HHS Public Access

Author manuscript

Chest. Author manuscript; available in PMC 2015 March 24.

Published in final edited form as:

Chest. 2001 July ; 120(1): 156–161.

Upper-Airway Collapsibility*:

Measurements and Sleep Effects

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Abstract

Study objectives—Obstructive sleep apnea (OSA) is characterized by repetitive pharyngeal collapse during sleep. Several techniques have been proposed to assess the collapsibility of the upper airway in awake humans, but sleep-wake comparisons have rarely been attempted and there are few studies comparing OSA patients to control subjects. We sought to compare two collapsibility measurement techniques between normal and apneic subjects, and between wakefulness and sleep.

Design—We conducted three studies. First, we examined whether collapsibility assessed by negative pressure pulses (NPPs) during wakefulness reflected values during sleep in 21 normal subjects. Second, we determined in these normal subjects whether collapsibility during sleep assessed by NPPs was predictive of collapsibility measured by inspiratory resistive loading (IRL). Finally, we compared upper-airway collapsibility between apnea patients (n = 22) and normal volunteers (n = 38) during wakefulness by NPPs.

Setting—Clinical and research laboratories at the Brigham and Women's Hospital.

Participants—Two populations of normal subjects (n = 21 and n = 38) and OSA patients (n = 22).

Measurements and results—Collapsibility during wakefulness, as measured by NPPs, correlated significantly with collapsibility during sleep ($r = 0.62$; $p = 0.003$). There was also a significant correlation between the two measures of collapsibility (IRL and NPP) during sleep ($r = 0.53$; $p = 0.04$). Both measures revealed a significant increase in pharyngeal collapsibility during sleep as compared to wakefulness. Finally, apnea patients had significantly greater pharyngeal collapsibility than control subjects during wakefulness ($p = 0.017$).

Conclusions—These data suggest that upper-airway collapsibility measured during wakefulness does provide useful physiologic information about pharyngeal mechanics during sleep and demonstrates clear differences between individuals with and without sleep apnea.

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Keywords

breathing; collapsibility; critical pressure; dilator; flow limitation; genioglossus; inspiratory resistive load; lung; negative pressure; obstructive sleep apnea; pharynx; resistance; upper airway

Obstructive sleep apnea (OSA) is a disease characterized by repetitive pharyngeal collapse during sleep. One variable used in describing the mechanics of the upper airway is to measure its propensity for collapse (collapsibility). Upper-airway collapsibility has been assessed by at least three techniques. These include the application of negative pressure pulses (NPPs; measuring the pressure drop across the pharynx),[1,2] the resistance change observed in response to externally applied inspira-tory resistive loading (IRL),[3–6] and the determination of the critical pressure (Pcrit) needed to induce a cessation of airflow.[7–10] Although several investigators have measured pharyngeal collapsibility during wakefulness, there are minimal data suggesting that collapsibility measured during wakefulness is predictive of collapsibility during sleep, or that the value differs between normal subjects and apnea patients. This substantially limits the potential usefulness of these techniques. If collapsibility during wakefulness could predict collapsibility during sleep, it might be clinically useful and would certainly suggest that sleep-induced changes in the airway characteristics do not totally explain apnea. Of the techniques described above, only the NPP can be reliably performed during wakefulness, as it can be accomplished rapidly, thus freeing it from behavioral influences. The measurement of Pcrit and collapsibility by the IRL technique could be substantially limited by these behavioral factors.

We hypothesized that inherent pharyngeal properties may influence collapsibility during both wakefulness and sleep. In order to test this hypothesis, we posed three questions: (1) Although the pharyngeal airway is more collapsible asleep than awake, does collapsibility measured awake predict collapsibility measured during sleep? (2) Do apnea patients have a more collapsible airway than normal control subjects when assessed during wakefulness, thereby indicating an airway abnormality that is present and quantifiable awake? (3) Do different measures of collapsibility yield similar results (*ie*, propensity for collapse)?

