Chest Wall Oscillation at 1 Hz Reduces Spontaneous Ventilation in Healthy Subjects During Sleep*

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Study objective: The objective was to determine whether external chest wall oscillation (ECWO) during sleep (1) reduced spontaneous ventilation while maintaining adequate gas exchange over several hours, (2) influenced the quality and distribution of sleep, and (3) increased the number of respiratory events.

Design: Prospective controlled study with counterbalanced order of intervention.

Setting: Pulmonary function sleep laboratory.

Participants: Seven healthy volunteers.

Intervention: One night of ECWO at 1 Hz (I:E=1:1; oscillation mean [SEM] from -11.1 [0.7] to 6.0 [0.7] cm H2O) and a night during which the cuirass was applied without ECWO.

Measurements and results: ECWO resulted in a significant decrease in spontaneous minute ventilation (Ve) in all stages of sleep. ECWO was associated with a reduction in the total sleep time and a reduction in rapid eye movement (REM) sleep. The number of stage changes and the sleep efficiency did not change significantly. The mean PCO2 was similar between the control and cuirass nights (44 to 46 mm Hg). There was a significant decrease in the mean P<sub>CO2</sub> during stage 1 (41 [2] mm Hg) and stage 2 (42 [2] mm Hg) sleep during the ECWO night. The mean arterial oxygen saturation (SaO<sub>2</sub>) was maintained at 96 to 97% throughout sleep during the control, cuirass, and ECWO nights. The apnea-hypopnea index increased (p<0.05) during ECWO mostly due to an increase in the number of hypopneas in stage 2 sleep. During ECWO, 18 of 30 respiratory events were associated with an arousal, whereas only 2 events were associated with an arousal during the control night.

Conclusions: ECWO can be tolerated for several hours and will assist ventilation while maintaining normal mean P<sub>CO2</sub> and mean SaO<sub>2</sub> during sleep. Monitoring of the apnea-hypopnea index and the SaO<sub>2</sub> is recommended at the time of application. Clinical trials to define the most appropriate indications for ECWO are now necessary.

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Key words: chest wall oscillation; gas exchange; sleep; ventilation

Abbreviations: AHindex=apneas and hypopneas per hour of sleep; ECWO=external chest wall oscillation; REM=rapid eye movement; SaO<sub>2</sub>=arterial oxygen saturation; Ve=minute ventilation

Although a number of studies<sup>1-8</sup> have described patients successfully ventilated in their homes for up to 20 years using noninvasive negative pressure ventilation, recent prospective controlled trials in subjects with COPD reported poor subject tolerance at night to the point that most subjects chose to apply negative pressure ventilation only during the day.<sup>9-11</sup> When healthy volunteers or subjects with COPD have been able to sleep during negative pressure ventilation administered at conventional frequencies (10 to 16 breaths·min<sup>-1</sup>), an increase in apneas and hypopneas attributable to upper airway obstruction has been observed.<sup>12,13</sup> The precise mechanism and site of this upper airway obstruction have not been clearly defined but are likely to relate at least in part to the amount of negative pressure applied. In healthy volunteers, airway collapse occurred when a threshold pressure of -12 cm H<sub>2</sub>O was superimposed on spontaneous breathing.<sup>14</sup> Conventional negative pressure ventilators utilize pressures well in excess of this threshold.

In contrast, external chest wall oscillation (ECWO) involves oscillating the chest wall at higher than conventional frequencies using negative and positive pressures within a cuirass. Recent reports have suggested that ECWO can provide effective ventilation among healthy adults<sup>15,16</sup> and patients with COPD<sup>17</sup> in the presence or absence of spontaneous breathing. In

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these studies, ECWO was effective and well tolerated at oscillatory frequencies of less than 2 Hz.

The external chest wall oscillator differs from a conventional negative pressure ventilator in that it achieves mechanical ventilatory support with less negative pressure. It is therefore possible that ECWO may not be associated with an increase in obstructive apneas and hypopneas. An alternative hypothesis is that even with a high operating frequency, a low chamber pressure span, and a positive expiratory assist, ECWO would still be associated with an increase in the apnea-hypopnea index because any airway exposure to negative pressure in the absence of spontaneous breathing will be associated with a predisposition to obstructive respiratory events.

