Original article

Role of the left atrial function on the pseudonormalization of the transmitral flow velocity pattern evaluated by two-dimensional tissue tracking technique

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A B S T R A C T

Background: Mechanisms of the pseudonormalization (PN) of the transmitral flow (TMF) velocity pattern have been mainly attributed to left ventricular diastolic function.

Purpose: To assess the influence of left atrial (LA) function on the PN with two-dimensional tissue tracking technique.

Methods: The subjects consisted of 21 healthy volunteers and 70 patients with various cardiac diseases. Images of one cardiac cycle in the apical four-chamber view were stored by the HITVISION 900 (Hitachi Medical, Chiba, Japan). The LA volume (LAV) loop was created using two-dimensional tissue tracking technique and LAV index (LAVI) at a given cardiac phase was calculated. A preload of 90 mmHg was applied using a customized lower body positive pressure (LBPP) system. Patients were divided into the PN group (n=18) with their early diastolic TMF velocity (E) increased and late diastolic TMF velocity (A) decreased, and the non-(N)-PN group (n=52) with both E and A wave velocities increased by LBPP.

Results: (1) During LBPP, the LAVI in both the groups increased significantly. (2) In the N-PN group, the LAVI (p<0.001), LAVI (p<0.01), and LAVI (p<0.0001) increased significantly. The dV/dt (p<0.0001) and dV/dt (p<0.0001) increased significantly with an increase in the dV/dt. On the other hand, there was no change in those parameters except LAVI (p<0.05) and dV/dt (p<0.05) significantly increased in the PN group. (3) As a result, the LAVI was significantly greater in the PN group than in the N-PN group (p<0.0001) during LBPP. The ratio of E velocity to early diastolic mitral annular velocity (E/E) during LBPP was significantly greater in the PN group than in the N-PN group (p<0.0001).

Conclusions: The lack of an increase in active LA emptying volume in response to an increase in preload leads to elevated LA pressure and the pseudonormalization of the TMF velocity pattern in patients with various cardiac diseases.

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Introduction

Left ventricular (LV) diastolic performance has been evaluated by Doppler echocardiography because LV diastolic function is regarded as important for LV filling [1–3]. Doppler assessment of transmitral flow (TMF) usually reveals early (E) and late (A) diastolic waves. A lower E velocity and a compensatory increase of the A velocity usually indicate impairment of LV relaxation. When the left atrial (LA) pressure and/or LV end-diastolic pressure increases, the velocity of the E wave increases and that of the A wave decreases, which has been called 'pseudonormalization' (PN) and is seen with various LV myocardial disorders. However, we should pay more attention to the important role of the LA in LV filling independent of LV myocardial function [4, 5]. The LA can be considered to have reservoir, conduit, and booster pump functions [6]. It is located upstream of the LV and collects blood from the pulmonary veins as its reservoir function. Then blood passes from the LA during early diastole mainly due to the pressure gradient between atrium and ventricle, although active LA contraction also supports LV filling. Therefore, abnormalities in LA compliance and pump function should have an important influence on LV filling independently of LV myocardial disorders [7].

Application of lower body positive pressure (LBPP) can increase the preload of the LA [8]. If the Frank–Starling mechanism...
operates ideally, LA output (forward stroke volume) should be increased by LBPP, resulting in an increase of the transmitral flow velocity (E and A waves). If this mechanism is abnormal, pseudonormalization of TMF with an increase of the E wave velocity and a decrease of the A wave velocity may be observed. Thus, LBPP could simulate the progression of congestive heart failure, which leads to pseudonormalization in the clinical setting.

The two-dimensional (2D) tissue tracking technique can be employed to automatically trace myocardial movement on 2D echocardiographic images using the block matching method [9]. With this method, LA volume (LAV) loops can be automatically constructed for single cardiac cycles by simply tracing the LA endocardium at the time of the R wave on the electrocardiogram (ECG) and then automatically calculating the LAV frame by frame [10].