Materials and Methods

Subjects/Patients

The study had two parts.

Part 1: Twenty-one normal subjects (mean ± SD age, 27.7 ± 1.1 years; body mass index [BMI], 23.3 ± 0.5 kg/m^2) underwent collapsibility measurements (NPP) during both wakefulness and sleep. In 16 of these subjects, both IRL and NPP measurements were obtained (awake and asleep). For this protocol, we studied subjects in the lateral decubitus posture, as IRL during sleep in the supine posture may lead to complete pharyngeal collapse, making measurement of resistance problematic (Table 1).

Part 2: The collapsibility of 22 patients with OSA (mean age, 47 ± 2 years; BMI, 38 ± 2 kg/m^2; respiratory disturbance index [RDI], 63 ± 7/h [minimum RDI, 20/h]) was compared with that of 38 control subjects (mean age, 50 ± 3 years; BMI, 25 ± 1 kg/m^2)

during wakefulness. All the control subjects were historically free from any medical or sleep complaints, and the apnea patients were otherwise healthy. We chose to study these individuals in the supine posture in order to maximize the differences in upper-airway collapsibility between normal and apneic subjects during wakefulness. Although collapsibility during wakefulness is relatively low, it is maximal in the supine position. Informed consent was obtained from each participant, with the protocol having the prior approval of the Human Subjects Committee of the Brigham and Women's Hospital.

Instrumentation and Techniques

Subjects wore a nasal mask (Healthdyne Technologies; Marietta, GA) connected to a two-way valve partitioning inspiration and expiration. Inspiratory flow was determined with a pneumo-tachometer (Fleish; Lausanne, Switzerland) and differential pressure transducer (Validyne; Northridge, CA), calibrated with a rotameter. The subject's breathing was exclusively nasal as ensured by mouth tape and video monitoring.

Polysomnography

In part 1 of the study, wakefulness/sleep was documented with a two-channel EEG, electro-oculography, and submental electromyography using standard techniques.[11,12] Subjects maintained the lateral decubitus posture throughout the study as verified by video camera. Although part 2 of the study was conducted only during wakefulness, all participants had undergone full polysomnography to confirm the presence or absence of apnea.

Pressure Measurements and Negative Pressure Applications

Airway pressures were monitored in the nasal mask (with a Validyne transducer) and at the level of the choanae (choanal pressure [Pcho]) and the epiglottis (epiglottic pressure [Pepi]), using pressure-tipped catheters (MPC-500; Millar; Houston, TX).[1,2] Negative airway pressure pulses were generated using a partially evacuated 50-L canister and a solenoid valve connected to the nasal mask, as described previously.[1,2] Each negative pressure application occurred during early inspiration (100 ms after the start of inspiratory flow) and had a rapid onset and offset for a total duration of < 0.5 s, and generated – 8 to – 13 cm H_2O pressure at the choanae, with a goal of – 10 cm H_2O. All signals were recorded and signal averaged for analysis. Collapsibility was quantified as the pressure difference between the choanae and the epiglottis during the pressure pulse. Since the magnitude of negative pressure applied can affect the collapsibility, we indexed this measure of collapsibility for the level of negative pressure applied using the following formula:

$$\text{Collapsibility} = ([Pcho - Pepi]/Pcho) \times 100$$

using the nadir negative pressures during the pulse.

