Effect of External Chest Wall Oscillation on Gas Exchange in Healthy Subjects*

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Effective gas exchange can be maintained in animals without the need for endotracheal intubation using external chest wall oscillation (ECWO). The clinical application of this technique has been limited by equipment which was either impractical or uncomfortable. We evaluated a prototype of a new oscillator in which an oscillatory profile of negative and positive pressure was imposed on a negative baseline pressure within a cuirass. In seven healthy subjects, we identified an oscillatory cuirass pressure that could effectively ventilate but would not result in severe hypocapnia over 5 min. We then measured the influence of changing the frequency of oscillation (fo) on PaCO₂ and spontaneous ventilation. Lastly, we evaluated the capability of this prototype to achieve targeted changes in chamber pressure. Subjects were ventilated with an inspiratory chamber pressure of ~20 ± 4 cm H₂O, an expiratory chamber pressure of 5 cm H₂O and an inspiratory-expiratory ratio of 1:1 at 9 oscillatory frequencies (fo: 1 to 5 Hz at 0.5-Hz increments). Each subject was ventilated for 5 min with consecutive periods of ECWO being separated from each other by 10 min of unassisted breathing. Oscillatory tidal volume (Vo) was sampled and PaCO₂ was determined from the expired carbon dioxide concentration (FeCO₂) measured at the mouth. The change in PaCO₂ (ΔPaCO₂) was the difference in PaCO₂ immediately before and after ECWO. We found that ΔPaCO₂ and Vo were inversely related to fo. At 1 Hz the ΔPaCO₂ was ~13 ± 1 mm Hg and Vo was 344 ± 34 mL in the absence of spontaneous breathing (fb=0). At 3 Hz and above, at the chamber pressures used, the ΔPaCO₂ was small (~1 to ~2 mm Hg) and the Vo was less than the predicted dead space. Subjects breathed spontaneously but at a frequency below that of their resting fb. With this prototype, chamber pressure changes up to 30 cm H₂O could be accurately achieved at 1, 2.5, and 4 Hz. In conclusion, ECWO can provide effective ventilation among healthy adults in the presence or absence of spontaneous breathing, and further studies are warranted to explore its effectiveness in a variety of clinical circumstances.

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| ECWO=external chest wall oscillation; fb=spontaneous frequency of breathing; FeCO₂=expired carbon dioxide concentration; fo=frequency of oscillation; HFV=high-frequency ventilation; I-E=inspiratory-expiratory ratio; ΔPaCO₂=change in arterial carbon dioxide pressure; Pe=expired pressure; ΔPe=change in chamber pressure; Pcc=inspiratory chamber pressure; Ppl=pleural pressure; ΔPpl=change in pleural pressure; Vo=oscillatory tidal volume |

Key words: external chest wall oscillation, gas exchange

Interest in alternative methods of ventilation has increased in recent years as the limitations of conventional ventilation become apparent. Conventional positive pressure ventilation may have detrimental cardiovascular or respiratory effects especially in the presence of severe airway or parenchymal dysfunction when high airway pressures and increased ventilatory volumes may be necessary for effective gas exchange. Alternatively, gas exchange can be maintained at low airway pressures and tidal volumes using high-frequency ventilation (HFV), but problems associated with its use include the need for endotracheal intubation, alterations in baseline lung volume, and dehumidification of the respiratory system.1-3 Even when used with an adaptive mouthpiece, difficulties in warming and humidifying the air persisted.4 To avoid the complications of both conventional positive pressure ventilation and HFV, attempts have been made to enhance gas exchange by external chest wall oscillation (ECWO).

Preliminary studies in which ECWO was applied to animals have reported that gas exchange could be enhanced despite a reduction in spontaneous ventilation.5-7 External chest wall oscillation has compared well with HFV in animals with normal lungs and may in fact be more effective than HFV in the presence of airway obstruction.8 It also may be more effective in enhancing gas exchange than conventional ventilation in the presence of increased elastic recoil.9

Attempts to externally ventilate human volunteers have met with varied success. Ventilation was decreased by 16 to 40% when ECWO was applied for brief periods in an oscillatory range of 3 to 10 Hz with either an inflatable rubber cuff10 or a sealed body box to which loud speakers were attached.4 The effect of such oscillations was comparable to that of HFV

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among healthy volunteers, but the methods were somewhat impractical. The study and further application of ECWO has been limited by the absence of commercially available oscillators.

