Does Positive End-Expiratory Pressure Ventilation Improve Left Ventricular Function?*

A Comparative Study by Transesophageal Echocardiography in Cardiac and Noncardiac Patients

Jean-Luc Fellahi, MD; Bruno Valtier, MD; Alain Beauchet, MD; Jean-Pierre Bourdarias, MD; and François Jardin, MD

Study objectives: Positive end-expiratory pressure (PEEP) has been proposed to improve cardiac output in patients with left ventricular (LV) dysfunction. This study was designed to compare quantitative global and regional LV performance in response to PEEP in patients with normal and poor LV function.

Design: A prospective clinical trial.

Setting: Adult medical ICU in a university hospital.

Patients: Twelve critically ill patients requiring respiratory support and divided into two groups according to baseline transesophageal echocardiographic (TEE) measurements: normal LV dimensions and fractional area of contraction (FAC=61±5%) (n=7) and dilated cardiomyopathy with reduced FAC (21±1%) (n=5).

Measurements and results: All patients were studied when two successive levels of PEEP (best PEEP as the highest value of respiratory compliance and high PEEP as best PEEP+10 cm H₂O) were applied. Global systolic LV performance and quantitative regional wall motion analysis performed by the centerline method were assessed on the TEE transgastric short-axis view. End-systolic wall stress (ESWS) was used as a reliable indication of LV afterload. PEEP reduced LV dimensions asymmetrically in both groups of patients and septal diameter significantly decreased without affecting global LV systolic performance. Additionally, high PEEP produced a significant improvement in septal kinetics as evidenced by the centerline method. High PEEP also decreased ESWS for all patients (~27% in normal group and ~23% in cardiac group, p<0.05) without significant improvement in global systolic LV performance (FAC: +2% in normal group and +0% in cardiac group; not significant).

Conclusions: PEEP cannot be recommended routinely to improve LV performance in patients with severe dilated cardiomyopathy.

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Key words: cardiomyopathy; hemodynamics; PEEP ventilation; transesophageal echocardiography

Abbreviations: C=chord; CRS=total respiratory compliance; EDA=end-diastolic area; ESA=end-systolic area; ESD=end-systolic diameter; ESWS=end-systolic wall stress; FAC=fractional area of contraction; PEEP=positive end-expiratory pressure; SA=stroke area; TEE=transesophageal echocardiography; Vt=tidal volume; WT=wall thickness; ZEEP=zero end-expiratory pressure

The effects of positive end-expiratory pressure (PEEP) on heart loading conditions have been well documented for many years.1–3 By an increase in pleural pressure, PEEP produces a decrease in left ventricular preload3 and afterload.4 Because the preload effect is predominant in patients with normal left ventricular function, the result is generally a drop in cardiac output and in systemic BP. In patients with cardiac dysfunction, clinical studies5–7 have suggested that PEEP could improve rather than impair cardiac output by decreasing left ventricular afterload despite decreasing left ventricular preload, in a manner similar to systemic vasodilator therapy.8 However, no data concerning PEEP-in-
duced regional wall motion abnormalities and its consequences on global left ventricular performance are reported in these studies. Using transesophageal echocardiography (TEE), the aim of the present study was to assess quantitative global and regional left ventricular performance in response to PEEP in patients with normal and poor left ventricular function and to determine whether PEEP is of beneficial hemodynamic effect in cardiac patients.

