LABORATORY INVESTIGATION
HEMODYNAMICS AND VENTRICULAR FUNCTION

Volume-dependent effects of positive airway pressure on intracavitary left ventricular end-diastolic pressure

ROY V. DITCHEY, M.D.

ABSTRACT To test the hypothesis that the effects of positive end-expiratory airway pressure (PEEP) on intracavitary left ventricular end-diastolic pressure (LVEDP) depend on the ventricular filling conditions under which PEEP is applied, the effects of PEEP on pressure in and around the left ventricle were determined before and after stepwise expansion of intravascular blood volume in 10 closed-chest dogs. Over a range of 0 to 20 cm of water, PEEP progressively increased both intrapericardial and intracavitary right ventricular end-diastolic pressures. These increases in pressure around the left ventricle were approximately linear and were relatively unaffected by volume loading. At the same time, PEEP always decreased transmural LVEDP by decreasing ventricular filling. However, transmural LVEDP fell more when ventricular volume was initially large, due to the nonlinear relationship between left ventricular transmural pressure and volume. As a result, intracavitary LVEDP (which reflected the sum of decreased transmural LVEDP and increased external pressure) increased when baseline ventricular volume was small and decreased when baseline ventricular volume was large. At intermediate volumes the fall in transmural pressure equaled the rise in external pressure, and intracavitary LVEDP did not change. These findings demonstrate that changes due to PEEP in intracavitary LVEDP are a complex function of increased intrathoracic pressure, decreased ventricular filling, and the operative level of left ventricular compliance.


POSITIVE end-expiratory airway pressure (PEEP) complicates hemodynamic monitoring by increasing pressure around the left ventricle. This is because ventricular filling is a function of transmural, rather than intracavitary, left ventricular end-diastolic pressure (LVEDP). As a result, intracavitary LVEDP and its correlates, mean left atrial and pulmonary artery wedge pressures, are inaccurate indexes of left ventricular preload in the presence of PEEP. Furthermore, an incomplete understanding of the effects of PEEP on these intravascular pressures has led to physiologically unsound recommendations for dealing with this problem in clinical practice. For example, it has been proposed that the magnitude of potential error in wedge pressure measurements can be estimated by comparing pressures measured in the presence and absence of PEEP. Since such comparisons frequently demonstrate only small pressure differences, it even has been suggested that correction for increased intrathoracic pressure is unnecessary. However, the following analysis not only demonstrates that the net effects of PEEP on intracavitary left ventricular filling pressures depend on variables other than altered intrathoracic pressure, but also suggests that these effects are functions of the left ventricular filling conditions under which PEEP is applied.

By definition, transmural LVEDP is equal to intracavitary LVEDP (LVEDP_{IC}) minus the pressure around the left ventricle. This means that changes in intracavitary LVEDP must equal the sum of changes in transmural LVEDP (LVEDP_{TM}) and "external" pressure (∆LVEDP_{IC} = ∆LVEDP_{TM} + ∆external pressure). Since PEEP increases the effective pressure around the left ventricle (a function of both intrapericardial pressure and right ventricular end-diastolic pressure [RVEDP]^5) but decreases left ventricular volume (and therefore decreases transmural LVEDP^4), net changes in intracavitary pressure are functions of the combined (and opposite) effects of PEEP on intrathoracic pressure and ventricular filling. Furthermore, since the left ventricular transmural pressure-volume relationship is nonlinear, comparable reductions in
filling (from any cause) decrease transmural pressure more when ventricular volume is initially large. This suggests that the magnitude and even the direction of changes due to PEEP in intracavitary LVEDP could vary, depending on ventricular volume and, therefore, the operative level of left ventricular compliance.

On the basis of these considerations, it was hypothesized that the effects of PEEP on intracavitary LVEDP are functions of the left ventricular filling conditions under which PEEP is applied. To test this hypothesis and to demonstrate the complex nature of changes in intracavitary LVEDP, the effects of PEEP on pressures in and around the left ventricle were determined under different baseline filling conditions in closed-chest dogs. The results demonstrate a fundamental series of physiologic interrelationships that present a conceptual challenge to current methods of estimating left ventricular preload in patients treated with PEEP.