IRL

Inspiratory resistance was added using a specially designed variable resistance device, placed distal to the inspiratory valve, as previously described.[13,14] Loads of 15 cm H_2O/L/s and 25 cm H_2O/L/s were applied for three breaths each and then removed. Pharyngeal

resistance (Rph) [between the choanae and the epiglottis] was determined prior to and during each loaded breath. Rph index (a measure of tendency for pharyngeal collapse) was defined as the ratio of peak Rph (at peak negative pressure) during loading to the baseline resistance prior to loading (Rph loaded/Rph baseline). Because flow limitation may develop during sleep while loading, the peak flow does not always coincide with the peak negative Pepi. Therefore, the point at which the peak negative (nadir) Pepi occurs was chosen, as this represents the peak resistance that develops in response to the applied load. However, we recognize that the measurement of resistance during flow limitation is somewhat complex, and thus we also determined this Rph index at 0.2 L/s inspiratory flow that occurred prior to the development of flow limitation. Furthermore, resistance measurements become impossible when the airway is totally occluded, as resistance approaches infinity.

Study Protocol

In part 1 of the study, all subjects reported to the laboratory in the evening and were fully instrumented as described above. Collapsibility was then measured awake using NPP and IRL. Subjects were then allowed to fall asleep, and collapsibility was assessed during stable nonrapid eye movement sleep (stages 2, 3, or 4). Each IRL was applied at least three times (for three breaths each), and 40 NPPs were applied during both wakefulness and sleep. Prior to each load application, a stable baseline (> 30 s) was documented. In part 2 of the study, 40 NPPs were applied during clear wakefulness (eyes open on video monitor) in all participants (apnea patients and normal control subjects).

All statistical analyses were performed with commercially available software (SigmaStat; SPSS; Chicago, IL, and Excel 97; Microsoft; Redmond, WA). Comparisons between groups, positions, and wake vs sleep were accomplished using two-tailed Student's *t* test. Correlation analyses were performed using standard least-squares linear regression techniques. For all analyses, α was set at 0.05 and results given as mean ± SEM.

Results

Pressures and collapsibility during NPPs are provided in Table 2, with representative raw data shown in Figure 1. Apnea patients had significantly greater pharyngeal collapsibility than control subjects during wakefulness (54.2 ± 4.4% vs 38.8 ± 4.0%; $p = 0.017$; Fig 2). Within the apnea group, however, there was no correlation between collapsibility and either RDI ($r = 0.34$; $p > 0.1$) or BMI ($r = -0.21$; $p > 0.1$). Collapsibility in normal control subjects tended to be higher in the group that was studied in the supine posture than the group studied in the lateral position (between group comparison), although the difference did not reach statistical significance (38.8% vs 27.5%; $p = 0.07$).

In the control group studied in the lateral posture, collapsibility during sleep was significantly greater than during wakefulness (54.4% vs 27.5%; $p < 0.01$; Table 2). In addition, the two NPP collapsibility measurements (awake and asleep) correlated significantly ($r = 0.62$; $p = 0.003$).

Rph index calculated at peak flow under baseline and loaded conditions during both wakefulness and sleep is presented in Table 3. Rph increased significantly in the two loading

conditions during sleep but not during wakefulness. Since loading was associated with flow limitation in eight of the subjects (50%) during sleep (but in none during wakefulness), the index was also calculated at a flow of 0.2 L/s, which represents an early measurement during the breath, on the linear part of the pressure flow curve, prior to the onset of flow limitation. The results of these measurements are presented in Table 4. As can be seen, Rph did not change significantly with loading during wakefulness, but increased significantly during sleep with an IRL of 25 cm H_2O/L/s. Finally, there was a significant correlation between collapsibility during sleep as assessed by NPP and Rph index (by IRL) at both 15 cm H_2O and 25 cm H_2O/L/s ($r = 0.53$ and $r = 0.51$, respectively; $p < 0.05$ in both; Fig 3).

Discussion

The results of this study suggest that the measurement of pharyngeal collapsibility during wakefulness using NPPs is somewhat predictive of collapsibility during sleep in normal subjects. This measure of collapsibility is also significantly higher in apnea patients when compared with age-matched control subjects. Finally, upper-airway collapsibility during sleep as assessed by NPPs applied through the nose correlated with pharyngeal collapsibility as measured by IRL when negative pressure is generated by the diaphragm.

