Influence of Positive End-Expiratory Pressure on Right and Left Ventricular Performance Assessed by Doppler Two-Dimensional Echocardiography

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The advent of modern Doppler two-dimensional ultrasound technology has overcome the need of invasive measurements of several important cardiac parameters. It allows estimation of preload, contractility, and afterload. Positive end-expiratory pressure (PEEP) is associated with a reduction in cardiac output. The responsible mechanisms are controversial. To evaluate the cardiovascular responses to PEEP, we employed different Doppler hemodynamic indices for the first time, combined with conventional two-dimensional echocardiography. Twenty-one healthy, young, and unsedated volunteers were admitted to the study. Under spontaneous respiration, PEEP level was increased stepwise (0, 5, 7.5, 10, 12.5 cm H2O). At each PEEP level, the following right and left ventricular parameters were assessed with Doppler two-dimensional echocardiography: two-dimensional variables: end-diastolic volume indices (EDVI), ejection fraction (EF), and left ventricular afterload - LaPlace relation (combined with cuff systolic pressure); Doppler variables: cardiac index (CI) (combined with two-dimensional measure of valve area), maximum velocity (Vmax), time velocity integral (TVI), acceleration time (AT), deceleration time (DT), deceleration rate (DR), ratio of early to atrial peak (E/A), ratio of isovolumic contraction time to ejection time (IVCT/ET), and maximum blood acceleration (dv/dt) in aorta and main pulmonary artery. Increasing PEEP resulted in a proportional decrease in biventricular EDVI. Moreover, PEEP application is also causing a drop of CI, which is determined from a decrease in Vmax and TVI, while EF, IVCT/ET, dv/dt, Doppler transatrioventricular parameters, and afterload stay in normal ranges. Employing Doppler hemodynamic indices for the first time in this study setting clearly supports data that the drop in EDVI and CI during PEEP is caused by reduction in ventricular filling due to decreased venous return. Using the Doppler parameters IVCT/ET and dv/dt, changes in myocardial contractility, as well as changes in afterload (LaPlace relation) can be ruled out.

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Since the introduction of controlled ventilation with positive end-expiratory pressure (PEEP), it has been widely recognized that an increase in intrathoracic pressure is associated with a decrease in cardiac output. One of the mechanisms behind this is a reduction of venous return1-4 in accordance with Starling’s law. Other mechanisms remain the subject of considerable debate. It has also been suggested that the reduction in cardiac output may be caused by an increase in right ventricular afterload,5 reflexly mediated depression of cardiac function with increased systemic afterload,6 or by altered myocardial metabolism.7 Furthermore, Jardin et al,8,9 using two-dimensional echocardiography during mechanical ventilation with PEEP, demonstrated a decreasing radius of interventricular septum in diastole leading to its leftward shift with impeding filling of the left ventricle. Nevertheless, the study of the mechanisms for decrease in cardiac output by PEEP application has been limited by the methods available to date.

We anticipated that new Doppler parameters would be very useful for examination of this problem. The goal of the present study was to investigate the impact of different PEEP levels on right and left ventricular function and hemodynamics using precordial two-dimensional echocardiography. Also, for the first time in such a setting, we wanted to investigate the Doppler parameters in an attempt to clarify some of the mechanism(s) responsible for the drop in cardiac output in healthy, awake volunteers.

Material and Methods

After approval by the ethics committee and receiving informed consent, 21 healthy volunteers, 12 men and 7 women, aged from 21 to 39 years (mean age, 29.2) were admitted to the study. The subjects were instructed to breathe with a tightly attached face mask (Rüsch) using a high flow (40 L/min) CPAP system (Dräger CF 800), that recorded airway pressure and tidal volume continuously. Heart rate (beats/min), end-tidal CO2 (mm Hg), and

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AT=acceleration time; CI=cardiac index; DR=deceleration rate; DT=deceleration time; dv/dt=maximum acceleration of the blood; E/A=ratio of early to atrial peak; EDVI=end-diastolic volume index; IVCT/ET=isovolumic contraction time/ejection time; LV=left ventricle; PEEP=positive end-expiratory pressure; RV=right ventricle; TVI=time velocity integral; Vmax=maximum velocity

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oxygen saturation (mm Hg) were monitored continuously (Siemens-Sirecust 961). Blood pressure was recorded with an electronic oscillometer (Dinmap).

