Continuous Positive-Pressure Ventilation Decreases Right and Left Ventricular End-Diastolic Volumes in the Dog

JAMES E. FEWELL, DANA R. ABENDSCHEIN, C. JEFFREY CARLSON, JOHN F. MURRAY, AND ELLIOT RAPAPORT

SUMMARY We investigated the mechanism(s) responsible for the decreased cardiac output during continuous positive-pressure ventilation (CPPV). Seven dogs were anesthetized with chloralose-urethane, intubated, and ventilated using a volume ventilator. We measured heart rate, stroke volume, and the determinants of stroke volume: left and right ventricular end-diastolic volumes, isovolumic and ejection phase indices of myocardial contractility, and pulmonary and systemic arterial pressures. Myocardial blood flow was estimated using radioactive microspheres. Variables were measured during a control period of intermittent positive-pressure ventilation (IPPV), 8-20 minutes after the initiation of CPPV using 12 cm H2O positive end-expiratory pressure (PEEP), and 8-20 minutes after the removal of PEEP. CPPV decreased cardiac output but did not affect total or regional myocardial blood flow or the ratio of subendocardial to subepicardial blood flow. Isovolumic and ejection phase indices of myocardial contractility, heart rate, and systemic arterial pressure did not change during CPPV. Right and left ventricular end-diastolic and end-systolic volumes decreased markedly during CPPV. We conclude that CPPV decreases cardiac output in accordance with Starling's law by decreasing preload.


CONTINUOUS positive-pressure ventilation (CPPV) is used to increase the arterial Po2 in patients with acute respiratory failure and hypoxemia refractory to usual oxygen therapy (Ashbaugh et al., 1969; McIntyre et al., 1969; Kumar et al., 1972; Falke et al., 1972). Although CPPV usually improves the arterial Po2, it also decreases cardiac output and may actually decrease the amount of oxygen transported to the tissues (Lutch and Murray, 1972).

Studies on animals (Tucker and Murray, 1972; Jones and King, 1973) and humans (Cournand et al., 1948; Powers et al., 1973) have demonstrated that CPPV decreases the cardiac output, but the mechanism is unclear. It generally was believed that cardiac output falls due to decreased venous return (Cournand et al., 1948; Ashbaugh and Petty, 1973; Qvist et al., 1975). If this were true, the effective or transmural filling pressure of the right atrium (right atrial pressure minus pleural pressure) would be expected to decrease during CPPV. However, several investigators have reported that transmural right and left atrial pressures, measured relative to lateral pleural or esophageal pressure, do not decrease, but stay the same or actually increase during CPPV (Scharf et al., 1977; Zarins et al., 1977; Cassidy et al., 1978). This suggests that decreased venous return is not the primary factor producing the decrease in cardiac output. A decrease in cardiac output in the presence of constant or increasing transmural atrial pressures may indicate a decrease in myocardial contractility. Lozman et al. (1974) and Powers and Dutton (1975) have suggested that ventricular dysfunction occurs during CPPV and may be related to decreased subendocardial blood flow.

The purpose of the present study was to clarify the mechanism(s) responsible for the decreased cardiac output observed during CPPV. Specifically, we measured stroke volume and its determinants: left and right ventricular end-diastolic volumes, isovolumic and ejection phase indices of myocardial contractility, and pulmonary and systemic arterial pressures. Furthermore, because of the suggestion that a decrease in subendocardial blood flow may produce ventricular dysfunction during CPPV, we also measured total and regional myocardial blood flow.

Methods

Animal Preparation

Seven mongrel dogs weighing 20–25 kg were anesthetized by intravenous injection of a mixture of chloralose (50 mg/kg) and urethane (500 mg/kg); additional anesthetic was administered every 15
minutes to maintain a constant level of anesthesia. Each dog was placed supine, and its trachea was intubated with a cuffed endotracheal tube. The cuff was inflated to a gas-tight fit, and the lungs were ventilated with a volume ventilator (Harvard Apparatus Respiration Pump, model 607) set to deliver a tidal volume of 15 ml/kg at a frequency of 12 breaths/min. The cephalic vein was cannulated for infusion of 0.9% sodium chloride (pH 7.4). A catheter was inserted into a femoral artery and advanced to the ascending aorta for measurement of systemic arterial pressure and heart rate.