Several previous studies have attempted to find measurable variables during wakefulness that predict apnea status during sleep. Imaging studies[15–18] have shown OSA patients to have a smaller pharyngeal airway and increased upper-airway soft tissue than normal subjects when assessed during wakefulness, although there is considerable overlap between groups. The findings of the present study reveal a similar phenomenon for our physiologic measures of collapsibility. Generally, subjects with higher upper-airway collapsibility during wakefulness had higher collapsibility during sleep as well, with apneic patients having greater collapsibility than normal subjects, although clearly overlap exists. These data support an earlier report by Suratt et al,[19,20] who observed 10 OSA patients to have pharyngeal collapse in response to continuous negative airway pressure between – 17 cm H_2O and – 40 cm H_2O, while collapse could not easily be induced in control subjects. As there was overlap between our normal control subjects and apneic patients awake, we do not suggest that this measure can be used clinically to diagnose OSA during wakefulness. However, the results imply that certain properties of the pharyngeal airway may predispose to or protect the pharynx from collapse during both sleep and wakefulness, although obviously collapsibility measured during wakefulness cannot fully explain the pathophysiology of OSA. Thus, additional physiologic events must clearly occur during sleep allowing for increased upper-airway collapsibility in both apneic patients and control subjects. Possibilities include loss of protective reflexes, decreased dilator muscle activation, and change in lung volume, among others.[2,12,21–23]

The results of this study are potentially helpful for several additional reasons. First, investigators in the field of sleep-disordered breathing have used a number of different methods to assess collapsibility, with few comparisons or validations of any of these techniques. By showing a relationship between the increase in resistance induced by IRL and the collapsibility as measured by the NPP technique, we have at least some assurance that the two techniques are measuring similar phenomena. Second, due to the substantial

instrumentation required to measure pharyngeal physiology, upper-airway research is commonly performed during wakefulness. For this reason, measurements during wakefulness are often the only available information regarding the propensity for upper-airway collapse during sleep. Our data suggest that the NPP technique awake does provide some measure of collapsibility during sleep. However, likely due to behavioral influences, the increase in Rph induced by IRL awake was not predictive of any measure of collapsibility during sleep. As a result, the NPP method appears to be a technique of reasonable validity for assessing upper-airway collapsibility during wakefulness.

As stated previously, the NPP technique quantifies the pressure drop between the choanae and epiglottis during NPPs. In theory, a perfectly rigid pharynx would transmit all of the applied pressure from the choanae to the epiglottis, while an extremely collapsible pharynx would transmit essentially none of it. The pressure difference between the choanae and the epiglottis is thus a function of the collapsibility of the pharynx. However, the measured collapsibility cannot exceed the applied choanal pressure and thus can be influenced by the strength of the stimulus applied. To control for this problem, we have elected *a priori* to analyze collapsibility both as the absolute pressure difference between the choanae and the epiglottis and by calculating this pressure difference as a percentage of the applied stimulus. Therefore, a collapsibility measured as 5 cm H_2O would reflect “100% collapsibility” if the applied stimulus were 5 cm H_2O, but only “50% collapsibility” if the applied stimulus were 10 cm H_2O. Total collapse (occluded airway) by this method would therefore be 100%. We believe this percent collapsibility may be a more accurate measure than the absolute pressure gradient, as it is less influenced by the arbitrarily determined stimulus.

One previous study used this NPP technique during wakefulness and sleep: Wheatley et al[2] measured collapsibility in a cohort of normal subjects during both wakefulness and sleep and found that collapsibility was considerably greater during sleep than during wakefulness. In addition, collapsibility was expressed as an absolute number and therefore influenced by the applied pressure stimulus as explained above. Our greater number of subjects and the use of a “collapsibility index” allowed for a more robust comparison of this relationship and demonstrated a significant correlation between wakefulness and sleeping values although the strength was only moderate. To our knowledge, no other studies have been published attempting to estimate collapsibility during sleep using measures obtained during wakefulness.

