SLEEP DISORDERED BREATHING

Effect of increased lung volume on sleep disordered breathing in patients with sleep apnoea

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Background: Previous studies have shown that changes in lung volume influence upper airway size and resistance, particularly in patients with obstructive sleep apnoea (OSA), and that continuous positive airway pressure (CPAP) requirements decrease when the lung volume is increased. We sought to determine the effect of a constant lung volume increase on sleep disordered breathing during non-REM sleep.
Methods: Twelve subjects with OSA were studied during non-REM sleep in a rigid head-out shell equipped with a positive/negative pressure attachment for manipulation of extrathoracic pressure. The increase in lung volume due to CPAP (at a therapeutic level) was determined with four magnetometer coils placed on the chest wall and abdomen. CPAP was then stopped and the subjects were studied for 1 hour in three conditions (in random order): (1) no treatment (baseline); (2) at ''CPAP lung volume'', with the increased lung volume being reproduced by negative extrathoracic pressure alone (lung volume 1, LV1); and (3) 500 ml above the CPAP lung volume(lung volume 2, LV2).
Results: The mean (SE) apnoea/hypopnoea index (AHI) for baseline, LV1, and LV2, respectively, was 62.3 (10.2), 37.2 (5.0), and 31.2 (6.7) events per hour ($p = 0.009$); the 3% oxygen desaturation index was 43.0 (10.1), 16.1 (5.4), and 12.3 (5.3) events per hour ($p = 0.002$); and the mean oxygen saturation was 95.4 (0.3)%, 96.0 (0.2)%, 96.3 (0.3)%, respectively ($p = 0.001$).
Conclusion: An increase in lung volume causes a substantial decrease in sleep disordered breathing in patients with OSA during non-REM sleep.

Obstructive sleep apnoea (OSA) is a common disorder occurring in approximately 4% of middle aged men and 2% of women.[1] It is characterised by repetitive pharyngeal collapse during sleep leading to sleep disruption, arousals, and arterial oxygen desaturation. However, the mechanisms leading to pharyngeal collapse are not completely understood. Previous investigators have suggested that this airway collapse involves the combination of an anatomically compromised pharyngeal airway and sleep induced decrements in pharyngeal dilator muscle activity.[2–5]

During sleep, in normal subjects, upper airway resistance increases and functional residual capacity decreases.[6–8] This sleep induced decrease in lung volume is believed to result in increased upper airway collapsibility and to contribute to inspiratory flow limitation, although the exact mechanisms have not been delineated. Animal data using mongrel dogs have suggested that thoracic inflation increases upper airway pharyngeal size and stiffness through caudal traction on the trachea independently of upper airway muscle activity.[9 10]

Our group recently showed that, in patients with OSA, increased end expiratory lung volume (EELV) decreased the CPAP level required to prevent upper airway flow limitation.[11] Similarly, when EELV is decreased, the required CPAP level to prevent flow limitation increased. This suggests that increments in lung volume have a stabilising effect on the upper airway during sleep in patients with OSA. We therefore sought to determine the influence of a stable increase in EELV on sleep disordered breathing and sleep architecture in OSA patients during non-REM sleep.

METHODS

Subjects

We calculated that we needed 12 subjects to have 80% power to detect four events (apnoeas plus hypopnoeas) per hour difference with a standard deviation of three events per hour and an alpha of 0.05. Patients with moderate to severe OSA determined by overnight diagnostic polysomnography using criteria defined by the American Academy of Sleep Medicine were recruited.[12] The subjects were currently being treated with continuous positive airway pressure (CPAP). The protocol was approved by the Human Subjects Committee at Brigham and Women's Hospital. All subjects provided informed written consent prior to participation in the study. Subjects with medical disorders potentially affecting chest compliance of the upper or lower airway (other than OSA and obesity) were excluded.