Most reports have described the use of continuous ECWO for only brief periods, usually less than 1 h, although preliminary clinical information has suggested that it may be tolerated for much longer. 18,19 As it is both effective and easy to apply, ECWO may provide a practical noninvasive means of mechanical support for patients in whom a high ventilatory demand, altered mechanical load, or compromised respiratory muscle function contributes to ventilatory failure. 15-17,20 Clearly, for those in whom ongoing support will be required for several hours or days, it is important to determine whether ECWO will be effective and well tolerated for longer periods.

The purpose of this study was to determine whether ECWO could be tolerated during sleep, whether adequate gas exchange could be maintained over several hours in the presence of a reduction in spontaneous ventilation, and whether subjects would develop an increase in the apnea-hypopnea index during sleep.

Materials and Methods

Subjects

The protocol was approved by the University’s Human Subjects Review Committee and the Hospital’s Medical Advisory Committee. All participating subjects consented to the study after the procedures and risks had been explained to them. The subjects were not taking any medication and did not consume alcohol or caffeine on the evening of a sleep study.

External Chest Wall Oscillation

External chest wall oscillation was applied using an oscillator (Hayek Oscillator; Breasy Medical Equipment; London, England) that included a power unit, control unit, cuirass, and back plate. The cuirass was a specially shaped plastic shell that spanned the anterolateral chest and abdomen from sternum to pubis. The oscillator frequency was set at 1 Hz. Oscillation at 1 Hz has been shown to provide an oscillatory volume adequate to lower the PCO2 by 15-17 or maintain eucapnia with cuirass pressure changes of less than 20 cm H2O. With these settings, the resulting esophageal pressure changes have been less than 10 cm H2O. 16

Minute Ventilation

The inductance plethysmograph (Respirtrac; Ambulatory Monitoring; Ardsley, NY) was calibrated by asking the subject to breathe into a rolling-seal spirometer (Morgan, Gillingham, England) for 2 min. The plethysmograph was calibrated during spontaneous breathing every night before sleep and in the morning after the subject awoke. On the night that ECWO was used, calibration was also done with and without ECWO. To determine spontaneous breathing during ECWO, the signal was digitally low pass filtered at 0.6 Hz to remove the higher-frequency oscillation. An example of calibration recordings during ECWO is shown in Figure 1. The limits of agreement 21 between the spirometer and the plethysmograph for tidal volume measurements were ±50 mL when repeated breaths were measured and ±150 mL for comparison of single breaths. The average spontaneous tidal volume and frequency of breathing was determined and spontaneous minute ventilation (Ve) was calculated as their product.

Sleep Studies

Each sleep study included full respiratory polysomnographic measurements. Standard sleep variables included an ECG, submental electromyogram, electro-oculogram, and EGG. Sleep stages were scored in 30-s epochs by standard criteria. 22 Air flow was recorded with an oronasal thermocouple (model 02-1010A, Pro-Tech Service Inc; Woodenville, Wash.).

Arterial oxygen saturation (SaO2) was measured with a pulse oximeter attached via a finger probe (Ohmeda Biox 3700; Louisville, Colo.). Pco2 was measured transcutaneously (Kontron Micro Gas 7640; Watford, England). Transcutaneous Pco2 accurately and consistently reflects arterial PCO2 under standardized conditions. 23,24 Measurements of SaO2 were made on an epoch-by-epoch basis. During each epoch, the highest and lowest values were recorded and the results were expressed as the mean, the mean high, and the mean low values for each sleep stage. Measurements of Pco2 were made on an epoch-by-epoch basis and were expressed as the mean, mean low, and mean high value for each sleep stage. All sleep data were recorded and stored on an optical laser disk. The calibrated cuirass pressure signal and the signal from the calibrated inductance plethysmograph were recorded and stored separately.

Apneas and hypopneas were identified for each sleep stage. An apnea was defined as a cessation of airflow for more than 10 s. A hypopnea was defined as a decrease of 50% in the airflow relative to the preceding breaths for greater than 10 s. Apneas and hypopneas were also expressed as an index (apneas and hypopneas per hour sleep [AH index]) for each sleep stage. The AHIndex was calculated as the sum of apneas and hypopneas per hour for each sleep stage. Arousals associated with apneas or hypopneas were counted and the lowest SaO2 associated with each apnea and hypopnea was recorded.

On nights without ECWO, an obstructive apnea was defined as cessation of airflow despite respiratory effort as shown by the inductance plethysmograph. When no such effort was evident, apneas were considered to be of central origin. Mixed apneas contained periods of both central and obstructive apneas. During ECWO, all apneas were considered obstructive since pressure changes, "efforts," were constantly induced by the oscillator.