The purpose of the present study was to assess the influence of LA function on pseudonormalization of TMF by evaluating dynamic changes of LAV during the cardiac cycle with the automatic 2D tissue tracking technique.

Methods

Study population

The subjects consisted of 21 healthy volunteers (mean age: 38 ± 10 years; range: 24–60 years) and 70 patients with various cardiac diseases, including 35 patients with previous myocardial infarction (64 ± 8 years; 47–80 years), 19 with hypertensive heart diseases (59 ± 12 years; 36–77 years), 7 with hypertrophic cardiomyopathy (66 ± 9 years; 52–78 years) and 9 with dilated cardiomyopathy (64 ± 11 years; 39–75 years). In patients with previous myocardial infarction and those with dilated cardiomyopathy, the affected coronary vessels and severity of myocardial degeneration were identified by cardiac catheterization and/or myocardial biopsy. Hypertensive heart disease was defined by a systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg with a mean LV wall thickness ≥11.5 mm. Hypertrophic cardiomyopathy was defined as asymmetric septal hypertrophy with an interventricular septal thickness ≥15 mm and a septal/posterior wall thickness ratio ≥1.3. Subjects were in New York Heart Association functional class I or II at rest. The patients were being treated with diuretics, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, Ca2+ antagonists, and/or β-blockers, and the blood pressure was well controlled. Patients with resting LV outflow tract obstruction were excluded to avoid the influence of increased after load. Patients with atrial fibrillation and patients with valvular heart diseases were also excluded from the study. The healthy volunteers and the patients all gave written informed consent to this study, and it was approved by the research Ethics Committee of Fujita Health University.

Conventional echocardiography

Transthoracic echocardiography was performed at baseline and during LBPP (as described below) using commercially available equipment, which was a HVISION 900 (Hitachi Medico, Chiba, Japan) with a 2–5 MHz phased array transducer (SS0A). The LV end-diastolic and end-systolic volumes and ejection fractions were calculated by the single plane Simpson’s rule from the apical four-chamber view. LV mass index was calculated with Penn’s formula [11]. Relative wall thickness was calculated as the sum of the end-diastolic interventricular septal and posterior wall thicknesses divided by the LV end-diastolic dimension. Pulsed Doppler measurements were performed according to the recommendations of the American Society of Echocardiography [12]. The velocity at the mitral annulus was recorded by pulsed Doppler in the apical four-chamber view, with the systolic and the early (E) and late diastolic velocities being measured. The E/E’ ratio was calculated as an estimate of LV filling pressure [13].

Lower body positive pressure

A preload of 90 mmHg was applied for three minutes using a customized LBPP system (DM-5000EX; Nitto Corp., Tokyo, Japan) (Fig. 1). On the basis of the TMF velocity pattern during LBPP, the patients were divided into two groups, which were a PN group with an increase of E wave velocity and a decrease of A wave velocity resulting in a pseudonormal pattern (E/A > 1), and a non-pseudonormalization (N-PN) group with an increase of both E and A wave velocities even if the resulting E wave velocity was greater than the A wave velocity and resembled the pseudonormal pattern (E/A < 1 or E/A > 1) (Fig. 2). The PN group included 15 patients with previous myocardial infarction, 2 with dilated cardiomyopathy, and 1 with hypertrophic cardiomyopathy (Table 1).

Automatic construction of LAV loops and measurement of LAV

LAV was measured by automatic LAV tracking based on the 2D tissue tracking technique [10]. Images of a complete cardiac cycle in the apical four-chamber view were stored by the same echocardiographic equipment, after which automatic construction of LAV loops was performed off-line using the E-tool viewer (Hitachi Medico). First, the LA endocardium was manually traced at the time when the R wave occurred on the ECG (Fig. 3, left), after which

Fig. 1. Lower body positive pressure system. A 90 mmHg of preload was increased for three minutes using a customized lower body positive pressure (LBPP) system (DM-5000EX, Nitto Corp., Tokyo, Japan).