A midsternal thoracotomy and pericardiotomy were performed. The heart and great vessels were instrumented as shown in Figure 1. High fidelity micromanometers (Konigsberg, P-17) were placed in the apex of the left ventricle and the lateral wall of the right ventricle for measurement of pressures. Electromagnetic flow probes (Biotronex Laboratory, Inc.) were placed around the ascending aorta and main pulmonary artery. Specially constructed thermocouples in 20-gauge needles (Bailey Instruments) were inserted into the ascending aorta and main pulmonary artery to measure changes in blood temperature. Catheters were inserted into the right and left atria and the coronary sinus. The edges of the pericardium then were apposed loosely, and a mushroom catheter was inserted into the pleural space at mid right atrial level. The sternum was stabilized and the skin sutured to produce an air-tight seal. The lungs were hyperinflated, and the pleural catheter was occluded to reestablish negative pleural pressure. The pleural catheter then was connected to a strain gauge manometer to measure pleural pressure.

Arterial blood gases and pH were measured intermittently to ensure adequate alveolar ventilation (Feigl and D'Alecy, 1972). Aortic blood temperature was maintained at 37°C by an external thermal blanket. Reference pressures in the pulmonary artery, thoracic aorta, and both atria were measured using fluid-filled catheters and Statham P23Db transducers. The gains of the ventricular micromanometer amplifiers were adjusted to equal the reference arterial pressure in systole and the appropriate atrial pressure in diastole. The rate of saline infusion was adjusted to maintain end-expiratory left atrial pressure at 4-6 mm Hg during the control period. The flow probes were calibrated in vivo using the radioactive microsphere technique to measure cardiac output.

Experimental Protocol

After completing the surgical preparation, the dogs were ventilated with intermittent positive-pressure ventilation (IPPV, 0 cm H₂O end-expiratory pressure) and allowed to stabilize for 60 minutes. Experimental variables then were measured during an initial control period of IPPV, during the interval 8-20 minutes after institution of CPPV using 12 cm H₂O positive end-expiratory pressure (PEEP) and during the interval 8-20 minutes after the expiratory pressure had been returned to zero. CPPV was accomplished by partial static inflation of an occlusive balloon manifold incorporated into the expiratory limb of the ventilator circuit. Variables were measured in the following sequence: blood gases and pH, cardiovascular and respiratory pressures, myocardial blood flow, and ventricular volumes.

Experimental Measurements

Blood Gases and pH

Arterial and coronary sinus blood samples were drawn into heparinized syringes and analyzed with a Corning 175 blood gas analyzer. Blood gas values were corrected to body temperature.

Cardiovascular and Respiratory Pressures

The following pressures were measured at end-expiration: right and left ventricular end-diastolic pressures, right and left ventricular peak systolic pressures, systemic arterial pressure, pulmonary arterial pressure, and lateral pleural pressure. These pressures were recorded on an Electronics for Medicine DR-8 optical recorder (Fig. 2). Transmural ventricular-filling pressure was calculated as end-diastolic pressure minus lateral pleural pressure. A spring-loaded manometer was incorporated into the ventilator circuit opposite the endotracheal tube port for continuous monitoring of proximal airway pressure. Left ventricular pressure, right ventricular pressure, aortic blood flow, and pulmonary blood
flow were recorded on a Hewlett Packard model 3960 tape recorder. Isovolumic and ejection phase indices of contractility were derived using an Electronics for Medicine ADV-23 analog data processor.

The derived isovolumic phase indices of contractility were: maximum dP/dt (Wallace et al., 1963; Mason, 1969; Furnival et al., 1970); ratio of maximum dP/dt to instantaneous developed pressure (dP/dt/IP); dP/dt at a developed pressure of 40 mm Hg (dP/dt/DP40, Davidson et al., 1974); and dP/dt at a developed pressure of 5 mm Hg (dP/dt/DP5). In all experiments, right ventricular maximum dP/dt preceded the onset of pulmonary artery blood flow.

