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Lung volume-related changes in the pharyngeal area of obese females with and without obstructive sleep apnoea

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ABSTRACT: The majority of male patients with obstructive sleep apnoea (OSA) have an abnormal pharyngeal structure and function, with episodic complete airway occlusion during sleep. Since OSA is less common in females than in males, it is possible that other abnormalities are active in female patients with OSA. Consequently, we measured pharyngeal area and its lung volume-related changes (LVRC) from functional residual capacity (FRC) to residual volume (RV) in overweight females, 14 with OSA and 14 without OSA. Pharyngeal areas were measured using the acoustic reflection technique. While there were no significant differences in pharyngeal area between the OSA and control groups at either FRC (mean±SD, 3.49±0.46 cm^2 *vs* 3.08±0.63 cm^2) or RV (2.86±0.47 cm^2 *vs* 2.67±0.49 cm^2), the reduction in pharyngeal area between FRC and RV was significantly greater in the OSA group (0.63±0.23 cm^2 *vs* 0.33±0.32 cm^2, $p<0.05$). Furthermore, although the expiratory reserve volume (ERV) was not significantly different between the two groups (0.4±0.2 *l vs* 0.4±0.3 *l*), LVRC, defined as the reduction in pharyngeal area normalized by ERV, was significantly higher in the females with OSA than in the non-apnoeic controls (2.68±2.24 $cm^2 \cdot l^{-1}$ *vs* 1.17±1.23 $cm^2 \cdot l^{-1}$, $p<0.02$). We conclude that females with OSA have abnormal pharyngeal mechanics similar to males with OSA.

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The pathophysiology of obstructive sleep apnoea (OSA) has been well studied in males, who are affected far more frequently than females by this disorder [1, 2]. While the pathogenesis of recurrent upper airway obstruction in males is multifactorial, pharyngeal narrowing and increased pharyngeal collapsibility have been identified as important factors rendering the pharynx susceptible to occlusion during sleep [3–6]. In contrast, little is known about the pathogenesis of OSA in females. Whereas obesity is a predisposing factor for the development of OSA, the much lower prevalence of OSA in females as compared to males [1] is surprising in view of the higher incidence of obesity among women [7]. This raises the question as to whether pharyngeal abnormalities observed in males with OSA are also present in apnoeic females.

Nevertheless, since pharyngeal collapse is the critical event in OSA, we hypothesized that pharyngeal dysfunction may play a role in the pathogenesis of OSA in females as is the case in males. In order to test this hypothesis, pharyngeal cross-sectional area and its changes with lung volume during continuous slow expiration from functional residual capacity (FRC) to residual volume (RV) were measured in overweight females with and without OSA using the acoustic reflection technique [3, 5, 6, 8]. Our findings suggest that in overweight females, just as in overweight males, abnormalities of pharyngeal function may play an important role in the pathogenesis of OSA.

Methods

Subjects

Subjects consisted of 14 consecutive female patients with OSA and 14 female control subjects without OSA. All patients and controls were overweight, as defined by a body mass index (BMI) greater than 24 $kg \cdot m^{-2}$ [9]. All patients were referred to the sleep laboratory because of clinical suspicion of sleep apnoea. Among the 14 control subjects, 10 were referred because of clinical suspicion of OSA, while the other 4 were referred for routine evaluation prior to gastroplasty for weight reduction. None of the patients or control subjects had any clinical or laboratory evidence of hypothyroidism. All apnoeic and non-apnoeic patients had a history of habitual snoring.

Sleep studies

Overnight sleep studies were performed in all 28 subjects using standard polysomnographic techniques [10]. Respiratory movements of the rib cage and abdomen as well as tidal volume were measured using a respiratory inductance plethysmograph (Respitrace, Ambulatory Monitoring, Ardsley, NY) calibrated by the simultaneous equations method. An ear oximeter (model 47201A, Hewlett-Packard Corp., Waltham, MA) was used for continuous monitoring of arterial oxygen saturation (Sao_2). All variables were recorded continuously on a polygraph (Model 78, Grass Instruments, Quincy, MA) at a paper speed of 15 $mm \cdot s^{-1}$.