**Methods**

Studies were performed in 10 mongrel dogs weighing 14.6 to 43.2 kg. A preliminary left lateral thoracotomy was performed to allow placement of intrapleural and intrapericardial balloon manometers. Each balloon was hand-constructed according to previously described methods. Briefly, condom rubber was cemented to both sides of a ring of silicone rubber sheeting (55 mm diameter, 1.6 mm thickness), with an inner chamber (23 mm diameter) containing the end of a 30 cm length of silicone rubber tubing (1.6 mm internal diameter) with multiple side holes. These balloon manometers approximate optimal design for the measurement of pressure between two contiguous surfaces. The pressure-volume characteristics of each balloon were determined before insertion, and the internal volumes necessary to maintain zero pressure were recorded (range 0.7 to 0.8 ml). In addition, a linear pressure response over the range of pressures encountered in each study was confirmed for each balloon in vitro. Balloons were placed through a small (approximately 2 cm) pericardial incision and positioned over the lateral surface of the left ventricle. The pericardial incision was then closed, and the silicone rubber tubing was securely attached at the point of entry into the pericardial space to prevent balloon migration. Intrapleural balloons were positioned over the upper lobe of the left lung in the mid-antero-posterior position. The distal balloon rim was attached to the lateral chest wall with a single suture to prevent migration. The silicone rubber tubes from the intrapericardial and intrapleural balloons were then exteriorized through small incisions in the lateral chest wall.

Physiologic studies were conducted on the third or fourth day after instrumentation. This limited interval was chosen to allow adequate time for return of intrathoracic pressures to physiologic levels, but to minimize the development of adhesions around the intrapericardial and intrapleural balloons. Dogs were anesthetized (pentobarbital 30 mg/kg), intubated, and mechanically ventilated with a Harvard respirator (tidal volume 400 to 500 ml). D-Tubocurare was given as a single dose (1 to 3 mg) followed by a continuous intravenous infusion (1 to 3 mg/hr) to minimize spontaneous changes in thoracic muscle tone and chest wall mechanics. In addition, propranolol (1 mg/kg) was administered intravenously to slow heart rate. This was done to facilitate identification of end-diastolic pressures. A No. 7F balloon flotation catheter (Swan-Ganz) was passed into the right ventricle via an external jugular vein. A No. 8F pigtail angiographic catheter was introduced through a femoral artery and passed retrograde into the left ventricle. In addition, an 18-gauge needle was introduced into the inspiratory ventilator hose and was attached to a 30 cm length of polyethylene pressure tubing. The right and left ventricular catheters were connected to Gould P23Db pressure transducers with zero reference set at the mid-chest level. The airway pressure tubing and both the intrapleural and intrapericardial balloon manometers were connected to Validyne MP45-871 low-range, differential pressure transducers, also set at the mid-chest level. The second port of each Validyne transducer was open to atmosphere. The intrapleural and intrapericardial balloons were filled to their predetermined optimal volumes, and each transducer was checked for baseline drift before each set of pressure recordings was taken. All pressures were recorded along with a standard limb lead electrocardiogram on a Beckman R611 eight-channel, forced-ink recorder. Pressure measurements were made with respiration suspended at end-expiration, at a time when all pressures were stable.

Airway, intrapericardial, intrapleural, and right and left ventricular pressures were recorded when end-expiratory airway pressure was varied from 0 to 30 cm of water in 2 cm increments. Pressure measurements then were repeated after intravenous administration of 10% dextran in a volume sufficient to raise intracavitary LVEDP before PEEP, first to between 10 and 15 mm Hg and then to greater than 15 mm Hg. In addition, pressure recordings were repeated under the same loading conditions in each dog to assess reproducibility. Finally, lung and chest wall pressure—volume relationships were determined under each set of study conditions by inflating the lungs to a volume 800 to 1000 ml above functional residual capacity with a spirometric calibration syringe and by recording pressures as lung volume was reduced in 100 or 200 ml increments.