Fig. 2. Patient classification. On the bases of the transmitral flow (TMF) velocity pattern during lower body positive pressure (LBPP), the patients were divided into the two groups, which were a pseudonormalization (PN) group with an increase of early diastolic TMF velocity (E) and a decrease of late diastolic TMF velocity (A) resulting in a pseudonormal pattern (E/A > 1), and a non-pseudonormalization (N-PN) group with an increase in both E and A wave velocities even if the resulting E wave velocity was greater than A wave velocity and resembled the pseudonormal pattern (E/A < 1 or E/A > 1).
the LAV for each frame was automatically calculated by the single plane Simpson’s rule and a LAV loop was constructed (Fig. 3, right). One cardiac cycle was represented by 60–80 frames in normal sinus rhythm and 2D echocardiographic images were acquired at 60–120 frames per second.

From the LAV loops thus obtained, the LAV at a given cardiac phase could be measured [14] (Fig. 3, right). Then the LAV index (LAVI) was calculated as the measured LAV divided by the body surface area [15]. The maximum LAVI (LAVImax) at end-systole, LAVI at the onset of the P wave on the ECG (LAVIp), and the minimum LAVI (LAVImin) at end-diastole were calculated. The active emptying LAVI (LAVIact) was defined as LAVIp − LAVImin; the passive emptying LAVI (LAVIpass) was defined as LAVImax − LAVIp, and the total emptying LAVI (LAVItotal) was defined as LAVImax − LAVImin.

The active LA emptying fraction (%LAVIact) was defined as the ratio of LAVIact to LAVIp, the passive LA emptying fraction (%LAVIpass) was defined as the ratio of LAVIpass to LAVImax, and the total LA emptying fraction (%LAVItotal) was defined as the ratio of LAVItotal to LAVImax.

Table 1: Demographic characteristics.

<table>
<thead>
<tr>
<th></th>
<th>Normal (n = 21)</th>
<th>N-PN (n = 52)</th>
<th>PN (n = 18)</th>
</tr>
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<tbody>
<tr>
<td>Male/female</td>
<td>21/0</td>
<td>38/14</td>
<td>14/4</td>
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<tr>
<td>Age [years]</td>
<td>38 ± 10</td>
<td>63 ± 10</td>
<td>62 ± 10***</td>
</tr>
<tr>
<td>Mean blood pressure (mmHg)</td>
<td>75.6 ± 6.2</td>
<td>81.4 ± 5.2</td>
<td>80.8 ± 4.2</td>
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<tr>
<td>LV mass index (g/m²)</td>
<td>107 ± 26</td>
<td>184 ± 87†</td>
<td>165 ± 55**</td>
</tr>
<tr>
<td>Relative wall thickness</td>
<td>0.40 ± 0.10</td>
<td>0.50 ± 0.21†</td>
<td>0.40 ± 0.15*a</td>
</tr>
<tr>
<td>Hypertensive heart disease</td>
<td>0</td>
<td>19</td>
<td>0</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>0</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>0</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>0</td>
<td>20</td>
<td>15</td>
</tr>
<tr>
<td>LV, left ventricular; N-PN, non-pseudonormalized group; and PN, pseudonormalized group.</td>
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<td>† p &lt; 0.05.</td>
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<td>†† p &lt; 0.01.</td>
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<td>††† p &lt; 0.001 vs normal.</td>
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<tr>
<td>* p &lt; 0.05 vs N-PN.</td>
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Statistical analysis

Data were expressed as the mean ± SD. Comparison of parameters among the groups was performed by analysis of variance (ANOVA) and statistical significance was confirmed with Sheffe’s F-test. A paired t-test was used for comparison of parameters measured at baseline with those measured during LBPP. LAVmax was measured by two independent observers. Bland and Altman method [16] was used for evaluating inter- and intraobserver variability. Variability was expressed as mean ± SD of the absolute difference between the 2 sets of measurements. In all analyses, p < 0.05 was considered to be statistically significant.

