Body Fat Distribution and Sleep Apnea Severity in Women*

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The contribution of body fat distribution to sleep-disordered breathing in women has not been examined in detail (to our knowledge). Fifty women under 65 years of age were diagnosed as having obstructive sleep apnea (OSA) by all-night polysomnography in a 6-month period. Twenty-five women underwent body fat measurements of skin folds and circumferences. The 12 premenopausal and 13 postmenopausal women did not differ in regard to apnea hypopnea index (AHI), SaO2 nadir, body mass index (BMI), or anthropometric measurements. The AHI for these 25 patients was related to the severity of obesity assessed by triceps and subscapular skin folds, the sum of the skin folds, waist circumference, and BMI. The SaO2 nadir correlated with triceps and subscapular skin folds, the sum of the skin folds, and neck skin fold. Clinical features of this same group of 25 women were then compared with those of 45 men with OSA previously described by our laboratory. The women, despite similar age, had less severe OSA than the men (AHI of 34.4 ± 5.4 vs 51.1 ± 4.9, p<0.05). Despite similar BMIs and waist circumference, the men had evidence of a greater degree of upper body obesity with a larger subscapular skin fold thickness, waist-hip ratio, and neck circumference. In addition, for a given degree of upper-body obesity, men had more severe sleep apnea. These findings may explain, at least in part, the greater severity of OSA in the men. (Chest 1995; 107:362-66)

AHI=apnea hypopnea index; BMI=body mass index; NHANES=National Health and Nutrition Examination Surveys; OSA=obstructive sleep apnea

Key words: body fat distribution; obesity; obstructive sleep apnea; women

Early descriptive studies of women suggested that obstructive sleep apnea (OSA) was a disease of postmenopausal women and that premenopausal women were spared because of the respiratory stimulant effects of circulating progesterone.1 Treatment with 30 mg/d of medroxyprogesterone, however, failed to significantly improve sleep-disordered breathing in postmenopausal women.2 More recent studies have shown that OSA indeed can occur in both premenopausal and postmenopausal populations.3-6 Massive obesity rather than structural abnormalities of the upper airway or hormonal differences has been believed to be the dominant factor for the appearance of OSA in women,3 though disease in older women may occur independent of obesity.5

One observation from these studies is that female patients with OSA seen at a sleep laboratory have tended to be more obese than their male counterparts, yet they have demonstrated a similar degree of sleep apnea severity.3,4,6 Guilleminault et al,5 in addition, found that the premenopausal women had more severe OSA than postmenopausal women despite equivalent body mass indices (BMIs).5 Rajala et al7 studied 13 men and 14 women with a BMI of at least 40 kg/m² and found the occurrence of OSA to be 76.9% in men and only 7.1% in the women. These observations suggest that the mere presence of obesity in itself is not the only cause of sleep apnea in women.

One factor that was not examined in these studies was the distribution of body fat. It is well recognized that men tend to have predominantly an upper body or android body fat distribution, while women tend to develop a lower body or gynecoid body fat distribution. Studies of mixed male and female OSA populations have shown a significant relationship between neck size and OSA severity.8-11 Detailed anthropometric evaluation of male patients with OSA from our laboratory demonstrated a high prevalence of upper-body obesity and a significant relationship between skin fold thickness and sleep apnea severity.12

One could postulate from these studies that body fat distribution in women is an important determinant for the development of OSA. We therefore studied our female OSA patients to (1) examine the cross-sectional relationship between upper-body obesity and disease severity and (2) compare the results...
with those of male patients with OSA to determine whether different body fat distributions may explain gender differences in sleep apnea severity. In addition, we attempted to determine whether body fat distribution differed between premenopausal and postmenopausal patients.

**METHODS**

All women 18 to 65 years of age were eligible to be candidates for the study if they had been given a diagnosis of OSA by all-night polysomnography at the Sleep Disorders Center of Rhode Island Hospital during the period from January to June 1992. All subjects had been referred for the evaluation of daytime sleepiness, loud snoring, or observed apneas during sleep. Women were classified as premenopausal if they were still having menstrual periods, and postmenopausal if both ovaries had been surgically removed or they had not had a menstrual period for at least 1 year.

Diagnostic polysomnography was performed as previously described. Apnea was defined as a cessation of airflow lasting more than 10 s, while a hypopnea was defined as a decrease in airflow for more than 10 s resulting in a 4% or greater fall in SaO2. Events were labeled as obstructive if respiratory effort continued despite a decrease of airflow. Central apneas consisted of an absence of airflow and respiratory effort. None of the patients had a predominance of central events. Patients with the symptoms noted above and an apnea hypopnea index (AHI) greater than 5 were considered to have OSA.