**Materials and Methods**

**Patients**

Twelve critically ill patients (mean age, 62 years) who required continuous mechanical ventilation with PEEP for acute hypoxic respiratory failure (PaO2/fraction of inspired oxygen < 200 mm Hg) were studied. Respiratory failure resulted from extensive bacterial pneumonia in two cases, acute exacerbation of COPD in five cases, and cardiogenic pulmonary edema in five cases (Table 1). Patients were divided into two groups according to baseline left ventricular echocardiographic measurements: group 1 patients (n=7) with normal left ventricular dimensions (end-diastolic area in a range between 5 and 12 cm²/m²) and systolic function (fractional area of contraction [FAC] > 50%) and group 2 patients (n=5) with dilated cardiomyopathy and markedly reduced systolic function (Table 2). All patients with cardiogenic pulmonary edema had history of cardiac dysfunction and were in group 2. The study was in accordance with the ethical regulations of our country (Huriet's law) and approved by the Comité consultatif de Protection des Personnes dans la Recherche Biomédicale of Ambroise Paré Hospital. At the time of the study, the lungs of all patients were being ventilated with a ventilator (7200 Series; Puritan Bennett; Carlsbad, CA) that delivered a constant inspiratory flow rate and respiratory frequency of 15 cycles/min, a tidal volume (Vt) of 10 mL/kg of body weight, with an end-inspiratory pause of 0.5 s and zero end-expiratory pressure (ZEEP). Inspired oxygen concentration was maintained at 50%. During the brief period of the study, the patients were sedated with midazolam and sufentanil and paralyzed with vecuronium. No change in baseline therapy and inotropic support was authorized throughout the study period.

**Measurements**

Measurements were performed after several days of respiratory support and hemodynamic stabilization. Airway pressures were obtained from the ventilator (Puritan Bennett 7200 Series). Using a side port of the tracheal tube, airway pressures were also displayed on the M-mode tracing to record the respiratory cycle during echocardiographic study. Total respiratory compliance (CRS) was computed as follows: CRS = Vt/PPPEEP, where Vt was inspired tidal volume, Pp the end-inspiratory plateau pressure, and PEEP, the total end-expiratory pressure (ie, including intrinsic PEEP measured with the end-expiratory occlusion hold of the ventilator). End-expiratory pleural pressure was measured before TEE examination by using an esophageal balloon advanced through the nose into the esophagus 35 to 40 cm from the nares, inflated with 10 mL air, and allowed to deflate spontaneously, resulting in a residual volume of approximately 0.2 mL air. Balloon placement was considered correct when the patient was briefly disconnected from the ventilator and a negative pleural pressure was recorded.

Systemic arterial BP was measured via an indwelling radial catheter with a transducer (Transpac; Abbott Laboratories; North Chicago, IL) positioned at the midaxillary line, with atmospheric pressure as a zero reference. Transmural systolic BP, defined as the peak arterial pressure minus pleural pressure, was used to approximate to end-systolic pressure for a noninvasive left ventricular meridional end-systolic wall stress (ESWS) calculation. Heart rate was obtained from patient’s ECG monitoring.

Echocardiographic measurements were performed by a single trained investigator (HP 77020A; Hewlett-Packard; Andover, MA). A transesophageal 5-MHz single-plane probe was positioned to obtain a short-axis cross-sectional view of the left ventricle at the midpapillary muscle level. Echocardiographic images were recorded during the protocol and reviewed in a blinded fashion for single-frame, stop-motion analysis. The end-diastolic frame was selected at the peak of the R wave on simultaneous ECG recording and the end-systolic frame was defined as the smallest ventricular dimension during the last half of the T wave. Using a microcomputer interfaced with the videotape player, end-expiratory stop-motion frames at end-diastole and end-systole were displayed on the microcomputer screen to digitize the endocardial outlines of the left ventricle. End-diastolic areas (EDA) and end-systolic areas (ESA) were automatically processed. Left ventricular anteroposterior and septolateral diameters at end-diastole were also computed during area measurements. The stroke area (SA) was calculated as EDA-ESA and the FAC was calculated as SA/EDA. FAC provided global assessment of left ventricular systolic performance as the net result of the integration of cardiac inotropic state and loading conditions. Left ventricular end-systolic wall thickness (WT) was measured by the leading edge-to-leading edge technique via M-mode recording. ESWS was calculated using the formula of Reichek et al.9

ESWS = 0.334 (transmural systolic BP/ESD)/WT/(1+WT/ESD) when ESD was left ventricular end-systolic diameter. Since some change in left ventricular geometry was expected to occur during the study, ESD was computed from measured ESA as an average value. ESWS provided a reliable indication of left ventricular afterload by reflecting the combined effects of peripheral loading conditions and intrinsic cardiac properties.10 Regional wall function was assessed using the centerline method described by Sheehan et al and usually applied to contrast ventriculography: regional wall motion was measured by the