Sleep stages were scored according to standard criteria [10]. Obstructive apnoeas were identified as the absence of tidal volume excursions for at least 10 s during which there were paradoxical movements of the rib cage and abdomen. Obstructive hypopnoeas were defined as reductions in tidal volume at least 50% below the baseline, lasting greater than 10 s and accompanied by paradoxical movements of the rib cage and abdomen [2, 6]. The number of apnoeas plus hypopnoeas per hour of sleep was calculated and termed the apnoea-hypopnoea index (AHI). The diagnosis of OSA was established if the AHI was greater than 10. The lowest nocturnal arterial oxygen saturation (low Sao_2) was also recorded.

Pulmonary function

Lung volumes and maximum expiratory flow rates were measured in all subjects by body plethysmography and spirometry.

Pharyngeal areas

These were determined in all 28 subjects using the acoustic reflection technique as described previously [5]. The basic principles of the technique, experimental

Table 1. – Anthropometric data, menopausal status, and sleep data

Subject	Age yrs	Weight kg	BMI $kg \cdot m^{-2}$	Menopausal status	AHI	Low Sao_2 %
OSA						
1	56	102	40	post	45	37
2	50	149	54	post	49	57
3	56	83	37	post	18	64
4	55	81	33	post	23	81
5	40	91	33	pre	56	62
6	29	127	51	pre	50	81
7	61	109	43	post	33	55
8	53	81	34	post	39	82
9	46	121	46	pre	13	72
10	51	110	46	pre	14	67
11	46	99	39	pre	21	92
12	39	102	51	pre	21	50
13	42	117	44	pre	52	76
14	58	100	37	post	21	58
Mean	49	105	42		33	67
±SD	9	19	7		16	15
Non-OSA						
1	60	84	35	post	7	40
2	36	96	37	pre	6	86
3	37	129	38	pre	0	87
4	40	135	47	pre	2	91
5	49	152	60	pre	9	65
6	28	135	48	pre	6	82
7	42	78	32	pre	2	95
8	55	132	53	post	7	80
9	50	121	49	post	0	91
10	56	133	53	post	0	85
11	45	122	44	pre	4	90
12	52	117	43	post	2	91
13	58	68	27	post	4	90
14	45	122	45	pre	3	88
Mean	47	116	44		4	83
±SD	9	25	9		3	14
p	NS	NS	NS		<0.001	<0.005

OSA: obstructive sleep apnoea; BMI: body mass index; AHI: apnoea-hypopnoea index; Low Sao_2: lowest nocturnal arterial oxygen saturation; NS: not significant.

Table 2. – Pulmonary function data

Subject	FRC		RV		ERV	FEV_1/FVC	
	l	% pred	*l*	% pred	*l*	ratio	% pred
OSA							
1	3.1	(119)	2.8	(165)	0.3	0.53	(71)
2	2.5	(78)	2.3	(121)	0.2	0.59	(92)
3	2.5	(106)	2.2	(145)	0.3	0.80	(108)
4	2.1	(84)	1.9	(119)	0.2	0.71	(95)
5	3.0	(100)	2.3	(135)	0.7	0.78	(102)
6	1.9	(70)	1.3	(93)	0.6	0.75	(95)
7	2.7	(108)	2.2	(129)	0.5	0.84	(115)
8	2.2	(88)	1.5	(94)	0.7	0.75	(100)
9	2.2	(69)	2.1	(117)	0.1	0.88	(116)
10	1.9	(76)	1.6	(100)	0.3	0.83	(110)
11	2.6	(105)	2.3	(163)	0.3	0.77	(101)
12	1.9	(110)	1.8	(178)	0.1	0.90	(116)
13	2.8	(91)	2.5	(141)	0.3	0.77	(101)
14	2.6	(83)	2.1	(105)	0.5	0.83	(115)
Mean	2.4	(92)	2.1	(128)	0.4	0.77	(103)
±SD	0.4	(16)	0.4	(26)	0.2	0.09	(12)
Non-OSA							
1	3.2	(139)	3.0	(200)	0.2	0.65	(88)
2	2.2	(85)	1.9	(136)	0.3	0.86	(110)
3	2.0	(51)	1.6	(76)	0.4	0.80	(102)
4	2.5	(80)	2.0	(115)	0.5	0.78	(102)
5	2.7	(96)	2.6	(153)	0.1	0.86	(114)
6	2.2	(71)	1.5	(94)	0.7	0.83	(105)
7	1.9	(73)	0.8	(53)	1.1	0.85	(111)
8	3.4	(126)	3.2	(192)	0.2	0.77	(103)
9	2.3	(78)	2.0	(114)	0.3	0.85	(113)
10	3.2	(111)	3.0	(165)	0.2	0.71	(96)
11	1.6	(63)	1.4	(92)	0.2	0.88	(115)
12	1.8	(60)	1.4	(78)	0.4	0.73	(98)
13	2.3	(85)	1.7	(100)	0.6	0.70	(95)
14	3.2	(106)	2.8	(160)	0.4	0.63	(82)
Mean	2.5	(87)	2.1	(123)	0.4	0.78	(102)
±SD	0.6	(25)	0.7	(45)	0.3	0.08	(10)

