple-lumen flow-directed Swan-Ganz catheter inserted via an antecubital vein and positioned within its tip in the pulmonary artery and the proximal lumen in the right atrium. Blood pressure was obtained either with an indwelling arterial line inserted in the brachial artery or with a mercury sphygmomanometer. Catheters were connected to pressure transducers, and pressures were recorded with a simultrace recorder with direct writer (Electronics for Medicine VR6). Heart rate was measured from the electrocardiogram. Cardiac output was measured by thermodilution with a Lyons desimeter-car-
diac output computer model DCCO.12

The zero reference level for pressure recording was 5 cm below the sternal angle. Mean pressures were obtained by electronic integration. The criteria for a satisfactory pulmonary artery wedge pressure were (1) a change from the typical pulmonary artery pressure waveform to the typical pulmonary artery wedge pressure waveform upon inflation of the Swan-Ganz balloon catheter, and (2) a mean pressure step-up upon deflation of the Swan-Ganz balloon catheter. The pulmonary artery wedge pressure was characterized by distinct a- and v-waves, with the v-wave occurring after the t-wave of the electrocardiogram. The following hemodynamic parameters, were measured during two control periods 15 minutes and immediately prior to administration of diazepam prior to cardioversion and immediately, one hour, three hours, and five hours after cardioversion: heart rate, blood pressure, mean right atrial pressure, mean pulmonary artery pressure, mean pulmonary capillary wedge pressure, and cardiac output. Stroke index and stroke work index were calculated by the following equations:

\[
SI = \frac{CI}{HR} \\
SWI = SI \times (BP - PCWP) \times 0.0136
\]

where SI = stroke index, HR = heart rate, SWI = stroke work index, BP = mean blood pressure, and PCWP = mean pulmonary capillary wedge pressure.

<table>
<thead>
<tr>
<th>Heart Rate (beats/min)</th>
<th>PCWP (mm Hg)</th>
<th>Stroke Index (ml/beat/m²)</th>
<th>Cardiac Index (liters/min/m²)</th>
<th>Stroke Work Index (gm-m/m²)</th>
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<tr>
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<td>Control 5 hours</td>
<td>Control 5 hours</td>
<td>Control 5 hours</td>
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<td>36.7 47.9</td>
<td>3.3 4.2</td>
<td>40.6 48.2</td>
</tr>
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</table>

PCWP = mean pulmonary capillary wedge pressure

*Remained in normal sinus rhythm at follow-up

Table 1—Hemodynamic Parameters in 15 Patients before and at 5 Hours after Cardioversion to Normal Sinus Rhythm

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Echocardiograms were performed in all 15 patients during the control periods and immediately, one hour, three hours, and five hours after cardioversion immediately following hemodynamic measurements. Echocardiography was performed with an ultrasonoscope (Ekoline-92) utilizing a 0.5 inch diameter 2.25 MHz transducer focused at 10 cm with a repetition rate of 1,000 impulses/sec and a multichannel recorder (Irex ContinuTrace 101). All echocardiograms were recorded with the subjects in a slight left lateral decubitus position with 15° elevation of the head. Echocardiograms were recorded simultaneously with the echocardiograms. The heart rates were measured from the electrocardiograms.

We selected a transducer position from which distinct and readily obtainable left ventricular dimensions could be obtained consistently. The transducer position giving the mitral valve echo was determined. Using the echoes obtained from the mitral valve apparatus as landmarks, reproducible left ventricular dimensions were obtained.13-14 Recordings were obtained at the constant respiratory phase of held mid-expiration. The LVESD was measured at the point of least separation of the septal and posterior wall endocardial echoes. The LVEDD was measured at the time of the peak of the R-wave in the electrocardiogram.

After recognition of the characteristic mitral valve echoes, the transducer was angled medially, posteriorly, and superiorly to obtain a recording of the aortic root and the left atrium. Measurements of LAD were made at ventricular end-systole and at ventricular end-diastole. Examination of identical areas of the left atrium was confirmed by vertical scanning and by identification of aortic leaflets as landmarks.