All subjects could maintain a tidal volume of 10 to 12 ml/kg at a respiratory rate between 12 and 14/min. Hemodynamic study was carried out at basal conditions and after incremental increase of PEEP levels: 5, 7.5, 10, and 12.5 cm H₂O with 15 min between each set-up.

Studies were performed by means of an ultrasound system (Hewlett-Packard) combining a two-dimensional phased-array sector scanner for imaging with a pulsed Doppler spectrum analyzer for velocity measurements. This apparatus has a software package for computerizing all investigated parameters. A 2.5-MHz transducer was used for imaging and velocity recording. Continuous videorecorded images were played back through videocassette system equipment. The frozen images were recorded on a glossy black and white paper at a speed of 100 mm/s. Patients were examined in the left semilateral decubitus position and standard transthoracic echocardiographic planes were used.

**Echocardiographic Image Acquisition**

Assessment of overall ventricular performance using two-dimensional echocardiography (end-diastolic volume, ejection fraction, and afterload) is based on geometric assumption of chamber size and shape. In contrast, Doppler echocardiography provides ventricular output data without a need for chamber geometric assumption. Estimation of cardiac output by Doppler technique requires knowledge of the cross-sectional area of the valve through which blood is flowing and the linear velocity of flow. The transaortic ventricular Doppler velocities were monitored by means of the apical four-chamber view. Calculation of effective mitral and especially tricuspid valve flow area was relatively complex due to the normal variation in size of the atrioventricular orifices during diastole. Whenever possible, they were also imaged in the parasternal position in addition to four chamber and diameter was averaged (D apical+D parasternal)/2. Measurements were taken from the inner edge of the lateral bright corner of the anulus to the inner edge of the medial corner just below insertion of the leaflets. Measurements from a minimum of three consecutive cardiac cycles were averaged. In the Doppler mode, the flow profile with the highest apparent velocities of mitral and tricuspid valves was recorded. It is assumed that the Doppler ultrasound beam is directed parallel to transatrio-ventricular blood flow in the apical four-chamber position (for incident angles less than 10°, the error will be less than 3 percent). Respiratory changes in cardiac position by PEEP application were minimal (similar in magnitude to the normal inspiration), confirmed by lack of any significant short axis rotation during a respiratory cycle, as judged by papillary muscle position.

**Doppler Two-Dimensional Echocardiographic Analysis**

1. End-diastolic volume index (EDVI) was estimated as end-diastolic chamber volume identified by the peak of the R wave of the ECG and expressed per body surface area (BSA) in milliliters per square meter.

2. Cardiac index (CI)\(^{10}\) = \(\frac{CSA \times TVI}{BSA} \times \frac{L/min}{m^2}\), where CSA is two-dimensional cross-sectional area of the interrogated valve in square centimeters, TVI is the Doppler time velocity integral of the same valve in centimeters.

3. Ejection fraction (EF) = \(\frac{EDV-ESV}{EDV}\), where EDV is ventricular end-diastolic volume and ESV is ventricular end-systolic volume corresponded to the image with smallest area following end-diastole.

4. Ratio IVCT/ET was obtained from continuous-wave Doppler by aiming the Doppler beam at an intermediate position between inflow and outflow, permitting recording of both atrioventricular and semilunar signals simultaneously. Thus, isovolumic contraction time (IVCT) is the time interval between the atrioventricular closing click and semilunar opening click on Doppler spectrum. Ejection time (ET) is the time interval laps between aortic opening click and aortic closing click on Doppler display (Fig 1). Because IVCT/ET is relatively independent from loading conditions and heart rate, this index is reliable for assessing myocardial contractility noninvasively.

5. Vmax is the maximal velocity of the Doppler transatrioventricular blood flow in centimeters per second.

6. Time velocity integral (TVI) is the area under the velocity curve for the entire diastolic period of the Doppler signal in centimeters.