Women identified as having OSA were asked to return to the laboratory for complete anthropometric measurements. Twenty-five women consented. The measurements were obtained using standard techniques by a single trained investigator (C.C.C.) who had performed all the anthropometric measurements in our previous study on male patients with OSA from our Sleep Disorders Center. Participants wore an examination gown when the measurements were taken. Height and weight were measured using a standard balance beam scale with a sliding height measure. The BMI was calculated and expressed in kilograms per square meter. Triceps, subscapular, and neck skin fold thickness were obtained using Lange skinfold calipers (Cambridge Scientific Instruments, Cambridge, Mass). The average of three readings to the nearest 0.1 cm was used as the measurement for each skin fold. The skin fold sum was determined by adding the individual values for triceps and subscapular skin fold thickness. Neck, waist, and hip circumferences were measured in duplicate using a metal tape and expressed in centimeters. The measurement for neck circumference was obtained just inferior to the laryngeal prominence. Waist circumference was measured at the narrowest point between the ribs and the iliac crest. Hip circumferences was determined at the level of maximum extension of the buttock.

The BMI and the sum of subscapular and triceps skin folds for the 25 women were also compared to normative data derived from the first and second National Health and Nutrition Examination Surveys (NHANES I and II) of the United States conducted during 1971 to 1974 and 1976 to 1980.12

The unpaired Student t test was used to compare premenopausal and postmenopausal groups for both the total population of women diagnosed as having OSA and for the subgroup that underwent anthropometric measurements. Comparisons were also made between the women who had anthropometric measurements and the men presented in our previous study. Pearson’s correlation coefficients were used to examine relationships between two variables. Multiple regression techniques were used to analyze relationships between two or more independent variables (age, BMI, and anthropomorphic measurements), and a dependent variable (AHI or SaO2 nadir). All statistical analysis was performed using software (Statview SE Graphics, Abacus Concepts Inc, Berkeley, Calif). A p value less than or equal to 0.05 was considered to be of statistical significance.

**RESULTS**

Of the 117 women in this age range examined in our laboratory during this 6-month evaluation period, 50 were diagnosed as having OSA. The characteristics of this group were as follows: age, 46.6 ± 1.4 years (mean ± SEM); BMI, 37.0 ± 1.3 kg/m²; AHI, 37.0 ± 4.0 events per hour; and SaO2 nadir, 84.7 ± 1.3%. All the women were white. Half of the women were actively menstruating; the other half were postmenopausal. Menopause had been induced surgically in nine of the women (36%). Only four of the women (16%) were currently receiving hormonal replacement. The premenopausal women were significantly younger than the postmenopausal women (39.5 ± 1.4 years compared with 53.0 ± 1.5 years, respectively, p<0.0001). They did not differ in regards to BMI or sleep apnea severity.

Half of the women were willing to come in for anthropometric measurements. There were 12 premenopausal and 13 postmenopausal women, and the clinical characteristics of these subgroups did not differ significantly from the original cohorts of 25 premenopausal and 25 postmenopausal women in regards to age, BMI, AHI, or SaO2 nadir. There were no differences in AHI, SaO2, BMI, or any skin fold or circumference measurement between the premenopausal and postmenopausal groups who underwent anthropometric measurements (Table 1). Because of the marked similarities between premenopausal and postmenopausal women, they were combined as a single group for the purpose of further analyses.

The BMI and skin fold sum for these 25 women were compared with NHANES I and II normative data. All the women were in the 50th percentile or above compared with national norms for BMI and...
60% were above the 85th percentile (Fig 1). In regards to skin fold sum, 88% of the women were in the 85th percentile. One women who was an active weightlifter fell in the 25th percentile.

Strong interrelationships between the various body fat measurements were found for the 25 women and are shown in Table 2. Using linear regression analysis, AHI correlated significantly not only with BMI, but also waist circumference, subscapular skin fold, triceps skin fold, and the sum of the skin folds (Table 3). The SaO2 nadir correlated best with the above skin fold measurements as well as neck skin fold. Neither variable correlated with neck circumference. With multiple stepwise regression, AHI correlated only with the sum of the skin folds (β=0.477, r²=0.223, p<0.05) and SaO2 nadir with triceps skin fold (β=-0.346, r²=0.205, p<0.05).

The 25 women were then compared with the 45 men previously described by our laboratory.12 The two groups were of a similar age (48.6 ± 2.1 years for the women compared with 48.2 ± 4.4 years for the men) and BMI (35.6 ± 1.6 kg/m² and 33.0 ± 0.8 kg/m², respectively). Despite these similarities, the men had more severe OSA with an AHI of 51.1 ± 4.9 compared with 34.4 ± 5.4 events per hour sleep for the women (p<0.05). The SaO2 nadir tended to be lower in the men at 80.0 ± 1.7% compared with 83.7 ± 2.1% for the women, but this comparison did not reach statistical significance. The men had some evidence of a greater tendency for upper body fat deposition in view of a smaller hip circumference (110.5 ± 1.8 cm in the men vs 120.8 ± 3.7 cm in the women, p<0.01), an increase in the waist to hip ratio (0.99 ± 0.02 vs 0.85 ± 0.02, respectively, p<0.01), and a larger subscapular skin fold (53.0 ± 1.7 mm vs 46.3 ± 2.7 mm, p<0.05). Neck circumference in the men was also significantly larger at 42.9 ± 0.5 cm compared with 38.7 ± 0.8 cm in the women (p<0.001). Waist circumference, however, did not differ between the two groups (109.7 ± 3.7 cm in the men vs 105.7 ± 3.7 cm in the women).