FRC: functional residual capacity; RV: residual volume; ERV: expiratory reserve volume; FEV_1: forced expiratory volume in one second; FVC: forced vital capacity; OSA: obstructive sleep apnoea; *l*: litres. No significant differences between variables for OSA or non-OSA.

set-up, mouthpiece construction, and software for data processing are identical to that described previously, and will be summarized here only briefly [3, 5, 6]. The technique is based on measurements of high frequency sound waves which are aimed at the mouth and reflected as they propagate along the respiratory tract. From the knowledge of reflected intensities and the time of arrival of reflections, the area at a given distance from the sensing microphone is computed, and a plot of airway cross-sectional area as a function of distance into the airway is constructed. We shall refer to such plots as the area-distance plots. The apparatus used in the present study consists of an acoustic emitter (loudspeaker), an acoustic transducer (microphone), and a 2 m long stainless steel wavetube. Appropriate amplifiers and filters condition the signal which is then processed to calculate and display the changes in airway area on the computer terminal screen. Simultaneous measurements of lung volume are obtained from the potentiometer attachment of the spirometer coupled to the wavetube. The system allows for the acquisition of 64 consecutive uninterrupted area-distance measurements as quickly as 5 times·s^{-1}.

Experimental design

Acoustic reflection studies were performed with an awake subject in the seated position, wearing a nose-clip, and coupled to the wavetube by means of a custom-made mouthpiece constructed from soft dental wax. Care was taken to ensure that the subject was comfortable and maintained the same neutral head position and neck configuration during all measurements. First, the subject comfortably breathed an 80% helium-20% oxygen mixture for 2–3 min from a

reservoir bag attached to the proximal end of the wavetube by means of a three-way valve. During this period of equilibration, three slow vital capacity manoeuvres were performed in order to standardize lung volume history. The measurements began by changing the position of the three-way valve, thereby enabling the subject to breathe through the wavetube into the spirometer which was also filled with the same gas mixture. In each individual we made simultaneous measurements of lung volume and pharyngeal cross-sectional area. Measurements were performed while the seated, awake subject exhaled slowly from total lung capacity (TLC) to residual volume (RV) and, in a second manoeuvre, during quiet tidal breathing at FRC. For each manoeuvre, 64 measurements of pharyngeal area and lung volume were obtained at intervals of 0.2–0.5 s, and two separate slow expiratory manoeuvres were performed. Mean pharyngeal cross-sectional area was determined at FRC (A_{FRC}) and at RV (A_{RV}) by integrating the total area between the mouthpiece and the glottis and dividing by the distance of integration [5, 6]. Up to 128 measurements of area were acquired at FRC and 12–24 measurements were available at RV. We chose to measure pharyngeal area at FRC and RV because these are the lung volumes between which patients would be breathing in the recumbent position during sleep when lung volume is at or below the upright FRC. The difference between the pharyngeal area at FRC and RV (*i.e.* A_{FRC}-A_{RV}) was calculated in all patients and control subjects.