Results

Reproducibility of LAV measurements

There were excellent intraobserver (r = 0.988, p < 0.0001) and interobserver (r = 0.965, p < 0.0001) agreement in the measurement of LAVmax. The intra- and interobserver variability for the LAVmax were −0.20 ± 5.07 and 0.19 ± 8.47, respectively. The percent intra- and interobserver variability (calculated as the difference between two observations divided by the mean of the two observations)
were 0.6% and 2.8%, respectively. From these results, the reproducibility of the automatic LAV measurement by the 2D tissue tracking method was considered to be reliable.

**Comparison of baseline demographic characteristics, two-dimensional echocardiographic parameters, and Doppler parameters**

The LV mass index was significantly larger in both patient groups than in the healthy controls, without a difference between the N-PN and PN groups (Table 1). The relative wall thickness was significantly larger in the N-PN group than in the normal control group, while it was significantly smaller in the PN group than in the N-PN group. There was no significant difference in baseline mean blood pressure among the groups. The baseline LV volume was significantly larger and the ejection fraction was significantly smaller in the two patient groups than in the healthy control group (Table 2). Assessment of TMF showed that the deceleration time of the E wave was significantly shorter in the PN group than in the N-PN group. All of the systolic, early diastolic, and late diastolic mitral annular velocities were significantly lower in both patient groups than in the normal control group, while there were no significant differences between the N-PN and PN groups. The baseline E/E′ ratio was the largest in the PN group.

**Changes in mean blood pressure, two-dimensional parameters, and Doppler parameters during LBPP**

During LBPP, the mean blood pressure was significantly increased in both groups of patients which promptly reflecting after load increase (Table 2). However, there was no significant difference between the N-PN and PN groups. Both the E and A wave velocities increased significantly in the healthy control group and the N-PN group, whereas the E wave velocity increased and the A wave velocity decreased significantly in the PN group, resulting in an E/A ratio >1. The deceleration time of the E wave was significantly shortened in the PN group during LBPP. Forward stroke volume showed a significant increase in the healthy control and N-PN groups, while there was no change in the PN group. While the E/E′ ratio was significantly increased in all three groups, that of the PN group was significantly larger than that of the N-PN group during LBPP.

**Baseline LAV in the N-PN and PN groups**

All of LAVmax, LAVp, and LAVmin were significantly larger in the two patient groups than in the healthy control group at baseline (Table 3). Active emptying LAV was significantly greater in the patient groups compared with that in the control group at baseline. However, due to the significantly smaller %LAVact, the %LAVtotal of the PN group was significantly smaller than that of the N-PN group at baseline. The early diastolic emptying rate was significantly larger and the late diastolic emptying rate was significantly smaller in the PN group compared with the N-PN group.

**Changes in the LAV during LBPP**

During LBPP, the LAVmax of both patient groups increased significantly, with a significantly larger increase in the PN group (Table 3). In the N-PN group, LAVp, LAVact, and LAVtotal all increased significantly. In contrast, LAVp decreased significantly in the PN group, whereas there was no significant change in LAVact or LAVtotal. Both %LAVact and %LAVp increased significantly in the N-PN group, while %LAVact decreased in the PN group. All of %LAVp, %LAVact, and %LAVtotal were significantly smaller in the PN group than in the N-PN group during LBPP. As a result, LAVmin
Fig. 5 shows representative recordings of the TMF velocities and LAV loops from the N-PN and PN groups. The E wave and A wave velocities both increased in the N-PN group during LBPP (Fig. 5, top left panel). On the other hand, the E wave velocity increased and the A wave velocity decreased during LBPP in the PN group, showing PN of the TMF velocity pattern (Fig. 5, top right panel). With regard to the LAV parameters, LAVI\textsubscript{max} increased in both patient groups during LBPP (both bottom panels). In the N-PN group, even though LAVI\textsubscript{max} was increased, the increase in LAVI\textsubscript{pass} and LAVI\textsubscript{act} meant that LAVI\textsubscript{min} returned to the baseline value and there was no excessive LA volume (Fig. 5, bottom left panel). In contrast, the PN group showed a decrease in both LAVI\textsubscript{pass} and LAVI\textsubscript{act}, leading to an increase in LAVI\textsubscript{min} (Fig. 5, bottom right panel) that could cause an increase in LA pressure.