**Table 1—Clinical Data and Outcome of Patients in Both Groups**

<table>
<thead>
<tr>
<th>Patient No./Age, yr/Sex</th>
<th>Diagnosis</th>
<th>Hemodynamic Therapy</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/76/M COPD</td>
<td>–</td>
<td>Nsurv</td>
<td></td>
</tr>
<tr>
<td>2/69/M COPD</td>
<td>–</td>
<td>Nsurv</td>
<td></td>
</tr>
<tr>
<td>3/69/M COPD</td>
<td>–</td>
<td>Nsurv</td>
<td></td>
</tr>
<tr>
<td>4/82/M COPD</td>
<td>Dopa=7 μg/kg/min</td>
<td>Nsurv</td>
<td></td>
</tr>
<tr>
<td>5/34/M Pneumonia</td>
<td>Dopa=5 μg/kg/min</td>
<td>Surv</td>
<td></td>
</tr>
<tr>
<td>6/40/M Pneumonia</td>
<td>–</td>
<td>Surv</td>
<td></td>
</tr>
<tr>
<td>7/61/F COPD</td>
<td>–</td>
<td>Surv</td>
<td></td>
</tr>
<tr>
<td>Group 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/60/M CPE</td>
<td>Doba=15 μg/kg/min</td>
<td>Nsurv</td>
<td></td>
</tr>
<tr>
<td>2/59/M CPE</td>
<td>Doba=12 μg/kg/min</td>
<td>Surv</td>
<td></td>
</tr>
<tr>
<td>3/61/M CPE</td>
<td>Doba=12 μg/kg/min</td>
<td>Nsurv</td>
<td></td>
</tr>
<tr>
<td>4/73/F CPE</td>
<td>–</td>
<td>Surv</td>
<td></td>
</tr>
<tr>
<td>5/67/M CPE</td>
<td>Doba=6 μg/kg/min</td>
<td>Nsurv</td>
<td></td>
</tr>
</tbody>
</table>

*CPE=cardiogenic pulmonary edema; Doba=dopamine; Doba=dobutamine; Surv=survivor; Nsurv=non survivor.
Table 2—Global Hemodynamic Parameters and Echocardiographic Data at Baseline (ZEEP) and Changes During PEEP Challenge for Both Groups of Patients*  

