Effects of Continuous Positive Airway Pressure/Positive End-Expiratory Pressure and Pressure-Support Ventilation on Work of Breathing, Using an Animal Model

Mark J Heulitt MD FAARC, Shirley J Holt RRT, Tracy L Thurman, Sterling Wilson MSc, and Renée A Hall MSc

OBJECTIVE: Evaluate the effects of continuous positive airway pressure (CPAP)/positive end-expiratory pressure (PEEP) and pressure support ventilation (PSV) on work of breathing (WOB).

METHODS: With 13 anesthetized lambs we measured WOB with an esophageal balloon and flow signals. All the animals were sedated, intubated, and ventilated, using 2 pediatric ventilators (Servo 300 and VIP Bird). Ventilator settings were CPAP of 0, 5, and 10 cm H2O and PSV of 5 and 10 cm H2O with PEEP of 0, 5, and 10 cm H2O. Data were analyzed with 2-way analysis of variance.

RESULTS: With the Servo 300 the total WOB (WOB_T) increased between CPAP/PEEP of 0 and 10 cm H2O (p < 0.0001) and between CPAP/PEEP of 5 and 10 cm H2O (p < 0.0002). With the Servo 300 the addition of PSV decreased WOB_T (p < 0.003). With the VIP Bird the WOB_T significantly increased between CPAP/PEEP of 0 and 10 cm H2O (p ≤ 0.02) and between CPAP/PEEP of 5 and 10 cm H2O (p ≤ 0.03). With PSV the WOB_T was lower only at PSV 10 cm H2O (p ≤ 0.0001). CONCLUSIONS: WOB_T increased as CPAP/PEEP was increased, and PSV lowered WOB_T. We hypothesize that in a healthy animal model increased CPAP/PEEP may cause alveolar overdistention. Key words: mechanical ventilation, ventilator, positive pressure, respiration, work of breathing. [Respir Care 2003;48(7):689–696. © 2003 Daedalus Enterprises]

Introduction

To achieve normal ventilation the body performs work, known as work of breathing (WOB), to overcome the elastic and frictional resistance of the lungs and chest wall. Total WOB (WOB_T) is the elastic work (WOB_E) plus the resistive work (WOB_R). WOB_E represents physiologic work, which includes the work to expand the lungs and chest wall. WOB_R is considered a measure of imposed WOB and includes work caused by the breathing apparatus (endotracheal tube [ETT], breathing circuit, and ventilator’s demand-flow system). The artificial airway is responsible for a large part of the imposed WOB_R, and the mechanical ventilator causes some of the remaining WOB_R.1

Clinicians have long recognized qualitatively that there is above-normal WOB in pediatric patients weaning from prolonged mechanical ventilation. Patient-related factors, equipment factors, and clinical decision-making affect weaning. Equipment factors relate to the mechanical ventilator’s ability to meet the patient’s needs. It was previously demonstrated with a lung model that the WOB depends on the device used.2 Equipment factors are more important with pediatric patients than with adult patients, because more WOB is imposed by the pediatric equipment.3–6

Continuous positive airway pressure (CPAP) and pressure-support ventilation (PSV) with positive end-expiratory pressure (PEEP) are commonly used ventilation modes for pediatric patients weaning from mechanical ventilation. However, little is known about the influence of their
combined use on WOB in pediatric patients. CPAP is the application of continuous positive pressure at the airway opening, via nasal prongs, face mask, or an ETT. CPAP is used with spontaneously breathing patients, and no mechanical positive-pressure breaths are delivered. Since its introduction by Gregory et al in 1971, CPAP has become a standard part of neonatal ventilator support.

PSV is an assisted ventilation mode. It is patient-initiated and supports spontaneous breathing. PSV decreases WOB during weaning from mechanical ventilation, with adults9 and children.10 In a study that used 3 different ventilators with 9 intubated adults undergoing weaning in an intensive care unit, Mancebo et al9 demonstrated that the inspiratory WOB was 38–64% less with the addition of PSV 15 cm H2O. With pediatric patients Tokioka et al10 found that the application of pressure support markedly reduced WOB. Six postoperative children were studied, using 3 PSV levels (0, 5, and 10 cm H2O). WOB decreased by 48% with PSV 5 cm H2O, and by 73% with PSV 10 cm H2O. In a study involving postoperative pediatric patients Takeuchi et al11 found that reducing PSV increased WOB and pressure-time product (PTP).

