Transesophageal Echocardiographic Prediction of Initially Successful Electrical Cardioversion of Isolated Atrial Fibrillation

Effects of Left Atrial Appendage Function

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SUMMARY

Left atrial appendage (LAA) flow velocities prior to electrical cardioversion were recorded using transesophageal pulsed Doppler echocardiography to predict initially successful cardioversion of isolated atrial fibrillation (AF). Patients with AF were placed into either a success group (19 patients) in which sinus rhythm was maintained for at least 2 days or a failure group (12 patients). The duration of AF was shorter in the success group. The maximum left atrial diameter was the same for the two groups. The maximum LAA area was smaller in the success group. The maximum forward and backward LAA velocities were greater in the success group, as were the mean forward and backward LAA velocities. In the patients with mean LAA flow velocities greater than 19 cm/sec, the success of cardioversion could be predicted with high sensitivity (80%) and specificity (88%). We conclude that the duration of AF, the maximum LAA area, and LAA flow velocities prior to cardioversion predict the initial recovery of sinus rhythm for isolated AF. (Jpn Heart J 1997; 38: 487-495)

Key words: Electrical cardioversion, Atrial fibrillation, Left atrial appendage function, Transesophageal pulsed Doppler echocardiography

In patients with atrial fibrillation (AF), the lack of active left atrial contraction causes a decrease in left ventricular filling during diastole. This lack of active contraction also increases the risk of thrombus formation through blood stagnation in the left atrium. Therefore, cardioversion of AF is effective in the prevention of cardiac dysfunction and systemic embolization. However, because
AF often recurs after cardioversion, it is clinically very important to predict the long-term success before performing cardioversion.

Recently, detailed studies of left atrial appendage (LAA) function and LAA flow velocity have been facilitated by the use of transesophageal echocardiography. As a result, we have been able to assess the mechanism of thrombus formation at the time of cardioversion and determine the indications for anticoagulant therapy, as well as determine LAA function and flow velocity patterns in normal volunteers and patients with AF. In this study, LAA flow velocity patterns in patients with isolated AF were recorded prior to electrical cardioversion by transesophageal pulsed Doppler echocardiography. These patterns were then used to predict the initial success of cardioversion in maintaining sinus rhythm.

**Patients and Methods**

**Study population:** The study was performed between July 1993 and February 1996 on 31 patients (22 men and 9 women; mean age, 57 ± 13 years) with chronic isolated AF who were referred to our hospital for transesophageal echocardiographic evaluation, and were receiving no treatment at the time of the study. All patients had no other cardiac disease as assessed by clinical examination and transthoracic echocardiography. None of the patients had evidence of a history of hypertension, left ventricular systolic dysfunction or left ventricular hypertrophy.

**Electrical cardioversion:** Two to 3 weeks prior to cardioversion, anticoagulant therapy with warfarin was started in all patients. Transesophageal echocardiography was performed after adequate anticoagulation was achieved, and the absence of left atrial thrombus was confirmed. Thiamylal sodium (4 mg/kg) and pentazocine (15 mg) were administered intravenously, and electrical cardioversion was performed. Cardioversion was performed using a defibrillator equipped with external paddles (Fukuda Electronics Inc., Tokyo, Japan). The initial cardioversion was attempted with 100 J of energy. If three attempts using 300 J were unsuccessful, cardioversion was regarded as a “failure”. The 31 patients with AF were divided into a success group (19 patients) in which sinus rhythm was maintained for at least 2 days following cardioversion (mean time in sinus rhythm: 17 ± 12 months) and a failure group (12 patients) in which cardioversion was either not successful or sinus rhythm was not maintained for at least 2 days following cardioversion (mean time in sinus rhythm: 6 ± 1.1 hours).