Protocol

During the introductory session, the negative pressure was adjusted so that eucapnia could be maintained during wakefulness with at least a 50% reduction in spontaneous Ve. Adequacy of ECWO was determined by the following: (1) subject comfort; (2) stable normal SaO2 (>90%); (3) stable normal Pco2 (equal to or no more than 5 mm Hg less than resting Pco2); and (4) a reduction (≥50%) in spontaneous Ve. The expiratory cuirass pressure was in the range of +1 to +10 cm H2O and the I:E was 1:1.

Every subject completed 5 sleep studies, each separated by at least 1 week. The first sleep study was the control night. During the second sleep study, the subject either slept while wearing the cuirass...
Analysis

For each dependent variable, a one-way repeated measures analysis of variance was used to determine if there were significant differences among the treatment nights (control, cuirass, and ECWO). If so, a Student-Newman-Keuls test was used for multiple pairwise comparisons of the treatment nights for each dependent variable. A p<0.05 was considered significant for all statistical tests. All values were expressed as mean (SEM) unless stated otherwise.

RESULTS

Subjects

Seven healthy volunteers between the ages of 29 and

Table 1—Quality and Distribution of Sleep

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Cuirass</th>
<th>ECWO</th>
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</thead>
<tbody>
<tr>
<td>Total sleep time, min</td>
<td>312 (19)</td>
<td>278 (29)</td>
<td>213 (17)*</td>
</tr>
<tr>
<td>Awake, % sleep period</td>
<td>12 (5)</td>
<td>10 (3)</td>
<td>20 (4)</td>
</tr>
<tr>
<td>Stage 1, % sleep period</td>
<td>9 (1)</td>
<td>8 (1)</td>
<td>12 (3)*</td>
</tr>
<tr>
<td>Stage 2, % sleep period</td>
<td>44 (4)</td>
<td>43 (6)</td>
<td>39 (4)</td>
</tr>
<tr>
<td>Stage 3, % sleep period</td>
<td>10 (1)</td>
<td>12 (2)</td>
<td>10 (2)</td>
</tr>
<tr>
<td>Stage 4, % sleep period</td>
<td>12 (2)</td>
<td>11 (2)</td>
<td>9 (2)</td>
</tr>
<tr>
<td>REM, % sleep period</td>
<td>11 (3)</td>
<td>10 (2)</td>
<td>5 (2)*</td>
</tr>
<tr>
<td>Stage changes, h⁻¹</td>
<td>23 (2)</td>
<td>26 (2)</td>
<td>24 (2)</td>
</tr>
<tr>
<td>Sleep efficiency, %</td>
<td>85 (3)</td>
<td>84 (3)</td>
<td>76 (6)</td>
</tr>
</tbody>
</table>

*Significantly different from control night, p<0.05.

41 years completed the 3 sleep nights. Their height, weight, and body mass index were 175 (2) cm, 71 (5) kg, and 23 (1) kg/m², respectively. External chest wall oscillation at 1 Hz was used with a nadir of negative pressure of -11.1 (0.7) cm H₂O and a peak of positive pressure of 6.0 (0.7) cm H₂O which resulted in cuirass pressure oscillation of 17.0 (0.9) cm H₂O and a mean cuirass pressure of -2.5 (0.5) cm H₂O. There was a significant effect of the treatment on the group's perceived amount of sleep estimated upon awakening (F=5.05; df=2,10). The subjects stated that their perceived amount of sleep after ECWO (190 [44] min) was less than their perceived amount of sleep after the first control night (323 [27] min).

Two subjects experienced moderate lower back pain during the cuirass and ECWO nights. A third subject experienced lower back pain during the ECWO night only. The back pain was relieved by supporting the shoulders with pillows, elevating the subject's knees, or by removing the cuirass for 1 h after which it was reapplied. One subject experienced a "stitch in his side" during ECWO that was relieved by stopping ECWO for 25 min. One subject experienced chest wall discomfort after 4 h of ECWO that ceased when ECWO was discontinued.

Sleep Summary

The quality and distribution of sleep were summarized in Table 1. The total amount of sleep differed significantly across treatment nights (F=4.99; df=2,12). It was significantly reduced during the ECWO night.
FIGURE 2. Spontaneous ventilation during different stages of sleep. The panels show the control night (no ventilatory apparatus), the cuirass night (cuirass without oscillation), and the ECWO night (oscillation). Asterisk = significantly different from the ventilation during the same stage of sleep of the control night (p<0.05). Dagger = significantly different from the ventilation during the same stage of sleep of the control and cuirass nights (p<0.05).