In the two parts of the study, we measured collapsibility in two different control groups, in different postures. As we applied IRL in the first protocol during both sleep and wakefulness, we wished to minimize complete pharyngeal collapse asleep, and thus used the lateral decubitus posture. As the second protocol was conducted only during wakefulness, we chose the supine posture to maximize pharyngeal collapsibility. We did find a trend toward greater collapsibility in the normal subjects studied supine when compared to another group of normal subjects studied in the lateral decubitus posture (38.8% vs 27.5%; $p = 0.07$). Although these were two different study populations, these results are consistent with previously published data[24–26] on positional dependence of upper-airway collapse.

This study had several limitations. First, the collapsibility measured during NPPs is valid only with the assumption that no airflow occurs during the NPPs (delivered during early inspiration). Although this assumption is not completely accurate, the small brief flow did not likely importantly influence the results. Second, although debated, many believe that the Pcrit represents the “gold standard” measure of upper-airway collapsibility. Although this technique has only been performed during sleep, it would have been of interest to have included this third measure of collapsibility during sleep. However, the difficulty of making multiple measurements in heavily instrumented normal subjects during sleep precluded our measuring Pcrit. In addition, as with the measures of collapsibility used in the present study, in previous studies[27] the measured Pcrit value has correlated poorly with the apnea hypopnea index. Finally, our apnea patients were not weight matched to the control subjects with whom they were compared. Thus, differences in observed collapsibility could have been a product of weight rather than apnea status. However, collapsibility did not correlate with BMI, and it is virtually impossible to find obese control subjects without at least some apnea. As a result, we believe that the conclusions are valid.

Conclusion

We believe this study supports the concept that the measurement of upper-airway collapsibility during wakefulness is predictive of what happens during sleep. However, these correlations are somewhat loose, emphasizing the importance of sleep-induced changes in pharyngeal physiology in predicting the propensity for airway collapse.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding for this project came from National Institutes of Health grants HL 48531 and HL 60292 and National Center for Research Resources grant RR02635.

Dr. Malhotra is funded by the Medical Research Council of Canada (Scientific Development Grant) and the American Heart Association (Beginner's Grant in Aid, Scientific Development Grant). Dr. Pillar received a Fulbright grant to conduct this research. Some subjects were recruited through the Harvard Cooperative on Aging.

Abbreviations

BMI	body mass index
IRL	inspiratory resistive loading
NPP	negative pressure pulse
OSA	obstructive sleep apnea
Pcho	choanal pressure
Pcrit	critical pressure
Pepi	epiglottic pressure

RDI	respiratory disturbance index
Rph	pharyngeal resistance

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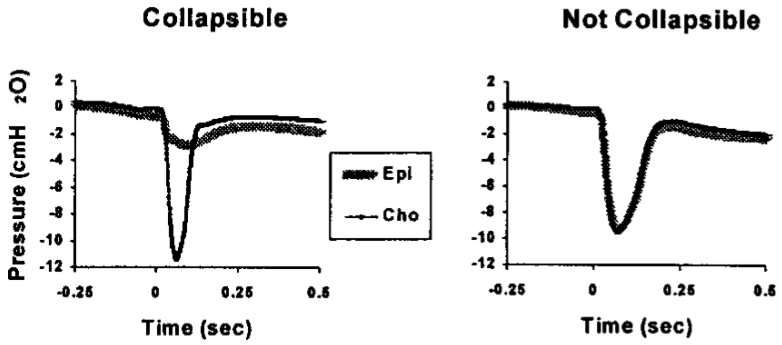

Figure 1.

Examples of individual subject's NPP determination of collapsibility. The “collapsible” subject has minimal pressure transmission from the choanae (Cho) to the epiglottis (Epi), whereas in the “noncollapsible” subject, essentially all of the pressure is transmitted from the choanae to the epiglottis.

