Assessment of Left Atrial Pressure and Volume Changes during Atrial Systole with Transesophageal Pulsed Doppler Echocardiography of Transmitial and Pulmonary Venous Flow Velocities

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SUMMARY
To determine whether transmitral and pulmonary venous flow velocity patterns can be used to evaluate left atrial pressure and volume changes during atrial systole, we performed transesophageal pulsed Doppler echocardiography and right heart catheterization in 85 patients (20 with hypertrophic cardiomyopathy, 20 with dilated cardiomyopathy, 30 with prior myocardial infarction, and 15 with mitral regurgitation), and 35 normal subjects. Pulsed Doppler variables from transmitral and pulmonary venous flow velocities during atrial systole were compared with mean pulmonary capillary wedge pressure (mean PCWP), pressure rise during atrial systole (PCWP-A), and left atrial volume change during atrial systole (∆LAV). The mean PCWP correlated significantly with the peak atrial systolic transmitral flow (r = -0.38, p < 0.05) and pulmonary venous flow (r = 0.40, p < 0.05) velocities in all patients. The PCWP-A correlated significantly with the peak atrial systolic transmitral flow (r = -0.39, p < 0.05) and pulmonary venous flow (r = 0.68, p < 0.0001) velocities in all patients. There was a particularly close correlation between the PCWP-A and the peak atrial systolic pulmonary venous flow velocities. The sum of the time-velocity integral of the atrial systolic transmitral and pulmonary venous flow velocities (TAI) correlated closely with the ∆LAV (r = 0.70, p < 0.0001) in all patients. Thus, the peak atrial systolic pulmonary venous flow velocity correlated well with left atrial pressure changes during atrial systole. Furthermore, the sum of the time-velocity integral of the atrial systolic transmitral and pulmonary venous flow velocities correlated well with left atrial volume changes during atrial systole. Therefore, transesophageal echocardiographic measurements of atrial systolic transmitral and pulmonary venous flow velocities are...
reasonable indicators of left atrial pressure and volume changes during atrial systole. (Jpn Heart J 1996; 37: 333–342)

Key words: Transmitral flow velocity Pulmonary venous flow velocity Atrial systolic wave LA hemodynamics

In the past, evaluation of left atrial function using left atrial pressure and volume relationship could be obtained only by invasive methods.1–4) Recently, it has become possible to record clearly pulmonary venous flow velocity patterns using transesophageal pulsed Doppler echocardiography.5–8) This waveform, together with transmitral flow velocity patterns, permit a less invasive evaluation of hemodynamic abnormalities of the left atrium and left ventricle.9–17) We hypothesized that left atrial pressure and volume changes during atrial systole can be estimated from transmitral and pulmonary venous flow velocities.

The purpose of this study was to examine the relationships between left atrial pressure and volume changes during atrial systole and the atrial systolic transmitral and pulmonary venous flow velocity patterns.

Methods

Patients: Eighty-five patients with sinus rhythm underwent cardiac catheterization within 3 hours following transesophageal echocardiography. There was no significant difference in heart rate and medications between the two evaluations in all patients. The patient population consisted of 15 with mitral regurgitation (range: 50–72 years, mean age: 56 ± 14 years), 20 patients with dilated cardiomyopathy (range: 24–68 years, mean age: 52 ± 18 years), 30 with prior myocardial infarction (range: 40–85 years, mean age: 56 ± 14 years), and 20 with hypertrophic cardiomyopathy (range: 34–72 years, mean age: 52 ± 13 years). These patients were compared with 35 normal subjects (range: 32–66 years, mean age: 52 ± 10 years), who had been referred with non-specific symptoms of chest pain, dyspnea and/or palpitation, and in whom there was no significant evidence of cardiovascular disease on clinical examination.

The 15 patients with mitral regurgitation had regurgitation of grade 3 or more, and the other 70 patients had no mitral regurgitation or regurgitation of grade 2 or less, as determined by Sellers’ classification using left ventricular angiography.18) The diagnosis of hypertrophic cardiomyopathy was established in each patient by echocardiographic demonstration of a hypertrophied left ventricle in the absence of any cardiac or systemic disease capable of producing left ventricular hypertrophy. All patients had asymmetric septal hypertrophy (ratio of septal to left ventricular posterior wall thickness ≥ 1.5). A diagnosis of prior myocardial infarction was confirmed by: 1) a history of previous myocardial
infarction; 2) normal serum concentrations of creatine kinase; 3) abnormal Q waves on the electrocardiogram (anterior precordial leads for anterior infarction, inferior limb leads for inferior infarction); and/or 4) recent coronary angiography. Twenty-five of the 30 patients with prior myocardial infarction had involvement of the left anterior descending coronary artery and 5 had involvement of the right and/or left circumflex coronary artery. All 20 patients with dilated cardiomyopathy had idiopathic congestive heart failure that could not be explained on the basis of coronary artery, hypertensive, or valvular heart disease. In these patients, normal coronary anatomy was demonstrated by angiography.