Transmural LVEDPs were calculated by each of two methods. First, mean intrapericardial pressure was subtracted from LVEDP. Second, the effective external pressure around the left ventricle (EP) was considered a function of both intracavitary RVEDP and mean intrapleural pressure according to the following relationship:

\[ EP = f \text{(intraventricular RVEDP)} + (1 - f) \text{(intrapericardial pressure)} \]

where \( f \) is the fractional surface area of the left ventricle acted on by the right ventricular pressure.\(^6\) The appropriate fraction \( f \) for each animal was determined at postmortem study by excising and separating the interventricular septum and left ventricular free wall and by determining their relative surface areas by planimetry. Transmural RVEDPs were calculated by subtracting intrapericardial pressure from intracavitary RVEDP. The contribution of intracavitary LVEDP to the effective pressure around the right ventricle was assumed to be relatively unimportant because of the marked differences between septal and right ventricular free wall thicknesses.

Satisfactory data were obtained over the full 0 to 30 cm of water range of airway pressures in all studies in six dogs. In four dogs, data obtained at airway pressures above 10 to 20 cm of water were excluded due either to technically inadequate pressure recordings or to small air leaks that precluded steady-state measurements. The slopes of the relationships between airway and intrapericardial, intrapleural, and intracavitary RVEDPs were estimated by standard linear regression methods. Since all pressure measurements are subject to unknown errors, the reduced major axis\(^9\) or line of symmetry was used as the line of best fit for each relationship.

Results are reported as mean values ± SD. Statistical significance was determined by Student’s \( t \) test for paired samples.
when duplicate slopes derived under the same conditions of intravascular volume were compared. All other comparisons involved at least three data groups and were tested for significance by analysis of variance with multiple comparison testing (Student-Newman-Keuls).\textsuperscript{11}

**Results**

The relationship between airway and intrapericardial pressures was nearly linear under all conditions ($r = .97$ to $>.99$; $p < .001$) (figure 1), although intrapericardial pressure appeared to plateau above airway pressures of 20 cm of water in two dogs (figure 2). Therefore, linear regression analyses were restricted to airway pressures between 0 and 20 cm of water.

The slope of the relationship between airway and intrapericardial pressures under baseline conditions of left ventricular filling averaged $0.425 \pm 0.103$ (range $.259$ to $.612$). Thus, PEEP increased intrapericardial pressure by an amount equal to approximately 43\% of airway pressure. Figures 1 and 2 demonstrate the reproducibility of this effect. Repeat pressure recordings under the same conditions of intravascular volume yielded mean slopes of $0.421 \pm 0.093$ and $0.437 \pm 0.093$, respectively ($p = \text{NS}$). Paired slopes from these two sets of measurements differed by .07 or less in all cases.

The effects of dextran infusion on left ventricular filling pressures before PEEP are summarized in table 1. Blood volume expansion progressively increased transmural LVEDP (measured in the presence of atmospheric airway pressure). The mean slopes of the relationship between airway and intrapericardial pressures for the three baseline ventricular filling conditions were $0.425 \pm 0.103$, $0.430 \pm 0.090$, and $0.426 \pm 0.084$, respectively ($p = \text{NS}$). Thus, ventricular volume influenced initial intrapericardial pressure (table 1), but

**FIGURE 1.** An example of an approximately linear end-expiratory airway–intrapericardial pressure relationship. Open and closed circles represent two separate sets of pressure measurements made under identical filling conditions in a single dog.

**FIGURE 2.** An example of a curvilinear end-expiratory airway–intrapericardial pressure relationship, demonstrating an apparent plateau above 20 cm of water. Open and closed circles represent two separate sets of pressure measurements made under identical filling conditions in a single dog.

### TABLE 1

<table>
<thead>
<tr>
<th></th>
<th>$V_1$</th>
<th>$V_2$</th>
<th>$V_3$</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDP-IC</td>
<td>$3.2 \pm 3.3$</td>
<td>$11.9 \pm 2.3^{A}$</td>
<td>$19.7 \pm 3.0^{A,B}$</td>
</tr>
<tr>
<td>P</td>
<td>$-1.4 \pm 1.2$</td>
<td>$-0.8 \pm 1.3$</td>
<td>$-0.5 \pm 1.7^{A}$</td>
</tr>
<tr>
<td>LVEDP-P</td>
<td>$4.6 \pm 4.0$</td>
<td>$12.7 \pm 2.6^{A}$</td>
<td>$20.1 \pm 3.4^{A,B}$</td>
</tr>
<tr>
<td>LVEDP-PR</td>
<td>$4.9 \pm 3.5$</td>
<td>$12.3 \pm 2.2^{A}$</td>
<td>$18.6 \pm 3.1^{A,B}$</td>
</tr>
</tbody>
</table>