**Transesophageal echocardiography:** Prior to cardioversion, transesophageal echocardiography was performed in all patients. Patients were studied in the fasting state following topical anesthesia of the hypopharynx with 1% lidocaine
Figure 1. Representative recordings of transmitral flow (TMF) and pulmonary venous flow (PVF) velocities and interatrial septal (IAS) motion using M-mode and pulsed Doppler transesophageal echocardiography. E = peak early diastolic velocity of TMF; PVS = peak systolic velocity of PVF; PVD = peak early diastolic velocity of PVF; LA = left atrium; RA = right atrium; IASa = maximum amplitude of interatrial septal motion; LAD = maximum left atrial diameter; ECG = electrocardiogram; PCG = phonocardiogram.

spray. The transesophageal probe was introduced into the esophagus with the patients lying in the left lateral decubitus position. The transmitral and pulmonary venous flow velocity patterns were recorded using the pulsed Doppler method. The peak early diastolic transmitral flow velocity was measured in the former, and the peak systolic and early diastolic pulmonary venous flow velocities were measured in the latter (Figure 1, left and middle). Interatrial septal motion was recorded in a horizontal plane that included the right and left atria using M-mode echocardiography. The maximum left atrial diameter and the maximum amplitude of the interatrial septal motion were also measured (Figure 1, right). For each measurement, the mean of measurements from five consecutive cardiac cycles during relatively stable R-R intervals were used.

Using a horizontal plane that included the left atrium and LAA, the LAA flow velocity pattern was recorded at a site in LAA 1 cm distal to its orifice (Figure 2, left). From this LAA flow velocity recording, the maximum forward and backward LAA flow velocities were calculated as the mean of maximum velocities in five consecutive cardiac cycles during relatively stable R-R intervals. Furthermore, the mean forward and backward LAA flow velocities from five random cardiac cycles at relatively stable R-R intervals were determined. Using a longitudinal plane that included the left atrium and LAA, the maximum and minimum LAA areas were measured (Figure 2, right), and the % change in area for the LAA, \[ \frac{\text{max LAA area} - \text{min LAA area}}{\text{max LAA area}} \times 100\% \], was
calculated according to the method of Pollick et al. The maximum and minimum LAA areas were visually identified, and the mean values calculated from five cardiac cycles at relatively stable R-R intervals were used. The presence or absence of thrombus or spontaneous echo contrast was examined in transverse and longitudinal planes that included the left atrium and LAA.

**Instruments:** Transesophageal echocardiography was performed using commercially available ultrasound diagnostic systems (Toshiba SSH-160A, Toshiba Corp., Tokyo, Japan and Aloka SSD-870, Aloka Co., Ltd., Tokyo) with a 5-MHz biplane transesophageal probe.

**Statistical analysis:** Values are expressed as mean ± standard deviation. Statistical analysis was performed using the unpaired Student’s t test. A p value < 0.05 was considered statistically significant.

**RESULTS**

**Age, heart rate, and duration of AF:** There was no significant difference in age between the success and failure groups. However, the heart rate prior to cardioversion was significantly higher in the success group than in the failure group (p < 0.01, Table). The duration of AF, based on clinical history and/or previous electrocardiograms, was significantly shorter in the success group than in the failure group (p < 0.001).

**Maximum left atrial diameter and maximum amplitude of interatrial
Table.  Key Parameters Prior to Cardioversion for the Success and Failure Groups