Equipment and measurements

Airway pressure was recorded at the level of the epiglottis with a pressure tipped catheter (Millar instruments Inc, Houston, TX, USA). Before insertion of the catheter both nostrils were decongested with 0.05% oxymetazoline hydrochloride and one nostril was anaesthetised with 1–2 ml of 4% lidocaine topical spray. Subjects breathed through a nasal mask (Respironics, Murraysville, PA, USA) with airflow measured with a pneumotachograph (Hans Rudolph, Kansas City, MO, USA) and pressure transducer (Validyne Corp, Northridge, CA, USA). End tidal carbon dioxide was sampled at the mask using a calibrated infrared carbon dioxide analyser (BCI Corp, Waukesha).

Wake/sleep states were determined using standard electroencephalography (EEG), chin electromyography (EMG) and electrooculography (EOG). Lung volume was manipulated with the subject lying supine in a head-out rigid shell (Portalung Inc, Murraysville, PA, USA) adapted with a vacuum/

Abbreviations: AHI, apnoea/hypopnoea index; BMI, body mass index; CPAP, continuous positive airway pressure; EELV, end expiratory lung volume; OSA, obstructive sleep apnoea; REM, rapid eye movement

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blower attachment (ShopVac, Williamsport, PA, USA) to increase or decrease extrathoracic pressure. Changes in EELV were measured with two pairs of magnetometers (EOL Eberhard, Oberwil, Switzerland) placed in the anteroposterior (AP) axis of the chest and abdomen using a standardised formula previously validated by Kono and Mead.[13] Calibration was performed during quiet breathing supine: the changes in chest wall and abdomen AP diameter were averaged over 12 breaths and combined with the pneumotachograph data. Each change in AP diameter was entered into the following equation describing the relationship between tidal volume and chest/abdominal excursion: TV = X (4 RC + AB). Tidal volume (TV) is determined using X, a coefficient determined in the calibration procedure for a given individual. All calibration manoeuvres were performed with subjects instrumented, lying supine in the rigid shell. This posture was maintained throughout the study.

Protocol

The CPAP was initially set at the patient's prescribed level. After achieving stable non-rapid eye movement (non-REM) sleep, the CPAP level was adjusted to the minimum required to prevent flow limitation. This was accomplished by adjusting the CPAP until the flow signal (pneumotachograph) and the pressure signal at the epiglottis consistently demonstrated similar inspiratory curves. Flow limitation was defined as at least a 1 cm H_2O decrement in epiglottic pressure without an associated increase in inspiratory flow.[14]

The increase in lung volume above the baseline EELV associated with this level of CPAP (defined as ''CPAP lung volume'') was determined with four magnetometer coils during non-REM sleep. CPAP was then stopped and the subjects were studied for 1 hour in each of three conditions (in random order): (1) without any treatment (baseline); (2) at ''CPAP lung volume'', the increased lung volume being reproduced by negative extrathoracic pressure alone without CPAP (lung volume 1, LV1); and (3) 500 ml above ''CPAP lung volume'' (lung volume 2, LV2).

Data analysis

The abovementioned signals were recorded on both a 16 channel polygraph (Grass model 78) and a personal computer. Each signal (EEG, EOG, EMG, EKG, end tidal CO_2, abdominal and rib cage magnetometers, flow, tidal volume, mask and epiglottic pressure) was analysed using signal processing software (Spike 2, CED Ltd, Cambridge, UK).

The recording at each lung volume condition (baseline, LV1 and LV2) was analysed in a blinded fashion. Blinding was achieved by splitting the recording into 1 hour segments for each condition. The 1 hour segments were given a unique name and the extrathoracic pressure channel was removed to prevent recognition of the condition. The segments were staged for sleep and breathing disturbances. The specific variables measured were: apnoea/hypopnoea index (AHI), 3% oxygen desaturation index, arousal index, mean oxygen saturation, and sleep stage distribution. Apnoeas and hypopnoeas were scored according to the Chicago criteria[12] with the respiratory effort being determined with the abdominal and thoracic magnetometers instead of bands. An arousal was defined as an abrupt shift in EEG frequency which includes theta, alpha, and/or a frequency higher than 16 Hz (but not spindles) of 3 seconds or greater duration. Arousals longer than 5 minutes and REM periods were not included in the recording time used to determine the indexes. A spectral analysis was also performed on the C3 A2 EEG channel which was digitised at 128 Hz frequency. The time in apnoea was estimated by the time spent with an inspiratory flow $\leqslant$0.1 l/s. The time spent with Sao_2 <90% was also determined for each condition using a custom script (Spike 2, CED Ltd, Cambridge, UK).

