Dynamic Upper Airway Soft-Tissue and Caliber Changes in Healthy Subjects and Snoring Patients

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BACKGROUND AND PURPOSE: The oropharyngeal airways are smaller in those who snore than in those who do not. We sought to determine which soft-tissue component surrounding the airways contributes to upper airway narrowing in those who snore.

METHODS: Ten control subjects and 19 snoring patients underwent CT, with 2-mm-thick axial sections obtained every 0.6 seconds during the respiration cycle at the same oropharyngeal level. We selected two sections with the widest and narrowest parts of the oropharyngeal airway to measure the anteroposterior and lateral dimensions of the airway as well as the thickness of the bilateral parapharyngeal fat pads, pterygoid muscles, and parapharyngeal walls. Mean values were calculated for each phase. For each subject, differences were calculated by subtracting the values in narrowest phase from those in the widest phase.

RESULTS: Changes in airway dimension (P < .05) and lateral parapharyngeal wall thickness (P < .01) were significantly different between snorers and control subjects. Changes in parapharyngeal wall thickness and transverse oropharyngeal airway diameter changes were significantly related (P < .01).

CONCLUSION: Airway narrowing predominantly occurs in the lateral dimension in people who snore. Changes in the lateral pharyngeal wall are more important than the parapharyngeal fat pads in airway calibration. Narrowing of the upper airway area at the end of the expirium and the beginning of the inspirium is thought to be the cause of snoring and due to augmented muscle mass and prolonged laxity rather than inadequate activation of the pharyngeal dilating muscles.

Snoring is a noisy inspiratory sound produced by vibrations and partial obstruction in the oropharynx. Some structural abnormalities can be associated with snoring (1). Snoring is not only an uncomfortable condition but also one of clinical importance. Snoring is closely linked with sleep apnea, and both conditions are risk factors for cardiovascular and cerebrovascular diseases (1, 2). Obesity, male sex, older age, smoking, alcohol, and some drugs (tranquilizers or muscle relaxants) are important risk factors for snoring (3, 4).

Some have reported that people who snore have oropharyngeal airways smaller than those of individuals who do not (5–8). Because dynamic airway changes during respiratory cycle can be demonstrated on cine CT, we have examined the upper airway with cine CT, as the other investigators did (8–12). In this study, we sought to detect which soft-tissue component surrounding the airways plays the most important role in this narrowing.

Methods

Nineteen snoring patients who were followed up by the otolaryngology department for snoring and 10 control subjects were evaluated. The volunteer control subjects had regular sleeping partners who were able to confirm that they did not habitually snore. A detailed medical and snoring history was taken. Snorers had a nightly snoring frequency and no episodes of cessation of breathing. Three patients had one or more symptoms of fatigue, tiredness, or sleepiness. The remaining 16 were symptom free.

Patients and control subjects who were found to have nasal, oral, or pharyngeal variations or diseases on CT scans were excluded. Their weights and heights were measured, and body mass indices (BMIs) were calculated by dividing their mass in kilograms by the square of their height in meters. The control group was not weight matched, as simply weight-matching subjects (i.e., those with the same BMI) may not be an appropriate control because fat distribution may still differ between groups (12).
An Xpress spiral CT scanner (GX model TSX 002a; Toshiba, Toshigi-Ken, Japan) was used for CT examinations. All subjects were examined in an awake state in the supine position with their head in neutral position. The Frankfort plane was perpendicular to the floor. Subjects were instructed to breathe exclusively through the nose and not to swallow or talk during scanning. A preview scan was performed from nasion to the epiglottis to exclude nasal, oral, or pharyngeal variations or diseases and to locate the oropharyngeal (retropalatal) anatomic level. Subsequently, 24 scans were obtained at the same oropharyngeal level in approximately four full respiratory cycles while subjects were breathing through the nose. Each of the CT sections were obtained with 2-mm thickness in 0.6 seconds without table increment. This technique provided cross-sectional dynamic viewing of the airway during various phases of respiration. A fixed window level of 50 H and window width of 350 H was used for standardization of all scans.

