Right Ventricular Function during Positive End-expiratory Pressure*

Thermodilution Evaluation and Clinical Application

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Right ventricular (RV) function was studied in 13 patients under controlled mechanical ventilation with positive end-expiratory pressure (PEEP) for adult respiratory distress syndrome. The assessment of RV function was made by the thermodilution technique. Calculations of RV ejection fraction (RVEF) and RV end-diastolic volume (RVEDV) were performed. In 11 patients, increasing PEEP was accompanied by a progressive decrease in blood pressure (BP), stroke volume (SV), RVEDV, and no change in RVEF. Increasing PEEP further was accompanied by a further decrease in RV preload. The remaining two patients exhibited a decrease in BP, SV, RVEF and an increase in RVEDV. One of these two patients exhibited a large decrease in cardiac output (CO). Thus, measurement of RVEDV (best parameter of ventricular preload) and RVEF are easily performed at the patient’s bedside using a special thermodilution technique. This allows selection of the best treatment of PEEP-induced decrease in CO.

While increasing oxygen content, mechanical ventilation with positive end-expiratory pressure (PEEP) may worsen oxygen transport by reducing cardiac output (CO). Thus, an understanding of the mechanisms involved in the reduced CO may help the physician improve the effectiveness of PEEP by choosing the best treatment when oxygen transport is impaired. It generally was believed that increased intrathoracic pressure generated by mechanical ventilation hinders venous return and thus reduces preload. However, recent observations of apparently increased ventricular transmural filling pressure together with reduced CO have suggested alternative explanations. A true dysfunction of both ventricles may occur as PEEP is applied. Santamore et al have suggested a decrease in the apparent distensibility of both the right and the left ventricles. The decreased compliance makes the ventricles harder to fill, accentuating the decrease in venous return. PEEP also could induce an interdependence between the left and the afterloaded right ventricles altering diastolic filling of the left ventricle. It also may be possible that the increase in juxtacardiac pressure is underestimated by the techniques used to measure intrathoracic pressure: pleural cannulas, pericardial catheters or esophageal balloons. In that case, filling pressures of both ventricles and preload would actually be reduced in an otherwise uncompromised heart. It is thus likely that PEEP decreases CO primarily by reducing right (RV) and left ventricular end-diastolic volumes (preload).

The right ventricle plays a major role in PEEP-induced decrease in CO. This can be explained by different possible mechanisms: (1) true dysfunction, (2) interdependence between the left and afterloaded right ventricles, causing restriction in left ventricular filling, and (3) decrease in RV preload, inducing a secondary decrease in left ventricular filling.

In clinical practice, we speculated that it would be of great interest to evaluate precisely the effects of PEEP on RV function. When RV preload (best appreciated by RV end-diastolic volume [RVEDV]) is decreased, modification in CO can be treated by blood volume expansion. In case of an increase of RVEDV (RV dysfunction), fluid challenge is not indicated to treat abnormalities in CO. To assess RV function, we used bedside thermodilution measurements of RV ejection fraction (RVEF) and RVEDV.

Patients, Material and Methods

Thirteen patients, free from cardiovascular antecedents and ranging in age from 36 to 67 years, were included in this study. Informed consent was obtained from the closest relative. The patients were hospitalized in the intensive care unit for adult respiratory distress syndrome. They were studied in the acute phase of their illness (first or second day from onset) on the first day that mechanical ventilation was required. Seven patients had acute pneumonia, four had hypoxia following peritonitis and the last two had hypoxia following blunt trauma. Chest radiography showed bilateral diffuse parenchymal opacities. The pulmonary capillary wedge pressure (PCWP) was below 12 mm Hg in all patients. The mean arterial oxygen tension (PaO2) at zero end-expiratory pressure (ZEEP) was 46.2 ± 3.3 mm Hg on a fractional inspired oxygen

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concentration (FIO₂) of 0.6. To avoid hemodynamic modifications due to hypoxia, FIO₂ was maintained throughout the study between 0.6 and 0.8. Patients were sedated with phenergan (1 mg/h), paralyzed with pancuronium bromide (2 mg/h) and received controlled mechanical ventilation (tidal volume 12 to 17 ml/kg) via an endotracheal tube. Respiratory rate was adjusted to maintain an arterial carbon dioxide tension between 35 to 40 mm Hg. At ZEEP, we obtained measurements of mean arterial blood pressure (BP), intravascular mean pulmonary arterial pressure (PAP), intravascular PCWP, intravascular RV end-diastolic pressure (RVEDP), and heart rate (HR).

