Effects of Positive End-Expiratory Pressure on Oscillated Flow Rate During High-Frequency Chest Compression*

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Objective: To investigate the effects of positive end-expiratory pressure (PEEP) on end-expiratory lung volume (EELV) and mean oscillated flow rate (Vosc) during high-frequency chest compression (HFCC) in normal subjects and patients with severe COPD.

Design: Comparative study.

Setting: Pulmonary function and lung mechanics laboratory, University of Alberta Hospitals.

Participants: Six normal subjects (five male; one female) and six patients with clinically stable COPD (five male; one female) with hypercapnia.

Interventions: A pneumatic vest system was operated at 10 Hz with a mean chest wall pressure of 16 cm H2O to provide the HFCC. A closed-circuit spirometer system permitted measurement of HFCC- and PEEP-induced changes in EELV that were expressed as per cent baseline functional residual capacity (FRC). An isothermic chamber connected near the mouthpiece permitted measurement of Vosc.

Results: For the normal subjects, HFCC caused a significant decrease in EELV to 82.0% of FRC (p≤0.01) and the addition of 4.8±0.5 cm H2O of PEEP during HFCC increased EELV to 97.5% FRC. In the COPD patients, HFCC decreased EELV to 92.3% of FRC (p≤0.01), and the addition of 3.7±1.0 cm H2O of PEEP increased EELV to 98.4% FRC. For the normal subjects, increasing EELV to near FRC caused Vosc during expiration to increase 14.0% (p≤0.01), but there was no significant effect on Vosc during inspiration (5.1% increase). In the COPD patients, PEEP increased Vosc during both inspiration (30.5%) and expiration (57.0%) (both, p≤0.01).

Conclusions: Addition of a modest amount of PEEP during HFCC prevents the decrease in EELV and increases Vosc during both phases of spontaneous breathing in COPD patients. This higher Vosc during HFCC+PEEP may improve the effectiveness of HFCC in clearing mucus from the lungs of patients with airway disease.

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Key words: chronic obstructive pulmonary disease; end-expiratory lung volume; high-frequency chest compression; positive end-expiratory pressure

Abbreviations: CF=cystic fibrosis; EELV=end-expiratory lung volume; FRC=functional residual capacity; HFCC=high-frequency chest compression; PetCO2=end-tidal partial pressure of carbon dioxide; PEEP=positive end-expiratory pressure; RV=residual volume; Vosc=mean oscillated flow rate; VoscE=mean oscillated flow rate during spontaneous expiration; VoscI=mean oscillated flow rate during spontaneous inspiration

In 1990, Hansen and Warwick1 described the first commercial device used for high-frequency chest compression (HFCC) and showed HFCC to be more effective than conventional chest physiotherapy for aiding mucus clearance from the lungs of patients with obstructive lung disease. The same authors later showed HFCC therapy improved lung function in 15 of 16 cystic fibrosis (CF) patients after 22 months of daily HFCC use.2 Subsequent studies confirmed HFCC to be as effective as other standard chest physiotherapy regimens for patient tolerance,3 hospital stay,4 improving lung function,5,4 and increasing sputum expectoration.3,4 Although HFCC is a promising alternative for the treatment of patients with obstructive lung disease, guidelines for optimizing treatments are needed as suggested by Butler and O'Neill.5

In 1995, Jones et al6 studied the short-term effects of HFCC on respiratory system mechanics in normal
subjects and CF patients. They found that an oscillation frequency of 10 to 15 Hz resulted in the highest oscillated flow, and a low vest pressure minimized the HFCC-induced decrease in end-expiratory lung volume (EELV). In CF patients, HFCC produced oscillated flows that were less than one half of those generated in normal subjects at similar external chest wall pressures. This was related to the higher airway resistance in CF patients since oscillated flow was significantly and positively correlated with FEV<sub>1</sub>. Therefore, the HFCC-induced decrease in EELV may exaggerate the high airway resistance in patients with airway obstruction. We believe this contributes to the low oscillated flows in these patients.

In the present investigation, we used positive end-expiratory pressure (PEEP) in patients with COPD to counteract the HFCC-induced decrease in EELV. We postulated that by maintaining EELV closer to functional residual capacity (FRC), airway resistance would be lower than without PEEP and that oscillated flow rate would increase.