A recently developed approach to ECWO uses a cuirass system to oscillate the anterolateral area of the chest and abdomen with negative and positive pressures in combination with a continuous negative baseline pressure. With this technique, the pressure changes cause both inspiratory and expiratory flow. Oscillatory tidal volume (Vo) as well as mean lung volume can be controlled, a mouthpiece is unnecessary, and inspired air is warmed and humidified naturally. We are currently evaluating a prototype oscillator (Breasy Medical Equipment, London, England) developed for clinical application. Before clinical studies, we determined the influence of the frequency of oscillation on carbon dioxide elimination and spontaneous breathing among healthy volunteers.

**Methods and Procedures**

**Methods**

The study subjects were seven healthy volunteers, six of whom were men and one of whom was a woman, who participated after giving their informed consent. Standard measurements of lung volumes, flows, airway resistance, and diffusing capacity were made, and anatomical dead space was predicted from body weight. Each subject was ventilated with ECWO using a Hayek Oscillator (Breasy Medical Equipment, London, England).

The oscillator consisted of a power unit, a control unit, and a chest cuirass. The cuirass was a specially shaped clear plastic shell that spanned the anterolateral area of the chest and abdomen from sternum to pubis. Additional support and a close seal was provided by a steel back plate secured to the cuirass. Thus, an airtight chamber could be created with the cuirass, chest, and abdominal wall. The chamber was connected to a power unit with flexible tubing. The power unit consisted of a piston that altered the chamber pressure from negative (inspiration) to positive (expiration). The chest compressions were superimposed upon a continuous negative pressure provided by the power unit. The control unit was connected to the chamber by a flexible narrow lumen tube. In this way, the control unit monitored the chamber pressure and appropriately adjusted the power unit to achieve the desired fo, inspiratory chamber pressure (Pic), expiratory chamber pressure (Pec), and inspiratory-expiratory ratio (I:E). Change in chamber pressure (APc) was calculated as the difference between Pic and Pec.

Chamber pressure (Pic) was measured with the pressure transducers present within the control unit of the oscillator. Pleural pressure (Ppl) was measured using an esophageal balloon catheter. A 5-cm latex balloon was secured over the perforated distal end of a 110-cm catheter (balloon volume, less than 0.5 mL), and it was positioned using an occlusion method to minimize cardiac and structural artifacts and to accurately reflect Ppl. Esophageal pressure closely reflects Ppl in the frequency range of 2 to 32 Hz. The frequency response of the measurement system (catheter and transducer) was flat to 13 Hz. The change due to oscillation or change in pleural pressure (APpl) was the difference between end-inspiratory and end-expiratory pressures.

A spontaneous breath was defined as an active clearing of dead space identified when expired carbon dioxide concentration (Fe-

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21708/)

**Figure 1.** Recording of Pc (upper tracing) and FeCO₂ (lower tracing) at the end of 5 min of ECWO. During ECWO at 1 Hz, there were no spontaneous breaths and the mean FeCO₂ was 4.3% (PaCO₂ was 30.1 mm Hg). During ECWO at 4.5 Hz, there were spontaneous breaths shown by an FeCO₂ of 0% associated with a positive inspiratory Pc; FeCO₂ was 6.3% (PaCO₂ was 44.0 mm Hg).

CO₂ declined to zero. The accompanying increase in thoracic volume reduced chamber volume, thereby increasing Pic. Spontaneous breaths also could be identified by low pass filtering of the esophageal pressure, which eliminated the high-frequency oscillation due to ECWO. The spontaneous frequency of breathing (fb) during ECWO was expressed as the number of spontaneous breaths per minute. Examples of ECWO without and with spontaneous breaths are shown in Figure 1, A and B, respectively.

Vo was measured at the mouth using a dry rolling seal spirometer (Morgan, Gillingham, England). The resistance characteristics of the spirometer have been determined using a high-flow pump capable of a 5-L stroke at a peak velocity of 35 L.s⁻¹. Resistance was 0.02 cm H₂O at 10 L.s⁻¹, and the frequency response was flat ±1% to 4 Hz with a 1-L excursion. The Vo was derived from the difference between the peak and the valley of the volume waveform during ECWO. Some measurements were made during apnea attributable to ECWO. When spontaneous breathing was present, Vo was determined during the end-expiratory pause before the next breath.