$V_1$, $V_2$, $V_3$ = baseline ($V_1$) and two levels of increased ($V_2$, $V_3$) left ventricular volume before PEEP; LVEDP-IC = intracavitary left ventricular end-diastolic pressure (mm Hg); P = intrapericardial pressure (mm Hg); LVEDP-P = transmural left ventricular end-diastolic pressure calculated as intracavitary left ventricular pressure minus intrapericardial pressure (mm Hg); LVEDP-PR = transmural left ventricular end-diastolic pressure calculated as intracavitary left ventricular pressure minus fractions of both intracavitary right ventricular end-diastolic and intrapericardial pressures (mm Hg) (see text for explanation).

$^{A}p < .05$ vs $V_1$.

$^{B}p < .05$ vs $V_2$. 
not to the extent to which that pressure was subsequently increased by PEEP (figure 3).

PEEP also caused linear increases in intrapleural and intracavitary RVEDPs (r = .78 to > .99; p < .001) (table 2) that were similar in magnitude to increases in intrapericardial pressure under all conditions (figure 4). However, the slope of the relationship between airway pressure and intracavitary RVEDP was slightly greater than that for airway and intrapericardial pressures before volume loading (table 2) and slightly less than that for airway and intrapleural pressure after volume loading. PEEP caused the difference between intracavitary RVEDP and intrapericardial pressure to increase slightly under baseline filling conditions, but had no effect on this pressure difference after the second increment in blood volume (table 3).

Transmural LVEDP fell progressively with PEEP under all three filling conditions (table 4), but decreased more when ventricular volume was initially large. For example, 30 cm of water of PEEP caused transmural LVEDP (calculated as intracavitary LVEDP minus fractions of both intrapericardial pressure and intracavitary RVEDP) to decrease by 3.6 ± 4.4, 7.9 ± 3.6, and 12.0 ± 4.1 mm Hg before and after two sequential increments in blood volume, respectively (all p < .05 vs each other, n = 6). In contrast, the effects of PEEP on intracavitary LVEDP were more uniform and highly dependent on baseline ventricular filling conditions (table 5). At low, intermediate, and high levels of initial transmural pressure, PEEP caused a slight increase, no change, and a slight decrease in intracavitary LVEDP, respectively. As a result, PEEP-induced changes in intracavitary and transmural LVEDP were invariably discordant in magnitude, direction, or both (figure 5). This was true whether transmural pressure was considered as a function of both intrapericardial pressure and intracavitary RVEDP or as intrapericardial pressure alone.

Lung and chest wall mechanics under each set of study conditions are summarized in table 6. Volume loading had no significant effect on lung or chest wall compliance.

Discussion

By increasing intrathoracic pressure and decreasing left ventricular volume, PEEP has opposite ef-

<table>
<thead>
<tr>
<th>TABLE 2</th>
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<tbody>
<tr>
<td>Effects of PEEP on intrapericardial, intrapleural, and intracavitary right ventricular end-diastolic pressures (n = 10)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>V₁</td>
</tr>
<tr>
<td>V₂</td>
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<td>V₃</td>
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</table>

Slopeₚ, Slopeᵢᵣ, and Slopeᵢᵣ = slopes of the linear relationships between end-expiratory airway pressure and intrapericardial, intrapleural, and intracavitary right ventricular end-diastolic pressures, respectively, over a range of airway pressures from 0 to 20 cm of water; other abbreviations as for table 1.

a p < .05 vs Slopeₚ,

b p < .05 vs Slopeᵢᵣ,

c p < .05 vs V₁ and V₂.
Effects on the two determinants of intracavitary LVEDP. Net changes in intracavitary pressure depend on the relative extent to which PEEP increases pressure around the left ventricle and decreases transmural LVEDP.