In order to account for the differences in expiratory reserve volume (ERV), we normalized the difference in mean pharyngeal cross-sectional area (A_{FRC}-A_{RV}) by ERV to obtain the lung volume-related change (LVRC) of the pharyngeal area. A two-tailed, unpaired Student's t-test analysis was used to compare variables between OSA and non-OSA groups. Statistical significance was established if $p<0.05$.

Table 3. – Pharyngeal area at different lung volumes

Subject	A_{FRC}	A_{RV}	A_{FRC}-A_{RV}	$\frac{A_{FRC}-A_{RV}}{A_{FRC}}$	LVRC
	cm^2	cm^2	cm^2	%	$cm^2 \cdot l^{-1}$
OSA					
1	3.40	2.75	0.65	19	2.17
2	3.40	2.40	1.00	29	5.00
3	3.90	3.00	0.90	23	3.00
4	3.80	3.05	0.75	20	3.75
5	3.90	3.45	0.45	12	0.64
6	3.65	3.10	0.55	15	0.92
7	3.50	3.10	0.50	11	0.80
8	3.35	2.90	0.45	13	0.64
9	2.90	2.45	0.45	16	4.50
10	4.30	3.50	0.80	19	2.67
11	2.55	1.80	0.75	29	2.50
12	3.25	2.40	0.85	26	8.50
13	3.90	3.25	0.65	17	2.17
14	3.05	2.90	0.15	5	0.30
Mean	3.49	2.86	0.63	18	2.68
±SD	0.46	0.47	0.23	7	2.24
Non-OSA					
1	2.85	2.75	0.10	4	0.50
2	4.25	3.10	1.15	27	3.83
3	3.60	3.05	0.55	15	1.38
4	3.80	3.60	0.20	5	0.40
5	2.45	2.25	0.20	8	2.00
6	2.90	2.85	0.05	2	0.07
7	3.20	3.00	0.20	6	0.18
8	2.80	2.50	0.30	11	1.50
9	1.65	1.55	0.10	6	0.33
10	3.35	2.65	0.70	21	3.50
11	3.00	2.90	0.10	3	0.50
12	3.15	2.50	0.65	21	1.63
13	2.65	2.65	0.00	0	0
14	3.50	3.25	0.25	7	0.63
Mean	3.08	2.76	0.33	10	1.17
±SD	0.63	0.49	0.32	8	1.23
p	NS	NS	<0.05	<0.05	<0.02

A_{FRC}: pharyngeal area at functional residual capacity (FRC); A_{RV}: pharyngeal area at residual volume (RV); LVRC: lung volume related change of pharyngeal area, *i.e.* A_{FRC}-A_{RV}/ERV (expiratory reserve volume); OSA: obstructive sleep apnoea; NS: not significant.

Results

The anthropometric data, menopausal status, pulmonary function tests, and sleep data for all subjects with and without OSA are shown in tables 1 and 2. There were no significant differences in age, BMI, FRC, RV, ERV, or forced expiratory volume in one second (FEV_1)/forced vital capacity (FVC) between the two groups. The lowest nocturnal oxygen saturation was significantly lower in the OSA group (67±15% *vs* 83±14%, p<0.005).

Representative area-distance plots derived from acoustic reflection both at FRC and RV in a patient with OSA (upper panel) and a control subject (lower panel) are shown in figure 1. The reduction in pharyngeal area from FRC to RV is more pronounced in the patient with OSA.