**Discussion**

The mechanisms involved in PN of the TMF velocity pattern have mainly been discussed on the basis of LV diastolic performance [1]. In contrast, the role of LA function in PN has not been sufficiently appreciated, partly because of limited measurement of the LAV. Only the maximum LAV at end-systole has been used for estimating LA pressure and outcomes in clinical studies [17–24]. We developed an automatic LAV measurement method based on the 2D tissue tracking technique. Using this method, we could measure dynamic changes of the LAV frame by frame and automatically construct the LAV loop for a complete cardiac cycle. Because LBPP increases the LA preload, we employed it to precisely evaluate changes in LAV performance in relation to changes in the TMF velocity pattern as preload was increased.

The present study demonstrated the following points. (1) Patients showed two responses in the TMF velocity pattern when LBPP was applied. In the N-PN group, both the E and A wave velocities were increased by LBPP, while the E wave velocity was increased and the A wave velocity was decreased in the PN group, leading to pseudonormalization. (2) In the N-PN group, the forward LA stroke volume increased during LBPP. On the other hand, the PN group showed no change in the LA stroke volume, but had a shorter deceleration time of the E wave and an increased E/E' ratio, which suggested the elevation of LV filling pressure. (3) In both patient groups, LAVI\textsubscript{max} increased significantly during LBPP. In the N-PN group, LAVI\textsubscript{min} returned approximately to baseline at each end of cardiac cycle due to an increase in the emptying LAV. In contrast, the PN group showed an increase in LAVI\textsubscript{min} during LBPP compared with baseline because the decrease in LAVI\textsubscript{act} might have caused an increase in the LV filling pressure and pseudonormalization of the TMF velocity pattern.

**Limited measurement of LAV in previous studies**

Detailed assessment of LA function should ideally be performed using the LA pressure–volume relationship, but measurement of LAV is hampered by technical problems caused by the anatomical features of the LA. It has been difficult to identify the LA endocardium because the LA is located between the pulmonary veins and the LV, and because the orifice of the LA appendage opens into the LA. Hoit et al. [25] constructed LAV loops by employing sonomicrometry in animal experiments. The changes in LAV were calculated electrically using crystals attached to the LA wall. Although this may be the best method of LAV measurement, it is invasive and cannot be applied to humans. Triposkiadis et al. [14,15]...
used echocardiography to evaluate LA function based on LAV data obtained by the single plane area–length method. This was considered to be the most precise and reliable of the past non-invasive LAV measurements. However, they only measured LAV at certain phases of the cardiac cycle and did not evaluate dynamic changes in LAV.

**Measurement of LAV based on the 2D tissue tracking technique**

We used an automatic LAV measurement method that was based on the 2D tissue tracking technique [9]. This method can automatically trace myocardial movement by the block matching method on 2D echocardiographic images and is independent of tissue velocity. After a point in the first frame of the 2D recording is selected, the algorithm searches for the point in the next frame that is assumed to be the closest to the first selected point based on the pixel intensity distribution. By applying this method, the LAV loop for an entire cardiac cycle could be constructed after simply tracing the LA endocardium at the time of the R wave, and then automatically calculating the LAV in each subsequent frame.

**LA response to the increase in preload by LBPP**

In both the N-PN and PN groups, LAVI max increased in response to the increase in preload during LBPP. The increased volumes would be ejected by a normal LA based on the Frank–Starling mechanism. Thus, if the normal physiological mechanism operates, LAVI total would increase with an increase in the forward stroke volume.

In the N-PN group, the forward stroke volume increased as evidenced by an increase in LAVIpass, LAVI act, and LAVI total. In particular, there was an increase in the LA systolic filling and passive emptying velocities, as shown by the increment of LA systolic filling and early diastolic emptying rates. In contrast, the PN group showed no significant changes in LAVI act and LAVI total, even though LAVIpass increased significantly. In fact, %LAVI act and %LAVI total rather decreased in the PN group despite %LAVI pass increasing significantly. All of %LAVI pass, %LAVI act, and %LAVI total were significantly smaller in the PN group than in the N-PN group during LBPP. As a result, LAVI min became significantly larger in the PN group than in the N-PN group. The fact that LAVI act did not increase and instead decreased was possibly related to deterioration of active LA pump function.