Since AHI for both the women and the men correlated best with the sum of the subscapular and triceps skinfolds, we plotted the relationship of AHI to skin fold sum for each gender as separate plots on the same graph. As shown in Figure 2, men had a greater AHI for the same degree of obesity as measured by

![Figure 1. Distribution of BMI (top) and subscapular skin fold sum (bottom) based on NHANES normative data for the 25 female patients who underwent anthropometric measurements.](image)

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<th>Table 2—Correlation Between Individual Anthropometric Variables*</th>
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*Subscap=subscapular skin fold; C=circumference; WHR=waist to hip ratio.
fp<0.01.
f1p<0.05.

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skin fold sum.

**Discussion**

In this study, we attempted to define the pattern of fat distribution in our female patients with OSA and also tried to determine whether there were any correlations between fat distribution and disease severity. Our female patients with OSA had, in general, higher BMIs and a greater degree of body fat as measured by skin fold sum than the general population described in the NHANES studies. Furthermore, this study demonstrated that upper-body obesity is a significant correlate of sleep apnea severity in women when one compares anthropometric measurements with polysomnographic indices of OSA.

There were significant correlations between AHI and waist circumference, subscapular skin fold, triceps skin fold, and the skin fold sum. The SaO2 nadir correlated with these skin fold measurements and neck skin fold measurements. As with our previous study in male patients with OSA, we did not demonstrate a direct correlation with AHI or SaO2 and neck circumference. One explanation is that skin fold thicknesses may reflect the extent of fat deposits surrounding the upper airway more accurately than neck circumference, which is dependent on lean muscle mass, frame size, and adiposity. It is also possible that there is more of a threshold effect relating neck circumference to AHI in patients with OSA rather than a linear relationship.

Premenopausal and postmenopausal patients with OSA had similar BMIs and anthropometric measurements as well as disease severity. They only differed as would be expected in their age. These findings contrast somewhat from Guilleminault et al who found that their premenopausal patients with OSA had more severe OSA than their postmenopausal patients despite similar BMIs. They postulated that the higher AHI in premenopausal women was related to hormonal status and arousal responses. Since anthropomorphic measurements were not performed in their study, we do not know whether there were variations in body fat distribution between the two groups that could possibly account for the differences in OSA severity.

Despite an equivalent degree of obesity as measured by BMI, our male patients with OSA had more severe sleep apnea than the female patients. This could be partly related to the greater degree of upper-body obesity seen in the men that reflects the general gender differences in body fat distribution found in middle-age populations. As compared with our female population, the men had higher waist hip ratios, subscapular skin folds, and neck circumferences, and smaller hip circumferences.

For a given skin fold sum, the men also had more severe sleep apnea than the female patients, which suggests that fat distribution around the pharynx may play a greater role in the development of OSA in men than in women. One possibility is that the addition of even small amounts of fat around the pharynx in male patients with OSA might augment normal gender differences in the mechanical properties of the pharynx. Male subjects without OSA have been found to have larger pharyngeal cross-sectional areas than normal women. In addition, normal men have also been shown to have a larger change in pharyngeal area with changing lung volume, thus implying a greater tendency for the airway to collapse. Schwab et al found similar results using cine computed tomography. They showed that the percent change in airway size during inspiration and expiration at the retropalatal regions is larger in normal male control subjects than in normal women. It is therefore possible that the difference in OSA severity observed between men and women could be due to a combination of body fat distribution and gender-specific mechanical properties of the pharynx.

An alternative explanation for the difference in sleep apnea severity between men and women is that...
there are gender-related differences in ventilatory control that predispose obese men rather than obese women to develop OSA. Kunitomo et al found that occlusion pressure and ventilatory responses to hypoxia and occlusion pressure responses to hypercapnia were greater in obese than in normal-weight female subjects. No such increase in ventilatory responsiveness was observed with obesity in men. The relatively depressed chemosensitivities in the men could potentially predispose them to developing apneas while asleep.

It is always possible that there could be differences due to anatomic abnormalities such as enlarged tonsillar tissue or retrognathia accounting for some of the differences in disease severity between men and women.

Differences in body fat distribution may partly explain the lower prevalence and lesser severity of OSA in obese women compared with men. More detailed studies such as cine computed tomography of the pharynx and neck and magnetic resonance imaging evaluation of body fat sites need to be performed in these populations to better understand airway dimensions and mechanics.

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