**Protocol:** Before beginning the studies, the examinations were explained to all subjects, and their consent was obtained. All subjects underwent right heart catheterization and transesophageal echocardiography to obtain the pulmonary capillary wedge pressure (PCWP) curve, the transmitral flow velocity pattern, the pulmonary venous flow velocity pattern, the interatrial septal motion and the left atrial echogram. Right heart catheterization was performed with a Swan-Ganz catheter, and pressures were recorded with a polygraph system MCS-5500 (Fukuda Denshi, Co., Ltd., Tokyo, Japan) at a paper speed of 50 and 100 mm/sec. Fluid-filled lines and COBE strain-gauge transducers were used. The mid-chest was used as a reference point for calibration. The PCWP was obtained by inflating the balloon of the Swan-Ganz catheter in both pulmonary arteries and advancing it until each wedge pressure recording was observed. Left atrial pressure was estimated by obtaining PCWP in both lungs and using the average. Echocardiography was performed using the commercially available Aloka UST-5233 S-5 (Aloka Co., Tokyo, Japan) and Toshiba-Machida ESB-375 SR (Toshiba Corp., Tokyo). A 5 MHz probe was used with the Aloka system and a 3.75 MHz probe was used with the Toshiba system. Based on these measurements, variables during atrial systole were calculated using the following methods.

**Pulmonary capillary wedge pressure curve.** The mean PCWP and the pressure rise during atrial systole from the PCWP tracing (PCWP-A) were calculated in millimeters of mercury (Figure 1, PCWP).

**Transmitral flow velocity.** A blood flow signal was recorded after a sample volume had been placed at the tip of the mitral valve leaflets on the long-axis view of the left ventricle. Using the blood flow patterns obtained, the peak velocities of the atrial systolic waves of the transmitral flow (TMF-A) and the time-velocity integrals of the atrial systolic waves (TMF-AI) were calculated (Figure 1, TMF).

**Pulmonary venous flow velocity.** A blood flow signal was recorded by placing the sample volume within 1–2 cm of the opening of the pulmonary vein within the left atrium after the left atrium and the left superior pulmonary vein had been visualized. From the blood flow patterns obtained, the peak velocities of the
Figure 1. Measurements of parameters from the pulmonary capillary wedge pressure (PCWP) tracing, transmitral flow (TMF), pulmonary venous flow (PVF) and interatrial septal motion (IAS). mean PCWP = mean pulmonary capillary wedge pressure; PCWP-A = pressure rise during atrial systole in PCWP; E = peak velocity of early diastolic wave of TMF; TMF-A = peak velocity of atrial systolic wave of TMF; TMF-AI = time-velocity integral of atrial systolic wave of TMF; S1 and S2 = peak velocities of the first and second systolic forward waves of PVF, respectively; D = peak velocity of diastolic forward wave of PVF; PVA = peak velocity of atrial systolic backward wave of PVF; PV-AI = time-velocity integral of atrial systolic backward wave of PVF; LADps and LADAs = left atrial dimension immediately prior to and following atrial systole, respectively; LA = left atrium; RA = right atrium.

backward waves during atrial systole (PVA) and the time-velocity integrals of the atrial systolic waves (PV-AI) were calculated (Figure 1, PVF). The sum of the time-velocity integral of both the atrial systolic waves of the transmitral and pulmonary venous flow \( TAI = (TMF-AI) + (PV-AI) \) was calculated.

Interatrial septal motion. Transesophageal M-mode echograms were recorded at the left atrial-interatrial septal-right atrial junction. Left atrial volume changes during atrial systole (\( \Delta LAV \)) were calculated using the following equation by Toma et al.: \( \Delta LAV = 0.94 \times (LADps^{1.24} - LADAs^{1.24}) \), where LADps and LADAs denote the left atrial dimension immediately prior to and following left atrial systole, respectively (Figure 1, IAS).

Statistical analysis: Results are expressed as the mean ± standard deviation. The correlation between two variables was determined by linear regression analysis. Statistical significance was set at a \( p \) value < 0.05.