The present study was designed to illustrate that mechanically ventilated pediatric patients, who require smaller ETTs and faster respiratory rates, have different needs than adult patients. The objective of this study was to evaluate the effects of CPAP/PEEP and PSV on WOB.

Methods

The Animal Use and Care Committee of the University of Arkansas for Medical Sciences approved this study. Animals were cared for in accordance with the standards for care and use of laboratory animals set forth by the University.

Animal Model

The experiments were performed with thirteen 4–5-week-old Dorsett/Western Cross lambs, each of which weighed approximately 10 kg. This animal model was selected because the animals’ respiratory rate, tidal volume (V1), and minute ventilation are comparable to those of pediatric patients.

Prior to the study day, vascular catheters were surgically placed via cutdown in the external jugular vein, for drug administration, and the internal carotid artery, for blood pressure monitoring. After surgery the animals were allowed to recover for 24–48 hours. On the study day all the animals were anesthetized (midazolam 0.25 mg/kg, propofol 3 mg/kg) and intubated with a 5.0 mm inner-diameter cuffed ETT. ETT location was confirmed by direct visualization with a bronchoscope, and the cuff was inflated to a minimal occlusion pressure. After intubation the animal was placed into a prone position in a temperature-controlled chamber. Gas humidification was maintained by a heat-and-moisture exchanger (HME) (Portex Humidivent 600, Keene, New Hampshire) positioned on the ETT, between the animal and the flow transducer. Supplemental intravenous anesthesia (midazolam 0.15 mg/kg/h, propofol 10 mg/kg/h) was administered continuously. The level of sedation was clinically monitored to maintain the animal in a light sleep, breathing spontaneously, arousable with interventions or stimulation, but easily returning to sleep. Heart rate, respiratory frequency, arterial blood pressure, arterial oxygen saturation measured via pulse oximetry (SpO2), and temperature were monitored continuously with a physiologic monitor.

Experiment Protocol

All animals were sedated, intubated, and ventilated with both the Servo 300 ventilator (Siemens Medical Systems, Danvers, Massachusetts) and the VIP Bird ventilator (Viasys Healthcare, Palm Springs, California), in specific modes and settings, according to the study protocol. The same 2 ventilators were used for all experiments. Animals were ventilated on systematically applied settings of CPAP 0, 5, and 10 cm H2O, and on PSV of 5 and 10 cm H2O with PEEP of 0, 5, and 10 cm H2O, using pressure-triggering with the VIP Bird and both pressure-triggering and flow-triggering with the Servo 300. Each animal was allowed to breathe spontaneously during each experiment, with 5-minute rest periods between experiments.

Ventilator Mechanics

The Servo 300 is a microprocessor-controlled ventilator that has neonatal, pediatric, and adult options, providing distinctive flow ranges for each patient group. All studies were performed using the pediatric range, in which the ventilator provides a bias flow of 1 L/min. When the demand valve is triggered, inspiratory flow of 30 L/min is available to maintain set pressure. During pressure-triggering the sensitivity can be set from 0 to –17 cm H2O. However, it is important to note that the animal must overcome the baseline bias flow in addition to the set negative pressure to initiate inspiratory flow. Flow-triggering is selected by turning the dial into the green or red area, and sensitivity increases as the dial is moved toward the red area. The expiratory flow transducer detects changes in air flow. At any setting within the green area a breath is initiated when the inspiratory effort results in a momentary change in flow. For these experiments sensitivity was set at –2 cm H2O for pressure-triggering and in the middle of the green zone for flow-triggering. CPAP is generated by the compression and relaxation of a silicone tube on the
expiratory side of the ventilator. A microprocessor-controlled stepper motor controls the constriction of the silicone tube. The expiratory pressure valve acts like a flow resistor. PSV is a spontaneous-breathing mode in which the patient must trigger each breath delivered by the ventilator. When the patient triggers the ventilator, a pressure-support breath is delivered at the preset value. Inspiratory rise time is set for patient comfort, and the pressure is regulated during inspiration so that it corresponds to the PSV level above PEEP. For these experiments inspiratory rise time was set at 5%, as recommended by the manufacturer.12

The VIP Bird is pneumatically activated, microprocessor-controlled, and can be time- or volume-cycled. It is designed to ventilate neonatal and pediatric patients. The VIP Bird was used with a volume monitor (Partner, Thermo Respiratory Group, Palm Springs, California). In this setup a pneumotachometer is placed at the proximal end of the ETT and the flow signal is transmitted to the ventilator. The sensor detects the inspiratory effort and to maintain the pre-selected pressure level, controls the continuous flow through the expiratory valve. The flow termination criterion can be selected as a percentage of the peak flow. Because flow-triggering is not available in pressure support with the VIP Bird, all studies with the VIP Bird were made with pressure-triggering.13 For these experiments a trigger sensitivity of –2 cm H2O was used.