Measurements of CO and RVEF were obtained by the thermodilution technique, which has been described in detail elsewhere. We used a modified pulmonary artery catheter with a proximal port located 13 to 15 cm from the tip. This allowed the injection of a cold index directly into the right ventricle. The proper position of the proximal port was checked by the presence of RV pressure-pulse tracings. The quantitative measurement of thermal washout in the pulmonary artery was recorded, amplified and interpreted by a system with a dynamic response fast enough to record beat-to-beat washout temperature plateaus (Gamada Company, Paris, France). The fast-response thermistor bead of the catheter was laterally mounted on the catheter wall with a minimum of potting material. This design attempt to minimize the thermal mass (catheter and potting material) surrounding the thermistor bead, which improves the response to a step change in temperature. The standard time constant (time to achieve 63 percent of the step change in temperature) of the catheter's mounted-thermistors used in this study ranged from 100 to 150 ms. The percentage of the step change achieved at 0.5- and 1.0-s time intervals ranged from 90 to 95 percent. This short response time (standard time constant of regular thermistor catheters is about 1,200 ms) was obtained for the whole system, after the thermistor had been mounted into the side of the Swan-Ganz catheter. These modifications were made by the manufacturer (Baltithermal thermal dilution catheter [rapid response] 73 R 6067 Electro-catheter Corp, Rahway, NJ). This allowed the recording of the staircase curve of the downstream temperature change after injection of 10 ml ice-cold glucose (<2°C). Plateaus of the rapid step changes on the descending limb of the curve were easily located from the simultaneous electrocardiogram (ECC) recording. When a R-wave was detected on the ECC recording, the corresponding point on the downslope of the thermodilution curve was located. Three to five successive determinations were obtained on each thermodilution signal, starting with the first R-wave occurring after 80 percent of the peak deviation on the descending slope of the thermodilution curve. We measured the height in millimeters (C1-C2-C3) between the basal blood temperature (Fig 1, TA) and the temperature at end-diastole located as just described. Successive residual fractions (RF) were calculated using the formula:

\[
RF = \frac{C3}{C2} \quad \text{and} \quad RF = \frac{C2}{C1}
\]

Then the successive values were averaged (RF̅) and RVEF was obtained by subtracting RF from unity:

\[
RVEF = 1 - \overline{RF}
\]

Knowledge of RF and SV permitted calculation of RVEDV by the formula:

\[
RVEDV = SV/1 - RF
\]

and by substituting RF̅ with 1 - RVEF:

\[
RVEDV = SV/1 - (1 - RVEF) = SV/RVEF
\]

Thermodilution CO and RVEF were measured by successive injection of four boluses of dextrose and the values were averaged. These determinations were made consistently at the end-expiratory phase of ventilation. Stroke volume was obtained by dividing CO with HR. The standard deviation of the mean of RVEF determinations was 5.8 percent. The mean worst error, determined by dividing the difference between the two outside determinations of RVEF by the mean, was 9.7 percent.

Then PEEP was applied by 5-cm H₂O increments. Measurements were obtained 45 min after the onset of each level to reach a steady state. At 15 cm H₂O level, nine patients needed blood volume expansion (BVE) with dextran because the decrease in SV was more

