High-Frequency Chest Wall Oscillation*
Assistance to Ventilation in Spontaneously Breathing Subjects

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In five supine normal subjects breathing spontaneously, we studied the effects of high-frequency chest wall oscillation (HFCWO), which was achieved by oscillating the pressure in an air-filled cuff wrapped around the lower thorax. Oscillations of 3.5 and 8 Hz (in randomized order) were applied for 15 minutes each at both maximal (mean of 90 to 102 cm H2O) and half-maximal peak tolerable cuff pressures. Fifteen minutes of control spontaneous ventilation preceded each HFCWO maneuver. The HFCWO resulted in a significant decrease in spontaneous minute ventilation (Ves) at maximal and half-maximal pressures by 35 and 40 percent, respectively, at 3 Hz and by 26 and 35 percent, respectively, at 5 Hz, with little change in Ves at 8 Hz. This occurred despite an unchanging arterial carbon dioxide tension at all frequencies. Arterial oxygen pressure increased at 3 Hz at maximal pressure but remained statistically unchanged at 3 Hz at half-maximal pressure and at 5 Hz and 8 Hz both at maximal and half-maximal pressures. We conclude that HFCWO may potentially assist ventilation in spontaneously breathing man without requiring an endotracheal tube.

Several groups of investigators have found that adequate gas exchange can be produced by high-frequency oscillation at the airway opening. This is generally accomplished by oscillating a continuous source of fresh gas presented to the endotracheal tube with tidal volumes smaller than the anatomic dead space. The mechanisms whereby adequate gas exchange is achieved appear to be a combination of diffusion and convection and may consist of one or more of the following: Pendelluft (literally, "oscillating air") between regions of uneven time constants, direct ventilation of alveoli close to the airway opening; asymmetric velocity profiles; and interaction of axial convection with radial transport caused by turbulence or molecular diffusion.

In conscious humans, high-frequency oscillation delivered by mouthpiece without an endotracheal tube has been found to produce gagging. This problem has led us to develop an alternative noninvasive system to produce gas oscillation without the requirement for an endotracheal tube. High-frequency chest wall oscillation (HFCWO) has been found to produce an oscillatory gas flow at the mouth and has been effective in maintaining gas exchange in anesthetized paralyzed dogs. With HFCWO, arterial gas tensions were found to improve in hypercapnic spontaneously ventilating dogs with airflow obstruction. In addition, clearance of mucus from the trachea as well as from the peripheral airways has been enhanced.

Because relatively small excursions of the chest wall are necessary to produce small oscillatory tidal volumes at the mouth at high frequency, it was reasoned that this may be a comfortable, yet effective noninvasive method of assisting ventilation in man. It was the purpose of this study to see how HFCWO can serve as a ventilatory assist in humans, as well as to observe any effect on gas exchange.

**Materials and Methods**

Three male and two female normal subjects, aged 33 to 43 years, were studied in the supine position. The study was approved by the hospital ethics committee, and informed consent was obtained from each subject.

The cuff applied to the chest for HFCWO has been described previously. This basically consists of a modified double blood pressure cuff with each bladder (12.5 x 27 cm) applied to the anterolateral part of the thorax and secured behind the thorax by a Velcro attachment. The lower border of the cuff was placed at the xiphisternum in the men, and the cuff was placed inframammary in the women. Large (2.54-cm inner diameter) tubing connected each bladder of the cuff to a modified air compressor which pumped air in and out of the bladders with a stroke volume of 680 ml. A flow of gas from a reservoir was fed into the cuff so as to keep the cuff partially inflated during the experimental runs. This gas was allowed to escape by a variable leak, thereby regulating the cuff pressure (measured by a pressure transducer Hewlett-Packard 267B).

An indication of the pressure transmitted into the thorax, esophageal pressure oscillations (Pes) were measured during HFCWO at functional residual capacity (FRC) using a 10-cm esophageal latex balloon filled with 0.5 ml of air and attached by 100 cm of tubing (PE 200) to a transducer (Hewlett-Packard 267B). The Pes was calibrated by oscillating the pressure in a sealed bottle by ± 10 cm H2O and was found to have a flat response to 7 Hz. The esophageal balloon was first positioned in the stomach and with-

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High-frequency Chest Wall Oscillation (Calverley et al.)
drawn 10 cm into the esophagus from the point at which reversal of pressure swings occurred during spontaneous ventilation.