The derived ejection phase indices of contractility were: systolic ejection period (obtained from the flow tracings); the ejection fraction (calculated as 1 - K, from the thermodilution measurements); and mean circumferential shortening rate (MCSR, calculated from end-diastolic volume, end-systolic volume, and the duration of the systolic ejection period, assuming a spherical shape for the left ventricle, Gorlin et al., 1964). Right ventricular MCSR was not calculated.

**Myocardial Blood Flow**

Total and regional blood flows were measured by the method of Heymann et al. (1977) using 15 ± 3 μm microspheres labeled with 153Ce, 51Cr, 85Sr, or 95Nb (3M). The microspheres (suspended in 10% dextran) were added to specially constructed injection vials. Before each injection, the vials were placed in an ultrasonicator for several minutes to dissociate microsphere aggregates. Approximately 900,000 microspheres were injected into the left atrium over a 30-second period. A reference blood sample was withdrawn from a femoral artery (phantom organ) at 8 ml/min during and 120 seconds after the injection. The radionuclides were injected in random sequence.

After the experiment, the heart was removed and placed in 10% formalin for 3–5 days. The atria and great vessels were discarded and the ventricles were cut into three transverse sections from base to apex. In each section, the left ventricular free wall and the septum were divided into four layers of equal thickness, and the right ventricular free wall was divided into two layers. One- to 4-g samples were placed in plastic counting vials with 10% formalin. Myocardial samples and reference blood samples were counted for 5 minutes (minimum of 10,000 counts per sample) in a Packard 3002 series automatic γ counter. Absolute total myocardial blood flow, regional myocardial flow per gram of wet tissue, and cardiac output were calculated using a Hewlett Packard 9830 computer.

**Ventricular Volumes**

Right and left ventricular volumes were measured by the thermodilution technique (Keroes and Rapaport, 1972). The residual fraction (K) was calculated from three to five successive heart beats.
starting with the second beat after the injection of cold saline. A typical thermodilution curve is shown in Figure 3. Knowledge of K and stroke volume (SV) permitted calculation of end-diastolic volume (EDV) by the formula, EDV = SV/1 - K. Ventricular end-diastolic volumes were calculated from the average of 8 to 10 successive thermodilution curves. Right ventricular volumes were calculated from thermal curves recorded from the pulmonary artery after right atrial injection of cold saline, whereas left ventricular volumes were calculated from thermal curves recorded from the aorta after left atrial injection of cold saline.

Statistical Analysis

Statistical analysis was performed using a one-way analysis of variance for repeated measures on the same factor (Winer, 1971). The Dunnett multiple range t-test then was used to determine which variable means were statistically different from the mean of the control period at the 0.05 level of significance (Steel and Torrie, 1960).

Results

The initial cardiovascular response to a 12-cm H2O change in expiratory pressure is presented in Figure 4. Pulmonary and aortic blood flow changed within two ventilatory cycles after the expiratory pressure was altered. Blood flow changes in the pulmonary artery preceded blood flow changes in the aorta by two to three heart beats. Systemic arterial pressure changed transiently but returned toward the control level by 3-5 minutes. Dramatic changes in pulse pressure were produced by altering expiratory pressure.

Respiratory variations in blood flow occurred during both IPPV and CPPV. Inspiration decreased pulmonary blood flow within one to two heart beats. Pulmonary blood flow continued to decrease for the next three to five heart beats and was lowest when pleural pressure reached maximum. On the other hand, aortic blood flow was constant or slightly increased during the initial phase of inspiration. The lowest aortic blood flow occurred three heart beats after pleural pressure reached maximum. The phase lag between pulmonary and aortic blood flow during tidal ventilation is similar to that described by Charlier (1967). We also observed variations in aortic blood pressure which were in phase with variations in aortic blood flow and pleural pressure during both IPPV and CPPV. However, there was one exception. Variations in aortic blood pressure were out of phase with variations in aortic blood flow and pleural pressure during the second and third tidal ventilations after the removal of PEEP. This is most likely the result of a reflex decrease in peripheral vascular resistance mediated by the arterial baroreceptors in response to the dramatic increase in systemic arterial pressure.