**Table 1—Effects of PEEP on Patients**

<table>
<thead>
<tr>
<th>PEEP (cm H₂O)†</th>
<th>RVEDP (mm Hg)†</th>
<th>RVEDV (ml)†</th>
<th>EF (%)†</th>
<th>SV (ml)†</th>
<th>HR (beats/min)†</th>
<th>BP (mm Hg)†</th>
<th>PAP (mm Hg)†</th>
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</thead>
<tbody>
<tr>
<td>0</td>
<td>8 ± 3.3</td>
<td>122 ± 13</td>
<td>45 ± 2</td>
<td>54 ± 6</td>
<td>106 ± 11</td>
<td>112 ± 14</td>
<td>26 ± 3</td>
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<tr>
<td>(n = 11)</td>
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<td>5</td>
<td>8.5 ± 4</td>
<td>117 ± 14</td>
<td>46 ± 2</td>
<td>52 ± 6</td>
<td>111 ± 6</td>
<td>110 ± 12</td>
<td>27 ± 3</td>
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<td>(n = 11)</td>
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<td>10</td>
<td>9.6 ± 6.2</td>
<td>106 ± 14‡</td>
<td>47 ± 1</td>
<td>49 ± 6‡</td>
<td>109 ± 6</td>
<td>103 ± 11‡</td>
<td>29 ± 4</td>
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<td>(n = 11)</td>
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<tr>
<td>15</td>
<td>12.5 ± 6.1</td>
<td>96 ± 13‡</td>
<td>46 ± 2</td>
<td>44 ± 5‡</td>
<td>112 ± 5</td>
<td>91 ± 13‡</td>
<td>31 ± 3‡</td>
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<td>(n = 11)</td>
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<tr>
<td>15 + BVE</td>
<td>15.7 ± 6.3‡</td>
<td>118 ± 10</td>
<td>46 ± 2</td>
<td>53 ± 4</td>
<td>108 ± 4</td>
<td>109 ± 10</td>
<td>34 ± 5‡</td>
</tr>
<tr>
<td>(n = 11)</td>
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</tbody>
</table>

*Those who displayed a similar evolution according to RV end-diastolic volume (RVEDV), ejection fraction (EF) and stroke volume (SV). All values mean ± SEM.
†PEEP, positive end-expiratory pressure; RVEDP, RV end-diastolic pressure (intravascular); HR, heart rate; BP, mean arterial blood pressure; PAP, mean pulmonary arterial pressure (intravascular); BVE, blood volume expansion.
‡p<0.001 from zero end-expiratory pressure.
FIGURE 2. Frank-Starling relationships between stroke volume (SV) and corresponding right ventricular end-diastolic volume (RVEDV). Data correspond with those in Table 1 and 2; SEM were averaged and are represented by cross bars. Open circles, values during PEEP 0, 5, 10, 15 cm H₂O (11 patients). Asterisk, values after blood volume expansion (BVE) (11 patients). Solid circles, values during PEEP 20 (eight patients) and 25 cm H₂O (six patients). Values during PEEP before and after BVE show no tendency to depart from a single curve.

than 30 percent of the ZEEP value. The PEEP was increased up to 20 cm H₂O in eight patients and to 25 cm H₂O in six patients because the ratio of PaO₂ to FIO₂ was less than 300.** All results are expressed as the mean ± SEM. Statistical analysis was performed using two-way analysis of variance and the Student t test for paired data. Right ventricular function curves (plotting SV against RVEDV) were analyzed using a curvilinear model. Based on the magnitude of the correlation coefficient, a second-degree polynomial (parabolic) model provided the best least-squares fit of the data when compared to linear model. A p<0.05 was considered significant.

**RESULTS**

All thirteen patients exhibited a decrease in SV when PEEP was added. Eleven patients showed a similar evolution (Table 1). Stroke volume, BP and RVEDV were reduced (p<0.001) with no change in RVEF and HR as PEEP was increased from 0 to 15 cm H₂O. Modifications of RVEDP (intravascular) and PAP (intravascular) reflect direct effects of PEEP on these parameters. According to the respective changes in RVEDV and RVEF, the reduction of SV was related to a decreased RV preload. A Frank-Starling SV/RVEDV function curve was constructed and the parabolic equation was calculated (RVEDV = 0.02 SV² + 1.04 SV + 17.4, r = 0.87, p<0.001). Individual values showed no tendency to depart from a single curve and SV decreased as RVEDV decreased (Fig 2). At a PEEP of 15 cm H₂O, because of the magnitude of change in BP and SV, patients were given intravenous fluids to restore RVEDV to baseline values. After RVEDV had been corrected by BVE (dextran, 875 ± 105 ml), values of SV returned to baseline (Table 1). A Starling function curve was constructed between the SV/RVEDV points before and after volume infusion at 15 cm H₂O PEEP and was not different from the Starling curve constructed at other levels of PEEP (RVEDV = 0.005 SV² + 1.11 SV + 30.5, r = 0.83, p<0.01 and NS from the previous curve) (Fig 2). The effects of BVE on PAO₂ and PCWP were as follows: PEEP 15: PaO₂, 76 ± 10 mm Hg; PCWP (intravascular), 14.3 ± 5.5 mm Hg; PEEP 15 + BVE: PaO₂, 72 ± 9 mm Hg; PCWP (intravascular), 17.6 ± 5.1 mm Hg.