To assess spontaneous tidal volume (Vrs) and respiratory frequency, a respiratory induction plethysmograph (RIP) was used (Respiracpe Corp.). This was used rather than a pneumotachygraph so as to have no dead space at the mouth. At the onset of the study, calibration was performed in the supine position using the isovolume technique to equalize rib cage and abdominal signals. The resultant sum signal was adjusted to equal a volume signal obtained simultaneously from a pneumotachygraph (Fleisch No. 2). During HFCWO, Vrs was measured from the middle of the oscillatory RIP signal at FRC to the midpoint of this signal at the end of inspiration.

With the RIP signal, it was not possible to measure the oscillatory tidal volume (Vto) produced at the mouth by HFCWO. This was because there was distortion of the induction coils of the RIP belts by the oscillating cuff on the thorax, as well as probable distortion of the chest wall and abdomen. Nevertheless, the Vrs could be reliably estimated (as discussed subsequently, Fig 1). To measure Vto, the subject was connected to a mouthpiece and the Fleisch No. 2 pneumotachygraph. This was always done after arterial blood gas levels had been drawn so that the arterial gas tensions would not be influenced by the added dead space at the mouth. The pneumotachygraph's flow and volume (integrated flow) signals were calibrated in a manner similar to that described previously.

The Vto was always measured at the end of expiration during spontaneous ventilation. An effective tidal volume (Vreff) for gas exchange during HFCWO was taken as the sum of Vrs and Vto.

Arterial blood gas tensions were measured by withdrawing blood from an indwelling radial arterial cannula. Blood was also withdrawn at the beginning and end of the study for analysis of serum creatine phosphokinase (CPK), hemoglobin, and haptoglobin. This was used to ascertain if HFCWO caused damage to the muscles of the chest wall or the red blood cells.

To determine the change in the FRC of the subject during HFCWO relative to spontaneous ventilation (ΔFRC), an "inspiratory capacity" (IC) maneuver was performed on both occasions, and the difference was calculated. The measured values were not the true ICs, as the subject was restricted from full expansion of the rib cage by the deflated cuff around the thorax. During HFCWO the cuff was suddenly deflated, and the oscillations were discontinued just after the onset of a spontaneous inspiration; the subject was instructed to continue to inspire in order to perform the IC maneuver.

An electrocardiogram was recorded throughout using three standard chest leads. All signals were recorded on paper (Hewlett-Packard 7758A recorder). During each period with and without HFCWO, arterial blood pressure was noted using a standard sphygmomanometer.

The experiments were always conducted in the following sequence: the control period before each HFCWO consisted of the subject resting quietly with the cuff deflated for at least 15 minutes. The Vrs and spontaneous respiratory frequency were measured by induction plethysmography, and a sample of arterial blood was drawn at the end of the 15-minute period. The subject then breathed through the pneumotachygraph for a further three minutes, during which time ventilation measured with the pneumotachygraph was compared with that measured by RIP. At the end of this period, an IC maneuver was performed. The subject then underwent HFCWO for a minimum of 15 minutes, and the previous measurements were repeated. Using the integrated pneumotachygraphic signal, it was now possible to obtain Vto.

Each HFCWO (at 3, 5, and 8 Hz in randomized order) was preceded by a control period of spontaneous ventilation alone, to which all measurements were related. Applied maximal cuff pressure was determined empirically at the outset of each testing period, with the subject indicating the maximum pressure he believed he could tolerate for 15 minutes. The HFCWO experiments were conducted at this pressure and, after a further 15 minutes of control measurements, were repeated at half of the maximum peak cuff pressure. A separate maximum peak cuff pressure was determined for each frequency. Any subjective side effects during the experiment and in the 24 hours after the study were noted.

Results presented below are means ± SE unless otherwise stated, and statistical significance was evaluated using a nonparametric

![Figure 1. Results comparing values for Vrs measured from integrated pneumotachygraph flow signal those measured by RIP.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21505/)

![Figure 2. Mean ± SE of cuff pressure, Pexp, peak flow and Vto with HFCWO at maximal (M) and half-maximal (0.5 M) cuff pressures.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21505/)
RESULTS

