Cardiorespiratory Effects of Pressure Controlled Ventilation in Severe Respiratory Failure*  

Edward Abraham, M.D., F.C.C.P.; and Gary Yoshihara, R.R.T.

Cardiorespiratory values were measured in ten patients with severe respiratory failure on volume controlled and pressure controlled ventilation. Tidal volume, respiratory rate, PEEP; auto-PEEP, inspiratory:expiratory ratio (I:E) and FlO₂ were maintained at the same value for both ventilatory modalities. Changing from VCV to PCV was associated with significant improvements in PaO₂, oxygen delivery, and tissue oxygen consumption. Peak inspiratory pressure fell. There were no significant changes in other cardiorespiratory values, such as arterial blood pressure, nor in ventilatory measurements, such as mean airway pressure, associated with the use of PCV. These results suggest that PCV may be a beneficial ventilatory modality in the treatment of severe respiratory failure since it results in improvement in arterial oxygenation, tissue oxygen delivery and utilization without any concomitant adverse effects on other hemodynamic or ventilatory factors.

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WF = wedge pressure; PIP = peak airway pressure; Paw = mean airway pressure; O₂, Est = oxygen extraction ratio; Ce = effective pulmonary compliance

Critically ill patients with multiple organ system failure (MOSF) frequently demonstrate abnormalities in tissue oxygen delivery and utilization.1,2 In septic syndromes, even though cardiac index, ÐO₂, and oxygen consumption already may be increased, further augmentation in ÐO₂ and CI through the use of inotropes or volume therapy often leads to additional increases in VO₂, demonstrating the presence of unmet tissue oxygen needs.3,4 A similar situation exists in patients with the adult respiratory distress syndrome where large tissue oxygen needs often are unmet by normal physiologic compensatory mechanisms, as shown by increases in VO₂, which occur when ÐO₂ is made to rise.5,9 Several studies10-12 have suggested that outcome in critically ill patients could be improved through early optimization in cardiorespiratory parameters, particularly through increasing ÐO₂ to meet tissue oxygen needs.

Several ventilatory modalities, capable of improving PaO₂ beyond the level achieved with volume controlled ventilation, are available for use in patients with severe respiratory failure. Unfortunately, there is often a conflict between the ability of these techniques to maintain adequate oxygenation and their deleterious effects on CI and ÐO₂. This problem is particularly apparent with the use of PEEP, where increased levels of PEEP, although resulting in improved PaO₂, also produce diminished CI and ÐO₂.14 Similar negative hemodynamic effects have been associated with pressure-controlled inverse ratio ventilation (PC-IRV).15,16

Intermittent positive pressure ventilation using preset pressure limits (PCV) previously was widely used in adults, and continues to be used in neonates with respiratory failure.17 However, because of risks of hypventilation secondary to changes in pulmonary compliance and problems with auto-PEEP, this ventilatory mode became less frequently employed when volume preset ventilators were developed. Recently, new ventilators able to provide PCV, but which continue to monitor tidal volume and the level of auto-PEEP have become available. With these new ventilators, the previous problems with the use of PCV have been largely eliminated, but the utility of this ventilatory mode in patients with severe respiratory failure remains unclear. PCV has been proposed to recruit closed alveolar units and improve oxygenation through changing the inspiratory flow pattern from a square wave as used with VCV to a rapidly exponentially decaying curve and through maintaining airway pressures at a constant level throughout the inspiratory phase.17,18 Despite these theoretic reasons for the use of PCV, which suggested that this ventilatory modality might result in improved oxygenation, little is known...
of the effects of PCV on cardiorespiratory variables in critically ill patients. We initiated the present prospective study to better examine the use and physiologic effects of PCV in patients with severe respiratory failure.

**Methods**

**Patients**

Ten patients (Table 1) with severe ARDS, as manifested by diffuse pulmonary infiltrates on chest roentgenograms, arterial hypoxemia with widened A-a gradients despite supplemental oxygen, pulmonary capillary wedge pressures less than 20 mm Hg, and decreased static and dynamic thoracic compliance, were entered in the study. In each case, the patient was placed on pressure controlled ventilation at the request of the attending physician, who judged the patient to be failing conventional volume controlled ventilation. In all cases, a flow-directed pulmonary artery (Swan-Ganz) catheter with a fiberoptic channel for the continuous measurement of mixed venous oxygen saturation had been placed previously for hemodynamic and cardiorespiratory monitoring. The mixed venous oxygen saturation determination from the pulmonary artery catheter was calibrated and verified by using a mixed venous blood sample in which the oxygen saturation was measured by COoximetry. All patients had indwelling arterial catheters and pulse oximeters. All patients previously had been placed on a Servo-controlled ventilator (Siemens 900C), operating in the volume controlled mode. All patients were sedated with appropriate doses of benzodiazepines and paralyzed with vecuronium by continuous intravenous infusion, after an initial bolus dose.