All data are reported as means and standard error of the mean (SE). A p value of <0.05 was considered significant. A repeated measure ANOVA (or an ANOVA on the ranks when data were not normally distributed) was performed to compare the AHI, arousal index, oxygen desaturation index, mean saturation, power spectrum, time spent in the different sleep stages, and the time spent in apnoea or <90% Sao_2. The p for each ANOVA represents the p value for heterogeneity. A post hoc Tukey test was used to determine if there was a significant difference between baseline, LV1, and LV2 for the main outcomes (AHI and 3% desaturation index).

RESULTS

Sixteen patients were recruited to the study. Three patients could not complete the protocol because they could not sleep inside the iron lung and one patient because she could not sleep without CPAP. Twelve patients (seven men) of mean (SD) age 47.5 (2.8) years (range 32–63) therefore completed the study (table 1). The mean AHI from the diagnostic polysomnogram was 41.5 (7.9) events/hour of sleep. The mean CPAP level required to prevent upper airway flow limitation was 12.9 (4.0) cm H_2O. At this CPAP level the mean increase in EELV was 770 (165) ml above the baseline EELV during sleep (without CPAP). The mean negative extrathoracic pressure required to induce the same EELV increase (LV1) was −8.9 (2.2) cm H_2O, and the mean negative extrathoracic pressure required to induce LV2 (an increase in EELV of 529 (30) ml above LV1 or ~1300 ml above baseline) was −14.0 (3.2) cm H_2O.

The results of the main outcomes are reported for each condition in table 2. With increased EELV there was a significant decrease in AHI (fig 1), arousal index, and 3% oxygen desaturation index (fig 2), as well as a significant increase in the mean Sao_2. The AHI and the type of respiratory events that occurred in each condition are shown in fig 1. For AHI and 3% oxygen desaturation index we also performed a post hoc Tukey test: there was a significant difference for both between baseline and LV1 ($p<0.05$) and between baseline and LV2 ($p<0.05$), but the difference between LV1 and LV2 was not significant.

For baseline, LV1 and LV2 conditions, respectively, the percentage of time spent with an inspiratory flow of $\leqslant$0.1 l/s was 22.9 (3.9)%, 15.3 (3.8)% and 13.2 (3.8)% ($p = 0.018$). The percentage of time spent with an Sao_2 <90% was, respectively, 4.7 (2.0)%, 0.8 (0.4)%, and 0.3 (0.3)% ($p = 0.012$).

When EELV was increased there was a significant decrease in the percentage of stage 1 non-REM sleep, as well as an increase in the percentage of stage 2 non-REM sleep (table 2). There was also a trend towards a reduction in the percentage of time awake (p value for heterogeneity = 0.071). Only four subjects had slow wave sleep (stage III/IV) during the recordings, which only occurred when the lung volume was increased and never at baseline. The power density (μv^2/Hz) of beta waves decreased ($p = 0.001$) and the power density of theta waves increased ($p = 0.004$) when EELV was increased. Delta power tended to increase and alpha power to decrease but these differences did not reach statistical significance.

Table 1 Demographic data for the 12 study subjects

	Mean	SE	Range
Age (years)	47.5	2.8	32–63
BMI (kg/m^2)	34.9	2.2	25.6–46.8
AHI (events/h)	41.5	7.9	15–99
Male/female	7/5		

BMI, body mass index; AHI, apnoea-hypopnoea index.

