Tranesophageal Echocardiographic Dimensional Analysis of Four Cardiac Chambers during Positive End-expiratory Pressure

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The authors examined the effects of positive end-expiratory pressure (PEEP) on cardiac function by dimensional analysis of the four heart chambers using M-mode transesophageal echocardiography (TEE). The accuracy of cardiac output (CO) calculated from TEE was confirmed by its close correlation (r = 0.97) with CO determined by the thermodilution technique. The reliability of TEE also was confirmed by excellent correlation (r = 0.95) between left ventricular end-diastolic (LVEDD) and end-systolic (LVESD) dimensions measured by the two-dimensional precordial echocardiography and those by TEE. With 10 cmH₂O PEEP LVEDD decreased from its level during zero end-expiratory pressure (ZEEP), and the calculated stroke volume also decreased. These decreases were greater during 15 cmH₂O PEEP, where heart rate increased slightly but significantly. Ejection fraction (EF) and fractional shortening (FS), as a whole, did not change significantly. Mean velocity of circumferential fiber shortening (mean Vcf) significantly increased and LVESD significantly decreased with PEEP. Although systolic blood pressure (SBP) significantly decreased, the (SBP-PEEP value)/LVEDD ratio was not changed with PEEP. Such measures of left ventricular systolic function as EF, FS, mean Vcf, and (SBP-PEEP value)/LVESD were not decreased. Right ventricular end-diastolic dimension decreased with PEEP. Right atrial end-diastolic dimension began to decrease immediately after PEEP was initiated, whereas left atrial end-diastolic dimension began to decrease a few seconds later, suggesting that left ventricular preload decreased as a result of a decrease in right ventricular preload. The authors therefore conclude that CO was decreased as a result of the decrease in right and left ventricular preload. (Key words: Equipment: echocardiograph, transesophageal. Heart: myocardial function. Ventilation: positive end-expiratory pressure.)

Positive end-expiratory pressure (PEEP) has been widely employed for treating adult respiratory distress syndrome. With PEEP, hypoxemia is improved, but cardiac output (CO) decreases. The mechanism by which CO is decreased remains controversial. The study of this mechanism has been limited by the methods available to date.

We anticipated that M-mode transesophageal echocardiography (TEE) would be very useful for the clinical study of cardiac function during PEEP. Unlike conventional precordial echocardiography with which technically adequate images may not be obtainable during PEEP, TEE permits good quality images of the four heart chambers, from which the evaluation of left ventricular systolic function even during PEEP is possible. The purpose of this study was to determine the mechanism of the decrease in CO during PEEP by dimensional analysis of the four heart chambers using TEE.

Materials and Methods

Sixteen patients receiving treatment at the Department of Traumatology, Osaka University Hospital, were selected for the study. Their ages ranged from 18 to 63 yr. All patients were mechanically ventilated for respiratory failure of various causes. Neither diuretics nor infusion of significant volume was permitted during the study period. No patient had evidence of myocardial injury at the time of this study, nor a clinical history of cardiac or pulmonary disease. Informed consent concerning the nature and purpose of this study was obtained in each case from the patients themselves or from their nearest relatives.

Patients were sedated with intravenous diazepam and paralyzed with pancuronium bromide and ventilated with mechanically (AHS, model CV 2000) via a cuffed endotracheal tube. Arterial oxygen tension (PaO₂) was greater than 80 mmHg on an inspired oxygen concentration (FiO₂) of 0.21–1.0 and at zero end-expiratory pressure. The FiO₂ was maintained constant throughout the study. In all patients, the ventilator was set at a constant tidal volume (15 ml/kg) at the respiratory rate needed to maintain arterial carbon dioxide tension at 30–40 mmHg.

Hemodynamic Measurement

In each patient a radial arterial catheter was inserted and connected to a Statham P-23 1D® fluid-filled pressure transducer to measure systolic arterial pressure (SBP). Heart rate (HR) was calculated from the electrocardiogram (ECG). To examine the accuracy of TEE, we determined CO by the thermodilution technique during the expiratory phase at least three times with a Gould® cardiac index computer (model ST1435) and compared this CO with that indirectly determined by TEE. Furthermore, to examine the reliability of TEE, we compared left ventricular end-diastolic dimension (LVEDD) and left ventricular end-systolic dimension (LVESD) measured by TEE with those obtained by two-dimensional precordial echocardiography in three patients in whom the latter measurements were successful.