(213 [17] min) as compared with the control night (312 [19] min), but the total amount of sleep during the cuirass night (278 [29] min) did not differ from the control night. During ECWO, analysis of variance showed significant differences across treatment nights in the amount of rapid eye movement (REM) sleep (F=5.07; df=2,12) and in stage 1 sleep (F=7.68; df=2,12). The amount of REM sleep decreased significantly and the amount of stage 1 sleep increased significantly. A trend toward wakefulness did not reach statistical significance. The sleep efficiency during the ECWO night (76 [6]%) was slightly less than that observed during the control night (85 [5]%).

Spontaneous Breathing

Analysis of variance showed that spontaneous VE was significantly different across treatment nights during wakefulness (F=33.6; df=2,11), and all stages of sleep (stage 1: F=35.5; df=2,11; stage 2: F=32.6; df=2,12; stage 3: F=7.5; df=2,10; stage 4: F=13.2; df=2,10; REM: F=42.2; df=2,9). During the control night, mean spontaneous VE was 4.8 [0.5] L·min⁻¹ during wakefulness and ranged from 4.0 [0.5] (REM) to 4.6 [0.9] L·min⁻¹ (stage 4) during different stages of sleep (Fig 2). Assisting ventilation with ECWO resulted in a significant decrease in spontaneous VE during wakefulness and all stages of sleep. During the ECWO night, the mean spontaneous VE was lowest during stage 2 sleep (0.7 [0.3] L·min⁻¹) when compared with stage 3 (1.8 [0.5] L·min⁻¹) and 4 (1.7 [0.5] L·min⁻¹) as well as REM sleep (1.5 [0.6] L·min⁻¹). In contrast, when the cuirass was applied without ECWO, spontaneous VE was highest during wakefulness (5.2 [0.5] L·min⁻¹) and REM sleep (5.3 [0.5] L·min⁻¹) and lowest during stages 1, 2, 3, and 4.

Oximetry and Carbon Dioxide Tension

Analysis of variance showed a significant treatment effect on the Pco₂ across nights during stage 1 (F=4.97; df=11,2) and stage 2 (F=4.02; df=11,2) sleep.
During the control night, the mean Pco2 increased from 44 [1] mm Hg during wakefulness to 46 mm Hg during non-REM and REM sleep. During the cuirass night, similar levels of Pco2 were observed. During ECWO, the Pco2 was 40 [2] mm Hg during wakefulness. The Pco2 during ECWO in stage 1 (41 [2] mm Hg) and stage 2 (42 [2] mm Hg) differed significantly from the control night. There was no difference in mean Pco2 between the control and ECWO nights during REM sleep (46 [1] mm Hg and 45 [2] mm Hg, respectively).

The mean SaO2 was maintained between 96 and 97% throughout wakefulness and sleep during the control, cuirass, and ECWO nights.

<table>
<thead>
<tr>
<th>Table 2—Apneas and Hypopneas by Sleep Stage</th>
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<tbody>
<tr>
<td>AH Index, h⁻¹</td>
</tr>
<tr>
<td>Control</td>
</tr>
<tr>
<td>Awake (SE)</td>
</tr>
<tr>
<td>(0.1)</td>
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<tr>
<td>Stage 1</td>
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<td>(SE)</td>
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<td>Stage 2</td>
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<td>Stage 4</td>
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<td>(SE)</td>
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<td>REM</td>
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<tr>
<td>(SE)</td>
</tr>
<tr>
<td>Total</td>
</tr>
<tr>
<td>(SE)</td>
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</tbody>
</table>

* n/a=not available.

1 Significantly different from control and cuirass nights, p<0.05.

Apneas and Hypopneas

The AHIndex for different sleep stages and the associated arousals and lowest SaO2 are shown in Table 2. The AHIndex during the ECWO night attributable mostly to an increase in hypopneas in stages 1 and 2. Figure 3 shows recordings of sleep variables from one subject during control, cuirass, and ECWO nights, including an example of ECWO in the absence of spontaneous ventilation and a respiratory event that occurred during stage 2 sleep. In stage 2 (F=5.16, df=12.2), the AHIndex during ECWO (17.4 [8.0] h⁻¹) was significantly greater than during the control night (1.5 [0.9] h⁻¹). During stage 2 sleep on the ECWO night, there was a wide range in the AHIndex (0 to 57 h⁻¹) but only in 3 subjects was the AHIndex above 10 h⁻¹. All subjects achieved stage 4 sleep without any respiratory events. Of the 5 subjects who achieved REM sleep, the only subject who experienced respiratory events during REM sleep (69 h⁻¹) had an AHIndex of 17 h⁻¹ during REM sleep on the control night.