<table>
<thead>
<tr>
<th>Parameter</th>
<th>ZEEP</th>
<th>Best PEEP</th>
<th>High PEEP</th>
<th>ZEEP</th>
<th>Difference Between Groups: p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP, mm Hg</td>
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<td></td>
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<tr>
<td>Group 1</td>
<td>140±5</td>
<td>133±5</td>
<td>119±5‡</td>
<td>138±5</td>
<td>NS</td>
</tr>
<tr>
<td>Group 2</td>
<td>130±9</td>
<td>123±9</td>
<td>110±9‡</td>
<td>129±9</td>
<td></td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Group 1</td>
<td>66±9</td>
<td>73±9</td>
<td>68±9</td>
<td>70±9</td>
<td>NS</td>
</tr>
<tr>
<td>Group 2</td>
<td>66±4</td>
<td>65±4</td>
<td>62±4</td>
<td>64±4</td>
<td></td>
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<tr>
<td>HR, beats/min</td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Group 1</td>
<td>103±7</td>
<td>105±7</td>
<td>106±7</td>
<td>105±7</td>
<td>NS</td>
</tr>
<tr>
<td>Group 2</td>
<td>102±10</td>
<td>103±10</td>
<td>104±10</td>
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<td></td>
</tr>
<tr>
<td>EDA, cm²/m²</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 1</td>
<td>10.6±1.2</td>
<td>8.7±1.3‡</td>
<td>7.9±1.3‡</td>
<td>10.3±1.1</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Group 2</td>
<td>24.6±3.4</td>
<td>23.2±3</td>
<td>20.4±3.8‡</td>
<td>24.2±3.2</td>
<td></td>
</tr>
<tr>
<td>ESA, cm²/m²</td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Group 1</td>
<td>4.0±0.4</td>
<td>3.6±0.5</td>
<td>2.7±0.4‡</td>
<td>3.6±0.4</td>
<td>NS</td>
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<tr>
<td>Group 2</td>
<td>19.4±2.7</td>
<td>18.4±2.5</td>
<td>16.2±3.2‡</td>
<td>19.0±2.5</td>
<td></td>
</tr>
<tr>
<td>SA, cm²/m²</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 1</td>
<td>6.6±1.0</td>
<td>5.2±0.9‡</td>
<td>5.2±1.0‡</td>
<td>6.7±1.0</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Group 2</td>
<td>5.2±0.8</td>
<td>4.8±0.7</td>
<td>4.2±0.7‡</td>
<td>5.2±0.7</td>
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<tr>
<td>FAC, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Group 1</td>
<td>61±5</td>
<td>58±4</td>
<td>63±4</td>
<td>63±5</td>
<td>NS</td>
</tr>
<tr>
<td>Group 2</td>
<td>21±1</td>
<td>21±2</td>
<td>21±1</td>
<td>22±2</td>
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</tr>
<tr>
<td>ESWS, kdyn/cm²/m²</td>
<td></td>
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</tr>
<tr>
<td>Group 1</td>
<td>33±4</td>
<td>34±4</td>
<td>24±4‡</td>
<td>33±4</td>
<td>NS</td>
</tr>
<tr>
<td>Group 2</td>
<td>96±14</td>
<td>88±14</td>
<td>74±14†</td>
<td>93±14</td>
<td></td>
</tr>
</tbody>
</table>

*SBP=systolic BP; DBP=diastolic BP; HR=heart rate; NS=nonsignificant. Data are expressed as mean±SEM.  
†p<0.05 vs ZEEP.

computer along 100 chords (c) constructed perpendicular to a centerline drawn midway between the end-diastolic and end-systolic contours on the short-axis view and expressed in terms of absolute displacement during systole normalized for diastolic dimension. With this method, performed clockwise on a short-axis view of the left ventricle obtained by a transthoracic approach, c90 to c10 approximately involve posterior wall, c10 to c35 lateral wall, c35 to c60 anterior wall, and c60 to c90 interventricular septum.

Protocol

After completion of a baseline set of measurements with ZEEP for each patient (No. 1), the best PEEP level, defined as PEEP providing the highest value of CRS according to the criteria of Suter et al.,12 was applied for 5 min and measurements were repeated (No. 2). Then a supplementary PEEP of 10 cm H₂O was added to the best PEEP, giving the high PEEP level, and measurements were repeated after 5 min (No. 3). These short stabilizing phases were sufficient because cardiovascular changes occur within seconds of PEEP application.13 Finally, ZEEP was restored and a second set of baseline measurements obtained (No. 4).

Statistical Analysis

Statistical significance was tested using statistical software (Statgraphics version 5.0, Univeware, Paris, France). Data are expressed as mean±1 SEM. Comparisons of means at each level of PEEP (No. 2 and 3) with the average of baseline ZEEP measurements (No. 1 and 4) within and between groups were performed using a two-way analysis of variance for repeated measurements, completed in case of significance by multiple comparisons tests using the contrast method. A test giving a p value of <0.05 was considered statistically significant.

RESULTS

Clinical data and outcome in both groups of patients are indicated in Table 1. Baseline left ventricular two-dimensional echocardiographic data (short-axis view) in both groups are indicated in Table 2 (ZEEP column). Whereas patients in group 1 had left ventricular dimensions and FAC in a normal range, patients in group 2 had a dilated left ventricle with reduced FAC.