**Pressure Control Trial**

Prior to the initiation of pressure controlled ventilation, all patients had been paralyzed, sedated and ventilated in a volume control mode, with inspiratory time of 33 percent, for at least two hours. In each patient, before being changed to pressure controlled ventilation, a full set of hemodynamic and cardiorespiratory variables was measured. This included measurement of arterial systolic and diastolic pressure, heart rate, right atrial pressure, pulmonary artery systolic and diastolic pressure, and pulmonary capillary wedge pressure. Cardiac output was measured in triplicate by the thermodilution technique at end expiration. The timing of injection for cardiac output measurement was supervised by one of the authors (E.A.) to verify that the injection was initiated at the same point in the respiratory cycle. Variability among the three cardiac output determinations was less than 10 percent. Determination of arterial blood gases (PaO₂, PaCO₂, pH), arterial oxygen saturation (SaO₂), measured directly by COoximetry, and mixed venous oxygen saturation (SvO₂) were made. Inspired oxygen concentration, respiratory rate, tidal volume, PEEP, peak airway pressures, mean airway pressure, and "auto-PEEP" (using an end-expiratory pause hold) were measured.

The patient then was placed on pressure controlled ventilation. The inspiratory pressure (above the level of PEEP) was adjusted to achieve a tidal volume equal to the tidal volume received on volume controlled ventilation. All other ventilatory parameters (ie, FIO₂, respiratory rate, PEEP, and percentage inspiratory time [33 percent]) were maintained. The auto-PEEP again was measured.

After a 60-minute stabilization period on pressure controlled ventilation, another full set of cardiorespiratory, ventilatory, and blood gas measurements, as described above, was obtained. All pressure control trials could be continued at least for the 60 minute measurement period. In patients with improved oxygenation and without hemodynamic compromise, the pressure control mode was used for periods as long as 72 hours. Administration rates for fluids and isotropic drugs were kept at the same level during the pressure control trial as had been used when cardiorespiratory measurements were taken during volume controlled ventilation, immediately prior to the institution of pressure control.

**Data Analysis**

Derived cardiorespiratory variables, mean arterial pressure, systemic vascular resistance, pulmonary vascular resistance, left and right work indices, arterial and mixed venous oxygen content, oxygen delivery, oxygen consumption, and oxygen extraction ratio were calculated using standard and previously described formulae. Where appropriate, cardiorespiratory and hemodynamic variables were normalized, using the calculated body surface area for each patient. Effective pulmonary compliance was defined as tidal volume/(PIF-PEEP), and was calculated for each patient on volume

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/Sex</th>
<th>Major Clinical Problems</th>
<th>PEEP (cm H₂O)</th>
<th>Auto-PEEP (cm H₂O)</th>
<th>FIO₂</th>
<th>TV, ml</th>
<th>RR</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>41/M</td>
<td>Chronic myelogenous leukemia, postbone marrow transplantation, GI bleed</td>
<td>12</td>
<td>0</td>
<td>1.0</td>
<td>700</td>
<td>40</td>
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<tr>
<td>2</td>
<td>47/F</td>
<td>Sepsis, renal failure</td>
<td>10</td>
<td>0</td>
<td>1.0</td>
<td>800</td>
<td>15</td>
</tr>
<tr>
<td>3</td>
<td>50/F</td>
<td>Aspiration pneumonia, postphip fracture, renal failure</td>
<td>10</td>
<td>0</td>
<td>1.0</td>
<td>800</td>
<td>30</td>
</tr>
<tr>
<td>4</td>
<td>25/M</td>
<td>Gunshot wound to neck, postcardiac arrest</td>
<td>22</td>
<td>0</td>
<td>1.0</td>
<td>800</td>
<td>20</td>
</tr>
<tr>
<td>5</td>
<td>42/F</td>
<td>Aspiration pneumonia, systemic lupus erythematosus</td>
<td>8</td>
<td>0</td>
<td>1.0</td>
<td>650</td>
<td>12</td>
</tr>
<tr>
<td>6</td>
<td>45/F</td>
<td>Sepsis, chronic myelogenous leukemia, postbone marrow transplantation</td>
<td>10</td>
<td>4</td>
<td>1.0</td>
<td>800</td>
<td>28</td>
</tr>
<tr>
<td>7</td>
<td>34/F</td>
<td>Metastatic melanoma</td>
<td>5</td>
<td>0</td>
<td>1.0</td>
<td>600</td>
<td>25</td>
</tr>
<tr>
<td>8</td>
<td>49/M</td>
<td>Rheumatoid arthritis, cytomegalovirus pneumonia</td>
<td>10</td>
<td>0</td>
<td>1.0</td>
<td>800</td>
<td>15</td>
</tr>
<tr>
<td>9</td>
<td>34/F</td>
<td>Chronic myelogenous leukemia, postbone marrow transplantation, viral pneumonia</td>
<td>5</td>
<td>11</td>
<td>1.0</td>
<td>800</td>
<td>35</td>
</tr>
<tr>
<td>10</td>
<td>72/M</td>
<td>Viral pneumonia</td>
<td>10</td>
<td>0</td>
<td>1.0</td>
<td>850</td>
<td>35</td>
</tr>
</tbody>
</table>

TV = tidal volume; RR = respiratory rate.
and pressure controlled ventilation.