<table>
<thead>
<tr>
<th></th>
<th>Success (n = 19)</th>
<th>Failure (n = 12)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>59 ± 13</td>
<td>55 ± 10</td>
<td>NS</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>88 ± 20</td>
<td>71 ± 8</td>
<td>p &lt; 0.01</td>
</tr>
<tr>
<td>AF duration (days)</td>
<td>111 ± 71</td>
<td>425 ± 330</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>LAD (cm)</td>
<td>3.8 ± 0.9</td>
<td>4.1 ± 0.9</td>
<td>NS</td>
</tr>
<tr>
<td>IAS (mm)</td>
<td>6 ± 1</td>
<td>7 ± 2</td>
<td>NS</td>
</tr>
<tr>
<td>E (cm/s)</td>
<td>74 ± 18</td>
<td>65 ± 13</td>
<td>NS</td>
</tr>
<tr>
<td>PVS (cm/s)</td>
<td>51 ± 14</td>
<td>47 ± 11</td>
<td>NS</td>
</tr>
<tr>
<td>PVD (cm/s)</td>
<td>49 ± 15</td>
<td>46 ± 11</td>
<td>NS</td>
</tr>
<tr>
<td>max LAAV (cm/s)</td>
<td>48 ± 24</td>
<td>23 ± 4</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>max LAAbV (cm/s)</td>
<td>46 ± 20</td>
<td>27 ± 5</td>
<td>p &lt; 0.002</td>
</tr>
<tr>
<td>mean LAAV (cm/s)</td>
<td>33 ± 15</td>
<td>14 ± 3</td>
<td>p &lt; 0.0001</td>
</tr>
<tr>
<td>mean LAAbV (cm/s)</td>
<td>35 ± 16</td>
<td>14 ± 3</td>
<td>p &lt; 0.0001</td>
</tr>
<tr>
<td>max LAA area (cm²)</td>
<td>3.3 ± 0.6</td>
<td>4.1 ± 1.4</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>% change in LAA area (%)</td>
<td>34 ± 13</td>
<td>31 ± 12</td>
<td>NS</td>
</tr>
</tbody>
</table>

Success and Failure = patients with successful and unsuccessful cardioversion, respectively; HR = heart rate; AF = atrial fibrillation; LAD = maximum left atrial diameter; IAS = maximum amplitude of interatrial septal motion; E = peak early diastolic velocity of transmitral flow; PVS and PVD = peak systolic and early diastolic velocities of pulmonary venous flow, respectively; max LAAV and max LAAbV = maximum forward and backward velocities of left atrial appendage flow, respectively; mean LAAV and mean LAAbV = mean forward and backward velocities of left atrial appendage flow, respectively; max LAA area = maximum area of left atrial appendage measured from the longitudinal section including the left atrium and left atrial appendage; NS = not significant.

**Figure 3.** Comparison of maximum and mean left atrial appendage (LAA) flow velocities before cardioversion between the success and failure groups. LAAV = forward LAA flow velocity; LAAbV = backward LAA flow velocity. *p < 0.002 and **p < 0.001 vs failure group; +p < 0.0001 vs failure group.
septal motion: The maximum left atrial diameter was slightly smaller in the success group than in the failure group, but this difference was not statistically significant (Table). There was no significant difference in the maximum amplitude of interatrial septal motion between the two groups.

Transmitral and pulmonary venous flow velocities: There were no significant differences in the peak early diastolic velocity of the transmitral flow, or the peak systolic and early diastolic velocities of the pulmonary venous flow between the two groups.

LAA flow velocity: The maximum forward and backward LAA flow velocities were significantly greater in the success group than in the failure group ($p < 0.001$ and $p < 0.002$, respectively, Table). The mean forward and backward LAA flow velocities were also significantly greater in the success group than in the failure group (both $p < 0.0001$). In patients with mean forward and backward LAA flow velocities greater than 19 cm/sec, the success of cardioversion could be predicted with a sensitivity of 80% and a specificity of 88% (Figure 3).

LAA area: The maximum LAA area was significantly smaller in the success group than in the failure group ($p < 0.05$, Table). The success group tended to have a slightly greater percent change in LAA area compared to the failure group, but this difference was not statistically significant.

Thrombi and spontaneous echo contrast: In all 31 patients, no left atrial or LAA thrombi were observed prior to cardioversion. Spontaneous echo contrast was observed prior to cardioversion in 4 patients from the failure group, and remained following cardioversion.