Data Acquisition

Airway pressure and flow were measured at the proximal end of the ETT, using a differential pressure transducer connected to a pulmonary monitor (BiCore CP100, VIASYS Healthcare, Palm Springs, California). The dead space within the transducer was 11 mL. Intrapleural pressure was inferred using an esophageal catheter (SmartCath, Thermo Respiratory Group, Palm Springs, California), which was also connected to the pulmonary monitor. Esophageal balloon placement was confirmed with the occlusion test.14

The pulmonary monitor measured and recorded airway pressure, esophageal pressure, and airway flow, and from those measurements calculated the airway occlusion pressure 0.1 s after the onset of inspiratory effort (P0.1) and the ratio of inspiratory time to total breathing cycle time (TI/TTOT).

Diaphragmatic contraction is a response to stimulation from the neural drive, and P0.1 is a measure of that drive.15 P0.1 represents work expenditure during the initiated ventilator breath and, if elevated, it may represent an increase in WOB. Thus, a low P0.1 value may indicate blunted respiratory drive. TI/TTOT is a measure of endurance: if respiratory muscles fatigue, more time is spent in inspiration, so TI/TTOT increases.16

Other calculated values that relate to WOB include the change in esophageal pressure as a result of ventilation...
PTP is calculated by integrating $P_{es}$ and time for the duration of contraction of the respiratory muscles. Optimal PSV and CPAP decrease PTP, which varies directly with total lung resistance. $R_{AWE}$ is the difference in transpulmonary pressure and compliance, which is then divided by flow. Increased $R_{AWE}$ may indicate increased WOB.

In addition to the data from the pulmonary monitor, 10 consecutive breaths were manually recorded for each phase of the experiment to measure WOBT, WOB$_R$, and WOB$_E$, using the Campbell loop, as described by Blanch and Banner. This procedure involves directly measuring chest wall compliance during mechanical inflation of the lungs and thorax, while the animal is paralyzed. Chest wall compliance is directly measured by paralyzing the animal with succinylcholine (2 mg/kg) at the start of the experiment, ensuring an adequate respiratory frequency and VT with a control mode of mechanical ventilation. Analysis of the esophageal pressure and VT loop (using the Campbell-diagram software) was integrated into the WOB calculation to measure chest wall compliance. After chest wall compliance was determined, esophageal pressure-volume ($P_{es}$-VT) loops were combined into a single composite graph and WOB was measured from its planimetry by the pulmonary monitor. Immediately after paralysis for chest wall-compliance measurement, and before initiation of the experiment, a rest period was allowed for the animal to recover from the succinylcholine. We considered the animal fully recovered from the succinylcholine when it had returned to its normal baseline state (respiratory rate, $S_{pO_2}$, and VT, and was triggering the ventilator spontaneously).

### Statistical Analysis

Values are reported as mean ± SD. WOB data were analyzed as the average of 10 breaths for each animal for a given CPAP/PEEP and PSV level. Other parameters related to WOB ($P_{0.1}$, $T_I/T_{TOT}$, PTP, $\Delta P_{es}$, and $R_{AWE}$) were analyzed as the average of 50 breaths. The standard deviations were calculated from the averaged values to reflect standard medical practice. Two-way analysis of variance was used to examine the effects of CPAP/PEEP and PSV on WOB and related measures for each ventilator separately, since the VIP Bird used only pressure-triggering. The interaction, which represents the effect on the outcome as CPAP/PEEP is increased while PSV is decreased, was included in the model, and Type III sums of squares were used. Hochberg’s method was used to make adjustments for multiple comparisons of least squares means in the post-hoc analysis (reported p values). This method adjusts for unbalanced data and is a less conservative way of adjusting for multiple comparisons than Bonferroni’s adjustment. WOB$_E$ was log-transformed to correct normality; all other variables fulfilled model assumptions. All analyses were conducted with statistics software (SAS/STAT software, version 8.2 for Windows).
SAS Institute, Cary, North Carolina). A p value ≤ 0.05 was considered statistically significant. Graphs were constructed with statistics software (SPSS 11.0, SPSS Inc, Chicago, Illinois).