Measurements of A-wave amplitude on the mitral valve echo gram were also made in patients developing A-waves after cardioversion.9 Patients were followed-up for one week to nine months after cardioversion. Changes in hemodynamics and in echocardiographic parameters were correlated with maintenance of normal sinus rhythm.

Student's t test for paired data was used to analyze the data.

Results

Table 1 indicates the heart rate, PCWP, stroke index, cardiac index, and stroke work index for each of the 15 patients in the control period (average of two control values) and at five hours after cardioversion to normal sinus rhythm.

Table 2 shows the mean values ± one standard deviation for the various hemodynamic parameters in the control period (average of two control values) and immediately, one hour, three hours, and five hours after direct current cardioversion of atrial fibrillation to normal sinus rhythm in the 15 patients aged 44 to 74 years. Table 2 also shows statistical levels of significance.

Table 3 indicates the LAD at systole and at diastole and the LVESD and LVEDD for each of the 15 patients in the control period (average of two control values) and at five hours after cardioversion to normal sinus rhythm.

Table 4 shows the mean values ± one standard deviation for LAD at systole and at diastole and for LVESD and LVEDD in the control period (average
atrial fibrillation to normal sinus rhythm in the 15 patients ages 44 to 74 years. No significant difference in LAD at systole or at diastole or in LVEDD or in LVESD occurred after cardioversion to normal sinus rhythm.

Eleven of the 15 patients (73 percent) had an A-wave on the mitral valve echogram at five hours after cardioversion. At follow-up one week to nine months after cardioversion, 10 of the 15 patients (67 percent) remained in normal sinus rhythm. The follow-up period was similar for the group with an A-wave and for the group without an A-wave on the mitral valve echogram at five hours after cardioversion. Nine of 11 patients (82 percent) with an A-wave on the mitral valve echogram at five hours after cardioversion remained in normal sinus rhythm. Neither changes in hemodynamic parameters, in LAD at systole or diastole, or in LVEDD or LVESD, nor the presence of an A-wave, nor the height of the A-wave correlated with the maintenance of normal sinus rhythm. Seven of nine patients (78 percent) with an enlarged LAD in the control period and three of six patients (50 percent) with a normal LAD in the control period remained in normal sinus rhythm. Six of eight patients (75 percent) with an enlarged LAD at five hours after cardioversion and four of seven patients (57 percent) with a normal LAD at five hours after cardioversion remained in normal sinus rhythm.

Figure 1 illustrates a representative echogram of an A-wave on the mitral valve echogram taken five hours after cardioversion. Figure 2 shows a representative echogram of absence of an A-wave on the mitral valve echogram taken five hours after cardioversion.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control</th>
<th>Immediate</th>
<th>1 hour</th>
<th>3 hours</th>
<th>5 hours</th>
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<td>4.3</td>
<td>4.3</td>
<td>4.3</td>
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<tr>
<td>(cm)</td>
<td>±0.6</td>
<td>±0.7</td>
<td>±0.7</td>
<td>±0.8</td>
<td>±0.7</td>
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<tr>
<td>Left atrial dimension at diastole</td>
<td>4.0</td>
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<td>3.8</td>
<td>3.7</td>
<td>3.9</td>
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<tr>
<td>(cm)</td>
<td>±0.6</td>
<td>±0.7</td>
<td>±0.6</td>
<td>±0.8</td>
<td>±0.6</td>
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<tr>
<td>Left ventricular end-systolic</td>
<td>5.0</td>
<td>5.2</td>
<td>5.2</td>
<td>4.9</td>
<td>5.0</td>
</tr>
<tr>
<td>dimension (cm)</td>
<td>±0.7</td>
<td>±0.7</td>
<td>±0.7</td>
<td>±0.7</td>
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<tr>
<td>Left ventricular end-diastolic</td>
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<td>5.9</td>
<td>5.9</td>
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<td>6.0</td>
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<tr>
<td>dimension (cm)</td>
<td>±0.7</td>
<td>±0.7</td>
<td>±0.8</td>
<td>±0.8</td>
<td>±0.7</td>
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</table>