**Materials and Methods**

This study was approved by the University of Alberta Hospital ethics committee and signed informed consent was obtained. Six normal subjects (five male; one female) and six patients (five male; one female) with COPD participated in the study. The normal subjects were nonsmoking volunteers and exhibited normal lung function. The COPD patients had severe airway obstruction with hypercapnia and were selected from our Respiratory Home Care Clinic. All patients were in a clinically stable phase of their disease and daily routine and medical treatments were unaltered for this study.

Baseline lung function, which included spirometry and lung volumes, was determined in all subjects. Lung volumes were measured with the helium dilution method using a system (Gould 2400 PFT system; Gould; Valley View, Ohio). Baseline FRC was used to determine changes in EELV during HFCC. Anthropometric and baseline lung function data are shown in Table 1<sup>8,9</sup> for both study groups.

A pneumatic vest system (ThAIRapy model 103; American Biosystems; St. Paul, Minn) was used for HFCC. The vest was attached by having the subjects inhale to total lung capacity, and then it was securely fastened in place. This permitted breathing with large tidal volumes if necessary. When the air pulse generator is turned on, the vest inflates to a positive background pressure over which pressure oscillations are superimposed at frequencies adjustable between 5 and 25 Hz. In this study, background vest pressure was set at a dial setting of 9 and the oscillation frequency was 10 Hz for all subjects. This combination produces a mean external chest wall pressure of approximately 16 cm H<sub>2</sub>O at the end of a spontaneous expiration (unpublished data).

A breathing circuit consisting of a spirometer (Ohio 840; Ohio Medical Products; Madison, Wis), breathing valve, oxygen supply, carbon dioxide absorber, and water-filled PEEP chamber was used (Fig 1). We could switch from zero end-expired pressure to PEEP by turning a three-way valve. A 20-L isothermic chamber was attached close to the mouth to measure oscillated volumes (amount of air moving in and out of the mouth per oscillator cycle). End-tidal F<sub>CO</sub>_2 (PETCO<sub>2</sub>) was also measured just downstream from the expiration valve.

Application of HFCC, with its attendant increase in external chest wall pressure, causes EELV to decrease. The magnitude of the change from FRC was measured from the recorded spirogram. Addition of PEEP during HFCC increased EELV to near FRC. The PEEP level required to return EELV to FRC was determined for each subject during a prestudy trial.

The oscillated volume created by HFCC was determined using the 20-L isothermic chamber loosely filled with steel wool and connected to a differential pressure transducer (Validyne MP-45-50 cm H<sub>2</sub>O; Northridge, Calif). The breathing circuit acted as a low-pass filter so the rapid pressure changes at the mouth were transmitted only a short distance. The isothermic chamber was calibrated so that the pressure fluctuations in the airway could be converted to oscillated volume. The oscillated volume measurement system was frequency dependent and

**Table 1—Anthropometric and Lung Function Data**

<table>
<thead>
<tr>
<th></th>
<th>Normal Subjects (n=6)</th>
<th>COPD Patients (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age, yr</strong></td>
<td>46.2±5.8</td>
<td>70.8±5.8</td>
</tr>
<tr>
<td><strong>Height, cm</strong></td>
<td>173.8±6.1</td>
<td>168.8±3.9</td>
</tr>
<tr>
<td><strong>Weight, kg</strong></td>
<td>81.7±12.4</td>
<td>79.2±11.7</td>
</tr>
<tr>
<td><strong>FEV&lt;sub&gt;1&lt;/sub&gt;, L</strong></td>
<td>3.51±0.3 (102.7±7.7)</td>
<td>0.78±0.2 (29.5±6.0)</td>
</tr>
<tr>
<td><strong>FVC, L</strong></td>
<td>4.54±0.5 (105.5±9.5)</td>
<td>2.19±0.4 (61.0±7.5)</td>
</tr>
<tr>
<td><strong>FEV&lt;sub&gt;1&lt;/sub&gt;/FVC, %</strong></td>
<td>77.7±7.0</td>
<td>35.8±6.0</td>
</tr>
<tr>
<td><strong>FRC, L</strong></td>
<td>2.92±0.3 (92.2±15.1)</td>
<td>4.05±0.9 (123.7±30.7)</td>
</tr>
<tr>
<td><strong>RV, L</strong></td>
<td>1.73±0.3 (100.5±19.7)</td>
<td>3.36±0.8 (179.2±43.5)</td>
</tr>
<tr>
<td><strong>TLC, L</strong></td>
<td>6.43±0.7 (103.7±7.0)</td>
<td>5.94±0.8 (103.7±18.8)</td>
</tr>
<tr>
<td><strong>PaO&lt;sub&gt;2&lt;/sub&gt;, mm Hg</strong></td>
<td>—</td>
<td>58.8±3.45</td>
</tr>
<tr>
<td><strong>PaCO&lt;sub&gt;2&lt;/sub&gt;, mm Hg</strong></td>
<td>—</td>
<td>47.6±7.1</td>
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<tr>
<td><strong>PETCO&lt;sub&gt;2&lt;/sub&gt;, mm Hg (no HFCC)</strong></td>
<td>39.1±4.0</td>
<td>40.3±7.8</td>
</tr>
<tr>
<td><strong>PETCO&lt;sub&gt;2&lt;/sub&gt;, mm Hg (HFCC)</strong></td>
<td>33.2±7.2</td>
<td>40.0±7.9</td>
</tr>
<tr>
<td><strong>PETCO&lt;sub&gt;2&lt;/sub&gt;, mm Hg (HFCC+PEEP)</strong></td>
<td>34.3±7.6</td>
<td>40.4±7.5</td>
</tr>
</tbody>
</table>