**Determination of Arterial Carbon Dioxide Pressure**

Arterial carbon dioxide pressure was derived from FeCO₂ measured at the mouth with a rapidly responding carbon dioxide monitor (SensorMedics, Anaheim, Calif) calibrated with known fractional concentrations of carbon dioxide of 0, 5, and 7%. Although resting end-tidal PaCO₂ accurately reflects PaCO₂ within 4 mm Hg, this method is dependent on tidal breathing. While application of the cuirass does not significantly change functional residual capacity, ECWO could have lowered Pa-
CO₂ resulting in apnea such that by the time spontaneous respiration occurred, the PaCO₂ would have changed. We therefore chose to monitor PaCO₂ using the forced expiratory maneuver first described by Haldane and Priestley.²² In this maneuver, the subject exhaled promptly from his resting lung volume. Three well-defined phases of expired gas could be identified during the forced exhalation. In the first phase, the FeCO₂ was zero as dead space was exhaled. In the second phase, the FeCO₂ rose promptly as dead space and exhaled alveolar gas mixed. In the third phase, the FeCO₂ rose slowly as only alveolar gas was expired until airway closure. The mean FeCO₂ was calculated from the midpoint of the rising phase 2 to the end of phase 3. In two subjects, this technique was validated during the oscillation protocol described below in this article. An arterial line was inserted via the radial artery. Repeated measurements of PaCO₂ from arterial blood were compared with the Pco₂ derived from the mean FeCO₂. The results are displayed in Figure 2. Over the range of 24 to 45 mm Hg, the mean difference was 0.6 mm Hg (95% confidence intervals, 0.1 and 1.1 mm Hg).

All variables were recorded continuously on an electrostatic recorder (Gould ES 1000 Recorder; Gould, Cleveland) or digitally sampled (100 Hz) and recorded on computer disk.

**Protocol**

The protocol consisted of four sessions separated by at least 1 day. The first session was an introductory session, and the next two sessions were used to determine the relationship between PaCO₂, Vo, and fo. The fourth session was used to determine the limitations of this particular prototype in achieving the targeted ΔPc. Subjects were asked to empty their bladder immediately before each session.

**Introductory Session:** The purpose of the introductory session was to familiarize the subjects with the equipment and to determine the cuirass pressures that they could comfortably tolerate. During this session, we identified a cuirass pressure that could ventilate effectively but would not cause severe hypocapnia over 5 min. Upon arrival, the subject was shown the oscillator and fitted with a cuirass. The subject then reclined in a chair and breathed through a mouthpiece (<5 mL dead space) with a carbon dioxide sampling tube directed to sample airflow. The subject rested quietly for 5 min until PaCO₂ had stabilized within ±1 mm Hg.

The fo for the first session was 1.67 Hz, PEC was 5.0 cm H₂O, I-E was 1.1, and PEC was started at −22.0 cm H₂O. The PEC chosen was similar to pressures used in previous reports of negative pressure ventilation applied to healthy volunteers.²³ External chest wall oscillation continued for 5 min. The subject was coached to relax. After 5 min, ECWO was stopped and the subject immediately exhaled to residual volume. The post-ventilation PaCO₂ was determined and the change in PaCO₂ (ΔPaCO₂) was calculated. The ΔPaCO₂ was the resting (unassisted) PaCO₂ minus the PaCO₂ following 5 min of ECWO. If PaCO₂ had decreased 5 to 10 mm Hg, the subject was given a 10-min rest and the protocol repeated at the same settings. If the PaCO₂ decreased more than 10 mm Hg, the PEC was made less negative by 2 to 5 cm H₂O. This was imposed to avoid severe hypocapnia. If the PaCO₂ decreased less than 5 mm Hg, the PEC was made more negative by 2 to 5 cm H₂O and the protocol was repeated. There was always a 10-min rest period between ECWO. This procedure was repeated until the lowest PEC that caused a 5 to 10 mm Hg decrease in PaCO₂ with 5 min of ECWO was determined.