A summary of the hemodynamic and respiratory data during CPPV appears in Table 1. Cardiac output decreased an average of 26%. This was the result of a decreased stroke volume because heart rate was unchanged. Although mean arterial pressure decreased transiently with the onset of CPPV, it returned toward the control level by 3-5 minutes and did not differ statistically from control at 8 minutes. Left and right ventricular end-diastolic pressures increased during CPPV and paralleled changes in lateral pleural pressure. Left and right ventricular end-diastolic and end-systolic volumes decreased markedly during CPPV.

Although cardiac output decreased, there were no significant changes in total or regional coronary blood flow during CPPV (Fig. 5). In Figure 6, myocardial blood flow to the subendocardium and subepicardium of the left ventricle, septum, and right ventricle is presented. There were no significant changes in absolute flow or the ratio of subendocardial to subepicardial flow in any region during CPPV.

The isovolumic and ejection phase indices of myocardial contractility during CPPV are summarized in Table 2. Although small changes in several of these measures were observed, they were not statistically significant.

The application of CPPV produced a decrease in arterial pH, but no significant changes in arterial or coronary sinus PO2 or PCO2 occurred. A slight metabolic acidosis continued after PEEP was removed.

Discussion

We have evaluated the determinants of cardiac output during CPPV and have demonstrated that CPPV decreases right and left ventricular end-diastolic volumes. We also have presented evidence that myocardial contractility and myocardial blood flow do not decrease during CPPV. Because left ventricular volume fell and systemic arterial pressure remained constant, afterload (defined as wall force during systole) was clearly less during CPPV than IPPV, a change which would tend to increase
rather than decrease stroke output. End-diastolic volume returned to the control level after the removal of PEEP despite a continued metabolic acidosis. We conclude that CPPV decreases cardiac output and stroke volume primarily by decreasing preload.

Manny et al. (1978) have suggested that a humoral factor decreases myocardial contractility and cardiac output during CPPV. These investigators plotted Starling's curves from left ventricular end-diastolic and end-systolic pressures measured in isolated canine hearts perfused by donor dogs. Application of 15 cm H$_2$O PEEP to donor dogs decreased contractility in the isolated hearts as evidenced by a downward shift in the Starling's curves. Although a humoral factor that decreases contractility in the isolated heart may be released during CPPV, this clearly was not the case in our studies using intact dogs. Furthermore, in our study, cardiac output changed within one to two ventilatory cycles after the airway pressure pattern was altered, and flow changes in the pulmonary artery preceded those in the aorta by two to three heart beats. The timing and sequence of flow changes that we observed do not support a humoral mechanism.

Cassidy et al. (1978) have suggested that hyperinflation of the lung during CPPV produces a reflex depression of ventricular function that decreases cardiac output. Glick and associates (1969) have demonstrated that lung inflation using 20 mm Hg airway pressure produced a transient decrease in myocardial contractility, heart rate, and total peripheral resistance in dogs on either right heart bypass or total cardiopulmonary bypass. However, the cardiovascular depression produced by positive pressure inflation of the lungs was relatively short lived. Contractility, heart rate, and total peripheral resistance fell within 3-5 seconds but returned toward control levels within 15-25 seconds. The transient nature of this response makes it difficult to