The nonlinear relationship between left ventricular transmural pressure and volume makes the magnitude and direction of these changes a function of baseline ventricular filling conditions. Whereas the effects of PEEP on pressures around the left ventricle are relatively unaffected by volume changes (table 2), comparable reductions in filling decrease transmural pressure more when ventricular volume is initially large. As a result, the operative level of left ventricular compliance largely determines the net effect of PEEP on intracavitary LVEDP (figure 6). When ventricular volume is initially small, PEEP increases external pressures more than it decreases transmural pressure, and intracavitary LVEDP increases. When ventricular volume is initially large, transmural pressure falls more than external pressures rise, and intracavitary LVEDP decreases. With intermediate volumes, the opposite effects of PEEP on external and transmural pressures offset each other, leaving intracavitary LVEDP unchanged.

This analysis is limited only slightly by the lack of direct volume measurements. Since transmural pressures were increased by administering dextran and decreased by applying PEEP (an intervention known to impair left ventricular filling), it is reasonable to assume that changes in transmural LVEDP were caused by similar directional changes in left ventricular end-diastolic volume. Logical analysis does not depend on the precise relationships between left ventricular transmural pressures and volumes or on whether those relationships were altered by PEEP. It is necessary to assume only that individual pressure-volume relationships were nonlinear and that PEEP decreased left ventricular filling. These assumptions neither require nor exclude an additional effect of PEEP on left ventricular compliance. It must be acknowledged, however, that the extent to which PEEP decreased ventricular filling under each set of study conditions is unknown. Theoretically, a larger decrement in ventricular volume could have contributed to the greater fall in transmural LVEDP observed when PEEP was applied after volume loading. However, the beneficial effects of blood volume expansion on pulmonary vascular resistance and mean circulatory pressure would be expected (if there is an effect at all) to lessen the impact of PEEP on left ventricular filling. It is possible, therefore, that operation from a steeper portion of the left ventricular transmural pressure-volume curve allowed a greater fall in transmural pressure despite a lesser change in volume under these conditions.

It is apparent that the effects of PEEP on intracavitary RVEDP and LVEDP differ considerably. Specifically, changes in intracavitary RVEDP closely approximate increases in intrapericardial pressure under all conditions (figure 4). This suggests that the magni-

### TABLE 3

<table>
<thead>
<tr>
<th>PEEP (cm of water)</th>
<th>0</th>
<th>10</th>
<th>20</th>
<th>30</th>
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</thead>
<tbody>
<tr>
<td>RVEDP-P (V1)</td>
<td>-1.4±2.0</td>
<td>-0.8±2.5</td>
<td>-0.1±2.6</td>
<td>1.1±2.3&lt;sup&gt;A,B&lt;/sup&gt;</td>
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<tr>
<td>RVEDP-P (V2)</td>
<td>1.6±2.3</td>
<td>1.6±2.2</td>
<td>2.3±2.4</td>
<td>3.3±2.2&lt;sup&gt;A,B&lt;/sup&gt;</td>
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<tr>
<td>RVEDP-P (V3)</td>
<td>5.5±3.0</td>
<td>5.0±2.1</td>
<td>5.3±2.1</td>
<td>5.0±2.8</td>
</tr>
</tbody>
</table>

RVEDP-P = transmural right ventricular end-diastolic pressure calculated as intracavitary right ventricular pressure minus intrapericardial pressure (mm Hg); other abbreviations as for table 1.

### TABLE 4

<table>
<thead>
<tr>
<th>PEEP (cm of water)</th>
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<th>20</th>
<th>30</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDP-PR (V1)&lt;sup&gt;A&lt;/sup&gt;</td>
<td>4.8±3.7</td>
<td>2.1±3.1&lt;sup&gt;C&lt;/sup&gt;</td>
<td>1.3±3.1&lt;sup&gt;C&lt;/sup&gt;</td>
<td>0.9±3.3&lt;sup&gt;C&lt;/sup&gt;</td>
</tr>
<tr>
<td>LVEDP-PR (V2)&lt;sup&gt;B&lt;/sup&gt;</td>
<td>11.9±2.7</td>
<td>7.6±2.0&lt;sup&gt;F&lt;/sup&gt;</td>
<td>5.0±2.2&lt;sup&gt;C,D&lt;/sup&gt;</td>
<td>4.0±2.0&lt;sup&gt;C,D&lt;/sup&gt;</td>
</tr>
<tr>
<td>LVEDP-PR (V3)&lt;sup&gt;C&lt;/sup&gt;</td>
<td>19.8±1.8</td>
<td>15.8±2.2&lt;sup&gt;C&lt;/sup&gt;</td>
<td>11.0±2.7&lt;sup&gt;C,D&lt;/sup&gt;</td>
<td>7.8±3.3&lt;sup&gt;C,D,E&lt;/sup&gt;</td>
</tr>
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</table>