*Values presented as means±SD. Values in parentheses are per cent predicted obtained from two references. TLC=total lung capacity.
1Two of the six patients were receiving supplemental oxygen.
1*p<0.01 compared with no HFCC.
correction factors were required to obtain true oscillated volume. These factors ranged from 1.00 to 1.55 at oscillation frequencies of 5 to 25 Hz, respectively. At 10 Hz (the frequency used in this study), the factor was 1.10. Therefore, measured oscillated volume was multiplied by 1.10 and then it was corrected to body temperature ambient pressure and saturated with water vapor. The corrected oscillated volume was then multiplied by 2× oscillation frequency to provide mean oscillated flow rate (Vosc). To reduce any effects of lung volume on lung compliance and airway resistance, Vosc was measured during the middle portions of spontaneous inspiration (VoscI) and expiration (VoscE).

The experimental protocol required 13 min of rebreathing from the closed circuit spirometer system (3 min baseline, 5 min HFCC, 5 min HFCC+PEEP). The initial fraction of oxygen in the closed circuit was approximately 0.40 and oxygen was added during rebreathing to maintain spirometer volume. The flow of oxygen required was determined during the 3-min baseline period. Oxygen saturation (Ohmeda Biox 3700; Ohmeda; Louisville, Colo.) was monitored and it remained between 90% and 100% in all subjects.

Statistics were performed using software (GraphPad Prism; GraphPad Software Inc; San Diego, Calif). Paired t tests with the Bonferroni correction were performed to determine if significant differences existed between HFCC and HFCC+PEEP. The significance level was set at p<0.01. All values are represented as means±SEM.

**RESULTS**

In the patients, PetCO₂ averaged 40 mm Hg without HFCC and it remained at 40 mm Hg during HFCC and HFCC+PEEP (Table 1). Therefore, there was no evidence for an effect of HFCC on supplemental ventilation at the vest pressure and oscillation frequency used in this study. However, in the normal subjects, both HFCC and HFCC+PEEP caused PetCO₂ to decrease significantly from baseline values. There were no significant differences in PetCO₂ between HFCC and HFCC+PEEP for normal subjects or patients.

The effects of HFCC and HFCC+PEEP on EELV are shown in Figure 2. HFCC caused a significant decrease in EELV to 82.0% of FRC in the normal subjects (p<0.01). The EELV during HFCC was increased to 97.5% FRC with 4.8±0.5 cm H₂O of PEEP, and the difference between EELV and FRC after the addition of PEEP was not significant. In the COPD patients, HFCC decreased EELV to only 92.3% of FRC (p<0.01), and the addition of 3.7±1.0 cm H₂O of PEEP increased EELV to 98.4% FRC. Although the HFCC-induced decrease in lung volume for the patients was smaller than in the normal subjects, the patients' EELV was only 114% of residual volume (RV), whereas in the normal subjects, EELV was 139% of RV. Therefore, the relatively small decrease in EELV in the COPD patients caused lung volume to approach RV.