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Table 2 Main results: mean (95% confidence interval, CI)

	Baseline supine	LV1	LV2	p value for heterogeneity
AHI (events/h)	62.3 (42.3 to 82.3)	37.2 (27.4 to 47.0)	31.2 (18.1 to 44.3)	0.009
Arousal (events/h)	63.8 (46.2 to 81.4)	43.5 (31.2 to 55.8)	38.2(27.2 to 49.2)	0.039
3% desaturation (events/h)	43.0 (23.2 to 62.8)	16.1 (5.5 to 26.7)	12.3 (1.9 to 22.7)	0.002
Mean O_2 saturation (%)	95.4 (94.8 to 96.0)	96.0 (95.6 to 96.4)	96.3 (95.7 to 96.9)	0.001
% awake	17.4 (10.1 to 24.7)	10.1 (6.0 to 14.2)	8.6 (4.1 to 13.1)	0.071
% stage 1	27.8 (20.9-34.7)	13.5 (9.6-17.4)	14.1 (5.7 to 22.5)	0.001
% stage 2	54.8 (41.1 to 68.5)	66.5 (54.0 to 79.0)	77.2 (67.0 to 87.4)	0.009
Beta power	11.5 (8.4 to 14.6)	8.6 (6.6 to 10.6)	7.9 (5.7 to 10.1)	0.001
Alpha power	8.5 (4.4 to 12.6)	7.1 (4.9 to 9.3)	7.2 (4.8 to 9.6)	NS
Theta power	10.9 (7.8 to 14.0)	14.9 (10.6 to 19.2)	14.7 (12.2 to 17.2)	0.004
Delta power	46.4 (33.7 to 59.1)	55.7 (35.5 to 75.9)	48.8 (35.1 to 62.5)	NS

AHI, apnoea hypopea index.
Beta, alpha, theta and delta power are defined in the text.

DISCUSSION

These results demonstrate the influence of changes in lung volume on apnoea severity and sleep architecture in OSA patients during non-REM sleep. When EELV was increased the AHI, arousal index, and oxygen desaturation index all decreased significantly. Moreover, ''light sleep'', as measured by the percentage of stage 1 and beta power, decreased when EELV was increased.

Previous animal and human studies suggest that the mechanism underlying these results is probably an increase in upper airway stiffness and size with increased lung volume due to caudal traction on the pharyngeal airway. Studies in normal adults (without OSA) have shown that, during wakefulness and sleep, changes in lung volume result in important variations in upper airway mechanics.[15–18] Moreover, pharyngeal airway size as measured by CT scanning[19 20] or acoustic reflection[21] increases with lung inflation and decreases at lower lung volume. Lastly, it has been shown that patients with OSA have greater lung volume related dependence of upper airway size than non-apnoeic individuals both in men[20] and women.[21 22]

Because the subjects we studied were overweight or obese (like most patients with OSA), we suspect that their diaphragm was pushed upwards (cranially) when lying on their back. The negative extrathoracic pressure applied in the lung almost certainly pulled the diaphragm and trachea to a more caudal position, thereby increasing the traction on the upper airway and making it less collapsible. Other authors have found that the AHI can be considerably reduced when obese OSA patients sleep in a semi-recumbent position,[23] which probably corroborates this hypothesis. In the present study there was also a trend toward a correlation between the extent of the reduction in AHI between baseline and LV2 and the BMI (correlation coefficient = 0.53, p = 0.077). This suggests that increases in lung volume may have a greater effect in more obese patients.

Another possibility is that an increase in EELV led to an overall increase in the Sa_{O_2} due to improved ventilation/perfusion matching that could lead to decreased Sa_{O_2} fluctuations. This reduction in chemical stimuli fluctuations may have stabilised respiratory control, decreasing the risk of cyclic breathing.

A previous study from our group showed that lung volume has an important effect on the CPAP level required to prevent upper airway flow limitation, which suggests that increments in lung volume may be one of the mechanisms by which CPAP eliminates disordered breathing during sleep. In the present study we have shown that an increase in lung volume alone (without CPAP) is able to decrease the apnoea and hypopnoea frequency significantly, which supports this hypothesis. The rest of the effect of CPAP is probably due to a splinting effect of positive airway pressure on the upper airway, maintaining a positive transmural pressure throughout the respiratory cycle.