7. Acceleration time (AT) and deceleration time (DT)\(^{11,12}\) represent the time intervals of the early rapid filling phase (E) of the transaortic inflow velocity (Fig 1) in milliseconds. These time intervals are corrected for heart rate according to

![Figure 1](https://example.com/figure1.png)
### Table 1—Results of Hemodynamic Variables (Mean ± SD) at Different Levels of PEEP*

<table>
<thead>
<tr>
<th>PEEP, cm H₂O</th>
<th>0</th>
<th>5</th>
<th>7.5</th>
<th>10</th>
<th>12.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right ventricle</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR</td>
<td>75 ± 11</td>
<td>73 ± 11</td>
<td>71 ± 18</td>
<td>74 ± 11</td>
<td>73 ± 12</td>
</tr>
<tr>
<td>BP syst</td>
<td>114 ± 15</td>
<td>110 ± 31</td>
<td>118 ± 13</td>
<td>115 ± 13</td>
<td>118 ± 16</td>
</tr>
<tr>
<td>BP diast</td>
<td>73 ± 11</td>
<td>74 ± 13</td>
<td>72 ± 13</td>
<td>72 ± 15</td>
<td>73 ± 14</td>
</tr>
<tr>
<td>EDVI</td>
<td>60.9 ± 11.2</td>
<td>56.7 ± 11.7</td>
<td>51.3 ± 11.3</td>
<td>45.6 ± 10.9</td>
<td>41.6 ± 9.8</td>
</tr>
<tr>
<td>CI</td>
<td>2.9 ± 0.55</td>
<td>2.7 ± 0.55</td>
<td>2.5 ± 0.52</td>
<td>2.3 ± 0.49</td>
<td>2.0 ± 0.47</td>
</tr>
<tr>
<td>EF</td>
<td>0.64 ± 0.07</td>
<td>0.62 ± 0.08</td>
<td>0.59 ± 0.09</td>
<td>0.57 ± 0.1</td>
<td>0.57 ± 0.1</td>
</tr>
<tr>
<td>Vmax</td>
<td>0.67 ± 0.12</td>
<td>0.61 ± 0.12</td>
<td>0.51 ± 0.13</td>
<td>0.44 ± 0.12</td>
<td>0.39 ± 0.12</td>
</tr>
<tr>
<td>TVI</td>
<td>18.9 ± 2.5</td>
<td>17.5 ± 2.8</td>
<td>16.3 ± 2.8</td>
<td>14.8 ± 2.8</td>
<td>13.5 ± 3.3</td>
</tr>
<tr>
<td>E/A</td>
<td>1.67 ± 0.12</td>
<td>1.64 ± 0.11</td>
<td>1.66 ± 0.11</td>
<td>1.65 ± 0.12</td>
<td>1.64 ± 0.11</td>
</tr>
<tr>
<td>AT</td>
<td>77.3 ± 10.3</td>
<td>80.0 ± 9.6</td>
<td>79.5 ± 10.3</td>
<td>81.1 ± 10.5</td>
<td>78.5 ± 11.7</td>
</tr>
<tr>
<td>DT</td>
<td>148.5 ± 31.6</td>
<td>149.6 ± 30.2</td>
<td>152.9 ± 34.2</td>
<td>153.8 ± 31.3</td>
<td>153.7 ± 29.6</td>
</tr>
<tr>
<td>DR</td>
<td>4.4 ± 0.8</td>
<td>4.4 ± 1.0</td>
<td>4.5 ± 0.9</td>
<td>4.5 ± 0.9</td>
<td>4.6 ± 0.9</td>
</tr>
<tr>
<td>IVCT/ET</td>
<td>0.25 ± 0.03</td>
<td>0.25 ± 0.04</td>
<td>0.26 ± 0.03</td>
<td>0.26 ± 0.04</td>
<td>0.26 ± 0.03</td>
</tr>
<tr>
<td>Left ventricle</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EDVI</td>
<td>62.5 ± 11.1</td>
<td>59.3 ± 12.5</td>
<td>52.9 ± 12.8</td>
<td>46.9 ± 12.5</td>
<td>43.7 ± 12.3</td>
</tr>
<tr>
<td>CI</td>
<td>2.94 ± 0.52</td>
<td>2.71 ± 0.55</td>
<td>2.53 ± 0.54</td>
<td>2.29 ± 0.54</td>
<td>2.15 ± 0.51</td>
</tr>
<tr>
<td>EF</td>
<td>0.64 ± 0.06</td>
<td>0.65 ± 0.08</td>
<td>0.63 ± 0.08</td>
<td>0.60 ± 0.10</td>
<td>0.59 ± 0.11</td>
</tr>
<tr>
<td>Vmax</td>
<td>0.75 ± 0.21</td>
<td>0.70 ± 0.20</td>
<td>0.61 ± 0.17</td>
<td>0.51 ± 0.15</td>
<td>0.50 ± 0.13</td>
</tr>
<tr>
<td>TVI</td>
<td>19.3 ± 2.9</td>
<td>17.5 ± 3.11</td>
<td>16.3 ± 3.41</td>
<td>14.7 ± 3.71</td>
<td>13.6 ± 3.65</td>
</tr>
<tr>
<td>E/A</td>
<td>1.6 ± 0.2</td>
<td>1.6 ± 0.2</td>
<td>1.6 ± 0.14</td>
<td>1.5 ± 0.1</td>
<td>1.6 ± 0.1</td>
</tr>
<tr>
<td>AT</td>
<td>83.4 ± 23.1</td>
<td>84.4 ± 22.8</td>
<td>84.6 ± 20.5</td>
<td>87.3 ± 24.6</td>
<td>87.7 ± 19.7</td>
</tr>
<tr>
<td>DT</td>
<td>151.8 ± 34.0</td>
<td>155.2 ± 35.2</td>
<td>154.8 ± 36.2</td>
<td>154.1 ± 33.2</td>
<td>157.0 ± 32.3</td>
</tr>
<tr>
<td>DR</td>
<td>4.2 ± 0.9</td>
<td>4.3 ± 0.9</td>
<td>4.3 ± 0.8</td>
<td>4.3 ± 0.9</td>
<td>4.4 ± 0.9</td>
</tr>
<tr>
<td>IVCT/ET</td>
<td>0.25 ± 0.03</td>
<td>0.28 ± 0.04</td>
<td>0.26 ± 0.04</td>
<td>0.27 ± 0.04</td>
<td>0.28 ± 0.04</td>
</tr>
<tr>
<td>dv/dt</td>
<td>17.6 ± 2.9</td>
<td>17.6 ± 3.0</td>
<td>17.6 ± 3.0</td>
<td>17.6 ± 3.2</td>
<td>17.6 ± 2.7</td>
</tr>
<tr>
<td>LaPlace</td>
<td>158.5 ± 38.1</td>
<td>163.4 ± 40.6</td>
<td>166.9 ± 39.9</td>
<td>169.7 ± 37.6</td>
<td>174.0 ± 39.4</td>
</tr>
</tbody>
</table>