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M-MODE TRANSESOPHAGEAL ECHOCARDIOGRAPHY

TEE was performed on patients in the supine position. The transducer was inserted orally into the esophagus and placed at a depth of about 35 cm from the mouthpiece. The mitral valve usually could be identified at this level. The transducer was pulled back slightly to allow visualization of the interatrial septum and right atrial free wall. This method was identical to that originally described by Toma et al.1 The transducer then was advanced a few centimeters from the mitral valve position and flexed upward so that a clear echogram could be obtained of the left ventricle at the chordae tendineae of the mitral valve. This level was defined as the point of measurement of the left ventricular dimensions by TEE (fig. 1). This echogram represents a mirror image of that recorded with the standard precordial method.2 In addition, the transducer was slightly manipulated in a clockwise rotation from the left ventricular dimension position to identify the right ventricle. The right ventricular dimension was measured at the level of the chordae tendineae of the tricuspid valve. In all cases, we chose the transducer positions that gave the maximal dimensions of both atria and both ventricles.

The transducer was fixed in position during measurement of each maximal dimension. When PEEP altered the position of the heart relative to the transducer and the endocardial echo of the chambers became obscured or when PEEP rotated that position and the anterior and the posterior wall protrusion became asymmetrical, the position and direction of the transducer were slightly adjusted so that the maximal chamber dimensions could be obtained with clear and symmetric endocardial definition. With a 3.5 MHz unfocused transducer of 10 mm in diameter, the TEE was recorded on a strip-chart recorder (Aloka SSD-110S). A phonocardiogram, lead II ECG, and carotid pulse tracing were recorded simultaneously. Both atrial end-diastolic dimensions and LVESD were measured at the initiation of the aortic component of the second heart sound on the phonocardiogram. Left and right ventricular end-diastolic dimensions were measured at the peak of the R wave of the ECG. All dimensions from the echocardiogram were determined with a digitizer (Graphitec®, model KD-4030), which had been programmed to plot the wall tracings and dimensions continuously during the cardiac cycle. An average of five consecutive heart beats during the inspiratory phase was used in all measurements (the coefficient of variation was 3.1 ± 1.3% on the average).

CALCULATION

Left ventricular end-diastolic volume (LVEDV) and left ventricular end-systolic volume (LVESV) were calculated from both ventricular dimensions (LVEDD and LVESD) by the formulas described by Gibson.3

\[
\text{LVEDV} = \pi /6 \times \text{LVEDD}^2 \times (0.98 \times \text{LVEDD} + 5.90)
\]

\[
\text{LVESV} = \pi /6 \times \text{LVESD}^2 \times (1.14 \times \text{LVESD} + 4.18)
\]

where LVEDD is the left ventricular end-diastolic dimension and LVESD is the left ventricular end-systolic dimension in centimeters. Stroke volume (SV) was obtained as the difference between LVEDV and LVESV. TEE CO was calculated as the product of SV and HR. Ejection fraction (EF) was SV/LVEDV. Fractional shortening (FS) was calculated as follows:

\[
\text{FS} = (\text{LVEDD} - \text{LVESD}) / \text{LVEDD}
\]

Mean velocity of circumferential fiber shortening (mean
Vcf) was FS/LVET, where LVET represents left ventricular ejection time, which was measured from the beginning of the upstroke to the onset of the dicrotic notch in the carotid pulse tracing.

All these measured values were expressed as the mean ± standard error. Statistical analysis was performed with the use of a two-way analysis of variance. When nonrandom variance was significant (P < 0.05), multiple comparisons were made by using the Bonferroni test.⁴

Results

Reliability of TEE

Cardiac output calculated by TEE was compared with that by thermodilution technique in 33 simultaneous measurements made at ZEEP and 10 and 15 cmH₂O PEEP in 11 patients. A reasonable correlation was observed between CO obtained by the thermodilution technique and by TEE (y = 0.88 X + 0.89, r = 0.97) (fig. 2A). The linear regression line was reasonably close to the identity line. Left ventricular dimensions (LVD: LVEDD and LVESD) measured by TEE were compared with those successfully obtained by the two-dimensional precordial method in 18 simultaneous measurements made at ZEEP, 10 and 15 cmH₂O PEEP in three patients. An excellent correlation was found between LVD values measured by TEE and the two-dimensional approach (y = 0.90 X + 0.32, r = 0.95) (Fig. 2B). The linear regression line was also reasonably close to the identity line.

Left Ventricular Performance

In 13 patients, TEE recordings of the left ventricle was obtained during PEEP. All measured and derived variables of left ventricular function are summarized in table 1.