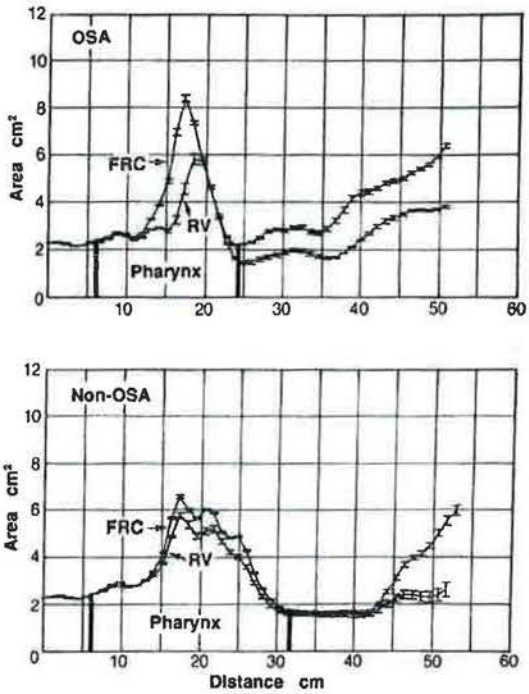

Fig. 1. – Airway cross-sectional area *versus* distance from the microphone in a patient with obstructive sleep apnoea (OSA) (upper panel) and a control subject (lower panel); the pharynx lies between two heavy vertical lines; the greater lung volume-related change of pharyngeal area in the patient with OSA is illustrated by the greater fall in pharyngeal area between functional residual capacity (FRC) and residual volume (RV).

Pharyngeal cross-sectional areas at different lung volumes are shown in table 3. While there were no significant differences in A_{FRC} and A_{RV} between the two groups, A_{FRC}-A_{RV} was significantly greater in the OSA group both in absolute (0.63±0.23 cm^2 *vs* 0.33±0.32 cm^2, p<0.05) and percentage terms (18±7% *vs* 10±8%, p<0.05). Moreover, the OSA group demonstrated a significantly larger LVRC of the pharyngeal area than the non-apnoeic group (2.68±2.24 $cm^2 \cdot l^{-1}$ *vs* 1.17±1.23 $cm^2 \cdot l^{-1}$, p<0.02).

Discussion

Our study demonstrates that, just as in males [5, 6], increased LVRC in the pharyngeal area distinguishes overweight females who snore and have OSA from those who snore and do not have OSA. This was so despite pharyngeal areas that were comparable at FRC and RV. It appears, therefore, that there is a functional abnormality of the pharynx in female patients with OSA which distinguishes them from snoring, non-apnoeic females. Accordingly, the findings of the present study suggest that the increased LVRC in the pharyngeal area of overweight females with OSA is one of the factors promoting occlusive apnoeas during sleep.

We found that non-apnoeic controls tended to have smaller pharyngeal areas (although not significantly so), than apnoeic patients, and yet they did not develop obstructive apnoeas upon assuming the recumbent posture and falling asleep. This indicates that the distinguishing feature between the two groups is not pharyngeal structure but function, as reflected by a higher LVRC of the pharyngeal area, present in the OSA group. It is of note that non-apnoeic women tended to be more obese than the apnoeic ones and this may account for the tendency towards a lower pharyngeal area in that group.

In this study we used LVRC of the pharyngeal area in awake, seated subjects during voluntary expiration as an index of pharyngeal function during sleep. The measurements obtained under these conditions may be different from those obtained during the involuntary inspiratory manoeuvres in supine, sleeping subjects, leading to pharyngeal occlusion during sleep in patients with OSA. However, under both circumstances the measurements of area reflect an integrated effect of pharyngeal muscles and intrinsic pharyngeal tissue properties, *i.e.* pharyngeal muscle tone and tissue linkage. Our finding of increased lung volume associated changes in pharyngeal area imply a reduction in the tone of pharyngeal muscles or a generalized laxity (*i.e.* higher compliance) in the awake patients with OSA as compared with non-apnoeic, snoring controls. The findings reported here would be expected to be even more prominent, rather than reversed, during sleep, when the muscle tone is further reduced. If we had not observed any differences in pharyngeal mechanics in the awake state between the two groups, we still could not reject the hypothesis that such differences exist during sleep, but are simply not present in the awake, sitting subject. Several previous studies by independent groups using different techniques have all demonstrated pharyngeal abnormalities in awake patients with OSA [4, 5, 11–13]. This indicates that, although obstructive apnoeas are confined to sleep, abnormalities of pharyngeal size and function, which may predispose to apnoea, can be detected during wakefulness.