Measurements

Axial images were played back in a closed-loop movie format so that real-time changes in cross-sectional area could be displayed (Fig 1). Two sections were chosen: one at the beginning of expiration, which had the widest airway area, and a second at the end of expiration and the beginning of the inspiration, where the narrowest airway area was demonstrated (12). On these two sections, anteroposterior and lateral dimensions of the airway and the thicknesses of left and right parapharyngeal fat pad, left and right pterygoid muscles, and left and right parapharyngeal walls were measured, and mean values were calculated for each section (Fig 2). For each subject, the difference of values in the widest and narrowest phases of the airway were calculated and used for statistical analysis.

Statistical Analysis

The paired t test was used to compare variables in the two groups. Pearson coefficients (r) were calculated to determine the relationship between variables.
There was no difference in mean age between two groups (P > .05). The mean BMI of snorers was 24.87 (normal weight, 18.5–24.9) in control subjects. The difference was significant (P = .01). The mean BMI of snorers was 2.34 mm in control subjects; this difference was not significant (P > .05). Changes in the lateral parapharyngeal wall thickness were 0.39 ± 0.99 mm in snorers and 0.59 ± 1.16 mm in control subjects. For lateral parapharyngeal fat pad thickness, changes were 2.34 ± 2.25 mm in snorers and 0.95 ± 2.03 mm in control subjects; the changes were not significantly different (P > .05) between the groups.

In snoring patients, changes in parapharyngeal wall thickness were significantly related to changes in transverse oropharyngeal airway diameter (r = 0.72, P < .01). The relationship between changes in parapharyngeal wall thickness and changes in anteroposterior oropharyngeal airway diameter was moderate (r = 0.66, P < .05), and transverse and anteroposterior oropharyngeal airway diameters were significantly related.
greater than that of healthy individuals (18). With weight loss, upper-airway volume significantly increases while the volume of the lateral pharyngeal walls is reduced (19). However, to display the upper airway at the beginning, mid, and end of both expiration and inspiration, each section must be obtained in at least 0.4–0.6 second at the same level. Thus, volumetric analysis is not possible on dynamic cross-sectional imaging. On the other hand, volumetric and dimensional measurements are correlated. Since the dimensions of the pharyngeal airway change during the respiratory cycle, we performed dynamic scanning at the same anatomic level. Many investigators have shown that the oropharyngeal level is the most-affected part of the pharynx (7, 12, 20); therefore, we performed our scanning at this level.

Few studies of dynamic width changes at the parapharyngeal space are reported in the literature. In the present study, we found that the airway narrowing in snorers is predominantly in the lateral dimension, similar to the findings of Schwab et al (21). Two important soft-tissue components seen lateral to the airway are the parapharyngeal fat pads and the parapharyngeal walls. Investigators have noted that the fat pads play an important role in airway configuration. The fat pads are wider in snorers and apneic people than in others; therefore, the pads are closer to the airway and compress the airway laterally (18, 22–25). However, we did not find any important difference in the parapharyngeal fat pads in narrowing of the lateral pharyngeal walls between snorers and control subjects, as Schwab et al noted (21). Contrary to previous studies (18, 22–25), ours showed the lateral pharyngeal fat pad was thicker in healthy people than in nonapneic snorers (Fig 1, F, G), and cine CT images suggested that the pads could easily compress the parapharyngeal walls into the airways.

Obesity is a known risk factor for sleep-disordered breathing, and weight loss is associated with improvements in the degree of the disorder (24, 26). None of our patients or control subjects was obese, and this may be the reason why we did not find narrowing of airways by the parapharyngeal fat pads in this study. Possibly, the mechanism between obesity and sleep-disordered breathing may have an alternative explanation, as Schwab et al offered (21). In addition, our patients with normal body habitus did not have this cause of pharyngeal narrowing. Rather, they had poor musculature dilatory mechanisms in the pharyngeal wall, as previous authors proposed. Some have noted that weight loss results in decreased muscle mass and a reduction in the size of the lateral pharyngeal walls (21).