Abbreviations as for table 1. *PEEP had similar effects when transmural LVEDP was calculated as intracavitary LVEDP minus intrapericardial pressure.

### TABLE 5

<table>
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<tr>
<th>PEEP (cm of water)</th>
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<th>20</th>
<th>30</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDP-IC (V1)&lt;sup&gt;A&lt;/sup&gt;</td>
<td>3.2±3.5</td>
<td>4.4±2.9</td>
<td>6.5±3.1&lt;sup&gt;A,B&lt;/sup&gt;</td>
<td>8.3±3.3&lt;sup&gt;A,B&lt;/sup&gt;</td>
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<tr>
<td>LVEDP-IC (V2)&lt;sup&gt;B&lt;/sup&gt;</td>
<td>11.6±2.2</td>
<td>11.0±2.4</td>
<td>11.3±2.7</td>
<td>12.6±1.7</td>
</tr>
<tr>
<td>LVEDP-IC (V3)&lt;sup&gt;C&lt;/sup&gt;</td>
<td>21.0±1.8</td>
<td>20.0±3.0</td>
<td>18.1±3.7&lt;sup&gt;A&lt;/sup&gt;</td>
<td>17.3±3.1&lt;sup&gt;A,B&lt;/sup&gt;</td>
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</table>

Abbreviations as for table 1. *PEEP had similar effects when transmural LVEDP was calculated as intracavitary LVEDP minus intrapericardial pressure.

*P < .05 vs 0 PEEP.

*P < .05 vs 0 PEEP.
tude and direction of changes in intracavitary RVEDP are determined primarily by the effects of PEEP on pressures around the right ventricle; i.e., either PEEP causes only minor changes in right ventricular end-diastolic volume or volume changes that do occur have little effect on transmural pressure. Since the right ventricle is more compliant than the left and since its distensibility is increased further when left ventricular volume is decreased,\textsuperscript{5, 6, 7, 16} comparable changes in ventricular volume due to PEEP undoubtedly alter transmural LVEDP more than transmural RVEDP. However, it also is important that PEEP has opposing effects on right ventricular volume.\textsuperscript{7} By increasing intrathoracic pressure, PEEP decreases the driving force for systemic venous return and right ventricular filling; however, by increasing pulmonary vascular resistance,\textsuperscript{5, 16} PEEP increases right ventricular afterload and provides a stimulus for compensatory right ventricular dilatation. As a result, PEEP causes only small net changes in right ventricular volume and transmural RVEDP.\textsuperscript{5} It is interesting that volume loading influences the direction of these changes (table 3), presumably by decreasing the effects of PEEP on pulmonary vascular resistance.\textsuperscript{16} Transmural RVEDP (i.e., the difference between intracavitary RVEDP and intracardiac pressure) increased slightly when PEEP was applied under baseline filling conditions, suggesting that changes in afterload caused a net increase in right ventricular volume. However, when PEEP was applied after volume loading, transmural RVEDP did not change significantly, suggesting that the effects of increased afterload on right ventricular volume were less important (and offset by the direct effects of increased intrathoracic pressure on systemic venous return) under these conditions.

To remain useful as indexes of left ventricular preload in the presence of PEEP, pulmonary artery wedge pressure and other more direct estimates of intracavitary LVEDP must be corrected for the effects of PEEP on pressure around the left ventricle. The observation

| TABLE 6  |
| Effects of volume loading on lung and chest wall compliance (n = 10)\textsuperscript{4} |
|          | V\textsubscript{1}       | V\textsubscript{2}       | V\textsubscript{3}       |
| Lung compliance | 158 ± 44                | 162 ± 46                | 161 ± 54                |
| Chest wall compliance | 133 ± 39                | 131 ± 39                | 122 ± 30                |

Abbreviations as for table 1.