Figure 3 shows the effects of HFCC and HFCC+PEEP on Vosc in the normal subjects and patients. In the normal subjects, the addition of PEEP to HFCC had no significant effect on Vosc compared with HFCC alone. In contrast, VoscE...
**Figure 2.** Effects of HFCC and HFCC+PEEP on EELV in (top, A) normal subjects and (bottom, B) COPD patients. The mean FRC is represented by the solid line and RV by the dotted line. Asterisk = p ≤ 0.01. Values are mean ± SEM.

**Figure 3.** Effects of HFCC and HFCC+PEEP on Vosc in (top, A) normal subjects and (bottom, B) COPD patients. Asterisk = p ≤ 0.01. Values are mean ± SEM.

(significantly increased with the addition of PEEP (p ≤ 0.01). In the patients, HFCC+PEEP significantly increased both Vosci and Vosce (both, p ≤ 0.01). The patients had a lower Vosc overall compared with the normal subjects, but the percent increase in Vosc with the addition of PEEP to HFCC was approximately fourfold greater in the patients than the normal subjects.

**Discussion**

Consistent with the previous study from our laboratory, we found HFCC decreased EELV below
FRC in both patients and normal subjects. Although the decrease in lung volume in the patients was smaller than in the normal subjects, the effect may be more significant since EELV decreased to near RV in the patients. As a consequence, we anticipate that airway resistance would be higher during HFCC and perhaps some airway closure might occur in the patients. Generally, patients with airway obstruction breathe at a high FRC and forcing them to breathe at a lower lung volume is undesirable because of the resultant increase in airway resistance. When PEEP is applied during HFCC, lung volume can be increased to FRC and this, in turn, should decrease airway resistance. We believe the postulated lower airway resistance during HFCC+PEEP compared with HFCC alone explains the increase in Vosc seen when PEEP was added during HFCC. Of course, increasing EELV back to FRC with PEEP still leaves the COPD patient with a higher than normal airway resistance, which explains the much lower Vosc in these patients.

The phase of spontaneous breathing has a considerable effect on Vosc with Vosce being only 47% of Vosci during HFCC in the COPD patients. In the normal subjects, Vosce was 75% of Vosci so the phase of spontaneous breathing affects Vosc regardless of whether airway obstruction does or does not exist. Therefore, spontaneous expiration appears to dampen the translation of external chest oscillations into oscillatory air movement in the airways. The Vosci-Vosce difference can be explained by the observation that airway resistance is higher during expiration than it is during inspiration in the normal subjects and especially in patients with airway obstruction. These changes in airway resistance may be due to the less negative pleural pressure during expiration resulting in relative compression of the larger airways compared with inspiration where pleural pressure is more negative. It is clear that pressure external to the airways, i.e., pleural pressure, affects the diameter of the airways. It is unlikely that compliance of the lung or chest wall is important in causing the Vosci-Vosce difference since Vosc was measured under isovolume conditions at mid-inspiration and mid-expiration.

Although there was a large difference between baseline PaCO₂ and PetCO₂ in the COPD patients, we made the assumption that changes in PetCO₂ reflected relative changes in PaCO₂. The decrease in PetCO₂ in the normal subjects was an unexpected finding since methods of chest wall oscillation that are designed to improve gas exchange are normally operated at <5 Hz. Therefore, it was interesting that subjects with normal airways seem to receive some supplemental ventilation at 10 Hz using the pneumatic vest system, which is primarily a device used to enhance mucus clearance. However, our COPD patients did not have a change in PetCO₂ during HFCC. This is consistent with HFCC, administered at 10 Hz, being of little use as a ventilation supplement in patients with airway disease.

It has been shown that oscillating air flow may act as a physical mucolytic augmenting the ability to clear mucus through coughing and long-term HFCC therapy improves lung function in CF patients. The addition of PEEP may increase the benefits of HFCC therapy by reducing airway resistance (through its effect on lung volume) and increasing the Vosc that is believed responsible for the mucolytic and mucus clearance effects of HFCC therapy.

CONCLUSIONS

The addition of <5 cm H₂O PEEP during HFCC restores lung volume to FRC. In both normal subjects and COPD patients, HFCC+PEEP improved oscillated air flow compared with HFCC without PEEP. This might be the result of PEEP decreasing airway resistance through its effect of increasing lung volume. This action of PEEP might improve the effectiveness of HFCC in clearing airway secretions.

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