The lateral pharyngeal wall has a complex structure made up of lymphoid tissue pharyngeal mucosa, and numerous muscles (hyoglossus, styloglossus, stylohyoid, stylopharyngeus, palatoglossus, palatopharyngeus, and pharyngeal constrictors) with varying functions. Although the changes in anteroposterior dimension were important in airway narrowing, the most significant airway changes occurred in the transverse dimension. Both of these diameter changes were more prominent in snorers than in control subjects. Decreased lateral dimension of the airways can be explained by the significant increase in width of the pharyngeal walls during the respiratory cycle in snorers; however, this dimensional change was not clear in the control subjects. Hudgel (27) speculated that edema or inflammation of the pharyngeal tissues might narrow the upper airway. If his presumption is valid, wall thickening and upper-airway narrowing should persist in snorers during all phases of the respiratory cycle, since the thickness of inflamed and edematous walls should not change significantly from beginning to end of the expirium. In contrary to his suggestion (20), our results showed that the lateral pharyngeal walls in snorers were thinner than or nearly equal to the lateral pharyngeal walls in control subjects at the largest phase, whereas they become larger at the end of the expirium, the narrowest phase of respiration (Table 1). The changes of thickness of the lateral pharyngeal wall between the beginning and the end of expirium in snorers (4.14 mm) were significantly higher than in control subjects (0.66 mm).

Our study indicated insufficient widening of upper airway at the end of the expirium of the lateral pharyngeal walls may be caused by the collapse from inflammation (30) or hypertrophy of the soft tissues (35). We believe that the collapse from inflammation in snorers with a positive history of sleep apnea is more probable because of the significant increase in width of the parapharyngeal fat pads (0.66 mm) and the lateral pharyngeal walls (4.14 mm) in snorers at the end of the expirium. Furthermore, snorers have a higher degree of negative inspiratory pressure, prolonged inspiratory time, and inspiratory flow limitation are increased total pulmonary resistance (30). The findings of our study are consistent with the normal consequences of sleep (2). Snorers have higher negative inspiratory pressure, greater pulmonary resistance, prolonged inspiratory time, and flow limitation than others (28, 29). Moreover, some authors found that critical pressures required to collapse the upper airway vary from markedly negative in healthy people to less negative in nonapneic snorers to slightly positive in subjects with frank sleep apnea (30, 31). Smirne et al (32) demonstrated that snorers have an increased percentage of hypertrophic type IIA fibers in their medium pharyngeal constrictor muscle. Postural muscle tone is highest in wakefulness, decreased in non-REM sleep, and minimal or absent in REM sleep (33). Obstruction occurring in REM is likely to be more severe because of a loss of muscle tone, and it may last longer because of impaired arousal mechanisms (34). Although upper airway muscle tone decreases dramatically in REM sleep, airway resistance does not increase beyond the levels found in non-REM sleep (27). Greater muscle laxity in snorers or an increase in muscle mass due to weight gain or the exercise of overcoming apnea might explain the increase in the size of the lateral soft tissues (35).

Positive intraluminal pressure that expands in early expiration abates toward the end of expiration with resultant narrowing of the airway. Mahadevia et al (36) have demonstrated that expiratory positive air-
way pressure alone can effectively treat obstructive apneas. Snore sounds at the end of the expirium result from narrowing of the airway, which is caused by less positive pressure due to respiratory parameters such as low tidal volume or increased muscle mass in its inactive phase. Snore sounds at the phase of early inspirium occur due to gradual forceful opening of the closed airway caused by the aforementioned factors. In early inspirium, increased negative pressure and prolonged pharyngeal muscle laxity due to reduced strength and increased latency of the reflex muscle activity (15) cause a delay in a adequate opening of the narrowed or obstructed airway; therefore, snore sounds occur.

Changes in the width of the pterygoid muscles were not significantly different between the two groups. We suggest that this tonus insufficiency did not affect this muscle group, and these muscles did not affect the airways.

**Conclusion**

In our study, changes in the thickness of the lateral pharyngeal wall was significantly related to airway diameter in snorers, whereas there was no notable change in the parapharyngeal fat pads. Narrowing of upper-airway area at the end of the expirium and the beginning of the inspirium, thought to be the cause of snoring, occurs because of augmented muscle mass in its inactive phase. Snoring sounds at the phase of early inspirium, increased negative pressure and prolonged pharyngeal muscle laxity due to reduced strength and increased latency of the reflex muscle activity (15) cause a delay in adequate opening of the narrowed or obstructed airway; therefore, snore sounds occur.


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