PEEP was increased up to 20 cm H₂O in eight patients and to 25 cm H₂O in six patients because the PaO₂/FIO₂ ratio was less than 300.** A further decrease in RVEDV and SV (p<0.015 from PEEP 15) with no change in RVEF and HR was noted (Table 2). A Frank-Starling curve was constructed between the SV/RVEDV points (15 ± BVE, 20, 25 cm H₂O PEEP) and was not different from the two previous curves (RVEDV = 0.09 SV² - 7.05 SV + 22.66, r = 0.78, p<0.05).

The other two patients showed a different evolution as PEEP was increased. Adding 15 cm H₂O PEEP caused a decrease in CO and RVEF together with an increase in RVEDV (Table 3). Since we only measured intravascular PAP, we did not calculate pulmonary vascular resistance. Thus, we cannot speculate on the mechanism involved: true ventricular dysfunction or increased afterload. Because of a significant decrease in CO, BP and urine output at PEEP 15 cm H₂O, one patient needed hemodynamic support. Dobutamine (8 μg/kg/min) improved RV performance and CO and RVEF increased with a concomitant decrease in RVEDV. Intravascular PAP was also lowered by dobutamine.

**DISCUSSION**

The use of controlled mechanical ventilation with

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**Table 2—Hemodynamic Data of Patients Who Received 20 or 25 cm H₂O PEEP**

<table>
<thead>
<tr>
<th>PEEP (cm H₂O)</th>
<th>RVEDP (mm Hg)</th>
<th>RVEDV (ml)</th>
<th>EF (%)</th>
<th>SV (ml)</th>
<th>HR (beats/min)</th>
<th>BP (mm Hg)</th>
<th>PAP (mm Hg)</th>
</tr>
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<tbody>
<tr>
<td>15 ± BVE</td>
<td>16.9 ± 8.2</td>
<td>118 ± 9.1</td>
<td>44 ± 1</td>
<td>54 ± 4</td>
<td>109 ± 9</td>
<td>108 ± 13</td>
<td>34 ± 5</td>
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<tr>
<td>(n = 5)</td>
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<tr>
<td>20</td>
<td>19.6 ± 7.4†</td>
<td>113 ± 9†</td>
<td>44 ± 1</td>
<td>50 ± 5†</td>
<td>100 ± 13</td>
<td>104 ± 14</td>
<td>36 ± 3</td>
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<td>(n = 5)</td>
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<tr>
<td>25</td>
<td>22.5 ± 8.3†</td>
<td>110 ± 9†</td>
<td>44 ± 2</td>
<td>47 ± 4†</td>
<td>120 ± 6</td>
<td>98 ± 6</td>
<td>38 ± 5</td>
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<td>(n = 6)</td>
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</table>

*PEEP, positive end-expiratory pressure; RVEDP, RV end-diastolic pressure (intravascular); RVEDV, RV end-diastolic volume; EF, ejection fraction; SV, stroke volume; HR, heart rate; BP, mean arterial blood pressure; PAP, mean pulmonary arterial pressure (intravascular); BVE, blood volume expansion. All values mean ± SEM. †p<0.05 from PEEP 15.
Table 3—Hemodynamic Changes in Two Patients Who Displayed a Decrease in Cardiac Output Ejection Fraction and an Increase in Right Ventricular End-Diastolic Volume