Maximal tolerable cuff pressures were similar for each subject at each of the tested oscillatory frequencies. For the three frequencies employed, the mean pressures for the five subjects ranged from 90 to 102 cm H$_2$O (Fig 2). On the back stroke of the piston pump, the residual positive pressure in the cuff was higher with maximal than half-maximal pressure; and the mean pressures at the three frequencies ranged from 8 to 13 cm H$_2$O at maximal pressure from 2 to 4 cm H$_2$O at half-maximal pressure. The peak positive Pes$_{\text{max}}$, measured at FRC, ranged from a mean of 9.5 to 13.8 cm H$_2$O. These applied pressures at maximal pressure resulted in a mean peak expiratory oscillatory flow ($\dot{V}_o$), measured near FRC, of 2.1 to 2.77 L/sec, which was usually greater than peak inspiratory flow rates. The $\dot{V}_o$, measured with the pneumotachygraph, had a mean value of 69 ml at 3 Hz, 46 ml at 5 Hz, and 39 ml at 8 Hz. In all five subjects, half-maximal tolerable cuff pressures produced changes in Pes, $\dot{V}_o$, and Vro very similar to those seen at maximal pressure.

With HFCWO, there were no significant changes in arterial oxygen pressure (PaO$_2$) or arterial carbon dioxide tension (PaCO$_2$) from control values at either maximal or half-maximal pressure (Fig 3), except at 3 Hz and maximal pressure, where PaO$_2$ increased significantly from a mean of 82.8 to 90.4 mm Hg ($p<0.05$). Although there was a similar trend at 5 Hz, it was not statistically significant. Despite the lack of change in PaCO$_2$, spontaneous minute ventilation ($\dot{V}_{es}$) decreased significantly by a mean of 35 percent at maximal pressure and 40 percent at half-maximal pressure at 3 Hz and by 26 percent and 35 percent at maximal and half-maximal pressure, respectively, with HFCWO at 5 Hz (Fig 4). At 8 Hz, there was no significant difference in $\dot{V}_{es}$ from control at both maximal and half-maximal pressures.

Table 1—Mean Changes in Spontaneous Breathing Pattern with HFCWO

<table>
<thead>
<tr>
<th>Group*</th>
<th>Respiratory Frequency, breaths per min</th>
<th>$\dot{V}_t$, Vts per min</th>
<th>Vreff</th>
<th>T$_1$, sec</th>
<th>$\dot{V}_t$/T$_1$, ml/sec</th>
<th>T$<em>1$/T$</em>{TOT}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>15.6</td>
<td>448</td>
<td>...</td>
<td>1.39</td>
<td>317</td>
<td>0.35</td>
</tr>
<tr>
<td>3 Hz, M</td>
<td>14.8</td>
<td>306†</td>
<td>375†</td>
<td>1.36</td>
<td>151†</td>
<td>0.35</td>
</tr>
<tr>
<td>Control</td>
<td>13.6</td>
<td>534</td>
<td>...</td>
<td>1.35</td>
<td>375</td>
<td>0.31</td>
</tr>
<tr>
<td>3 Hz, 0.5 M</td>
<td>14.6</td>
<td>366†</td>
<td>435†</td>
<td>1.36</td>
<td>159†</td>
<td>0.32</td>
</tr>
<tr>
<td>Control</td>
<td>13.8</td>
<td>468</td>
<td>...</td>
<td>1.32</td>
<td>313</td>
<td>0.33</td>
</tr>
<tr>
<td>5 Hz, M</td>
<td>14.0</td>
<td>356</td>
<td>402</td>
<td>1.50</td>
<td>285</td>
<td>0.31</td>
</tr>
<tr>
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<td>14.4</td>
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<td>...</td>
<td>1.71</td>
<td>298</td>
<td>0.35</td>
</tr>
<tr>
<td>5 Hz, 0.5 M</td>
<td>16.8</td>
<td>373</td>
<td>433</td>
<td>1.36</td>
<td>285</td>
<td>0.30</td>
</tr>
<tr>
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<td>12.8</td>
<td>531</td>
<td>...</td>
<td>1.67</td>
<td>357</td>
<td>0.31</td>
</tr>
<tr>
<td>8 Hz, M</td>
<td>17.6</td>
<td>313</td>
<td>352</td>
<td>1.10</td>
<td>285</td>
<td>0.30</td>
</tr>
<tr>
<td>Control</td>
<td>9.8</td>
<td>429</td>
<td>...</td>
<td>1.32</td>
<td>428</td>
<td>0.30</td>
</tr>
<tr>
<td>8 Hz, 0.5 M</td>
<td>19.8</td>
<td>304</td>
<td>342</td>
<td>1.14</td>
<td>274</td>
<td>0.35</td>
</tr>
</tbody>
</table>