**Results**

Overall we found that the interaction between CPAP/PEEP and PSV was always nonsignificant; however, CPAP/PEEP and PSV independently affected WOB.

Figures 1-3 show WOB_T for all the CPAP/PEEP and PSV levels with the Servo 300 with pressure-triggering and flow-triggering, and with the VIP Bird with pressure-triggering.

When we evaluated the effects of CPAP/PEEP and PSV on WOB_T we found that with the Servo 300, with both pressure-triggering and flow-triggering, WOB_T did not differ between CPAP/PEEP 0 cm H₂O and CPAP/PEEP 5 cm H₂O; however, WOB_T increased between CPAP/PEEP 0 and CPAP/PEEP 10 cm H₂O (p ≤ 0.0001) and between CPAP/PEEP 5 and CPAP/PEEP 10 cm H₂O (p ≤ 0.0002). In addition, an increase in PSV decreased WOB_T with the Servo 300 with both pressure-triggering and flow-triggering (p ≤ 0.003).

With the VIP Bird we found no differences in WOB_T between CPAP/PEEP 0 cm H₂O and 5 cm H₂O; however, there was a significant increase in WOB_T between CPAP/PEEP 0 cm H₂O and 10 cm H₂O (p ≤ 0.02) and between CPAP/PEEP 5 cm H₂O and 10 cm H₂O (p ≤ 0.03). When we evaluated PSV we found no differences in WOB_T between CPAP and PSV 5 cm H₂O; however, WOB_T was lower at PSV 10 cm H₂O (p ≤ 0.0001).

### Table 1. Resistive Work of Breathing with the Servo 300 and VIP Bird Ventilators

<table>
<thead>
<tr>
<th>Setting (cm H₂O)</th>
<th>Servo 300 (pressure triggering)</th>
<th>Servo 300 (flow triggering)</th>
<th>VIP Bird (pressure triggering)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPAP 0</td>
<td>0.52 ± 0.19</td>
<td>0.48 ± 0.19</td>
<td>0.63 ± 0.12</td>
</tr>
<tr>
<td>CPAP 5</td>
<td>0.55 ± 0.18</td>
<td>0.50 ± 0.19</td>
<td>0.64 ± 0.15</td>
</tr>
<tr>
<td>CPAP 10</td>
<td>0.68 ± 0.19</td>
<td>0.56 ± 0.14</td>
<td>0.75 ± 0.15</td>
</tr>
<tr>
<td>PSV 5, PEEP 0</td>
<td>0.43 ± 0.13</td>
<td>0.29 ± 0.12</td>
<td>0.69 ± 0.38</td>
</tr>
<tr>
<td>PSV 5, PEEP 5</td>
<td>0.47 ± 0.22</td>
<td>0.37 ± 0.15</td>
<td>0.73 ± 0.35</td>
</tr>
<tr>
<td>PSV 5, PEEP 10</td>
<td>0.59 ± 0.23</td>
<td>0.54 ± 0.24</td>
<td>0.89 ± 0.37</td>
</tr>
<tr>
<td>PSV 10, PEEP 0</td>
<td>0.29 ± 0.07</td>
<td>0.13 ± 0.09</td>
<td>0.43 ± 0.17</td>
</tr>
<tr>
<td>PSV 10, PEEP 5</td>
<td>0.31 ± 0.07</td>
<td>0.21 ± 0.12</td>
<td>0.43 ± 0.21</td>
</tr>
<tr>
<td>PSV 10, PEEP 10</td>
<td>0.44 ± 0.16</td>
<td>0.36 ± 0.19</td>
<td>0.55 ± 0.24</td>
</tr>
</tbody>
</table>

*Values are mean ± standard deviation.
CPAP = continuous positive airway pressure
PSV = pressure support ventilation
PEEP = positive end-expiratory pressure