**Influence of Frequency of Oscillation on Arterial Carbon Dioxide Pressure:** We then determined the effects of ECWO at 1 to 5 Hz on PaCO₂ and spontaneous breathing. The PEC determined previously to elicit a 5 to 10 mm Hg ΔPaCO₂ at a fo of 1.67 Hz was used throughout. The PEC was always +5 cm H₂O and ΔPc was PEC minus PEC. The fo was changed after each of the 5 min of ECWO. The fo was varied from 1.0 to 3.0 Hz by 0.5-Hz increments presented in random order. The subject was given at least 10 min to rest between each of the 5 min of ECWO. During ECWO, Vo was measured with a spirometer for approximately 15 s. All measurements were repeated on another day with the order of presentation of fo values being randomized again.

**The Influence of Chamber Pressure:** This session was to determine the effect of changing PEC, PEC, and fo on Vo and ΔPp. We wished to evaluate the limitations of this particular prototype in achieving the targeted ΔPc at various frequencies. A balloon catheter was introduced transnasally into the subject’s esophagus. The PEC, PEC, and I-E were set and the fo varied as before. Two minutes of ECWO was alternated with 2 min of rest. The Vo and ΔPp were measured. After ECWO was performed at the nine different fo values, the same procedure was used with different combinations of PEC, PEC, and fo. The targeted PEC was either −20.0 or −30.0 cm H₂O, the targeted PEC was either +5.0 or +10.0 cm H₂O, and the fo was 1.0, 2.5, or 4.0 Hz, resulting in 12 different combinations performed in random order.

**FIGURE 2.** The difference between the estimate of PaCO₂ (PcCO₂) using an expiratory maneuver and PaCO₂ determined from arterial blood samples is plotted vs PaCO₂. Samples were taken before and after ECWO at nine different frequencies in two subjects. Dashed reference lines represent the mean difference (0.6 mm Hg) and the 95% confidence intervals of the difference (0.1 and 1.1 mm Hg).
Table 1—Anthropometric, Pulmonary Function, and Estimated Dead Space Data for Each Subject*

<table>
<thead>
<tr>
<th>Subject</th>
<th>Sex</th>
<th>Age, yr</th>
<th>Height, cm</th>
<th>Weight, kg</th>
<th>FVC, L</th>
<th>FEV1/FVC Ratio, %</th>
<th>DCO, mL·min⁻¹·mm Hg⁻¹</th>
<th>Vd, mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>m</td>
<td>30</td>
<td>177</td>
<td>76</td>
<td>4.97</td>
<td>73</td>
<td>43.0</td>
<td>167</td>
</tr>
<tr>
<td>2</td>
<td>f</td>
<td>26</td>
<td>170</td>
<td>63</td>
<td>4.24</td>
<td>89</td>
<td>23.8</td>
<td>139</td>
</tr>
<tr>
<td>3</td>
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<td>85</td>
<td>4.62</td>
<td>79</td>
<td>35.3</td>
<td>187</td>
</tr>
<tr>
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<td>183</td>
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<tr>
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<td>29</td>
<td>179</td>
<td>79</td>
<td>5.44</td>
<td>82</td>
<td>46.1</td>
<td>174</td>
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<tr>
<td>6</td>
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<td>185</td>
<td>75</td>
<td>6.21</td>
<td>85</td>
<td>39.2</td>
<td>165</td>
</tr>
<tr>
<td>7</td>
<td>m</td>
<td>28</td>
<td>169</td>
<td>75</td>
<td>5.63</td>
<td>82</td>
<td>31.8</td>
<td>165</td>
</tr>
<tr>
<td>mean ± SD</td>
<td></td>
<td>30 ± 3</td>
<td>177 ± 6</td>
<td>77 ± 7</td>
<td>5.34 ± 0.78</td>
<td>82 ± 5</td>
<td>35.8 ± 7.7</td>
<td>169 ± 16</td>
</tr>
</tbody>
</table>

*FVC, forced vital capacity; DCO, diffusing capacity for carbon monoxide; Vd, anatomic dead space volume.