Table 4—Mean Values ± 1 Standard Deviation of Echocardiographic Measurements before and after Cardioversion of Atrial Fibrillation.
DISCUSSION

Conflicting results regarding improvement in hemodynamic function following cardioversion of atrial fibrillation to normal sinus rhythm have been reported by various investigators.1-4 Graettinger and associates1 reported no significant improvement in cardiac output following cardioversion of atrial fibrillation to normal sinus rhythm. Morris and associates,2 Reale,3 Rowlands and co-workers,4 Kaplan and associates,5 and Khaja and Parker6 demonstrated an improvement in cardiac output following cardioversion of atrial fibrillation to normal sinus rhythm. Killip and Baer4 showed there was improvement in cardiac output following cardioversion of atrial fibrillation to normal sinus rhythm in patients with valvular heart disease, but no significant improvement in cardiac output in patients with benign atrial fibrillation. Shapiro and Klein7 found no significant change in resting cardiac output but an improvement in cardiac output during exercise in patients following cardioversion of atrial fibrillation to normal sinus rhythm.

Our data obtained in patients without primary valve disease and in patients who have had atrial fibrillation for less than one year showed significant improvement in cardiac index (P < 0.005), in stroke index (P < 0.005), and in stroke work index (P < 0.025) after cardioversion. Although the slight but insignificant decrease in resting heart rate at five hours after cardioversion of atrial fibrillation to normal sinus rhythm might have contributed to an increased stroke index, an improved atrial contraction contributing to ventricular filling was probably a more important factor in the increase in stroke index.

With improvement in hemodynamic function following cardioversion of atrial fibrillation to normal sinus rhythm, one might have expected improvement in ventricular dimensions and possibly improvement in LAD. However, our data showed an insignificant rise in LVEDD, no significant change in LVESD, and an insignificant decrease in LAD.

These results differ from those reported by DeMaria and associates,8 who demonstrated after cardioversion a reduction in LAD, a rise in LVEDD, no significant change in LVESD, and a rise in stroke volume. It should be pointed out, however, that the LVEDD, LVESD, and LAD values were higher in our patients than in the patients reported by DeMaria and associates. The difference in baseline characteristics between these two groups may possibly explain the different results. The decrease in heart rate after cardioversion contributed to the results observed by DeMaria and associates. Furthermore, an insignificant rise in LVEDD associated with no change in LVESD and an insignificant de-
crease in LAD—changes in the right direction—were observed in our study. The sensitivity of M mode echocardiography is also such that our small, though statistically significant hemodynamic changes, escaped detection.

Our data showed that neither changes in hemodynamics, LAD, LVESD, nor LVEDD correlated with the maintenance of normal sinus rhythm. Six of our eight patients (75 percent) with an enlarged LAD at five hours after cardioversion and four of seven patients (57 percent) with a normal LAD at five hours after cardioversion remained in normal sinus rhythm.

We recorded A-waves on the mitral valve echoogram in 11 of 15 patients (73 percent). In agreement with DeMaria and associates, we found that the A-wave amplitude on the mitral valve echoogram could not be correlated with changes in left atrial and left ventricular chamber size. In contrast to the findings of DeMaria and associates, our findings showed no correlation between the presence or amplitude of the A-wave and maintenance of normal sinus rhythm in the small number of patients in this study. However, we observed reversion to atrial fibrillation in 50 percent of patients (two of four) with no A-wave on the mitral valve echogram at five hours after cardioversion and in 18 percent of patients (two of 11) with an A-wave on the mitral valve echogram at five hours after cardioversion.

In conclusion, our data are in agreement with investigators reporting significant improvement in hemodynamic function following cardioversion of atrial fibrillation to normal sinus rhythm. We showed by echocardiography an insignificant rise in LVEDD, no change in LVESD, and an insignificant decrease in LAD after cardioversion. Neither the presence nor amplitude of the A wave in the mitral valve echogram, the size of the left atrium, nor changes in hemodynamics, LAD, LVESD, or LVEDD correlated with the persistence of normal sinus rhythm.

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