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**Figure 4** Hemodynamic response to application (upper panel) and removal (lower panel) of 12 cm H$_2$O positive end-expiratory pressure. Abbreviations are the same as in Figure 2.
TABLE 1  Effect of Continuous Positive-Pressure Ventilation on Cardiovascular and Respiratory Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>IPPV</th>
<th>CPPV</th>
<th>IPPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intrapleural pressure (mm Hg)</td>
<td>-1.9 ± 1.12</td>
<td>1.0 ± 0.86*</td>
<td>-2.1 ± 0.72</td>
</tr>
<tr>
<td>Cardiac output (ml/min)</td>
<td>3204 ± 518.1</td>
<td>2356 ± 789.0*</td>
<td>3087 ± 948.7</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>136 ± 18.0</td>
<td>147 ± 9.9</td>
<td>132 ± 14.8</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>23.9 ± 5.08</td>
<td>16.1 ± 5.41*</td>
<td>23.6 ± 7.78</td>
</tr>
<tr>
<td>Aortic pressure (mm Hg)</td>
<td>125.0 ± 14.93</td>
<td>120.3 ± 16.92</td>
<td>123.3 ± 16.52</td>
</tr>
<tr>
<td>Pulmonary artery pressure (mm Hg)</td>
<td>15.7 ± 2.11</td>
<td>19.3 ± 3.55*</td>
<td>15.3 ± 3.38</td>
</tr>
<tr>
<td>Transmural pulmonary artery pressure (mm Hg)</td>
<td>17.5 ± 2.16</td>
<td>18.4 ± 3.80</td>
<td>17.4 ± 3.32</td>
</tr>
<tr>
<td>Left ventricle</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>End-diastolic pressure (mm Hg)</td>
<td>4.0 ± 2.34</td>
<td>6.2 ± 2.76*</td>
<td>4.4 ± 2.20</td>
</tr>
<tr>
<td>Transmural end-diastolic pressure (mm Hg)</td>
<td>5.8 ± 2.19</td>
<td>5.7 ± 3.52</td>
<td>6.5 ± 2.28</td>
</tr>
<tr>
<td>End-diastolic volume (ml)</td>
<td>57.4 ± 8.50</td>
<td>40.2 ± 12.53*</td>
<td>55.0 ± 21.79</td>
</tr>
<tr>
<td>End-systolic volume (ml)</td>
<td>33.5 ± 7.25</td>
<td>24.1 ± 7.40</td>
<td>31.4 ± 14.51</td>
</tr>
<tr>
<td>Right ventricle</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>End-diastolic pressure (mm Hg)</td>
<td>3.6 ± 2.33</td>
<td>5.9 ± 2.50*</td>
<td>3.1 ± 1.83</td>
</tr>
<tr>
<td>Transmural end-diastolic pressure (mm Hg)</td>
<td>5.4 ± 2.82</td>
<td>5.0 ± 2.56</td>
<td>5.2 ± 1.71</td>
</tr>
<tr>
<td>End-diastolic volume (ml)</td>
<td>51.3 ± 12.30</td>
<td>33.8 ± 10.17*</td>
<td>51.1 ± 16.39</td>
</tr>
<tr>
<td>End-systolic volume (ml)</td>
<td>27.4 ± 7.80</td>
<td>17.8 ± 4.99*</td>
<td>27.5 ± 8.72</td>
</tr>
</tbody>
</table>

Values are means ± 1 SD for seven experiments. * Indicates a significant difference, at the 0.05 level of significance, from the mean of the control period.

believe that a neural mechanism plays a major role in decreasing cardiac output during CPPV. Qvist et al. (1975) have shown that CPPV can decrease cardiac output for up to 8 hours. Furthermore, several investigators (Marotta and Harner, 1962; Maulsby and Hoff, 1962; Wong et al., 1967; Scharf et al., 1977) have demonstrated that bilateral cervical vagotomy, which interrupts most afferent nerve fibers from the lungs, does not alter the cardiovascular response to CPPV.

Figure 5  Effect of continuous positive-pressure ventilation on total and regional myocardial blood flow. Values represent means ± 1 SD. S = septum. No statistically significant differences were observed.

Figure 6  Effect of continuous positive-pressure ventilation on myocardial blood flow to the subendocardium and subepicardium of the left ventricle, septum, and right ventricle. Values represent means ± 1 SD. Abbreviations are the same as in Figure 5. No statistically significant differences in absolute flow or in the ratio of subendocardial to subepicardial flow were observed in any region.
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TABLE 2  Effect of Continuous Positive-Pressure Ventilation on Isovolumic and Ejection Phase Indices of Contractility