Results

The correlation coefficient between the atrial systolic transmitral and pulmonary venous flow velocities, the left atrial volume change during atrial systole, and the atrial systolic PCWP curves in all patients are shown in the Table. In the normal group, the peak atrial systolic transmitral and pulmonary venous flow
ATRIAL SYSTOLIC WAVES OF TRANSMITRAL AND PULMONARY VENOUS FLOW

Table  Correlation Coefficients between Atrial Systolic Transmitral and Pulmonary Venous Flow Velocities, Left Atrial Volume Change, and Pulmonary Capillary Wedge Pressure Curves in 85 Patients with Different Forms of Heart Disease

<table>
<thead>
<tr>
<th></th>
<th>TMF-A</th>
<th>TMF-AI</th>
<th>PVA</th>
<th>PV-AI</th>
<th>TAI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean PCWP</td>
<td>-0.38*</td>
<td>-0.37*</td>
<td>0.40*</td>
<td>0.12</td>
<td>-0.28</td>
</tr>
<tr>
<td>PCWP-A</td>
<td>-0.39*</td>
<td>-0.34</td>
<td>0.68**</td>
<td>0.37*</td>
<td>0.24</td>
</tr>
<tr>
<td>ΔLAV</td>
<td>0.28</td>
<td>0.30*</td>
<td>0.13</td>
<td>0.21</td>
<td>0.70**</td>
</tr>
</tbody>
</table>

*p < 0.05, **p < 0.0001  Abbreviations are same as in Figure 1.

Figure 2. Correlation between the peak atrial systolic pulmonary venous flow velocity (PVA) and pressure rise during atrial systole (PCWP-A) using pulmonary capillary wedge pressure tracings in 85 patients with different forms of heart disease.

DCM = dilated cardiomyopathy; MR = mitral regurgitation; HCM = hypertrophic cardiomyopathy; OMI = prior myocardial infarction.

velocities, the left atrial volume change during atrial systole, and the mean PCWP and PCWP-A were 45.3 ± 8.2 cm/sec, 20.4 ± 9.2 cm/sec, 12.6 ± 2.4 cm³, 10.2 ± 3.8 mmHg and 4.0 ± 1.2 mmHg, respectively.

Transmitral atrial systolic wave: The peak atrial systolic transmitral flow velocity correlated roughly with the mean PCWP and PCWP-A (r = -0.38, p < 0.05; r = -0.39, p < 0.05, respectively), but not with left atrial volume change during atrial systole. Similarly, there was a rough correlation between the time-velocity integral of the transmitral atrial systolic wave and the mean PCWP (r = -0.37, p < 0.05) and the left atrial volume change during atrial systole (r = 0.38, p < 0.05). However, there was no significant correlation between the time-velocity
Figure 3. Correlation between the left atrial volume change during atrial systole (ΔLAV) and the sum of the time-velocity integral of the atrial systolic transmitral and pulmonary venous flow velocities (TAI) in 85 patients with different forms of heart disease. Abbreviations are same as in Figure 2.

integral of the transmitral atrial systolic wave and the PCWP-A.

**Pulmonary venous atrial systolic wave:** The peak atrial systolic pulmonary venous flow velocity correlated significantly with the mean PCWP (r = 0.40, p < 0.05) and with the PCWP-A (r = 0.68, p < 0.0001) (Figure 2). However, there was no significant correlation between the peak atrial systolic pulmonary venous flow velocity and the left atrial volume changes during atrial systole. A rough correlation existed between the time-velocity integral of the pulmonary venous atrial systolic wave and the PCWP-A (r = 0.37, p < 0.05). The time-velocity integral did not correlate significantly with the mean PCWP or left atrial volume changes during atrial systole.

**Transmitral and pulmonary venous atrial systolic waves:** A close correlation was observed between the sum of the time-velocity integral of both the atrial systolic waves and the left atrial volume changes during atrial systole (r = 0.70, p < 0.0001) (Figure 3). However, there was no significant correlation between the sum of the time-velocity integral and other variables.
DISCUSSION

The left atrium serves as a pump during active contraction of the atrial myocardium, a blood reservoir during ventricular systole, and a conduit for blood flow from the pulmonary veins to the left ventricle during early to mid-diastole.\textsuperscript{1-4} Clear recordings of pulmonary venous flow velocity patterns now have become feasible using transesophageal pulsed Doppler echocardiography.\textsuperscript{5-9} There have been several reports describing the relationship between the left atrium and ventricle based on detailed analyses of transmitral and pulmonary venous flow velocities.\textsuperscript{6-17} Important factors regulating transmitral and pulmonary venous flow include left ventricular and atrial contractility and diastolic function,\textsuperscript{7-10,14} loading conditions,\textsuperscript{5,11,12} mitral regurgitation,\textsuperscript{20,21} heart rate, and an increase in left atrial pressure.\textsuperscript{6,13,15-17} In this study, we evaluated the relationship between the transmitral and pulmonary venous flow velocities during atrial systole and the pulmonary capillary wedge pressure (PCWP) curves, and examined whether changes in left atrial pressure and volume during atrial systole can be estimated from transmitral and pulmonary venous flow velocity patterns.