*M, Maximal cuff pressure; and 0.5 M, half-maximal cuff pressure. †p<0.05.
Spontaneous tidal volumes recorded with RIP agreed well with those from the pneumotachograph recorded both at rest and during HFCWO throughout the study (correlation coefficient \( r = 0.91; p<0.001 \)) (Fig 1). At both maximal and half-maximal pressures and at all frequencies studied, the main change from control in the pattern of breathing was a decrease in \( V_{T} \), \( V_{T_{eff}} \), and mean inspiratory flow rate (\( V_{T_{eff}} \) divided by inspiratory time [\( T_{I} \)] (Table 1). No significant change occurred in spontaneous respiratory frequency at 3 and 5 Hz, but an increased frequency occurred at 8 Hz. The duty cycle \( T_{I} \) divided by total respiratory time (\( T_{T_{tot}} \)) remained unchanged. There was a consistent fall in FRC during HFCWO; the mean decrease ranged from 130 to 270 ml at the different applied frequencies (Fig 5). The changes were least marked at 3 Hz. Again, maximal and half-maximal pressures produced approximately equivalent changes in ΔFRC.

There were no changes in the serum CPK, hemoglobin, and haptoglobin levels measured at the end of the experiment. The blood pressures and pulse rates remained stable throughout the study, and all of the subjects were able to undergo the entire study. Four out of the five subjects found compression at 8 Hz with maximal pressure uncomfortable. In contrast, no side effects were attributed to compression at half-maximal pressure at any frequency. No serious sequelae, eg, pneumothorax or cutaneous blistering, occurred in any of the subjects.

**Discussion**

We studied normal subjects who breathed spontaneously with superimposed HFCWO. Our studies show that at 3 and 5 Hz, HFCWO can reduce \( V_{es} \) and maintain gas exchange in normal man. These effects occurred equally at both maximal and half-maximal tolerable cuff pressures. They were not associated with significant side effects except for some mild chest wall discomfort during HFCWO at 8 Hz at maximal tolerable cuff pressures. This may in part account for the more rapid shallow breathing seen at 8 Hz. The rapid shallow breathing would result in a greater ratio of dead space to tidal volume and may counteract the beneficial effect in terms of gas exchange of the addition of HFCWO. Although the periods of application of HFCWO were relatively short, the subjects believed that the applied pressures, especially the half-maximal tolerable pressure, could have been employed for much longer periods of time.

The mechanism whereby a \( V_{ro} \) is produced by HFCWO would be as follows: the driving forces producing oscillatory expiratory flow would be a result of the cuff acting on the thorax, the passive elastic characteristics of the respiratory system, and any active expiratory muscular effort. The restoring forces for inspiratory oscillatory flow would be the inspiratory muscles throughout spontaneous inspiration and the passive elastic recoil of the respiratory system below the subject's usual FRC.

The periods of breathing through the pneumotachograph after each blood sampling served to validate the stability and accuracy of the calibration of the RIP (Fig 1) for the measurement of \( V_{T} \) and confirmed that the HFCWO did not change the RIP signal. Although maximal high-frequency swings in \( P_{es} \), ranged up to a mean of 13.8 cm H\(_2\)O, the volume change at the mouth during each oscillatory cycle was less than 100 ml. The possible explanations for this apparent incongruity are as follows: (1) the measurement of \( P_{es} \) was inaccurate as a result of the supine position due to the influence of mediastinal contents (both the mediastinum and esophagus have an unknown frequency response); (2) local application of chest wall pressure caused very nonuniform pleural and transpulmonary pressure changes; and (3) with the rapid acceleration and deceleration of airway gas caused by HFCWO, a high internal resistance to airflow would occur. This would result in alveolar gas compression and decompression and would, with relatively little volume appearing at the airway opening, cause relatively large changes in \( P_{es} \). Independent of the magnitude of swings in \( P_{es} \) caused by HFCWO, the oscillatory volumes of magnitude less than 100 ml were responsible for the physiologic changes reported herein.