The best PEEP level for the whole group was 9.6±0.2 cm H₂O, resulting in an average increase in pleural pressure of 3.3±0.2 mm Hg (from −2.4±0.2 to 0.9±0.5 mm Hg, p<0.05). The high PEEP level for the whole group was 19.6±0.2 cm H₂O and induced an average increase in pleural pressure of 5.6±0.2 mm Hg when compared with ZEEP (3.2±0. 8 vs −2.4±0.2 mm Hg, p<0.05). Barotrauma was not observed in any patient in the study. As a prerequisite, CRS significantly improved at the
first level of PEEP (51±7 vs 44±7 mL/cm H₂O, p<0.05). However, with a higher level of PEEP, CBS was significantly impaired (28±3 vs 44±7 mL/cm H₂O, p<0.05).

The hemodynamic effects of the two successive levels of PEEP (best PEEP and high PEEP as defined earlier) for the two groups of patients are indicated in Table 2. Systolic BP was reduced by PEEP with a significant drop at the highest level in both groups, whereas diastolic BP was unaffected. Heart rate remained unchanged during the study. In group 1, PEEP caused a reduction in diastolic dimensions and in SA, which was already significant at the best PEEP level. This reduction was associated with a significant decrease in ESA at the high PEEP level. Similar behavior in EDA, ESA, and SA during high PEEP was observed in group 2. FAC was not affected by PEEP in both groups. In addition, calculated ESWS was significantly reduced at the highest level of PEEP in both groups of patients.

Analysis of left ventricular segmental wall motion for the whole group during PEEP is represented in Figure 1. As evidenced in this figure, a significant impairment in septal (c60 to c90) kinetics was produced by the high PEEP level. Additionally, the reduction in left ventricular diastolic dimensions by PEEP was mainly produced by a reduction in septolateral diameter, whereas anteroposterior diameter was not significantly affected, as illustrated in Figure 2. An example of the left ventricular distortion caused by PEEP and a prominent decrease in septolateral diameter with relative constancy of anteroposterior diameter in a patient with normal left ventricular dimensions (group 1) is shown in Figure 3.

**Discussion**

Changes in pleural pressure have been shown to influence left ventricular afterload. Indeed, a nega-
tive pleural pressure increases the left ventricular afterload\textsuperscript{14,15} while a positive pleural pressure decreases it.\textsuperscript{4,10} In the present study, the increase in pleural pressure induced by two successive levels of PEEP was slight and probably trivial in terms of afterload changes in patients with normal left ventricular function. However, it might be significant in cardiac patients because a dilated left ventricle is more sensitive to small afterload changes.\textsuperscript{15} Unfortunately, despite a documented reduction in left ventricular ESWS (a more reliable index of left ventricular afterload than systolic vascular resistance index\textsuperscript{10}) when the highest PEEP level was used, the present study failed to demonstrate any improvement in global left ventricular systolic performance in both groups of patients in response to an increase in pleural pressure by 5.6 mm Hg on average. One can doubt that such a small increase in pleural pressure might actually affect left ventricular function.\textsuperscript{17} Nevertheless, this finding is somewhat different from those reported in previous studies. Indeed, a decrease in left ventricular afterload is expected to improve left ventricular function in patients with severe cardiac failure.\textsuperscript{5,7} Moreover, Grace and Greenbaum\textsuperscript{6} and Mathru et al\textsuperscript{7} used a low level of PEEP (5 cm H\textsubscript{2}O) to improve cardiac output. Even if no data concerning PEEP-related pleural pressure changes were available, we can assume that they were minimal. In the absence of an accurate estimation of left ventricular afterload, the beneficial effects of low levels of PEEP on cardiac performance reported in these two studies could be attributed to a mechanical compression of the heart by the lungs in the cardiac fossa rather than a reduction in left ventricular ejection pressure, as Pinsky\textsuperscript{18} suggested recently. However, this assumption has not yet been proved.