Heart Rate and Stroke Volume. Heart rate showed no statistically significant change from zero to 10 cmH₂O PEEP. However, a significant increment in HR was found from zero to 15 cmH₂O PEEP (P < 0.05), although the increment was small. On the other hand, SV markedly and significantly decreased as PEEP was increased (P < 0.05) (fig. 3A).

Cardiac Output. Cardiac output calculated by TEE significantly decreased as PEEP was increased (P < 0.05).

Left Ventricular End-diastolic Dimension. Left ventricular end-diastolic dimension at 10 cmH₂O PEEP was significantly less than that at ZEEP (P < 0.05). At 15 cmH₂O LVEDD became significantly less than at 10 cmH₂O PEEP (P < 0.05). Stroke volume decreased with a decrease in LVEDD in all patients (fig. 3B).

Left Ventricular Systolic Function. Ejection fraction and FS tended to increase from ZEEP to 10 and 15 cmH₂O PEEP. However, these increases, as a whole, were not changed. An increase also was seen in mean Vcf from ZEEP to 10 and 15 cmH₂O PEEP (P < 0.05, respectively). LVESD significantly decreased from ZEEP to 10 and 15 cmH₂O PEEP (P < 0.05, respectively). The (SBP-PEEP)/LVESD ratio was not significantly changed with PEEP, although a significant decrease was seen in SBP.
Table 1. Mean ± SE of All Measured and Derived Variables for Left Ventricular Function during Application of PEEP in 13 Patients

<table>
<thead>
<tr>
<th></th>
<th>ZEEP</th>
<th>10 cm H₂O PEEP</th>
<th>15 cm H₂O PEEP</th>
<th>ZEEP</th>
</tr>
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<tbody>
<tr>
<td>SBP (mmHg)</td>
<td>143 ± 6</td>
<td>139 ± 4</td>
<td>132 ± 6*</td>
<td>141 ± 6</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>100 ± 5</td>
<td>104 ± 5</td>
<td>108 ± 6*</td>
<td>99 ± 5</td>
</tr>
<tr>
<td>LVEDD (cm)</td>
<td>4.9 ± 0.2</td>
<td>4.5 ± 0.2*</td>
<td>4.3 ± 0.2*</td>
<td>4.9 ± 0.2</td>
</tr>
<tr>
<td>LVESD (cm)</td>
<td>3.6 ± 0.2</td>
<td>3.2 ± 0.2*</td>
<td>3.1 ± 0.2*</td>
<td>3.6 ± 0.2</td>
</tr>
<tr>
<td>LVEDV (ml)</td>
<td>158 ± 12.6</td>
<td>115.1 ± 10.2*</td>
<td>102.1 ± 10.2*†</td>
<td>157 ± 14.0</td>
</tr>
<tr>
<td>LVESV (ml)</td>
<td>59.3 ± 8.8</td>
<td>44.8 ± 6.2*</td>
<td>40.7 ± 6.5*</td>
<td>60.5 ± 10.1</td>
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<tr>
<td>LVET (s)</td>
<td>0.24 ± 0.01</td>
<td>0.22 ± 0.01*</td>
<td>0.21 ± 0.01*</td>
<td>0.24 ± 0.01</td>
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<tr>
<td>SV (TEV) (ml/beat)</td>
<td>78.7 ± 5.5</td>
<td>70.3 ± 5.5*</td>
<td>61.4 ± 5.4*†</td>
<td>77.0 ± 6.1</td>
</tr>
<tr>
<td>CO (TEE) (l/min)</td>
<td>7.8 ± 0.6</td>
<td>7.2 ± 0.6*</td>
<td>6.5 ± 0.6*†</td>
<td>7.5 ± 0.7</td>
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<tr>
<td>EF</td>
<td>0.591 ± 0.030</td>
<td>0.623 ± 0.026*</td>
<td>0.617 ± 0.031</td>
<td>0.594 ± 0.035</td>
</tr>
<tr>
<td>FS</td>
<td>0.278 ± 0.021</td>
<td>0.300 ± 0.019*</td>
<td>0.297 ± 0.024</td>
<td>0.281 ± 0.025</td>
</tr>
<tr>
<td>mVcf (circ/s)</td>
<td>1.18 ± 0.11</td>
<td>1.35 ± 0.12*</td>
<td>1.39 ± 0.15*</td>
<td>1.11 ± 0.10</td>
</tr>
<tr>
<td>ESPm/LVESD (mmHg/ml)</td>
<td>4.2 ± 0.3</td>
<td>4.3 ± 0.3</td>
<td>4.2 ± 0.4</td>
<td>4.1 ± 0.3</td>
</tr>
</tbody>
</table>

Abbreviations: ZEEP = zero end-expiratory pressure; SBP = systolic blood pressure; HR = heart rate; LVEDD = left ventricular end-diastolic dimension; LVESD = left ventricular end-systolic dimension; LVEDV = left ventricular end-diastolic volume; LVESV = left ventricular end-systolic volume; LVET = left ventricular ejection time; SV = stroke volume; CO = cardiac output; EF = ejection fraction; FS = fractional shortening; mVcf = mean velocity of circumferential fiber shortening; ESPm = observed SBP-PEEP.