**Fig. 3.** Total work of breathing (WOB_T) with the Servo 300, with pressure-triggering (PT) and flow-triggering (FT), and the VIP Bird with pressure-triggering, with pressure support of 10 cm H₂O and set positive end-expiratory pressure (PEEP) of 0, 5, and 10 cm H₂O. Values plotted are approximate 95% confidence intervals.
Table 1 shows the results for WOB<sub>R</sub> manually recorded from the pulmonary monitor. With the Servo 300, with pressure-triggering and flow-triggering, we found no significant interaction between CPAP/PEEP and PSV; however, both CPAP/PEEP and PSV independently affected WOB<sub>R</sub>. There was a significant increase in WOB<sub>R</sub> between CPAP/PEEP 0 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.0004) and between CPAP/PEEP 5 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.01). When we examined the effects of PSV on WOB<sub>R</sub> with the Servo 300 with pressure-triggering, we found no significant difference between CPAP and PSV 5 cm H<sub>2</sub>O; however, WOB<sub>R</sub> significantly decreased between CPAP and PSV 10 cm H<sub>2</sub>O (p ≤ 0.0001) and PSV 5 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.001). With the Servo 300 with flow-triggering there was a significant decrease in WOB<sub>R</sub> with the addition of PSV (p ≤ 0.02).

With the VIP Bird we found no differences in WOB<sub>R</sub> at the different PEEP levels; however, WOB<sub>R</sub> significantly decreased between CPAP and PSV 10 cm H<sub>2</sub>O (p ≤ 0.01), and between PSV 5 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.0001). Table 2 shows the results for WOB<sub>R</sub> manually recorded from the pulmonary monitor. We found no significant interaction between CPAP/PEEP and PSV; however, both CPAP/PEEP and PSV independently affected WOB<sub>R</sub> with the Servo 300 with both pressure-triggering and flow-triggering. With the VIP Bird only PSV significantly affected WOB<sub>E</sub>; As with WOB<sub>T</sub> and WOB<sub>R</sub>, WOB<sub>E</sub> decreased with the addition of PSV with the Servo 300 with both pressure-triggering (p ≤ 0.0001) and flow-triggering (p ≤ 0.0001) and with the VIP Bird (p ≤ 0.0001). WOB<sub>E</sub> significantly increased at CPAP/PEEP 10 cm H<sub>2</sub>O with the Servo 300 with both pressure-triggering (p ≤ 0.0001) and flow-triggering (p ≤ 0.0001) and with the VIP Bird (p ≤ 0.0001).

When we examined the effects of CPAP/PEEP and PSV on P<sub>TP</sub> on PTP, we found that only PSV significantly affected PTP. The addition of PSV decreased PTP with the Servo 300 with both pressure-triggering (p ≤ 0.0001) and flow-triggering (p ≤ 0.0001). With the Servo 300 with flow-triggering we found that P<sub>TP</sub> decreased between CPAP and PSV 10 cm H<sub>2</sub>O (p ≤ 0.0001) and between PSV 5 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.0001). With the Servo 300 with pressure-triggering we found that P<sub>T</sub> decreased between CPAP/PEEP 0 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.0001) and between CPAP/PEEP 5 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.0001). With the Servo 300 with pressure-triggering we found that P<sub>T</sub> decreased between CPAP/PEEP 0 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.0001) and between CPAP/PEEP 5 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.0001).

When we examined the effects of CPAP/PEEP and PSV on R<sub>W</sub> we found that with the Servo 300 with pressure-triggering R<sub>W</sub> increased significantly between CPAP/PEEP 0 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.01). PSV did not affect R<sub>W</sub>. With the Servo 300 with flow-triggering R<sub>W</sub> increased between CPAP/PEEP 0 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.01) and between CPAP/PEEP 5 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.02). R<sub>W</sub> decreased between PSV 5 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.03). With the VIP Bird only CPAP/PEEP significantly affected R<sub>W</sub>; it increased between CPAP/PEEP 0 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.001).

When we examined the effects of CPAP/PEEP and PSV on R<sub>W</sub> we found that with the Servo 300 with pressure-triggering R<sub>W</sub> increased significantly between CPAP/PEEP 0 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.01). PSV did not affect R<sub>W</sub>. With the Servo 300 with flow-triggering R<sub>W</sub> increased between CPAP/PEEP 0 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.01) and between CPAP/PEEP 5 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.02). R<sub>W</sub> decreased between PSV 5 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.03). With the VIP Bird only CPAP/PEEP significantly affected R<sub>W</sub>; it increased between CPAP/PEEP 0 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.001).