Statistical Analysis

The effect of the independent variable fo on the dependent variables ΔPaCO₂, Vo, and fb was determined for each subject and repeated on a separate day. Therefore, the study was a two-factor design (6 × 2) with repeated measures on the factor “day.” Analysis of variance was used on each of the dependent variables to determine if there was a significant effect among fo values between days. Since there was not a significant interaction of fo and day for any of the dependent variables, the main effect of fo was analyzed collapsed across day. When the main effect of fo was significant, post hoc analysis with paired t tests was used to determine if there was a significant difference at each fo relative to baseline. A probability value of 0.05 with a Bonferroni correction was used to determine significant differences for all statistical tests. All values are presented as mean and standard error unless indicated otherwise.

Results

Seven subjects completed the study and their heights, weights, pulmonary function data, and predicted anatomic dead space are presented in Table 1. All subjects tolerated ventilation well, and the ventilatory settings for PIC, PEC, and I:E determined during the introductory session are presented in Table 2.

Analysis of variance showed a significant effect of fo on ΔPaCO₂. As fo increased the ΔPaCO₂ after 5 min of ventilation decreased (Fig 3, A). Similarly, there was a significant effect of fo on Vo. As fo was increased, the Vo decreased (Fig 3, B). As shown in Figure 3, C, the ΔPaCO₂ occurred in the absence of spontaneous breathing (fb=0) at 1 and 1.5 Hz. As fo was increased, fb increased and was greater than 5 breaths·min⁻¹ at frequencies greater than 2.5 Hz (Fig 3, C).

In four subjects, ΔPpl was measured. The PIC and PEC and therefore ΔPC were held constant across the nine different frequencies as in the second and third sessions. There was no significant difference in ΔPpl with increasing fo. The mean ΔPC for the four subjects was 27 ± 3 cm H₂O and ranged from 23 to 30 cm H₂O. The mean ΔPpl for the four subjects was 7 ± 2 cm H₂O and ranged from 4 to 12 cm H₂O. In this session, PIC and PEC also were varied. A target ΔPC of 30 cm H₂O was achieved at each of the frequencies tested (1, 2.5, and 4 Hz). At higher targets (35 and 40 cm H₂O), the pressures achieved were less than those targeted. When ΔPC of 40 cm H₂O was targeted, the mean ΔPC achieved was 38 ± 1 cm H₂O. This was achieved only at 4 Hz. At 1 and 2.5 Hz, the pressures achieved were 31 ± 3 and 37 ± 2 cm H₂O, respectively. To obtain a high ΔPC, it was necessary to tighten the cuirass that in turn reduced the time during which the subject could tolerate ECWO.

Discussion

An advantage of ECWO is that it may provide adequate ventilation with relatively small tidal volumes, thus avoiding the detrimental effects associated with conventional mechanical ventilation. The oscillator model that we evaluated was easily applied and required no more than 10 min to be fitted. Movement was somewhat restricted in the supine and semirecumbent positions because of the cuirass and back plate, but we are aware (personal communication, Breasy Medical) that the design is being modified to increase mobility and comfort. At PCs of up to 30 cm H₂O, oscillation was well tolerated within the time frame of our measurements.

We intended to evaluate PC changes above 30 cm H₂O. Unfortunately the prototype we tested was not well tolerated at high PCs, since the cuirass needed to be tightly applied to minimize the compliance of its hollow foam lining. It was less comfortable when tightened, and subjects occasionally experienced bladder stimulation when fo was above 2 Hz.

We found that the increase in carbon dioxide...
elimination with ECWO at a constant oscillatory pressure was inversely related to the $f_0$. At frequencies of 1 to 2 Hz, a decrease in PaCO$_2$ could be achieved in the absence of spontaneous ventilation. Although spontaneous ventilation occurred as the $f_0$ was increased beyond 3 Hz, the subjects remained normocapneic, and their frequency of spontaneous breathing was reduced to below that of their resting unassisted respiratory rate.

Calverley et al.\textsuperscript{10} obtained similar results when inducing external chest wall oscillation at 3, 5, and 8 Hz using a pressure cuff. The method used in the study of Calverley et al.\textsuperscript{10} did not control lung volume and may have been less effective in promoting gas exchange as inspiration was achieved by the passive recoil of the thorax. As the $f_0$ was increased, they observed a decrease in the Vo. Spontaneous minute ventilation was reduced with oscillations of 3 and 5 Hz, but PaCO$_2$ did not change.