* Significant difference with respect to ZEEP value (P < 0.05).
† Significant difference with respect to 10 cm H₂O PEEP value (P < 0.05).

Discussion

ADVANTAGE AND RELIABILITY OF TRANSESOPHAGEAL ECHOCARDIOGRAPHY DURING PEEP

Echocardiography is a practical and noninvasive technique for evaluation for left ventricular performance, particularly in critically ill patients. Ventricular volume measurement enables us to evaluate ventricular preload and left ventricular systolic function in a different manner from intraventricular pressure measurement.

**DIMENSIONAL CHANGES OF OTHER THREE CHAMBERS**

During this study the RVEDD was measured in six patients and RAD and LAD in seven patients by TEE.

**Right Ventricular End-diastolic Dimension.** In all six patients, RVEDD was decreased as PEEP was increased, as shown in figure 4A.

**Right Atrial End-diastolic Dimension and Left Atrial End-diastolic Dimension.** In all seven patients, both dimensions were also decreased following PEEP, as shown in figures 4B and C.

Fig. 3. Heart rate versus SV (A) and SV versus LVEDD (B). Decrease in SV was more prominent than the increment in HR. The decrease in LVEDD induced a corresponding decrease in SV during the addition of PEEP. HR = heart rate; SV = stroke volume; LVEDD = left ventricular end-diastolic dimension.
During mechanical ventilation, especially during PEEP, the feasibility of using the standard precordial echocardiography is often technically difficult because the transducer moves with the respiratory chest movement and the expanded lung interferes with the ultrasound beam. Furthermore, it is difficult to measure the maximal dimension of the right ventricle and it is impossible to measure both RAD and LAD simultaneously. By contrast, TEE enables continuous cardiac evaluation, even during PEEP. Because the TEE transducer can be placed behind the heart, the echograms of all four heart chambers are obtained without hindrance from lung tissue. Another advantage of TEE is that it can clearly measure both RAD and LAD simultaneously.\textsuperscript{1}

Matsumoto et al. reported the validity and reproducibility of TEE in evaluating left ventricular performance during open heart surgery\textsuperscript{5} and dynamic exercise.\textsuperscript{6} Our good correlations between TEE CO and thermodilution CO and between LVED by TEE and LVED by the two-dimensional method also indicate the feasibility of TEE for the evaluation of left ventricular dimensional changes even during PEEP. Therefore, we are confident that our measurements of left ventricular dimensions and calculated volumes are reasonably accurate.

The dimensional change of the right ventricle is more difficult to analyze than that of the left ventricle because of the asymmetric shape of the right ventricle. However, from the changes of the dimension alone at ZEEP and PEEP, we could detect changes in the size of right ventricle. Toma et al. reported that LAD and RAD measured from M-mode TEE correlated well with angiographic volumes during spontaneous respiration.\textsuperscript{1} We could also obtain LAD and RAD at ZEEP and PEEP. Therefore, in this study we assumed that even during PEEP the values for RVEDD and both atrial dimensions by TEE were reliable after proper manipulation of the transducer, although we did not test their reliability as for the measurements of the left ventricular dimensions.

**LEFT VENTRICULAR PERFORMANCE**

With addition of PEEP, CO is known to decrease prominently in both experimental animals\textsuperscript{7} and human beings.\textsuperscript{8} In this study, too, CO decreased markedly under PEEP. As shown in figure 3A, while HR increased only slightly, SV decreased prominently. Therefore, the decreased CO under PEEP is primarily a result of the decrease in SV.

There are two possible factors causing the decrease in SV. One is a decreased left ventricular preload and the other is a depressed left ventricular systolic function. Left ventricular end-diastolic dimension is a reasonable index of left ventricular preload. As shown in figure 3B, the relation between LVEDD and SV is proportional: a decrease in LVEDD was associated with a decrease in SV.