EDVI=end-diastolic volume index (mL/m²); CI=cardiac index (L/min/m²); EF=ejection fraction; Vmax=maximum velocity of transatrioventricular inflow (m/s); TVI*time velocity integral of transatrioventricular inflow (cm); E/A=ratio of early rapid filling peak to atrial peak filling of transatrioventricular inflow; AT=acceleration time of peak E (ms); DT=deceleration time of peak A (ms); DR=deceleration rate of peak A (cm/s²); IVCT/ET=ratio of isovolumic contraction time to ejection time; dv/dt=maximum acceleration of the blood after semilunar valve (m/s²) and LaPlace relation (dyn-cm²).

1p<0.05.
2p<0.01.
3p<0.005.
4p<0.001 for comparison with end-expiratory pressure (PEEP)=0 cm H₂O.

Bazett’s formula:

\[
\text{corrected interval} = \frac{\text{measured interval}}{\sqrt{R-R}}
\]

8. Deceleration rate (DR)=10.12 of E was measured as the slope of a straight line drawn through the peak of E in centimeters per square second (Fig 1).

9. Ratio E/A represents the quotient of early (E) to atrial (A) peak of transatrioventricular Doppler display (Fig 1).

10. Afterload was calculated as LaPlace relation:

\[
\sigma = 0.334 \times \text{P} \times \text{EDSD} / \text{h} \times (2 \text{h} / \text{EDSD})
\]

where P is cuff systolic arterial pressure in dynes (mm Hg·0.334=10³ dynes), ESD is the end-systolic left ventricular (LV) dimension in centimeters, and h is the systolic chamber wall thickness represented by the average of the anterior and posterior measurements in centimeters, σ is measured in 10³ dyn-cm².

