Velopharyngeal Anatomy in Patients With Obstructive Sleep Apnea Versus Normal Subjects

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Purpose: Obesity can cause disturbed breathing and is one of the most significant risk factors for obstructive sleep apnea (OSA). However, the anatomic basis of OSA and, specifically, the anatomic mechanisms leading from obesity to OSA are still unclear. We examined the anatomic features of the velopharynx in patients with OSA versus those without in correlation with the body mass index (BMI), age, history of snoring, and OSA severity and re-evaluated the contribution of adding a frontal view to the cephalometric analysis of patients with OSA.

Materials and Methods: Lateral and frontal cephalometric measurements were taken to assess the velopharyngeal anatomic features of 306 men with various degrees of OSA and 64 men without OSA and without a history of snoring. The demographic, polysomnographic, and cephalometric features were compared.

Results: The patients with OSA had an increased pharyngeal length, thicker velum, a thicker posterior pharyngeal wall, a reduced pharyngeal width, and a consequent narrowing of the pharyngeal lumen. As the BMI increased, the OSA severity increased. Also, in parallel, the velum and posterior pharyngeal wall thickness increased and the pharyngeal width decreased. Three types of velopharyngeal narrowing, with an increased occurrence in severe degrees of OSA, were identified: bottle shape, hourglass shape, and tube shape. These aerodynamically unfavorable changes might cause increased upper airway resistance, explaining the development of both OSA and hypoventilation syndrome in obese patients.

Conclusions: Velopharyngeal thickening and lumen narrowing were shown to be features of obese men with OSA. However, these features developed only above a threshold BMI value. The combination of frontal and lateral cephalometry is important for comprehensive evaluation of patients with OSA.

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Obesity is probably the most common cause of obstructive sleep apnea (OSA) in adults.[1] An increased body mass index (BMI), when characterized by an increased neck size, has been shown to correlate with OSA.[2-5]

Weight gain can gradually impede breathing function during sleep, although this effect can be reversible after weight loss.[6-8] Four mechanisms that underlie this association have been suggested. The first is fat

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deposits around the retropalatal airspace and in the parapharyngeal space.[9] Although fat deposits have been shown on magnetic resonance imaging (MRI) studies,[9-12] no statistically significant correlation was found between the size of these deposits and overweight status. The second mechanism is narrowing of the airway because of increased thickness and collapsibility of the lateral pharyngeal walls.[11-13] The third is mass loading of excessive adipose tissue in the neck,[2] thus, producing a gravitational load effect on the airway and causing changes in airway configuration and function, especially when the patients are in a supine position. The fourth mechanism is lung restriction resulting from an accumulation of adipose tissue in and around the ribs, abdomen, and diaphragm that reduces the compliance of the thoracic cage.[14]

Inspiratory collapse of the pharyngeal walls occurs when the forces developed by the contraction of the pharyngeal dilator muscles are less than the negative inspiratory pressure. According to Bernoulli's principle, the narrower the upper airways, the greater the inspiratory transmural pressure gradient and the greater the instability of the upper airways. Therefore, any reduction in the upper airway cross-sectional area, because of structural abnormalities or space-occupying lesions, can contribute to the collapse. In addition, changes in the relative relationship among the connective, muscular, and adipose tissue can alter the stiffness of the velopharyngeal wall and increase its elasticity. Structural changes that decrease airway volumes can also directly increase compliance and collapsibility.[11,15] Weight loss can result in decreased upper airway collapsibility.[16] It has previously been demonstrated that both severe snorers and patients with apnea have smaller airway volumes than nonsnorers.[11,17] The upper airway dimensions depend on both the craniofacial structure and the velopharyngeal soft tissue characteristics. Polo et al[18] found that the minimal cross-sectional area at the pharyngeal level decreased and the severity of nocturnal breathing disturbances increased consistently as a function of an increased BMI. However, this correlation between obesity and OSA severity has not been consistently found.[19-21] Moreover, a significant regression model using cephalometric variables could not be obtained for the obese patients to determine the apnea–hypopnea index (AHI).[22] This lack of significance can be attributed to the inherent limitation of the traditional lateral cephalometric investigation, which examines a 3-dimensional object using a 2-dimensional technique.

Finkelstein et al,[23] who first introduced frontal cephalometry to augment the traditionally lateral view, provided additional information on the transverse dimensions of the velopharynx and skull base. Using frontal and lateral cephalometry, the investigators showed that OSA is associated with statistically significant changes in a number of cephalometric measurements of the velopharynx and tongue. Given that the pathophysiology of snoring and OSA syndrome is related, at least in part, to local anatomic factors, it is safe to assume that these cephalometric findings reflect the upper airway soft tissue structural changes that are responsible for the development of OSA in obese patients.

In the present follow-up study of frontal and lateral cephalometry in patients with OSA,[23] we sought to examine the anatomic features of the velopharynx in patients with OSA versus normal subjects in correlation with the BMI, age, a history of snoring, and OSA severity and to clarify the structural effects of obesity in the etiology of OSA.

Materials and Methods

The present study followed the Declaration of Helsinki on medical protocol and ethics, and the regional ethical review board of Meir Medical Center approved the study.

PATIENTS

A total of 306 white men with OSA, who had been referred to the Palate Surgery Unit, Department of Otolaryngology-Head and Neck Surgery, Meir Medical Center, formed the study group. All patients had undergone an otolaryngologic examination, including flexible fiberoptic nasopharyngoscopy of the nose, pharynx, and larynx. Symptom onset was determined from detailed history taking and family or bed-partner reports relevant to upper airway obstruction obtained in structured interviews. All patients underwent a standard polysomnographic examination, including documentation of the AHI, arterial oxygen saturation, and snoring intensity. The latter was measured using a microphone located above the patient's head at a distance of 1 m. The patients were classified by OSA severity into 3 subgroups: *1)* mild OSA, AHI 6 to 20; *2)* moderate OSA, AHI 21 to 30; and *3)* severe OSA, AHI greater than 30. A total of 64 white men, who were selected from the hospital records and had undergone routine videofluoroscopy of their swallowing mechanism, formed the control group. These nonsnoring patients had a good quality of life and no clinical evidence of OSA, which was also ascertained from their family members and bed partners. The inclusion criterion was the use of polysomnography for the study group and family confirmation for the control group. The exclusion criteria were a personal or family history of neuromuscular disease, major malocclusion or tonsillar hypertrophy, cleft lip or cleft palate, craniofacial syndromes, and previous pharyngeal or maxillomandibular surgery.