On the other hand, PEEP did not decrease EF and mean Vcf. This seems to reflect that left ventricular systolic function is not depressed. These variables, however, may have been affected by changes not only in left ventricular preload but also in HR. Fractional shortening is an index of left ventricular performance and is known not to be influenced by change in HR.\textsuperscript{9} Fractional shortening also indicates that left ventricular systolic function is not decreased. The end-systolic pressure/dimension ratio (ESP/LVESV) is thought to be independent of preload, linear over a wide range of afterload and sensitive to change in the left ventricular inotropic state.\textsuperscript{10} For this variable we did not directly measure the transmural left ventricular end-systolic pressure. Observed SBP was not the true left ventricular afterload during PEEP. When SBP is to be used as an approximate equivalent of the left ventricular end-systolic pressure with the pressure outside the heart, which equals the value of PEEP, the lowest possible transmural end-systolic pressure is reasonably estimated to be the difference between observed SBP and the PEEP value.\textsuperscript{11} As shown in table 1, this transmural pressure/dimension ratio (ESP\textsubscript{Tm}/LVESD) showed no statistically significant change with PEEP. Therefore, we speculate that left ventricular systolic function is not depressed by PEEP. If there had been no change in left ventricular preload, SV would have been increased by the decrease in LVESV. Therefore, we conclude that the main cause of the decreased SV is a greater decrease in left ventricular preload than the decrease in LVESV.

**DIMENSIONAL ANALYSIS OF OTHER THREE CHAMBERS**

Decreased left ventricular preload may be caused by any of several factors, including a decreased systemic venous return,\textsuperscript{12} an increased right ventricular afterload,\textsuperscript{13} and a restriction of the left ventricular filling by the leftward displacement of the interventricular septum.\textsuperscript{14} Figure 4 shows decreases in RVEDD, RAD, and LAD with PEEP. These results indicate that the preload of the three cardiac chambers was also decreased. This diminished the likelihood that the decreased left ventricular preload was caused by a increased right ventricular afterload or restriction to left ventricular filling. The remaining possibility for the decreased left ventricular preload would be a decreased systemic venous return.

In figure 5, typical echograms of the left and right atria are presented under conditions of spontaneous breathing (upper panel), ZEEP and 15 cmH\textsubscript{2}O PEEP (middle panel), and ZEEP and 10 cmH\textsubscript{2}O PEEP (lower panel). During spontaneous breathing, RAD and LAD showed rhythmic changes with the cardiac cycle. This change, however, was not influenced prominently by the respiratory cycle. At ZEEP both atrial dimensions changed with the respiratory cycle (middle and lower panels). The cyclic change
**Fig. 4.** Changes in RVEDD, RAD, and LAD during PEEP. All values at ZEEP decreased following the addition of PEEP. RVEDD = right ventricular end-diastolic dimension; RAD = right atrial end-diastolic dimension; LAD = left atrial end-diastolic dimension.

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**Fig. 5.** Left and right atrium view by TEE. During spontaneous breathing (*upper panel*), ZEEP and 15 cmH₂O PEEP (*middle panel*), and ZEEP and 10 cmH₂O PEEP (*lower panel*). LA = left atrium; IAS = interatrial septum; RA = right atrium; RAFW = right atrial free wall; ECG = electrocardiogram; PCG = phonocardiogram; E = in expiratory phase; I = in inspiratory phase.
in RAD was more prominent than that in LAD. Namely, in the inspiratory phase RAD was shortened, while in the expiratory phase it was widened. Under PEEP, however, widening of RAD was not observed in the expiratory phase, and when PEEP was eliminated, that widening was reproduced (middle panel). TEE also showed that RAD began to decrease immediately after PEEP, while LAD decreased after a short time lag (lower panel). This phenomenon is similar to the effects of Valsalva maneuver. Therefore, we conclude that the decreased right atrial filling that occurred earlier than the decreased left atrial filling was the primary cause for the reduced CO.

In summary, TEE was confirmed to be a valid and reliable technique for the evaluation of cardiac function, even during PEEP. The application of TEE may be limited for an extremely horizontal heart in which the proper view of the heart chambers cannot be recorded. However, there is no radiation exposure, and TEE can be performed at the bed side more easily than by the other methods presently available. Therefore, we conclude that TEE is a suitable technique for evaluation of cardiac function, especially in critically ill patients. Using TEE, we found that the decreased CO during PEEP was caused by a decrease in SV and was associated with a decreased left ventricular preload, which was due to a decreased right ventricular preload. On the other hand, PEEP did not depress left ventricular systolic function as assessed by EF, mean Vcf, FS, and (SBP-PEEP value)/LVESD.

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References