11. Maximum acceleration of the blood (dv/dt) in meters per square second in the aorta and pulmonary artery,11,14,15 was obtained by means of electronic differentiation of Doppler flow profile across the semilunar valve. Maximum blood flow acceleration is independent from preload.

Statistical Analysis

Values are mean±standard deviation. Data obtained at 0 PEEP were compared with data obtained at any other level of PEEP. Statistical analysis was performed using the two-way analysis of variance test and standard least-square linear regression analysis. A p value <0.05 was considered significant.

RESULTS

Table 1 summarizes the baseline hemodynamic data and during application of different PEEP levels. For each stepwise increase in PEEP, EDVI, CI, Vmax, and TVI decreased proportionally. The ventricular response to PEEP calculating a right-left ventricular end-diastolic ratio stayed constant throughout the study period. Changes in CI are reflected by diminution in TVI, since heart rate is not changed significantly. In the normal inspiratory phase, Doppler TVI, as well as Vmax, were decreased, while in the expiratory phase they were increased. Under PEEP, however, augmentation of TVI and Vmax were not observed in the expiratory...
phase, and when PEEP was eliminated, that augmentations were reproduced. We also observed that RV TVI and Vmax began to decrease immediately after PEEP, while the same LV parameters did so after a short time lag. An illustrative example of Doppler two-dimensional changes at baseline and after application of PEEP 12.5 cm H2O are given in Figure 2. The relationship between the levels of PEEP and RV EDVI are given in Figure 3 and between PEEP and RV CI in Figure 4. Each data point represents the mean ± SD.

Table 2 shows the correlation coefficients calculated by means of linear regression analysis using PEEP levels as independent variables and two-dimensional Doppler echocardiographic indices as dependent variables. The highest degree of correlations was found among Vmax, TV1, EDVI, and CI on one hand and PEEP at 10 and 12.5 cm H2O. Contractility indexes, which are independent from loading conditions, such as IVCT/ET and dv/dt, stay
ventricular end-diastolic volume indices seems to be the most important factor in drop of CI. The effect of PEEP is a reduction in venous return produced by an increase in intrathoracic and pleural pressures which decrease the pressure gradient for systemic venous return.\(^1\) This effect is expected to reduce first RV preload and then as a result, LV preload. Furthermore, we separately assessed the ventricular response to PEEP calculating a right-left ventricular end-diastolic ratio, which remained constant throughout the study period, indicating absence of ventricular interdependence. Moreover, we did not find an increase in radius of interventricular septal curvature on two-dimensional echocardiography which is contrary to with findings of Jardin et al.\(^8,9\) The same authors also suggested that PEEP shifts the interventricular septum leftwards in diastole and thus restricts left ventricular filling.

Kooren et al.\(^2\) using transesophageal echocardiography, did not observe a leftward septal displacement during PEEP. In addition, we did not find a difference in filling pattern of right and left ventricles, since Doppler transatrioventricular flow parameters Vmax and TVI gradually and in parallel decreased in both ventricles with the increase of PEEP.

Another possibility for reduction of CI is that right and left ventricular pressure volume characteristics are altered as by increased stiffness.\(^18\) The results of our trial deny this hypothesis, because Doppler transatrioventricular parameters of both ventricles, reflecting myocardial distensibility (AT, DT and especially DR) stay in normal ranges during PEEP. Left ventricular contractile state defined as a Doppler echocardiographic ratio of time intervals IVCT/ET and Doppler flow acceleration dv/dt in the aorta and pulmonary artery was not changed during PEEP, a finding consistent with others using systolic time intervals\(^19\) or systolic function parameter dp/dt/DT.\(^20\)

Some authors have reported that overdistending the lung will cause the fall in contractile ventricular properties through involving cardiopulmonary mechanoreceptors with vagal afferents.\(^6,21\) Other investigators,\(^22\) however, have demonstrated that bilateral cervical vagotomy does not alter hemodynamic response to PEEP. Tittley et al.\(^7\) reported altered myocardial metabolism with effect on LV by normal RV function. Changes in CI in our experiments appeared one ventilatory cycle after PEEP was started or stopped. These data are consistent with those of Fewell et al\(^1\) who observed that flow changes in the pulmonary artery precede those in the aorta and do not support a humoral mechanism affecting ventricular contractility state during PEEP.