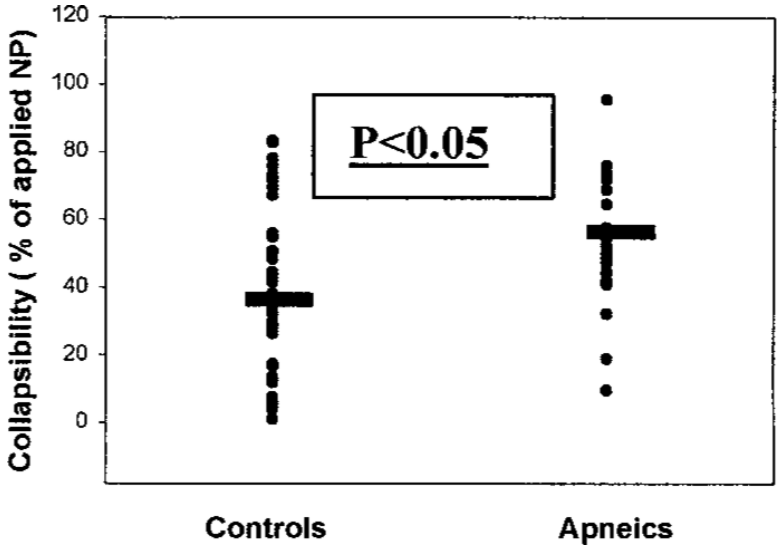

Figure 2.

Comparison of pharyngeal collapsibility during wakefulness between apneic patients and control subjects, all in the supine posture. Pharyngeal collapsibility is calculated as the pressure difference between the choanal and the epiglottic levels (during administration of brief NPPs), as a percentage of applied pressure (see text). The apnea patients had significantly greater collapsibility than control subjects ($p < 0.05$ between groups). The horizontal line represents the mean value for each group.

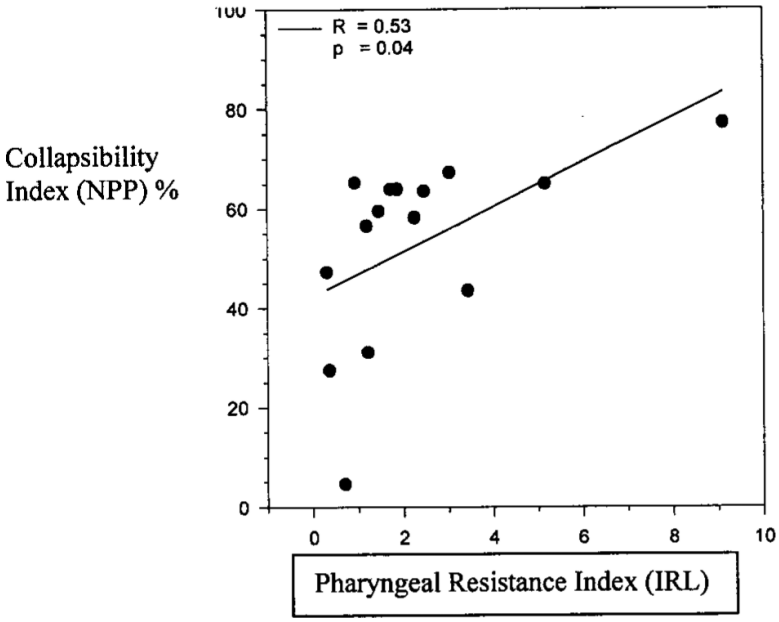

Figure 3.

Correlation between two different measures of upper-airway collapsibility during sleep in control subjects. The NPP technique uses the pressure drop between the choanae and epiglottis, while the Rph index uses the induced Rph observed during IRL. See text for complete definitions of collapsibility indexes.