During ECWO, 18 of 30 respiratory events were associated with an arousal whereas only 2 events were associated with an arousal on the control night. During the control night, the minimum SaO2 associated with apneas or hypopneas was 94% for the group and during the cuirass night the minimum SaO2 for the group was 92 to 94%. During ECWO, the minimum SaO2 was 85% with 4 of 6 subjects having an SaO2 of less than 90% during at least 1 event. The 1 subject with an elevated AHIndex during the control night reached a nadir in SaO2 of 66% during ECWO. In other stages of sleep, the minimum SaO2 associated with apneas or hypopneas was greater than 90%.

Discussion

In healthy volunteers, ECWO provided adequate gas exchange in the presence of a reduction in spontaneous ventilation over several hours and therefore can be considered among the options for noninvasive ventilatory support. However, it was also associated with a reduction in the total sleep time and an increase in the number of apneas and hypopneas when compared with control measures.

The reduction in sleep quality might well have been modified had the subjects had more exposure to the oscillator. For most subjects, it was only their second experience with ECWO and the first during which the intent was to sleep. Neither the sensation of the cuirass on the chest wall nor the obligation to sleep in a supine position resulted in a sleep profile that differed from control (unencumbered) measures.

ECWO was well tolerated by the subjects and a substantial amount of sleep was achieved. The reduction in REM sleep (two of seven subjects did not achieve REM sleep) and the trend toward increased wakefulness and decreased sleep efficiency were small.
Furthermore, ECWO compared well with more conventional negative pressure ventilation which induced a 20% reduction in sleep efficiency among 5 healthy subjects. The absence of changes in the other indicators of sleep quality such as movement arousals, stage changes, and the amount of slow-wave sleep suggested that subjects could easily become accustomed to ECWO.

Whereas the inductance plethysmograph has been well validated among spontaneously breathing subjects, it was important to ensure valid measures of spontaneous ventilation in the setting of ECWO. This was achieved by low-pass filtering of any oscillations attributable to ECWO (Fig 1) and confirmed by calibrating with a rolling-seal spirometer both with and without ECWO at the beginning and at the end of each night. Negative pressure ventilation may have increased the functional residual capacity; therefore, we calibrated the inductance plethysmograph over a wide (3 L) volume. A mean chamber pressure of 2.5 cm H2O applied to a respiratory system with normal compliance was unlikely to have resulted in changes in volume outside the range of calibration. The good agreement (±50 mL) between the calibrated plethysmograph volume and the spirometer volume was also helped by maintaining a supine posture throughout the night.

Negative pressure ventilation at conventional frequencies has been associated with an increase in the number of apneas and hypopneas during sleep. Although ECWO at 1 Hz did not avoid an increase in the AHIndex, the increase in apneas and hypopneas during ECWO occurred predominantly during stage 2 sleep and individual responses were quite varied. Contrary to previous findings with negative pressure ventilation, subjects did not experience respiratory events during stage 4 sleep with ECWO. Furthermore, the one subject who experienced respiratory events during REM sleep had a relatively high AHIndex during REM sleep on the control night. The predisposition to an increase in apneas and hypopneas during ECWO in subjects with an elevated AHIndex (in the absence of ECWO) requires further assessment. These concerns are especially important if ECWO is used among individuals who may be predisposed to upper airway obstruction.

The clinical relevance of apneas and hypopneas likely depends on their association with sleep fragmentation or hypoxemia. Whereas on the control night 2 respiratory events were associated with an arousal, this increased to 18 with ECWO. The mean value for the minimum SaO2 was less than 90% during ECWO with the subject with the highest AHIndex during the control night reaching a nadir of 66% SaO2 during ECWO. A similar case was reported during negative pressure ventilation at a lower frequency. Although the clinical implications of arousals of this frequency and desaturations of this magnitude are unclear, subjects should be monitored overnight when ECWO is first used.