The HFCWO resulted in a lower FRC (ΔFRC) by a mean of 130 to 270 ml at the three different applied frequencies when compared to spontaneous ventila-
tion. The ΔFRC was measured by the difference in "inspiratory capacity" from a stable FRC. The subjects did not attain their true total lung capacity (TLC), as they were limited by the presence of the deflated cuff. The fact that the IC during spontaneous ventilation had a variance of only 0.05 percent implies that the "TLC" attained was reproducible. The lowered FRC with HFCWO did not result in impaired gas exchange. The maintenance of a constant PaCO₂ despite a significant decrease in V̇es is undoubtedly a result of enhanced elimination of carbon dioxide by the externally applied HFCWO. This was most likely due to central regulation of the amount of spontaneous ventilation necessary to eliminate the carbon dioxide present in the lung. As the carbon dioxide load to be eliminated by spontaneous ventilation is reduced by HFCWO, it is reasonable to have a decreased Vt/TI as described by Phillipson et al., who saw similar but more drastic falls in ventilation in sheep ventilated by a carbon dioxide membrane lung. The reduced ventilatory drive is consistent with results observed during HFCWO in spontaneously breathing but obstructed dogs which became apneic after the PaCO₂ dropped below control values. Other authors have reported apnea during HFO/AO. This phenomenon is probably the result of some reflex mechanism.

The fact that PaO₂ did not decrease despite a decrease in FRC may be explained by the fact that the normal subjects tested most likely had small closing capacities, as the age range was 33 to 43 years. The improvement in PaO₂ at 3 Hz with maximal cuff pressure and the tendency to improved PaO₂ at 5 Hz may be a result of the improved efficiency of gas transport during HFCWO despite the fact that V̇eff during HFCWO was less than V̇rs without HFCWO. Harf et al. have recently demonstrated that in normal subjects where V̇rs and respiratory frequency were held constant, the addition of HFCWO resulted in a more rapid nitrogen washout. This was believed to be a result of HFCWO reducing the effective dead space. Other mechanisms as described by Chang may also be operative.

The same peak positive Pes, at FRC was achieved at half-maximal pressure at all tested frequencies (Fig 2). The fact that values for V̇rs were the same at half-maximal and maximal pressure would suggest that the peak Pes, was representative (although probably inaccurate) of the pressure transmitted from the cuff to within the thorax. As FRC during HFCWO declined by only a small amount, it is unlikely that the change in passive elastic characteristics of the chest wall accounted for the fact that Pes, was similar at maximal and half-maximal pressure. One possible explanation for this was that the chest wall was stiffening by active muscular contraction in response to the higher applied cuff pressure so as to allow only a certain maximal pressure to enter the thorax. Another possible explanation is that the cuff was inflated in such a way that it became more rounded in the cephalad-caudad direction at maximal pressure than at half-maximal pressure, thus having a reduced area of apposition to thorax at maximal pressure. This may be related to the particular cuff used. Since we were limited by the available equipment and since we were interested more in the physiologic consequences than in the engineering aspect of HFCWO, no further investigation was made on the effect of peak pressure on the chest wall. A third explanation was that the respiratory system was highly alinear so that when an increased pressure was applied to the thorax, there was little change in V̇rs.

Despite the considerable pressures applied, the subjects tolerated HFCWO well and experienced no discernible side effects. This suggests that HFCWO can be used for even longer periods. This was not done in the present experiment, as further modifications of the thoracic cuff should improve its efficiency eg, increasing its area of apposition to the thorax. As all of the subjects in this study preferred the lower frequencies, a prolonged use of HFCWO should probably be confined to relatively low frequencies, eg, 3 Hz. The results in our study of spontaneously ventilating dogs with induced airflow obstruction showed that PaCO₂ fell from hypercapnic to hypocapnic levels during HFCWO. This raises the possibility that HFCWO can be of benefit to patients with hypercapnic chronic obstructive pulmonary disease. The results of the present study show that HFCWO can assist ventilation in conscious normal subjects for short periods of time.

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REFERENCES
1 Lunkenheimer PP, Raffenbeul W, Keller H, Frank I, Dickhut HH, Fuhrmann C. Application of transtracheal pressure oscillations as a modification of "diffusing respiration." Br J Anaesth 1972; 44:627-38
8 Hazleton FR, Scherer PW. Bronchial bifurcations and respiratory mass transport. Science 1980; 208:69-71
15 Phillipson EA, Duffin J, Cooper JD. Critical dependence of respiratory rhythmicity on metabolic CO₂ load. J Appl Physiol Respir Environ Exercise Physiol 1981; 50:45-54
16 Chang HK, Harf A. High-frequency ventilation: a review. Respir Physiol 1984; 57:135-52

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