Data concerning afterload, blood pressure, and vascular resistance during PEEP application are

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### Table 2—Correlations Between Different Levels of PEEP and Two-Dimensional Doppler Parameters for Right Ventricular (RV) and Left Ventricular (LV) Performance

<table>
<thead>
<tr>
<th>PEEP, cm H(_2)O</th>
<th>RV EDVI</th>
<th>RV CI</th>
<th>RV Vmax</th>
<th>RV TVI</th>
<th>LV EDVI</th>
<th>LV CI</th>
<th>LV Vmax</th>
<th>LV TVI</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>-0.25</td>
<td>-0.21</td>
<td>-0.54</td>
<td>-0.56</td>
<td>-0.23</td>
<td>-0.27</td>
<td>-0.50</td>
<td>-0.51</td>
</tr>
<tr>
<td>7.5</td>
<td>-0.70</td>
<td>-0.68*</td>
<td>-0.73</td>
<td>-0.71</td>
<td>-0.65</td>
<td>-0.68*</td>
<td>-0.67</td>
<td>-0.69</td>
</tr>
<tr>
<td>10</td>
<td>-0.81</td>
<td>-0.771</td>
<td>-0.831</td>
<td>-0.871</td>
<td>-0.751</td>
<td>-0.721</td>
<td>-0.811</td>
<td>-0.821</td>
</tr>
<tr>
<td>12.5</td>
<td>-0.811</td>
<td>-0.851</td>
<td>-0.841</td>
<td>-0.841</td>
<td>-0.841</td>
<td>-0.801</td>
<td>-0.866</td>
<td>-0.877</td>
</tr>
</tbody>
</table>

*\(<0.05.\)*

\(†<0.01.\)

\(‡<0.005.\)

\(§<0.001.\)

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normal throughout study period. Also, transatrioventricular flow parameters (AT, DT, DR, and E/A), representing myocardial diastolic properties, remained unchanged. Finally, LV afterload assessed noninvasively according to the LaPlace relation did not show statistical significant modification.

Two minutes after removal of PEEP, all cardiac hemodynamic changes returned to control levels.

**DISCUSSION**

The results of the present study showed that a stepwise increase of PEEP under spontaneous ventilation induced a proportional decrease in CI, which is in agreement with several authors,\(^1,6,8,9,13,16,21\) but they give quite different and controversial explanations. In patients with uncompromised cardiorespiratory function, the drop of CI due to PEEP application does not limit its use in daily anesthesiologic practice, because it can be corrected with volume expansion. In patients with moderate heart decompensation, administration of positive inotropic agents may be necessary. However, in patients with severe decompensation, a drop in cardiac output appears to be critical and the two therapeutic interventions mentioned above may worsen the imbalance between alveolar ventilation and pulmonary blood flow.\(^3\) Moreover, in widely employed PEEP treatment of patients with adult respiratory distress syndrome, decreases in CI may counteract its positive effect on arterial oxygenation. Thus, it is important to understand the mechanism(s) responsible for the decrease in CI due to PEEP. From a pathophysiologic standpoint, the diminution in CI might be mediated by one or several of the following variables: (1) decreased preload (Frank-Starling mechanism), (2) depressed contractility, and (3) augmented afterload.