When we examined the effects of CPAP/PEEP and PSV on R<sub>W</sub> we found that with the Servo 300 with pressure-triggering R<sub>W</sub> increased significantly between CPAP/PEEP 0 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.01). PSV did not affect R<sub>W</sub>. With the Servo 300 with flow-triggering R<sub>W</sub> increased between CPAP/PEEP 0 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.01) and between CPAP/PEEP 5 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.02). R<sub>W</sub> decreased between PSV 5 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.03). With the VIP Bird only CPAP/PEEP significantly affected R<sub>W</sub>; it increased between CPAP/PEEP 0 cm H<sub>2</sub>O and 10 cm H<sub>2</sub>O (p ≤ 0.001).
With all the animals there were no changes in heart rate, respiratory frequency, arterial blood pressure, $S_{\text{pO}_2}$, or temperature.

Discussion

The principle finding of this study is that if healthy animals are allowed to breathe spontaneously while being supported with CPAP and PSV, using ventilator settings similar to those used with children in the pediatric intensive care unit, WOB is lowered with the addition of PSV.

CPAP is used only during spontaneous ventilation. CPAP maintains positive pressure within the circuit throughout the respiratory cycle. The total mechanical inspiratory WOB during CPAP depends not only on the subject’s respiratory mechanics but also on the CPAP system.\(^{19-21}\) The method by which the ventilator provides CPAP may influence inspiratory WOB by either failing to provide sufficient instantaneous fresh gas during inspiration, or by requiring a substantial pressure drop in the ventilator circuit before gas flow occurs.

Although the effects of various CPAP levels have been explored in human studies,\(^{2,22,23}\) there have been conflicting results regarding the effect of increasing CPAP on the inspiratory work with various CPAP machines using lung models.\(^{24,25}\) In human studies the decreased WOB found with increasing CPAP may relate to lung recruitment and improved compliance with higher CPAP. However, in patients whose pulmonary compliance does not improve with increased CPAP there are reasons (relating to the performance of various PEEP valves) why higher CPAP may increase inspiratory WOB, especially when using a continuous-flow system.\(^{26}\) In our study, increasing CPAP increased WOB\(_T\) with both ventilators. Torres et al\(^{27}\) studied the effects of CPAP and inspiratory pressure support on diaphragmatic function in spontaneously breathing sheep. They found that CPAP or inspiratory pressure support affected diaphragmatic function, but the combination of CPAP and inspiratory pressure support minimized those effects. In our study we did not measure diaphragmatic function but did measure $T_I/T_{TOT}$, which is a measure of endurance. For increasing levels of PEEP/CPAP, we found that CPAP/PEEP and PSV independently affected $T_I/T_{TOT}$.

Our study was potentially limited by the use of the HME. The HME was used primarily to protect the expiratory pneumotachometer from moisture, and since the animals were ventilated for not more than 3 hours at any one time, the potential for increased resistance due to saturation of the HME was minimal. One study suggested that changing the HME during extended periods of ventilation has little impact on HME resistance,\(^{28}\) However, Ploysongsang et al\(^ {29}\) demonstrated increased resistance with an HME. This could make it more difficult for the animal to trigger the ventilator during pressure-triggering than during flow-triggering because of the potential pressure drop across HME. Ploysongsang et al postulated that this pressure drop was clinically important only in patients with poor pulmonary reserve and when used for long periods of time. Those conditions do not apply to our animal model.

Another limitation of our study was that for CPAP of 0 cm H\(_2\)O, the Servo 300 guarantees 2 cm H\(_2\)O pressurization of the system with each breath. Thus, breathing on the Servo 300 during CPAP without this pressurization is not available. Ideally, adding pressure support of 2 cm H\(_2\)O to CPAP of 0 cm H\(_2\)O during pressure-triggering could have offset some of the differences we found, but this could only be done in pressure-triggering, since PSV is not available in flow-triggering on the VIP Bird.

Conclusions

PSV is an effective mode for decreasing WOB, especially when using PEEP/CPAP, which can increase WOB. Since there is increasing interest in lung recruitment strategies and the use of higher PEEP in pediatric patients with respiratory failure, the clinician must consider the effects of higher PEEP on WOB.

REFERENCES