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 and 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) in those who snored but not in control subjects.

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 of 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, 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).
CT Examinations

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 their nose and not to swallow or talk during scanning. The 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 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.
Results

There was no difference in mean age between the groups (P > .05). The mean BMI of snorers was 24.87 (normal weight, 18.5–24.9) and 28.2 (overweight, 25–29.9) in control subjects. The difference was not significant (P > .05). Changes in pterygoid muscle 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 re-

Table 1: Measures of the oropharyngeal airway and surrounding planes

<table>
<thead>
<tr>
<th>Group</th>
<th>Transverse Airway Diameter</th>
<th>Pharyngeal Wall Width</th>
<th>Parapharyngeal Fat Pad Width</th>
<th>Pterygoid Muscle Width</th>
<th>Anteroposterior Airway Diameter</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Narrowest</td>
<td>Widest</td>
<td>Narrowest</td>
<td>Widest</td>
<td>Narrowest</td>
</tr>
<tr>
<td>Controls</td>
<td>18.21</td>
<td>20.03</td>
<td>12.67</td>
<td>12.01</td>
<td>13.89</td>
</tr>
</tbody>
</table>

Note.—Data are in millimeters. Narrowest indicates measures in the narrowest phase of the oropharynx; widest, measures in the widest phase.

Table 2: Parameters and results for snorers and controls

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Snorers (n = 19)</th>
<th>Controls (n = 10)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Age (y)</td>
<td>40.42</td>
<td>10.75</td>
<td>39.10</td>
</tr>
<tr>
<td>BMI</td>
<td>24.87</td>
<td>0.7</td>
<td>28.2</td>
</tr>
<tr>
<td>Changes (mm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anteroposterior airway diameter</td>
<td>3.73</td>
<td>3.08</td>
<td>1.16</td>
</tr>
<tr>
<td>Transverse airway diameter</td>
<td>6.85</td>
<td>5.39</td>
<td>1.81</td>
</tr>
<tr>
<td>Pharyngeal wall width</td>
<td>−4.14</td>
<td>2.69</td>
<td>−0.66</td>
</tr>
<tr>
<td>Parapharyngeal fat pad width</td>
<td>2.34</td>
<td>2.25</td>
<td>0.95</td>
</tr>
<tr>
<td>Pterygoid muscle width</td>
<td>0.39</td>
<td>0.99</td>
<td>0.59</td>
</tr>
</tbody>
</table>

In this study, we tried to understand what leads to airway narrowing and which soft-tissue component is dominant in the pharyngeal-airway changes. Most of the studies in literature were not done dynamically at the same anatomic level. Volumetric MR imaging studies show that upper-airway volume is less in snorers and apneic patients than in healthy subjects (17). At the same time, the volume of adipose tissue adjacent to the pharyngeal airway in patients with apnea is
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 predominately 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). However, we did not find any importance of the parapharyngeal fat pads in narrowing of the airways, as Schwab et al noted (21). Contrary to previous studies (18, 22–25), ours showed that the parapharyngeal fat pad was thicker in the lateral pharyngeal walls. This enlargement may be caused by the significant increase in width of the lateral pharyngeal wall between the beginning and end of the inspiratory phase (Table 1). The changes of thickness of the lateral pharyngeal wall at the beginning and end of the inspiratory phase were more prominent in snorers than in control subjects (4.14 mm) were significantly higher than the changes in control subjects (0.66 mm).

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). Our findings suggest that insufficient widening of the upper airway at the end of the expirium may be caused by the significant increase in width of the lateral pharyngeal wall. This enlargement may be caused by the significant increase in width of the lateral pharyngeal walls during the respiratory cycle in snorers. Increased negative inspiratory pressure, increased total pulmonary resistance, prolonged inspiratory time, and inspiratory flow limitation are 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. 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 muscles 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.

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 and prolonged laxity rather than inflation of the pharyngeal dilating muscles.