STAUFFER *et al.* [12] have also shown a functional abnormality of the pharynx in patients with OSA compared to control subjects despite comparable pharyngeal size. In that study, transpharyngeal airflow resistance on inspiration was greater in patients with

OSA than in controls, even in the presence of similar pharyngeal cross-sectional area as measured at FRC by computerized tomography scanning. They concluded that increased inspiratory pharyngeal resistance may be related to an impaired ability to dilate the pharyngeal lumen during inspiration in patients with OSA. Presumably, this would predispose them to pharyngeal collapse on inspiration during sleep. These findings, together with the present data, emphasize that an abnormal function of the pharynx may be of equal or greater importance than anatomical pharyngeal narrowing in the pathophysiology of upper airway collapse in OSA. Further evidence in support of this view is that upper airway collapse is state-related and occurs only during sleep, presumably because of the superimposition of a sleep-related reduction in pharyngeal dilator muscle tone upon an intrinsically collapsible pharynx [15, 16].

Independently of sleep state, recumbency is also associated with a further reduction in pharyngeal area [17, 18], as well as a concomitant fall in ERV [19]. The effect of posture on pharyngeal area and distensibility in patients with and without OSA has been specifically addressed in a recent study [18]. It was found that assuming the supine posture results in a 15–20% reduction in pharyngeal area in apnoeic and non-apnoeic individuals. Furthermore, pharyngeal distensibility is significantly higher in the apnoeic patients, that in the non-apnoeic controls, independent of the posture. Thus, it appears that the abnormalities in pharyngeal structure and function present in the sitting position are preserved, and, in fact, are further accentuated in the supine position. It is possible that at some point a critical narrowing is reached so that the balance of forces begins to favour pharyngeal collapse upon exposure to negative inspiratory pressure during sleep [15], particularly in the presence of increased pressure and LVRC in the pharyngeal area [3, 5, 6]. Although the former was not measured in the present study, previous measurements in males with OSA have demonstrated an association between LVRC and increased pressure-related changes of pharyngeal area [3, 20]. Assuming that this relationship is also present in females, our results indicate that females with OSA have "floppier" pharynxes than control subjects. Clearly, this conclusion is an approximation since we have no measurements of intra-pharyngeal pressures to calculate pharyngeal distensibility. Consequently, while the absolute pharyngeal area did not differ between the two groups, higher LVRC and higher pharyngeal collapsibility among patients with OSA would favour pharyngeal collapse during sleep with the generation of negative intra-pharyngeal pressure at the onset of inspiration [15].

Although we found that females with OSA have abnormal pharyngeal mechanics, just as in males with this disorder [5, 6], this is probably not the sole abnormality responsible for airway occlusion during sleep. Closer examination of our results (table 3) reveals that among 14 females with OSA there were 5 with relatively low LVRC (below 1.00 $cm^2 \cdot l^{-1}$); nevertheless, these patients had severe sleep apnoea with a mean AHI of 40. It is possible that abnormalities at other anatomical sites - both proximal and distal to the pharynx - are responsible for promoting airway obstruction in these patients. For example, it is known that upper airway collapse in males may occur at the level of the nasopharynx [21]. However, nasopharyngeal area cannot be measured by acoustic reflection *via* the mouth. The results of acoustic reflection measurements of the pharynx described here and elsewhere [3, 5, 6, 20] are the mean of measurements made between the end of the mouthpiece (*i.e.* at the end of the hard palate) and the glottis. Accordingly, whereas we found no difference in mean pharyngeal area at either FRC or RV between the two groups, it is possible that there were differences in nasopharyngeal area that we could not detect. Furthermore, RUBINSTEIN *et al.* [22] have recently demonstrated that patients with severe OSA may have paradoxical inspiratory narrowing of the glottis as the only abnormality of the upper airway. Although we did not examine nasopharyngeal function or glottic movements in our patients, it is possible that abnormalities at these sites may have contributed to their sleep apnoea.

It is possible that structural differences in the pharyngeal airway between OSA and non-OSA females do exist, but cannot be resolved by the acoustic method. In this study we measured the average pharyngeal area, obtained by integrating 1 cm sections. Any differences in pharyngeal area occurring over a distance less than 1 cm cannot be resolved by this technique. GUILLEMINAULT *et al.* [23] used X-ray cephalometry and found subtle, but definite narrowing of the posterior airway space in females with OSA as compared to the non-apnoeic controls.