Even at baseline, the PN group had a larger LV volume, smaller ejection fraction, shorter deceleration time of the E wave, and larger E/E′ ratio than the N-PN group, suggesting that LV filling pressure was already greater in the PN group. When preload was increased further by LBPP, the decreased LA reservoir and pump function in the PN group led to stagnation of blood in the LA that resulted in an increase of LAVImax, which could further increase LA pressure. These findings suggest that operation of the Frank–Starling mechanism in the LA is impaired in the PN group. The increase in LA pressure caused an increase in E wave velocity. The decrease in A wave velocity indicated deterioration of active LA pump function and was not only due to elevation of LV end-diastolic pressure. The lack of an increase in total emptying LAV was mainly due to no increase in the active emptying LAV and the consequent increase in LA pressure caused PN of the TMF velocity pattern.

Because there were various cardiac diseases included in the patient group, the LV diastolic function itself surely influenced the LA after load. In the healthy subjects, LA does not always need to actively support LV filling because LV elastic recoil and diastolic suction may sufficiently respond to preload increase. However, LA should be activated when any intrinsic LV diastolic kinetics would insufficiently complete LV filling. The present study has clearly demonstrated the deterioration in the active LA pump function by directly showing decreased active LA emptying volumes.

**Limitations**

There was no direct assessment of the pressure and/or volume change during LBPP because this was a non-invasive echocardiographic study. The baseline LA pressure may influence the changes in the TMF velocity pattern. However, the importance might be
in the intrinsic LA compliance and active functions. In the N-PN group, there were also patients with LV dysfunction with higher E/E' included, indicating relatively higher baseline LA pressure. However, if those LA compliance and active functions were still preserved, the PN would not be presented.

There is also no experimental evidence about the effects of the LBPP system that we used in the present study. In this system, both legs of a subject are placed in pneumatic boots (Fig. 1) and inflating the boots applies 90 mmHg of preload according to the manufacturer (Nitto Corp., Tokyo, Japan). Oki et al. [8] reported that a 40 mmHg increase in preload due to LBPP was sufficient to cause PN of the TMF velocity pattern. Therefore, we consider that the changes in the TMF velocity pattern in the present study reflected the increase in preload. In order to show whether LBPP provided the same level of increase in preload between patients and healthy controls, the changes in inferior vena cava diameter may support the results. However, we did not evaluate them in the present study because of the limited study protocol.

There have been the problems that the orifices of the pulmonary veins and the LA appendage opening into the LA make it difficult to identify the LA borders. We determined the LA borders by avoiding these orifices carefully and traced the endocardium circumferentially. The block matching algorithm that we employed precisely tracked pixels frame by frame and the resultant LAV loops provided reliable volumes for any given cardiac phases similar to those reported by Triposkiadis et al. [14,15].

We used the single plane Simpson’s rule for calculating LAV in each frame. The biplane Simpson’s rule might be more accurate for evaluating LAV, but the LA endocardial outline obtained from the 2-chamber views was not always clear enough. Also, Lester et al. [26] reported that there was no difference between LAVs obtained with single plane and biplane methods.

Clinical implications

Pseudonormalization of the TMF velocity pattern has traditionally been discussed from the viewpoint of LV function and pressure. The present study highlighted the participation of LA reservoir and active emptying functions as well. Automatic LAV monitoring based on the 2D tissue tracking method enabled more precise assessment of LV filling in relation to LA mechanical function.

Conclusions

Under physiological conditions, the forward stroke volume is increased by an increase in LA emptying volumes as the preload increases. In contrast, the lack of an increase in active LA emptying volume in response to an increase of preload leads to the stagnation of blood in the left atrium and pseudonormalization of the TMF velocity pattern.

References