In patients with significant increases in left ventricular end-diastolic pressure, the peak atrial systolic velocity of the transmitral flow is suppressed, and retrograde flow from the left atrium into the pulmonary veins increases.\textsuperscript{8,9,16} However, as demonstrated by Kuecherer et al\textsuperscript{7} and Oki et al,\textsuperscript{8,9} if the increase in PCWP or left ventricular end-diastolic pressure is mild to moderate, the peak atrial systolic velocity of the transmitral flow increases, and the peak atrial systolic velocity of the pulmonary venous flow also increases in some cases. However, since both V- and A-wave heights in the left atrial pressure curve contribute to the mean PCWP, it is not adequate to evaluate left atrial hemodynamics using the mean PCWP in all patients, including patients with mitral regurgitation. This may explain the absence of a close correlation between the mean PCWP and the peak atrial systolic transmitral or pulmonary venous flow velocity in our study. In certain patients with dilated cardiomyopathy, an increase in the PCWP does not accompany an increase in the peak atrial systolic pulmonary venous flow velocity due to left atrial pump dysfunction caused by left atrial myocardial failure.\textsuperscript{8} Since factors that affect transmitral and pulmonary venous flow velocities vary under different conditions in patients with heart disease,\textsuperscript{5,11,12} it is not surprising that a close correlation was not found between the mean PCWP and the peak atrial systolic transmitral and pulmonary venous flow velocities.

In this study, we examined the relationship between the pressure changes during atrial systole using the PCWP curve and the peak atrial systolic transmitral and pulmonary venous flow velocities. There was a close positive correlation between the A-wave pressure change on the PCWP tracing and the
peak atrial systolic pulmonary venous flow velocity. A weak but significant correlation was found between the A-wave pressure change on the PCWP tracing and the peak atrial systolic transmitral flow velocity. Goto et al.\(^{22}\) have reported that the pulmonary venous system during diastole has a capacity two to three times larger than the left ventricle. It is known that the transmitral flow velocity in many patients with severe left heart failure pseudonormalizes.\(^{8-10}\) In these patients, effective blood flow into the left ventricle during atrial systole is suppressed. However, retrograde blood flow into the pulmonary veins is likely to occur because of the high capacity. Our study suggests that left atrial pressure changes during atrial systole are directly reflected by the peak atrial systolic pulmonary venous flow velocity in patients with a “pseudonormalization” of the transmitral flow velocity pattern, excluding patients with left atrial myocardial failure.\(^{8}\)

A close positive correlation was found between the left atrial volume change during atrial systole and the sum of the time-velocity integral of both atrial systolic waves. In general, cineangiography or two-dimensional echocardiography is widely accepted as a reliable method for determination of cardiac volume changes. In this study, interatrial septal and left atrial echograms were obtained by transesophageal M-mode echocardiography. Furthermore, left atrial volume changes during atrial systole were calculated using an equation described by Toma et al.\(^{19}\) Although there are certain problems using M-mode echograms for estimating left atrial volume changes, the echogram is made at the proper level of the left atrial cavity-interatrial septum-right atrial cavity by transesophageal echocardiography. In this view, changes in left atrial diameter represent interatrial septal motion, and are proportional to changes in left atrial volume. Yonezawa et al.\(^{23}\) have reported that interatrial septal motion during atrial systole depends upon active contraction of the left atrial myocardium. This observation is based upon simultaneous recording of interatrial septal echoes and left and right atrial pressures. Thus, it is thought that the effects of right atrial pressure during atrial systole can be ignored in our subjects.

In this study, we used a fluid-filled pulmonary capillary wedge catheter instead of a high fidelity manometer-tipped catheter to approximate left atrial pressure. The measurement of the rise in left atrial pressure with atrial contraction is bound to be damped by passage through the pulmonary capillaries and the fluid-filled catheter. Although it is clinically acceptable as a measure of mean left atrial pressure, many hemodynamic researchers do not accept PCWP because of its multiple assumptions. However, this substitution is allowable because patients with diseases such as mitral stenosis which cause pressure gradient between the left atrium and ventricle were excluded from this study, and because many studies have utilized PCWP as an estimate of left atrial pressure in the past.\(^{16,24,25}\)
Although there are some methodological problems with our study, the peak atrial systolic pulmonary venous flow velocity correlated closely with the pressure rise during atrial systole using PCWP curves, and the sum of both atrial systolic time-velocity integrals also correlated closely with left atrial volume changes during atrial systole using transesophageal M-mode echocardiography. Furthermore, these findings were observed in patients with different clinical conditions.

**CONCLUSION**

A semi-invasive evaluation of both atrial systolic transmitral and pulmonary venous flow velocities by transesophageal pulsed Doppler echocardiography provides important information regarding left atrial pressure and volume changes during atrial systole in patients with different forms of heart disease.

**REFERENCES**