Séries *et al* conducted a study in which the EELV was increased by 500 ml during sleep in OSA subjects.[24 25] They observed a decrease in the severity of oxygen desaturation

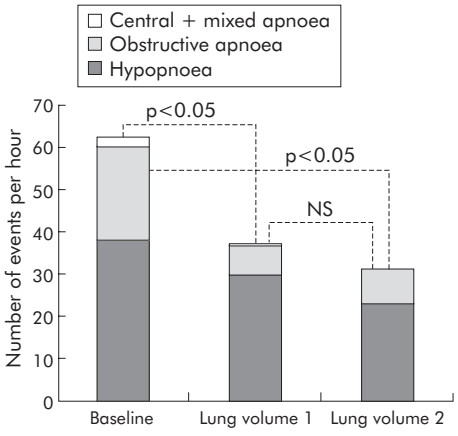


Figure 1 Type and number of respiratory events per hour at each lung volume.

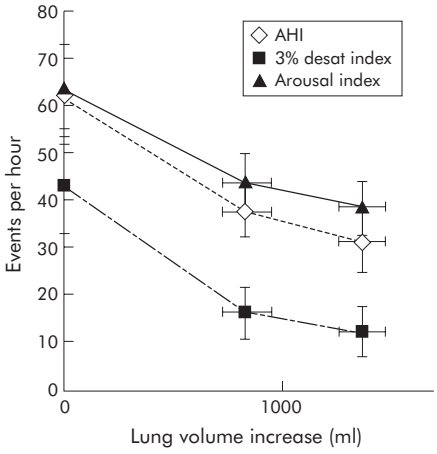


Figure 2 Mean (SE) number of respiratory events per hour (AHI), arousals per hour, and number of 3% oxygen drops per hour at each lung volume.

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which is confirmed by our results. However, in contrast to our results, they found no effect on sleep structure and a non-significant decrease in AHI when EELV was increased, despite studying their subjects for a longer period of time in each condition. There are several possible explanations for this discrepancy. Firstly, we induced a larger increase in lung volume (770 and 1300 ml compared with 500 ml) which probably achieved a greater traction on the trachea and on the upper airway. Secondly, we did not use the same technique to measure the changes in lung volume. Séries *et al* determined the negative pressure required to induce a 500 ml increase in lung volume while awake and then applied this negative pressure during the night. It is possible that there were behavioural influences during the awake manoeuvre. In our study we constantly monitored the lung volume during sleep to be sure that the increase in lung volume was maintained during the night; this may have allowed us to achieve a more precise and constant increase in lung volume. Thirdly, the population we studied had 90.7% obstructive apnoeas at baseline (7.9% mixed and 1.4% central apnoea) whereas the subjects studied by Séries *et al* had only 56.3% obstructive apnoeas (36.8% mixed and 6.9% central apnoea), even though the definition of central and mixed apnoea may have evolved between 1989 and 2005. It is therefore possible that upper airway mechanics was the main cause of sleep apnoea in our population, and that instability of respiratory control made a greater contribution to apnoea in the population studied by Séries *et al*. Indeed, one of our subjects who had 14% central sleep apnoeas at baseline had an increase in time spent in apnoea when EELV was increased, despite the fact that the AHI decreased and Sao_2 improved. It is possible that, for a subset of sleep apnoea patients with a high ventilatory instability, an increase in lung volume could trigger a ''Hering Breuer'' reflex inhibiting inspiration and thus extending the time in apnoea. Finally, our population was probably more obese (mean BMI 34.9) than that studied by Séries *et al* (124% of ideal body weight). This could have induced a more important effect of increased lung volume in our study because the diaphragm of obese patients is pushed more rostrally by abdominal fat than in less obese patients. However, further work will be needed to define which subgroups of patients with sleep apnoea respond best to changes in lung volume.

There was a significant decrease in AHI between baseline and both LV1 and LV2, but the decrease in AHI between LV1 and LV2 did not reach significance and some patients even had a higher AHI at LV2 than at LV1. This may be due either to a ceiling effect on AHI of increasing EELV or to arousals secondary to the discomfort of higher negative pressure in the iron lung which could have induced some respiratory instability in these patients.