<table>
<thead>
<tr>
<th>PEEP (cm H₂O)</th>
<th>Value</th>
<th>0</th>
<th>5</th>
<th>10</th>
<th>15</th>
<th>15+</th>
<th>Dobutamine</th>
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<tbody>
<tr>
<td>RVEDV (ml)</td>
<td></td>
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<tr>
<td>Patient 1</td>
<td>84</td>
<td>109</td>
<td>110</td>
<td>126</td>
<td>136</td>
<td>138</td>
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<tr>
<td>Patient 2</td>
<td>150</td>
<td>145</td>
<td>182</td>
<td>250</td>
<td>310</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EF (%)</td>
<td></td>
<td></td>
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<tr>
<td>Patient 1</td>
<td>50</td>
<td>45</td>
<td>50</td>
<td>30</td>
<td>46</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patient 2</td>
<td>35</td>
<td>45</td>
<td>37</td>
<td>22</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>CO (L/min)</td>
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<td></td>
<td></td>
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<tr>
<td>Patient 1</td>
<td>4.3</td>
<td>4.7</td>
<td>5.5</td>
<td>3.6</td>
<td>4.1</td>
<td></td>
<td></td>
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<tr>
<td>Patient 2</td>
<td>7.4</td>
<td>7</td>
<td>7.7</td>
<td>6.6</td>
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<td>HR (b/min)</td>
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<tr>
<td>Patient 1</td>
<td>98</td>
<td>95</td>
<td>95</td>
<td>93</td>
<td>104</td>
<td></td>
<td></td>
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<tr>
<td>Patient 2</td>
<td>126</td>
<td>107</td>
<td>114</td>
<td>116</td>
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<tr>
<td>PAP (mm Hg)</td>
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<tr>
<td>Patient 1</td>
<td>25</td>
<td>27</td>
<td>30</td>
<td>35</td>
<td>39</td>
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<td></td>
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<tr>
<td>Patient 2</td>
<td>31</td>
<td>31</td>
<td>35</td>
<td>38</td>
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</table>

*PEEP, positive end-expiratory pressure; RVEDV, RV end-diastolic volume; EF, ejection fraction; CO, cardiac output; HR, heart rate; PAP, mean pulmonary arterial pressure (intrapulmonary).

PEEP decreases CO and the underlying mechanisms continue to be the subject of considerable controversy. A main source of this controversy arises as a consequence of the inability to reliably estimate changes in ventricular end-diastolic volumes from end-diastolic pressures when intrathoracic pressures are altered. Intravascular pressure must be related to the mediastinal pressure adjacent to the left and right heart chambers. In clinical practice, this problem is extremely difficult to overcome. Assessment of paracardiac pressure by placing a catheter in the pericardial space is a hazardous technique. Substitution of pleural or esophageal pressures for pericardial pressure may give erroneous information on the pressure that surrounds the heart. One way to overcome this problem is to measure ventricular volumes more directly, and it is worth noting that RVEDV is the best index available for RV preload.

In this study we used the thermodilution technique to measure RVEDV. Availability and reproducibility are two great advantages of this technique, and RVEDV and EF can be measured any number of times at the patient's bedside without a large and expansive monitoring system. This technique is no more invasive than the insertion of a conventional thermodilution catheter. In the study of Kay et al, it was demonstrated that measurements of EF were reproducible at ± 5 percent (70 percent confidence limit) and that correlation with multigated blood pool imaging technique and first-pass technique were excellent. Our own results showed that the mean worst error was 9.7 percent (see Patients, Material and Methods section).

A limitation of the thermodilution technique is that the catheter body of the pulmonary artery catheter may have a blunting effect on the fast response thermistor, thus giving RVEF below true value. In our study, we did not use any correction because the blunting of a thermistor's response is reproducible for a given catheter and we analyzed the relative evolution of RVEF with PEEP and not the absolute value. Other methods are available to measure RV volume or dimensions and RVEF: echography, radionuclear angiography and contrast angiography. A major limiting factor is that those procedures require an expensive imaging system and specialized personnel; they are not repeatable bedside procedures and this precludes their use for routine measurements of RVEDV and EF in critically ill patients.

In our study, SV was found to decrease markedly during mechanical ventilation with PEEP and we speculated that measurement of RVEDV and EF could be helpful for managing fluid or drug therapy in our patients. Altered RV performance may be caused by (1) decreased venous return to the right (and then to the left) heart, (2) true dysfunction with a depressed inotropism, (3) increased afterload, (4) altered ventricular compliance.