\textsuperscript{4}Compliance values (ml/cm of water) did not differ significantly between loading conditions.

FIGURE 6. A. Theoretical way in which the fall in transmural left ventricular end-diastolic pressure (ΔTMP) caused by a reduction in left ventricular filling (ΔV) depends on baseline ventricular volume. Because the left ventricular transmural pressure–volume relationship is nonlinear, comparable volume changes cause greater changes in transmural pressure (ΔTMP\textsubscript{L}) when ventricular volume is initially large. B. Combined effects of volume-dependent decreases in transmural left ventricular end-diastolic pressure (TMP) and volume-independent increases in the effective external pressure (EP) around the heart on intracavitary left ventricular end-diastolic pressure (ICP) under the three initial filling conditions (V\textsubscript{1}, V\textsubscript{2}, and V\textsubscript{3}) described in A. ICP increases when ΔEP exceeds ΔTMP (V\textsubscript{1}), decreases when ΔTMP exceeds ΔEP (V\textsubscript{2}), and is unchanged when ΔEP = ΔTMP (V\textsubscript{3}).

FIGURE 5. Effects of PEEP on intracavitary and transmural LVEDPs before (volume 1) and after (volumes 2 and 3) two different levels of volume loading. Transmural pressure was calculated by subtracting fractions of both intrapacardial and intracavitary right ventricular pressures from intracavitary left ventricular pressure (see text for explanation). Data points represent average values obtained in six dogs studied over the full range of 0 to 30 cm of water of PEEP.
that PEEP commonly causes only minor changes in pulmonary artery wedge pressure does not mean that changes in intrathoracic pressure are not important. The effects of PEEP on intravascular left ventricular filling pressures are a complex function of increased intrathoracic pressure, decreased ventricular volume, and the operative level of left ventricular compliance. If pulmonary artery wedge pressure (or intracavitary LVEDP) does not change with PEEP, it means only that the increase in intrathoracic pressure caused by PEEP was offset by a volume change that decreased transmural filling pressure. The suggestions that left ventricular filling in the presence of PEEP can be evaluated accurately by discontinuing PEEP briefly when measuring wedge pressures or by comparing pressures with and without PEEP to quantitate potential errors in wedge pressure measurements are incorrect. Since PEEP decreases left ventricular volume, pressure measurements made in the presence and absence of PEEP represent two different conditions of left ventricular filling. Measurements made in the presence of PEEP can be related to true preload only by accounting for increases in pressure around the left ventricle.

The results of this study suggest that changes in intracavitary RVEDP could be used to estimate the effects of PEEP on these external pressures. This is an intriguing possibility, since intracavitary right heart filling pressures can be measured easily and since esophageal manometry (which is the only practical method of estimating intrathoracic pressure in clinical practice) may not indicate changes in pressure around the left ventricle accurately in the presence of PEEP. Assuming that discrepancies between pulmonary artery wedge and left atrial pressures can be avoided and that PEEP lacks an important effect on left ventricular compliance, correction for changes in intracavitary right heart filling pressure might strengthen the relationship between wedge pressures and true left ventricular preload in the presence of PEEP. However, the close association between the effects of PEEP on intracavitary RVEDP and intrapericardial pressure is largely a chance occurrence, dependent on offsetting changes in right ventricular volume. It remains to be determined whether the balance between these opposing effects is altered significantly by diseases that change either right ventricular compliance or the effects of PEEP on intrathoracic pressure and pulmonary vascular resistance.

I would like to express my appreciation to Stephen Bell, Patricia Schor, and Jane Smoak for their expert technical assistance, and to Mary Kohl, Barbara Breckenridge, and Jeanne Boschi for typing the manuscript.

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Volume-dependent effects of positive airway pressure on intracavitary left ventricular end-diastolic pressure.
R V Ditchey

Circulation. 1984;69:815-821
doi: 10.1161/01.CIR.69.4.815

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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