<table>
<thead>
<tr>
<th>Variable</th>
<th>Ventilatory pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IPPV</td>
</tr>
<tr>
<td></td>
<td>CPPV</td>
</tr>
<tr>
<td></td>
<td>IPPV</td>
</tr>
<tr>
<td>Isovolumic indices</td>
<td></td>
</tr>
<tr>
<td>Left ventricle</td>
<td></td>
</tr>
<tr>
<td>(dP/dt) (mm Hg/sec)</td>
<td>2778 ± 596.1</td>
</tr>
<tr>
<td>(dP/dt/IP) (sec(^{-1}))</td>
<td>30 ± 5.3</td>
</tr>
<tr>
<td>(dP/dt/DP40) (sec(^{-1}))</td>
<td>1628 ± 305.9</td>
</tr>
<tr>
<td>(dP/dt/DP5) (sec(^{-1}))</td>
<td>384 ± 89.7</td>
</tr>
<tr>
<td>Right ventricle</td>
<td></td>
</tr>
<tr>
<td>(dP/dt) (mm Hg/sec)</td>
<td>491 ± 103.8</td>
</tr>
<tr>
<td>(dP/dt/IP) (sec(^{-1}))</td>
<td>40 ± 6.3</td>
</tr>
<tr>
<td>(dP/dt/DP5) (sec(^{-1}))</td>
<td>269 ± 28.1</td>
</tr>
<tr>
<td>Ejection indices</td>
<td></td>
</tr>
<tr>
<td>Left ventricle</td>
<td></td>
</tr>
<tr>
<td>ejection time (msec)</td>
<td>142 ± 13.4</td>
</tr>
<tr>
<td>ejection fraction</td>
<td>0.42 ± 0.078</td>
</tr>
<tr>
<td>MCSR (circ/sec)</td>
<td>17.6 ± 2.99</td>
</tr>
<tr>
<td>Right ventricle</td>
<td></td>
</tr>
<tr>
<td>ejection time (msec)</td>
<td>210 ± 12.9</td>
</tr>
<tr>
<td>ejection fraction</td>
<td>0.47 ± 0.041</td>
</tr>
</tbody>
</table>

Values are means ± 1 SD for seven experiments. No significant differences, at the 0.05 level of significance, from the control means were observed.

It is likely, then, that CPPV decreases cardiac output primarily by reducing right and left ventricular end-diastolic volumes. Although the present study was not designed to determine the mechanism producing the decreased end-diastolic volumes during CPPV, we can speculate on some possible causes. If lateral pleural pressure is an accurate reflection of the actual pressure surrounding the heart during CPPV, then it appears that the decreased end-diastolic volume of both ventricles was the result of a decrease in ventricular end-diastolic compliance; i.e., decreased end-diastolic volume without a change in transmural ventricular end-diastolic pressure. However, if one considers the factors that have been shown to alter the diastolic pressure-volume relationship, this explanation seems unlikely. Variations in heart rate have been demonstrated to alter ventricular end-diastolic compliance (Braunwald et al., 1960), but in our study, heart rate did not change during CPPV. CPPV may alter neurohumoral stimuli, but evidence indicates that changes in circulating catecholamines and autonomic discharge to the heart do not decrease ventricular end-diastolic compliance (Wiggers, 1927; Mitchell et al., 1960; Hefner et al., 1961; Wildenthal et al., 1969a; Wildenthal et al., 1969b). Changes in coronary perfusion pressure or coronary blood flow can alter ventricular end-diastolic compliance (Cross et al., 1961), but these variables did not change during CPPV in our study. Therefore, it seems unlikely that decreased ventricular end-diastolic compliance decreases end-diastolic volumes during CPPV.

If lateral pleural pressure underestimates the pressure around the heart during CPPV, then an alternate explanation for our findings is that decreased end-diastolic volumes may have resulted from a decrease in transmural ventricular end-diastolic filling pressures. Brookhart and Boyd (1947) measured lateral pleural pressure and lateral pericardial pressure in dogs during continuous positive-pressure breathing (spontaneous breathing with positive end-expiratory pressure) and found that changes in lateral pleural pressure did, indeed, underestimate changes in lateral pericardial pressure. Although our evidence does not differentiate between the two possibilities, we favor the suggestion that the decrease in end-diastolic volumes during CPPV is the result of a decrease in transmural ventricular end-diastolic filling pressures and not from increased stiffness of the myocardium.

A decrease in transmural ventricular end-diastolic pressures during CPPV could result from amplified transmission of increased pleural pressure to the heart. The greater increase in pressure around the heart could result from compression of the heart by the expanded lungs or from pericardial traction due to the depressed diaphragm.

The decrease in end-systolic volumes during CPPV may be related to the effects of PEEP on the pressure around the heart as postulated by Summer et al., (1979). When PEEP is applied, the increase in pressure around the heart relative to the pressure in the aorta enhances ventricular ejection, which allows the heart to empty more completely.

Acknowledgments

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