Reduced ventricular filling induced by restricting venous return to the right heart has already been described with PEEP. This mechanism is evident from our results. Eleven of our patients exhibited a decrease of their SV and RVED in RVEF with PEEP was added. This indicates that the decrease in SV was mediated through a decrease in RV preload, and SV decreased along the same Frank-Starling curve, even at high levels of PEEP (20 and 25 cm H₂O) (Fig 2). Patients were given fluid intravenously which restored RVEDV and SV to baseline values. In these patients we did not find any evidence of a depressed inotropism. If this mechanism had been involved, restoration of SV after fluid challenge would have been accompanied by an increase in RVEDV. The fact that ventricular function is not impaired with PEEP is confirmed by other clinical and experimental studies. Thus, thermodilution allowed determination of the main cause of the decreased SV observed in our patients: the decrease in venous return and RVEDV induces a secondary decrease in left ventricular preload, which is easily corrected by BVE. The CO maintenance with BVE during PEEP 15 did not alter the beneficial effect of PEEP on PaO₂, probably because of a redistribution of pulmonary blood flow toward the "normal" V̇A/Q ratio units.

It also has been reported that PEEP-induced expansion of the lungs may alter filling of both ventricles by directly compressing right and left ventricles over the lateral wall. This induces an increase in the diastolic...
stiffness of both ventricles with decreased end-diastolic volume and thereby contributes to the reduction in SV. Evaluation of this mechanism would require plotting end-diastolic volumes vs transmural filling pressures, which was not done in this study. Consequently, we cannot speculate on this possible mechanism.

The use of thermocilation alone does not allow us to determine a possible effect of PEEP on RV end-diastolic compliance, which is a limitation of this technique. This requires simultaneous determinations of RVED and transmural pressures, which allows us to examine changes in ventricular distensibility. Santamore et al demonstrated such a mechanism in dogs. In their study, plots of RVEDV vs RVED transmural pressures showed that PEEP decreased the apparent distensibility of the RV. This would make the RV harder to fill, accentuating the effect of PEEP on venous return. The most probable cause for the decrease in RV distensibility could be an interaction between the inflated lung and the lateral wall of the RV, this mechanism being demonstrated for the left ventricle. In clinical practice, transmural pressures are derived by subtracting the lateral pleural or the esophageal pressures from the ventricular pressures. These measurements can be questioned since juxta-cardiac pressures (the most accurate to measure) are extremely difficult to obtain, and measurements made at other sites with different techniques can only be considered of the true pressure surrounding the heart. The latter usually rises higher during PEEP than measured elsewhere in the thorax by usual techniques.

Two of our patients exhibited hemodynamic modifications, which cannot be related to a decreased RV preload. The PEEP was associated with a decrease in BP and CO, accompanied by an increase in RVEDV and a decrease in RVEF. Right ventricular failure caused by increase in afterload already has been documented in patients ventilated with PEEP. A secondary left ventricular failure was observed in these studies because of a restriction of the left ventricular filling by the leftward displacement of the interventricular septum. Another explanation for the RV failure observed would be a decrease in ventricular inotropic state, due to endocardial blood supply impairment or a release of a negative inotropic agent secondary to lung inflation. However, impaired inotropic state is an unlikely explanation for the decrease in CO caused by PEEP. In our two patients, the first hypothesis (increased afterload of RV) was retained. Treatment was instituted in one patient because of the magnitude of change in CO. Vasodilator therapy would have theoretically been indicated, but because of low blood pressure, dobutamine was chosen since this drug has been demonstrated effective in acute right ventricular failure.

In summary, thermocilation technique is valuable for the evaluation of RV function during PEEP. In most of our patients we found that the decrease in SV was accompanied by a decrease in RVEDV and no change in RVEF. From this information, an appropriate therapy using fluid challenge was instituted. Only two patients exhibited a RV dysfunction with an increased RVEDV and a decreased RVEF. Whatever the origin, (increased afterload or negative inotropic state), dobutamine was found to be effective.

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Seventh Asian Congress of Cardiology

The Philippine Heart Association will sponsor the 7th Asian Congress of Cardiology at the Midtown Ramada Hotel, Manila, February 24-27, 1988. For information, contact the Secretariat: 7th Asian Congress of Cardiology, Philippine Heart Association, Inc., Philippine Heart Center, East Avenue, Quezon City, Philippines 1630.