Mean ± standard error of the mean was calculated for each of the cardiorespiratory, ventilatory, and blood gas variables, for the volume controlled and pressure controlled measurement periods. Comparison between variables before and after initiation of pressure controlled ventilation was performed by a paired Student’s t-test with Bonferroni corrections. Differences were considered to be significant for p < 0.05.

### Results

Table 1 summarizes the characteristics of the patient population studied. Four men and six women were included. The average age was 48 ± 6 years.

Arterial blood gas values, arterial and mixed venous oxygen saturation, airway pressures and compliance before and after institution of PCV are presented in Table 2. All patients had severe pulmonary compromise, with decreased compliance, elevated peak and mean airway pressures, and markedly widened A-a gradients.

### Table 2—Ventilatory and Blood Gas Factors with Volume and Pressure Controlled Ventilation*

<table>
<thead>
<tr>
<th></th>
<th>Volume Control</th>
<th>Pressure Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO₂, mm Hg</td>
<td>80 ± 9</td>
<td>92 ± 8†</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>44 ± 3</td>
<td>41 ± 3</td>
</tr>
<tr>
<td>pH</td>
<td>7.38 ± 0.03</td>
<td>7.38 ± 0.02</td>
</tr>
<tr>
<td>SaO₂, %</td>
<td>93 ± 1</td>
<td>95 ± 1</td>
</tr>
<tr>
<td>SvO₂, %</td>
<td>70 ± 3</td>
<td>72 ± 3</td>
</tr>
<tr>
<td>PIP, cm H₂O</td>
<td>62 ± 5</td>
<td>45 ± 3†</td>
</tr>
<tr>
<td>Pao2, cm H₂O</td>
<td>20 ± 1</td>
<td>21 ± 1</td>
</tr>
<tr>
<td>Ce</td>
<td>17 ± 2</td>
<td>23 ± 2†</td>
</tr>
</tbody>
</table>

*pValues are ± SEM.
tp < 0.05 vs volume controlled ventilation.
†p < 0.01 vs volume controlled ventilation.

PaO₂ rose in eight of ten patients after initiation of PCV, with the range of improvement being 1 to 54 mm Hg. Although SaO₂ rose with the use of PCV in the eight patients with increased PaO₂, the change generally was small since the initial SaO₂ was 89 percent or better in all patients. The PIP decreased in nine patients with pressure control, resulting in a significant increase in CE. Mean airway pressures were similar with both ventilatory modes. There was no change in the level of auto-PEEP in any patient after the institution of PCV.

Cardiorespiratory parameters with volume and pressure controlled ventilation are shown in Table 3. Pulmonary artery pressures, pulmonary vascular resistance, and shunt fraction (Qsp/Qt) were elevated in all patients, consistent with their severe respiratory failure. Right cardiac work index was increased in all patients, secondary to the high pulmonary artery pressures and vascular resistance.

Both Do₂ and VO₂ increased significantly with the initiation of PCV. The Do₂ rose in seven patients after PCV was instituted, and cardiac output rose in all of these. In contrast, two patients increased Do₂ with PCV despite a fall in PaO₂ and CaO₂. In all patients with increased Do₂ on PCV, a rise in VO₂ also was found.

There were no apparent adverse cardiorespiratory effects associated with the institution of PCV. No patient had a greater than 10 percent change in blood pressure when switched from VCV to PCV. In patients 6 and 10, CI decreased by approximately 15 percent when PCV was begun, but because of increased SVRI, neither patient demonstrated a fall in blood pressure.

Two patients, numbers 3 and 6, had a decrease in PaO₂ with the initiation of PCV. In patient 3, PaO₂ fell (from 105 mm Hg on VCV to 78 mm Hg with PCV). Despite this, Do₂ rose (from 285 ml/min/m² on VCV to 370 ml/min/m² on PCV), and VO₂ also increased (from 106 ml/min/m² to 137 ml/min/m²), reflecting an improvement in CI (from 2.3 L/min/m² to 3.0 L/min/m²) with the change to PCV. In contrast, in patient 6, cardiorespiratory changes associated with the decrease in PaO₂ (from 146 mm Hg on VCV to 114 mm Hg on PCV) included decreases in CI (from 5.4 L/min/m² to 4.6 L/min/m²), Do₂ (from 819 ml/min/m² to 866 ml/min/m²), and VO₂ (from 120 ml/min/m² to 84 ml/min/m²).