In our study, at oscillatory frequencies less than 2.5 Hz, the PaCO$_2$ decreased as the Vo was equal to or greater than the predicted anatomical dead space. In this frequency range, carbon dioxide elimination is highly dependent on bulk flow.\textsuperscript{24} At frequencies greater than 2.5 Hz, ECWO reduced dead space and allowed subjects to remain normocapneic at a lower frequency of spontaneous breathing. One can predict from the general equation of alveolar ventilation described by Venegas et al.\textsuperscript{24} that at 4 Hz a Vo of at least 130 mL would be required to maintain normocapnia in the absence of spontaneous breathing. Our measured Vo at 4 Hz was well below this value. It was not surprising therefore that spontaneous respiration continued. The maximum Pc changes that we could achieve at 4 Hz were 38 to 40 cm H$_2$O. At higher Pcs, spontaneous ventilation may well have been eliminated at higher frequencies. Thus, the characteristics of this ECWO device would be enhanced by a larger pump that would adequately compensate for cuirass compliance as well as leaks, and some of the useful properties of high-frequency ventilation on gas exchange could be used.

We could not identify an ideal frequency for ECWO between 1 and 5 Hz among healthy volunteers. Carbon dioxide elimination was directly related to the Vo achieved. As the $f_0$ increased, the Vo decreased. This decrease in Vo could be simply attributed to a decrease in the time for each oscillation (inspiration time or expiration time) because the Ppl amplitude did not decrease significantly over the range of frequencies. The Ppl amplitude remained constant as $f_0$ increased, changing only in association with changes in the amplitude of oscillatory Pc.

Resonant frequencies found during airway oscillations or high-frequency jet ventilation have ranged from 4 to 7 Hz among relaxed, healthy volunteers or

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure3.png}
\caption{The independent variable in each graph is the $f_0$ at constant Pi, Pe, and I-E. Plots are (A) the difference between the PaCO$_2$ after 5 min of ECWO and the resting (unassisted) PaCO$_2$; (B) shows tidal volume due to oscillation (Vo); bottom graph (C) indicates measured spontaneous respiratory frequency (fb). Mean values (±SEM) are presented for seven subjects. Asterisk denotes values significantly different from baseline (p<0.05).}
\end{figure}
paralyzed subjects.25,26 The frequency response of normal subjects to ECW0 may be different as both inspiration and expiration during ECW0 are forced. It may very well be that since inspiratory and expiratory oscillations are both active, Vo is a reflection of the time for which a pressure is applied across the chest wall.

Whether this method of ventilation is effective in patients with respiratory disease remains to be determined. Blood gas values were improved and endotracheal intubation was avoided in three of 9 patients in respiratory failure when ECW0 was used, although the oscillation frequency was limited to 1 Hz.27 At 5 Hz, chest wall oscillation has been shown to be effective in increasing intrapulmonary gas mixing as well as alveolar ventilation and Harf et al.28 have suggested that it might be of benefit to patients with COPD. When Piquet et al.29 superimposed oscillations at 5 Hz during exhalation only, he had limited success in lowering the spontaneous minute ventilation in patients with COPD. However, a slight increase in carbon dioxide elimination was achieved in association with a slower and deeper pattern of breathing. Oscillation in which both inspiration and expiration are active oscillatory processes may be more effective in such patients. Furthermore, by controlling the mean Pc at a negative value, the baseline lung volume may be increased, thus enhancing gas exchange by keeping the airways open. A preliminary report has suggested that changing the mean Pc by −15 cm H2O will increase the functional residual capacity by 580 mL.21

Our evaluation has been of a new externally applied ventilatory-assist device that functioned well among healthy volunteers at frequencies above those conventionally used. However, the characteristics of the pump and cuirass limit its use at the high Pcs required to achieve normocapnia in the absence of spontaneous respiration at frequencies greater than 3 Hz. Clearly, there will be considerable interest in the applicability of this technique to patients in whom respiratory impedance may be higher than in healthy volunteers as a result of conditions that affect the chest wall or the lung parenchyma.29,30 Whether or not the conclusions drawn from this report can be applied to such individuals will be the focus of further studies. In conclusion, we have evaluated a prototype of a commercially available ECW0 that can provide effective ventilation among healthy adults in the presence or absence of spontaneous breathing. Further studies are warranted to investigate the application of this technique in patients with impaired respiratory function.

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