Factors involved in LVRC of the pharyngeal area have not been precisely determined. Possible mechanisms include reflex activation of pharyngeal dilator muscles *via* pulmonary stretch receptors during inspiration as described for the larynx, trachea, and the upper airway muscles [24–26]. The expiratory fall in pharyngeal area may be either passive and related to the intrinsic muscle-tissue linkage or compliance of the pharyngeal walls [3], or active and related to the expiratory activation of the pharyngeal constrictors. Active pharyngeal constriction, rather than a passive reflex related to changes in lung volume, is favoured by a recent study of FOUKE and STROHL [17] which failed to demonstrate any changes in pharyngeal area with passive increases in lung volume produced by a negative pressure ventilator. Why some obese females but not others should demonstrate greater LVRC in the pharyngeal area is not clear. Differences in the distribution of the adipose tissue in the neck and pharyngeal walls with fatty infiltration rendering the pharynx more "floppy" is one possible explanation.

While previous studies have suggested a role for the menopause in the pathogenesis of OSA in some females, the bulk of the evidence indicates that the menopause *per se* is not of major pathophysiological significance [7, 27–30]. GUILLEMINAULT *et al.* [23] found no difference in static upper airway dimensions between

premenopausal and postmenopausal women with OSA. In the present study, OSA and control women were very closely matched for both age and weight, and it is very unlikely that differences in menopausal status accounted for the presence or absence of OSA or for the observed differences in pharyngeal function between the two groups.

In summary, the present findings indicate that OSA in overweight females is associated with an abnormality of pharyngeal function which is similar to that seen in overweight males suffering from this disorder. This functional abnormality may be characterized by increased LVRC of the pharyngeal area, which implies a greater tendency of the pharynx to collapse at low lung volumes, presumably in response to normal negative inspiratory pressures generated during sleep. Although the precise mechanism responsible for these abnormalities cannot be deduced from our measurements, they imply that high LVRC in the pharyngeal area is an important factor in the pathogenesis of OSA in overweight women.

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Modification de la surface pharyngée en relation avec les volumes pulmonaires chez les femmes obèses avec ou sans syndrome d'apnée du sommeil (SAS). I. Rubinstein, Y. Hoffstein, T.D. Bradley.

RÉSUMÉ: La majorité des patients de sexe masculin atteints de syndrome d'apnée obstructive du sommeil ont une fonction et une structure pharyngées anormales, permettant des occlusions complètes épisodiques de leur voie aérienne pendant le sommeil. Comme le SAS est moins fréquent chez les femmes que chez les hommes, il est possible que des anomalies étrangères aux propriétés pharyngiennes interviennent chez les femmes atteintes de SAS. Nous avons donc mesuré la surface pharyngée et ses variations en rapport avec les volumes pulmonaires, depuis la CRF jusqu'au volume résiduel chez 14 femmes obèses avec SAS et chez 14 femmes obèses sans SAS. Les surfaces pharyngées ont été mesurées par la technique de réflexion acoustique. Alors que la surface pharyngée était similaire dans le groupe SAS et le groupe contrôle à la CRF (moyenne±SD 3.49±0.46 cm^2 contre 3.08±0.63 cm^2) et au VR (2.86±0.47 cm^2 contre 2.76±0.49 cm^2), la réduction de surface pharyngée entre la CRF et le VR était significativement plus marquée dans le groupe SAS que chez les contrôles (0.63±0.23 cm^2 contre 0.33±0.32 cm^2, $p<0.05$). De plus, quoique le volume de réserve expiratoire ne soit pas significativement différent entre les deux groupes (0.4±0.2 *l* contre 0.4±0.3 *l*), la modification de la surface pharyngée en rapport avec les volumes définie comme la différence de surface pharyngée entre la capacité résiduelle fonctionnelle et le volume résiduel normalisé par le volume de réserve expiratoire, s'avère plus élevée chez les femmes atteintes de syndrome d'apnée du sommeil, par comparaison avec les contrôles non apnéïques (2.68±2.24 $cm^2 \cdot l^{-1}$ *vs* 1.17±1.23 $cm^2 \cdot l^{-1}$, $p<0.02$). Nous concluons que les femmes atteintes de syndrome d'apnée du sommeil ont une mécanique pharyngée anormale semblable à celle des hommes, et que celle-ci peut contribuer à la physiopathogénie du syndrome d'apnée du sommeil.

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