### Discussion

Increases in Paw and auto-PEEP have been suggested as mechanisms for the improved oxygenation found with pressure-controlled inverse ratio ventilation.9,21 In the present study, no alterations in auto-PEEP occurred when patients were switched from VCV to PCV. Similarly, no significant changes in Paw were found between VCV and PCV. These results...
show that the improvement in PaO\textsubscript{2} with PCV was not due to effects on either auto-PEEP or P\textsubscript{aw}.

In this study, altering the ventilation mode from VCV to PCV resulted in increased PaO\textsubscript{2}, DO\textsubscript{2}, and Vo\textsubscript{2}. Conventional I:E ratios of 1:2 were maintained for both VCV and PCV, and tidal volume, respiratory rate, PEEP, auto-PEEP, F\textsubscript{1}\textsubscript{O\textsubscript{2}}, and minute ventilation were kept at the same values for each ventilatory modality. Because no changes in SvO\textsubscript{2} nor in Qsp/Qt were found, the improvement in PaO\textsubscript{2} associated with the use of PCV appeared to result from a decrease in the degree of ventilation/perfusion (V/Q) mismatching.

The improvement in PaO\textsubscript{2} associated with the use of PCV probably resulted from the decelerating inspiratory waveform required for this ventilatory modality. In PCV, inspiratory flow is initially high (230 to 250 L/min), so that the preset pressure is reached rapidly. Airway pressure is then maintained throughout the inspiratory phase, with decreasing rates of flow. A constant inspiratory flow rate, with gradually increasing airway pressure, is used with VCV to attain the preset volume. It has been postulated that the high initial peak flow associated with the decelerating inspiratory flow pattern used in PCV can result in recruitment and improved ventilation of alveoli with prolonged time constants. Jansson and Jonson\textsuperscript{29} performed a theoretic analysis of ventilator flow patterns, using computer modeling of airway anatomy and resistance, and found that a decelerating inspiratory flow pattern produced a more even distribution of ventilation than was present when constant or accelerating flow patterns were used. In studies\textsuperscript{30,34} comparing the effects of decelerating and constant inspiratory flow waveforms in patients with respiratory failure, significant improvement in PaO\textsubscript{2}, static and dynamic compliance, as well as V\textsubscript{d}/V\textsubscript{T} occurred with the use of a decelerating waveform when tidal volume, inspiratory time, I:E ratio and respiratory rate were kept constant.

A significant increase in Vo\textsubscript{2} followed the institution of PCV, occurring in the same patients with improvement in Do\textsubscript{2}. This finding was not unexpected in the patients studied, all of whom had severe respiratory failure, and most of whom also had dysfunction of other organ systems. Previous studies have shown dependence of Vo\textsubscript{2} on Do\textsubscript{2} in the setting of ARDS, sepsis, and multiple organ system failure, consistent with inadequate tissue perfusion in these states.\textsuperscript{1,6}

The effects of PCV on patient outcome were not examined in this study, since we were initially unsure if PCV would be associated with any beneficial physiologic effects. The patients included had severe respiratory failure with multiple organ system dysfunction and because of these problems would have been expected to exhibit a high mortality rate.\textsuperscript{35,36} Increased mortality was, in fact, demonstrated in the present study since only two of the ten patients survived to leave the hospital. It is unlikely that institution of PCV late in these patients' clinical courses, after multiple organ system dysfunction already was manifest, would have had a meaningful impact on outcome. Because most evidence suggests that optimization of tissue oxygenation improves morbidity and mortality in critically ill patients only if utilized early in their clinical course, before multiple organ system failure develops, future studies examining the role of PCV probably will require use of this ventilatory modality at an early stage in patients' hospitalizations, when respiratory failure and overall physiologic state is less severe than that examined in this series.

All patients in this series were studied with pulmonary artery catheters in place and were both sedated and paralyzed. The lack of adverse hemodynamic consequences with PCV would suggest that invasive monitoring is not necessary when this mode of ventilation is utilized. Similarly, although some unparalyzed patients do not tolerate the high flow rates and respiratory pattern of PCV, further experience with this form of ventilation indicates that paralysis is not always necessary to use PCV.

Achievement of adequate tissue perfusion and oxygen delivery to meet cellular metabolic needs is an important physiologic goal in critically ill patients. The present study demonstrates that changing the pattern of mechanically delivered breaths in patients with severe respiratory failure by using PCV in the place of VCV can produce improvement in PaO\textsubscript{2}, DO\textsubscript{2} and Vo\textsubscript{2}, while decreasing PIP, and without changing P\textsubscript{aw}. Further studies will be necessary to determine if these beneficial physiologic effects of PCV will result in improved outcome in critically ill patients.

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