**DISCUSSION**

It has been reported that a lack of active left atrial contraction in patients with AF causes left atrial blood stagnation and a high incidence of left atrial thrombi and systemic emboli. In particular, patients with mitral stenosis are known to have a high incidence of AF, and these thromboembolic complications may be due to left atrial and LAA enlargement and increases in left atrial pressure. Transesophageal echocardiography has facilitated examination of the left atrium and LAA. Therefore, there have been a number of reports on the relationship between LAA flow velocity patterns and thrombus formation, and on the mechanism of thrombus formation at the time of cardioversion. In general, LAA flow velocity shows a 4-peak pattern in subjects in sinus rhythm, but an irregular serrated pattern is observed in patients with AF. The maximum LAA flow velocity is reported to be lower in patients with thrombi or spontaneous echo contrast in the LAA compared to patients without either. Thus, blood stagnation in the LAA is closely related to thrombus formation.
Electrical cardioversion of AF was first described by Lown et al. in 1962. Since then, a number of studies have established that factors such as underlying disease, duration of AF, and left atrial size determine the maintenance of sinus rhythm following cardioversion. Among these factors, duration of AF is considered to be particularly important. Based on pathologic findings in patients with mitral valve disease complicated by AF, Bailey et al. have reported that if AF is prolonged, the left atrial myocardium becomes fibrotic and there is a decrease in the number of myocytes because of disuse atrophy. Similarly, in patients with isolated AF, the incidence of successful cardioversion decreases with prolonged AF. Patients with AF often show left atrial enlargement irrespective of the underlying disease, and the enlargement becomes more marked with longer durations of AF. Recently, studies using the pulsed Doppler method indicate that greater peak atrial systolic velocity of the transmitral flow and greater atrial contribution to left ventricular filling immediately after cardioversion are closely related to the maintenance of sinus rhythm after cardioversion. On the other hand, Dittrich et al. have shown that the duration of AF and left atrial size are useful in predicting the maintenance of sinus rhythm 1 month after cardioversion, but they are not useful in predicting the recovery of sinus rhythm immediately after cardioversion. These studies also show that the outcome of cardioversion depends on the underlying disease and the duration of AF. In the present study using patients with isolated AF, the duration of AF, the maximum LAA area, and the maximum and mean LAA flow velocities were found to be good parameters for predicting the initial recovery of sinus rhythm for at least 2 days after cardioversion. In particular, we believe that LAA flow velocities are potentially useful because of their high sensitivity and specificity.

The appearance of the pattern of irregular serration in the LAA flow velocity in AF corresponds to the pattern of depolarization recorded on intracoronary sinus electrocardiograms. Further, the pattern seen in the flow velocity is also found as a reflected pattern in the left atrial pressure curve. Recently, transmitral and pulmonary venous flow velocities have been reported to be important parameters in the evaluation of left atrial and ventricular dysfunction. However, since these flow velocities are influenced by both left ventricular diastolic and systolic dysfunction, they do not reflect left atrial function alone. On the contrary, LAA flow velocity is believed to reflect more closely abnormal left atrial hemodynamics. Therefore, in patients with isolated AF in whom the left atrial pressure is rarely elevated, the LAA flow velocity more closely reflects hemodynamic changes in the left atrial main cavity, even in the setting of normal interatrial septal motion and transmitral and pulmonary venous flow velocities.

In the present study, the percent change in LAA area cannot be used as a parameter for the prediction of initially successful cardioversion. The reason this
is true is because ideally the LAA ejection fraction should be calculated using LAA area measurements during sinus rhythm. However, because patients with AF lack active atrial contraction, the percent change in LAA area was calculated using the maximum and minimum LAA areas from one cardiac cycle in this study. Using this method, it is difficult to assess accurately changes in the LAA area in one cardiac cycle.

In this study, no changes in thrombus formation or spontaneous echo contrast were observed after cardioversion. However, it has been reported that some patients with spontaneous echo contrast prior to cardioversion had a transient increase in the amount of spontaneous echo contrast immediately after cardioversion or formed left atrial thrombi. These findings suggest that the recovery of sinus rhythm cannot be expected after cardioversion in patients with markedly low LAA velocities, even though thrombus formation or spontaneous echo contrast are not detected in the left atrium and/or LAA prior to cardioversion. Therefore, the indications for cardioversion require careful consideration.

In conclusion, the duration of AF, and maximum and mean velocities of the forward and backward LAA flow prior to cardioversion, are good indicators for predicting initially successful cardioversion in patients with isolated AF.

**REFERENCES**