Table 1

Demographics*

	Control Population		
	1	2	Apneic Patients
Patients, No.	21	38	22
Male/female gender, No.	11/10	18/20	17/5
Age, yr	27.7 ± 1.1	50.0 ± 2.7	47 ± 2.4
BMI, kg/m^2	23.1 ± 0.62	25.3 ± 0.6	38 ± 2.1
AHI events/h	NA	3.8 ± 0.5	63 ± 7

NA = not applicable; AHI = apnea-hypopnea index.

* Data are presented as mean ± SEM unless otherwise indicated.

Table 2

Pressure Measurements and Collapsibility by Negative Pressure Applications for the Two Protocols*

Variables	Pcho, cm H_2O	Pepi, cm H_2O	Collapse, cm H_2O	Collapsibility Index, %
Apneic patients (supine)	–10.4 ± 0.4	–4.6 ± 0.4	5.6 ± 0.5†	54.2 ± 4.4†
Control subjects (supine)	–9.6 ± 0.2	–5.8 ± 0.4	3.9 ± 0.4	38.8 ± 4.0
Normal subjects on side (awake)	–8.8 ± 0.2	–6.3 ± 0.3	2.5 ± 0.4	27.5 ± 3.9
Normal subjects on side (asleep)	–8.9 ± 0.2	–3.9 ± 0.4‡	4.8 ± 0.5‡	54.4 ± 4.7‡

*Data are presented as mean ± SEM. See “Materials and Methods” section for definition of collapsibility index.

†$p < 0.02$ (apneic patients supine vs control subjects supine).

‡$p < 0.01$ (Normal-side group, asleep vs awake).

Table 3

Pressure Measurements and Collapsibility as Assessed by Response to Externally Applied Load in the Normal Group Measured at Peak Pepi (Peak Resistance), Lateral Posture*

Variables	Pcho, cm H_2O	Pepi, cm H_2O	Flow, L/s	Rph, cm H_2O/L/s	Rph Index
Awake					
Baseline	–2.07 ± 0.2	–2.39 ± 0.2	0.54 ± 0.0	0.73 ± 0.3	
IRL = 15 cm H_2O	–7.36 ± 0.8	–7.59 ± 0.8	0.44 ± 0.1	0.65 ± 0.2	0.89 ± 0.3
IRL = 25 cm H_2O	–10.35 ± 1.0	–10.68 ± 1.0	0.40 ± 0.1	0.86 ± 0.3	1.17 ± 0.4
Asleep					
Baseline	–0.8 ± 0.6	–3.2 ± 0.5	0.44 ± 0.02	6.1 ± 1.9	
IRL = 15 cm H_2O	–3.7 ± 1.0	–7.9 ± 1.2	0.29 ± 0.03†	18.9 ± 6.0†	3.10 ± 0.5
IRL = 25 cm H2O	–5.2 ± 1.3	–9.9 ± 1.3	0.24 ± 0.02†	27.1 ± 8.5†	4.44 ± 0.6

*Data are presented as mean ± SEM. The Rph index was determined by the division of peak Rph at a given IRL by peak Rph at baseline.

†$p < 0.05$ (loading vs baseline).

Table 4

Pressure Measurements and Collapsibility as Assessed by Response to Externally Applied Load in the Normal Group Measured at Flow of O.21/s, Lateral Posture*

Variables	Pcho, cm H_2O	Pepi, cm H_2O	Flow, L/s	Rph, cm H_2O/L/s	Rph Index
Asleep					
Baseline	–0.8 ± 0.1	–1.2 ± 0.1	0.2	0.77 ± 0.2	
IRL = 15 cm H_2O	–2.9 ± 0.3	–4.0 ± 0.6	0.2	2.23 ± 1.6	2.4 ± 1.3
IRL = 25 cm H_2O	–4.6 ± 0.6	–6.4 ± 0.9	0.2	4.3 ± 2.4	4.9 ± 2.6

* Data are presented as mean ± SEM. Only data during sleep are shown, since flow limitation was not observed during wakefulness.