In our study, reduction of preload estimated with two-dimensional echocardiography as right and left
conflicting. Sellden et al found a statistically significant rise in blood pressure and systemic vascular resistance, while Jardin et al demonstrated the opposite effect. Other investigators found changes in pulmonary vascular resistance and they assumed that reduction in vena caval blood flow could also have resulted from an impairment in RV systolic function due to an increase in RV output impedance. Calculating LaPlace relation in our study, we did not find any alteration in LV afterload. An increase in RV afterload (defined as wall stress during systole) indirectly can be ruled out since RV dimensions decrease progressively by increase of PEEP without twodimensional echocardiographic evidence for augmentation of pulmonary artery diameter (ie, pulmonary hypertension). Hence, the decrease in CI during PEEP cannot be related to changes in afterload. Thus, several studies, using a variety of techniques in animals, sedated subjects, patients with acute respiratory distress syndrome, or healthy, awake volunteers have given controversial conclusion about the mechanisms of decreased cardiac output by PEEP application. The apparent disparity among these studies may have been caused by methodologic

Doppler echocardiography has become increasingly important characterizing ventricular filling pattern during diastole. The pattern of ventricular filling depends on the proper interplay of multiple factors such as atrial pressure, rate of relaxation, myocardial stiffness, pericardial constraint, and intrathoracic pressure. Several investigators have studied the reliability of Doppler transatrioventricular parameters for assessment of ventricular diastolic filling. Although left atrial and left ventricular pressures are not available, the mitral flow velocity is dependent on pressure interaction, and thus can be used to assess changes in filling pressures.

Rokey et al compared left ventricular diastolic filling parameters determined by Doppler ultrasound with those obtained by cineangiography. A significant correlation was noted between Doppler echocardiographic and angiographic peak filling rates and normalized peak filling rates. A significant correlation has been found between DR and maximum negative dp/dt and time constant (TC) of myocardial relaxation. In this study, we used several indices from transatrioventricular blood flow (Vmax, TV1, AT, DT) reflecting changes in diastolic filling. A significant correlation was obtained between the levels of PEEP and Vmax and TV1 (Table 2), confirming noninvasively that filling pressure is decreased by application of PEEP. Moreover, DR and E/A reflecting myocardial relaxation and stiffness remained unchanged throughout the study period, which is in contrast to the results of Raper and Sibbald that ventricular diastolic properties are altered by PEEP in animal experiment.

Recently, it has been shown that peak blood flow acceleration dv/dt in aorta and pulmonary artery, can be used as an index of systolic function of the left and right ventricle. Studies by Stein and Sabbah showed that blood acceleration was intrinsic to the rate of change in power developed by the left ventricle and related closely to it. Peak blood acceleration was also shown to be sensitive to alteration in the inotropic state, but was less affected by an augmentation of preload and afterload. In our study, we did not find changes in blood acceleration in the aorta and pulmonary artery, supporting the view that left and right ventricles are not altered in the contractility state during PEEP application. In agreement with other authors, we observed statistically significant differences in acceleration of blood between aortic and pulmonary artery (7.5 m/s² vs 6.7 m/s², p<0.0001).

These data indicate that in normal subjects, despite a five times higher systemic resistance compared with the pulmonary circuit, blood is accelerated two and half times more rapidly in the ascending aorta than in the main pulmonary artery, confirming that ventricular systolic pressure and/or contractile properties of the ventricle are the most important determinants for the magnitude of dv/dt.

Interestingly, the lack of a compensatory rise in heart rate, as CI decreases, reported by almost all authors remains unclear, but may contribute to the overall adverse effect of PEEP on cardiac output.

The general limitations of transthoracic two-dimensional Doppler echocardiography are described elsewhere. In some subjects, an additional limitation may be met when exactly measuring ventricular wall thickness and calculating the LaPlace relation, due to poor lateral resolution, but we think that with advances in technology and development of high-resolution devices, this limitation will be abolished. Furthermore, we observed an overlapping of the heart by the inflated lungs during inspiration at a PEEP level of 12.5 cm H₂O in 2 of the 21 volunteers. Increasing the PEEP up to 15 cm H₂O, the quality of echo images was worsening and volunteers became increasingly uncomfortable.

While a final conclusion is hardly to be made at the present time, this experimental study strongly suggests that the major hemodynamic effect of PEEP is a reduction in venous return produced by an increase in pleural pressure, leading to a fall in biventricular EDVI and cardiac output. Furthermore, employing new Doppler parameters we can rule out that PEEP alters systolic and diastolic ventricular function. Also, PEEP did not change afterload significantly as

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Influence of PEEP on Right and Left Ventricular Performance (Huemer et al)
judged by two-dimensional hemodynamic LaPlace relation.

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