During wakefulness, subjects may actively entrain their breathing with ECWO. Evidence of this entrainment was suggested by the substantial decreases in PCO2 during wakefulness even when the pressure oscillation was very modest. Furthermore, this entrainment persisted despite a PCO2 less than the apneic threshold. The persistence of respiratory activity during assisted ventilation despite hypocapnia has been observed previously.

We speculate that hypocapnia during wakefulness
influenced the increase in the AHindex during the lighter stages of sleep. With the onset of sleep, as the cortical influence was lost, entrainment ceased at a time when the PCO2 was below the apneic threshold. At this time, the absence of upper airway dilator muscle activity rendered the subjects more susceptible to airflow obstruction during ECWO. Although spontaneous ventilation associated with an arousal from sleep terminated the obstruction, the subjects once awake were predisposed to resume the entrainment and repeat this cycle of events. In retrospect, among our subjects, the lowest mean PCO2 during sleep immediately preceding wakefulness (40 [2] mm Hg) was significantly greater than the lowest mean PCO2 during wakefulness (35 [2] mm Hg).

The increase in hypopneas was unlikely to have been attributable to sleeping in a supine position as the index was similar between the control and the cuirass-only nights. Nevertheless, modifications to the cuirass that would allow subjects to sleep on their side could increase subject comfort and thereby their tolerance for prolonged use of the equipment.

An attractive feature of the ECWO system is its ability to impose an oscillatory profile on a baseline negative chamber pressure. This has been observed to increase the operational lung volume, which could benefit some patients. However, the upper airway is also exposed to the subatmospheric pressure generated within the thorax. This has the potential of narrowing the upper airway, thereby increasing its resistance. The increase in airway resistance would necessitate more negative ventilating pressures and predispose the subject to airflow obstruction during sleep. In healthy volunteers, a static pressure of -16 cm H2O superimposed on spontaneous inspiration induced upper airway collapse. Although the mean (-2.5 cm H2O) and nadir (-11.1 cm H2O) of the cuirass pressures experienced by our subjects did not approach this level, the negative intrathoracic pressures in the presence of attenuated upper airway dilator muscle activity might still have contributed to a reduction in airflow caliber.

Negative airway transmural pressure also has a dynamic component that is dependent on the airflow. Upper airway closure occurred in healthy volunteers exposed to an additional brief (200 ms) transthoracic pressure of -12 cm H2O during spontaneous inspiration. Although we used ventilating pressures that ranged from -8.8 to -14.3 cm H2O, the time to the nadir of pressure during ECWO was 500 ms. The mean and peak flows during ECWO were, therefore, lower so that this potential complication was likely avoided. Had it been present, respiratory events would have been observed in all sleep stages. In fact, the respiratory events were predominantly in stages 1 and 2 with few apneas or hypopneas in stages 3, 4, or REM sleep. By further increasing the inspiratory time, the same oscillatory tidal volume could be achieved at even lower transmural pressures. However, the mean and peak expiratory flows would then increase. This would promote dynamic hyperinflation and increase intrinsic positive end-expiratory pressure, especially in the presence of small airways disease.

It is remarkable that clinicians continue to evaluate the effectiveness of negative pressure ventilation in the treatment of patients with acute and chronic respiratory failure despite the availability of noninvasive positive pressure ventilators that would appear to be simpler to use. Although upper airway obstruction is less likely to occur with positive pressure ventilation, negative pressure ventilation clearly remains a viable alternative for those in whom tolerance of positive pressure ventilation is limited by discomfort with the face mask or nasal pillows, or the presence of gastric distention.

During wakefulness, both ECWO at 1 to 2 Hz and negative pressure ventilation at conventional frequencies can support gas exchange for short periods in the absence of spontaneous ventilation. One might speculate that the difficulty in synchronizing spontaneous breathing with negative pressure ventilation at conventional frequencies contributed to the poor subject tolerance of negative pressure ventilation. At 1 Hz, subjects were not required to synchronize their breathing with the oscillator. A comparison between these two modalities of ventilatory support is necessary to establish whether the higher frequency and lower mean chamber pressure associated with ECWO accounted for the relatively good subject tolerance during sleep that we observed.

In conclusion, in healthy volunteers, ECWO maintained gas exchange for prolonged periods while allowing sleep. The quality of sleep was reduced in comparison to a control night and a night during which only the cuirass was used. It is possible that these effects might be attenuated with repeated exposure. ECWO was associated with an increase in the AHindex among healthy subjects and a substantial increase in one subject in whom the control AHindex was elevated. When ECWO is used for prolonged periods, it should be monitored at least initially during sleep.

References

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