Because the best PEEP level was unable to decrease left ventricular afterload in our patients, we applied a level of PEEP as high as 20 cm H\textsubscript{2}O. Despite this, we did not find any improvement in global left ventricular performance in cardiac patients. This result could be explained by the detrimental effect of high PEEP on septal kinetics. Indeed, PEEP reduced left ventricular diastolic dimensions asymmetrically in both groups of patients. Septal displacement decreased left ventricular septolateral diameter whereas anteroposterior diameter remained constant. This phenomenon was evidenced in previous studies by our group that documented via combined thermodilution and transthoracic two-dimensional echocardiography a leftward shift in the interventricular septum with a simultaneous increase in right ventricular afterload resulting in depressed right systolic function when a high level of PEEP was used.\textsuperscript{3,19} In addition, we clearly demonstrated in the

![Figure 3](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21811/)

**Figure 3.** An illustrative example of left ventricular deformation observed in a patient with normal left ventricular dimensions (group 1) during PEEP challenge by a transgastric approach in short-axis view, with end-diastolic (top) and end-systolic (bottom) frames. From left to right: baseline ZEEP, best PEEP level, high PEEP level, and return to ZEEP.
present study that PEEP induced a true septal dysfunction by quantitative assessment of left ventricular regional wall kinetics. The use of a biplane or omniplane probe providing both midpapillary long-axis and short-axis views would be useful to slightly improve the sensitivity for analysis of ventricular wall motion, but it is unlikely that it would markedly alter the major finding of the study. Regional wall motion abnormalities could counterbalance the decrease in left ventricular afterload and prevent any improvement in global left ventricular systolic performance when a high level of PEEP is used in cardiac patients. This result also emphasizes the role played by ventricular interdependence in PEEP-related left ventricular filling reduction.

The consequences of PEEP on left ventricular preload have been well elucidated in the past by experimental1,2 and clinical3 studies. PEEP reduces left ventricular preload5,20,21 and this reduction appears mediated by a complex mechanism, associating a decrease in systemic venous return1,21 and an increase in right ventricular afterload.2 Furthermore, Takata and Robotham22 suggested, in an experimental study, that the lungs could exert a direct left atrial and ventricular external constraint during PEEP and thus contribute to the reduction in left ventricular stroke output. However, the same clinical studies1,20 ruled out any detrimental effect of PEEP on left ventricular contractility, as previously suggested on the basis of experimental data.23 The present study confirmed these results: PEEP reduced left ventricular preload and SA in both groups of patients and global systolic left ventricular performance was not affected. A significant difference, however, was evidenced between groups. At the best PEEP level, preload was reduced in group 1 and unchanged in group 2. When a dilated cardiomyopathy is present, the left ventricle at end-diastole acts on the vertical part of its pressure/volume relationship. Thus, a substantial reduction in left ventricular filling pressure, in response to PEEP, is associated with only a small decrease in left ventricular diastolic dimensions. Moreover, we previously demonstrated that the cyclic increase in airway pressure during intermittent positive pressure breathing produced a cyclic increase in left ventricular end-diastolic dimensions, presumably caused by a mechanical squeeze of blood present in the capillary bed.24 This concept was later corroborated by experimental studies conducted by Versprille et al.25 Thus, in a patient with a dilated left ventricle and congested pulmonary capillary bed, a sustained increase in distal airway pressure by PEEP may well displace blood from the capillary bed toward the left ventricular cavity, precluding a significant preload reduction in response to the best PEEP level.

Obviously, it was debatable to increase PEEP instantaneously by 10 cm H2O above optimal PEEP without fluid administration or additional inotropic support. Nevertheless, these therapies are not without side effects in cardiac patients and no global hemodynamic untoward effects occurred during the application of the high PEEP level. Finally, our study emphasizes the inability of PEEP to improve cardiac performance in patients with poor left ventricular function solely by the reduction in left ventricular ejection pressure.

In conclusion, PEEP reduces left ventricular preload in mechanically ventilated patients with normal and poor cardiac function. Only a high level of PEEP, not used routinely, can significantly reduce left ventricular afterload by an increase in pleural pressure. This high PEEP level also induces a decrease in SA and a detrimental effect on septal kinetics that probably offsets the beneficial effect of afterload reduction expected on global systolic performance in cardiac patients. We postulate that PEEP cannot be recommended routinely for its favorable effects on left ventricular afterload in patients with dilated cardiomyopathy and poor left ventricular function.

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