There are several limitations to this study. Firstly, because the subjects were studied only during supine non-REM sleep, these results may not apply to other body positions or to REM sleep. The supine posture is usually the position in which the greatest respiratory disturbances are recorded, possibly in part due to abdominal fat applying pressure on the diaphragm leading to decreased lung volume and decreasing the traction on the upper airway by the trachea. If this assumption is correct, it is possible that, when subjects lie on their side or are prone, the effect of negative extrathoracic pressure would be smaller.

Secondly, while Séries *et al* studied their subjects for the whole night in each of the two conditions (baseline, +500 ml EELV), we studied our subjects for 1 hour in each of the three different conditions (baseline, +770 ml, +1300 ml). However, with our sample size (12 subjects) we had 80% power to detect a difference of 4 (3) events per hour between the three conditions using an ANOVA. Moreover, this design gave us homogeneous data collected in the exact same conditions during the same night. In addition, the randomisation of the order of the conditions prevented a ''time of the night'' bias. Thus, although this is a potential problem, we doubt it influenced our results.

Finally, a direct effect of the iron lung negative pressure on the neck and upper airway cannot be completely excluded. A decrease in the pressure around the neck when lung volume was increased could have ''unloaded'' the upper airway making it less collapsible. However, when the pressure in the iron lung was decreased, the webbing forming the seal around the neck was shifted inside the chamber which would only further decrease the neck area potentially exposed to the negative pressure in the lung. We therefore doubt that this contributed to the reduction in sleep disordered breathing

In summary, these results show that an increase in lung volume causes a decrease in sleep disordered breathing and improves sleep architecture in patients with sleep apnoea during non-REM sleep. Although an iron lung may well be more cumbersome than nasal CPAP therapy, our data suggest that increments in lung volume may be one of the mechanisms by which sleep disordered breathing is improved by CPAP. Thus, increased lung volume could be a direct therapeutic target for patients with sleep apnoea using, for example, an expiratory resistance as suggested by Mahadevia *et al*.[26]

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LUNG ALERT

Is there a gold standard test for diagnosing exercise induced asthma in elite athletes?

▲ Dickinson JW, Whyte GP, McConnell AK, Harries MG. Screening elite winter athletes for exercise induced asthma: a comparison of three challenge methods. *Br J Sports Med* 2006;**40**:179–82

Exercise induced asthma (EIA) is a common yet often unrecognised condition that occurs in both known asthmatics and otherwise healthy individuals. Misdiagnosis—both over- and under-diagnosis—frequently occurs. In order to diagnose EIA accurately, a bronchoprovocation challenge test must be performed. The provocation tests used to identify subjects with EIA include exercise (laboratory or field), eucapnic voluntary hyperventilation (EVH), and pharmacological agents (hypertonic saline or mannitol powder).

Bronchial provocation using pharmacological agents are less reliable and, while field exercise is more sensitive than laboratory exercise in the diagnosis of EIA, the major limitation of exercise tests is the control of variables such as environmental factors and challenge intensity. EVH is a laboratory based indirect provocation challenge that enables minute ventilation and environmental conditions to be controlled and thus greatly enhances the reliability and validity of the challenge test.

In this study the authors examined whether exercise tests (sport specific and laboratory) are as efficacious as EVH in diagnosing EIA in asymptomatic elite winter athletes. Fourteen athletes were studied, including two known asthmatics. All study participants completed exercise tests as well as EVH. The cut off point for diagnosis of EIA, as recommended by the International Olympic Committee, is a fall of 10% in forced expiratory volume in 1 second (FEV_1) from baseline. In all, 10 athletes were found to have a positive test. In comparing the three challenge tests, the authors found that EVH was best for diagnosing EIA (10 athletes), followed by sport specific exercise testing (3 athletes) and laboratory exercise testing (0 athletes).

In spite of the small number in the study group, this work complements previous studies in this field and suggests that it is time to adopt EVH as the gold standard test for the diagnosis of EIA in elite athletes.

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