Upper Airway Changes in Snorers and Mild Sleep Apnea Sufferers After Uvulopalatopharyngoplasty (UPPP)*

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Objectives: We used upper airway (UA) imaging in 20 patients to determine (1) whether an effective enlargement of the UA is obtained after uvulopalatopharyngoplasty (UPPP), and (2) whether UA modifications explain the results of such surgery.

Methods: Cephalometric measurements were made to assess the posterior airway space, the length and width of the soft palate, and the distance between the hyoid bone and the mandibular plane. Pharyngeal CT measured the airway cross-sectional area (CSA) at each 10-mm slice from 10 mm above (−10) to 40 mm below (+40) the hard palate. Polysomnography was performed before and after surgery (10±10 [SD] months). Good responders were defined by an apnea-hypopnea index (AHI) of <10 postsurgery or, in patients whom AHI was initially <10, a reduction of AHI >50% of the initial AHI.

Results: Twenty patients (age=45±11 years) were studied. For the whole group, the mean body mass index (26±4 kg/m²) and AHI (14±13 vs 18±16/h) were unchanged after UPPP. The results of the surgery were mediocre with 7 good responders (35%) and 13 nonresponders (65%) defined by polysomnographic criteria. The only changes on UA imaging for the group as a whole after UPPP were decrease in length (40±6 vs 29±5 mm, p≤0.0006) and increase in width of the soft palate (11.5±2.7 vs 13.6±3.5 mm, p≤0.006). The increase or decrease in minimal CSA at the oropharyngeal (OP) level after UPPP was significantly correlated with the change in AHI (r=−0.54, p<0.02). Moreover, the changes in CSA obtained at the OP level were significant only in the patients who responded favorably to UPPP (7 vs 13 nonresponders).

Conclusions: Postoperative OP enlargement is associated with a good outcome of UPPP. Persistent narrowing in nonresponders could be due to the increase in soft palate width after surgery.

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Key words: cephalometry; obstructive sleep apnea syndrome; pharyngeal CT scan; snoring; upper airway imaging; uvulopalatopharyngoplasty (UPPP)

Abbreviations: AHI=apnea-hypopnea index; BMI=body mass index; CPAP=continuous positive airway pressure; CSA=Cross-sectional area measured by pharyngeal CT scan; MP-H=distance between hyoid bone and mandibular plane; OSA=obstructive sleep apnea; PAS=posterior airway space; PNS=P=length of the soft palate; UA=upper airway; UPPP=uvulopalatopharyngoplasty; W=length of the soft palate

Uvulopalatopharyngoplasty (UPPP) was introduced by Fujita et al1 in 1981 as a treatment for snoring and obstructive sleep apnea (OSA) syndrome. It is performed on many thousands of patients per year throughout the world. In Australia, the number of UPPP operations has increased from 250 in 1991 to 1992 to about 2,000 in 1994 to 1995.2 UPPP improves apnea-hypopnea index (AHI) in 41% of apneic patients3 and this effect is attributed to an enlargement of the oropharynx. However, only a few studies4-6 have examined whether such surgery does increase the size of the oropharynx, and to our knowledge, none has used both cephalometry and CT of the pharynx to examine the morphologic changes induced by surgery.

In addition, many studies have tried to analyze the site of pharyngeal narrowing preoperatively, but few of these studies have attempted to establish the site of pharyngeal narrowing postoperatively in those
patients in whom surgery fails. Failure of surgery was initially thought to be associated with persistence of or appearance postsurgery of airway collapse at the level of the hypopharynx. In fact, upper airway (UA) pressure measurements during sleep post-UPPP in small numbers of patients reported by various authors7–11 have shown a persistence of airway collapse posterior to the residual soft palate, ie, at the oropharyngeal level in most patients.

The present study aimed to evaluate the effects of UPPP on the dimensions of the UA and to determine whether enlargement of the oropharynx was obtained after surgery. Furthermore, could the outcome of UPPP be explained in terms of the morphologic modifications produced by the surgery and the site of airway narrowing that persisted?

**Materials and Methods**

**Patients and Protocol**

This was a retrospective case note study of 20 snorers (18 men, 2 women) with or without mild OSA. Patients had undergone initial polysomnography, cephalometry, and pharyngeal CT scan for investigation of symptoms suggestive of OSA. Ten ± 10 months after UPPP, these patients had follow-up polysomnography, cephalometry, and pharyngeal CT scan. Patient symptoms were assessed from the clinical case notes. Patient mean age was 45 ± 11 years, and mean body mass index (BMI) was 26 ± 4 kg/m². Operations were performed by three different surgeons of the same team without significant difference both in term of surgical techniques and clinical results.

**UA Imaging**

**Anatomic Definitions**: The inferior border of the nasopharynx was defined by the lowest extent of the hard palate, and the lower margin of the oropharynx was identified by the tip of the uvula. The hypopharynx extends from the tip of the uvula to the epiglottis.

**Cephalometry**: Lateral cephalometric radiographs were obtained using the technique described by Riley et al.12 Briefly, the patient was seated with his head in a neutral position with the gaze parallel to the floor and the teeth together. The x-ray plate was placed next to the left side of the face, and the cone was 1.5 m from the patient. Exposures were taken with the patient remaining still while slowly exhaling a moderately deep breath. Each study gave 4.7 mGy of radiation to the skin. The following measurements were made from the radiographs (as described by Riley et al.13) (1) MP-H: distance from the mandibular plane (a plane constructed from gnathion through gonion to the hyoid bone; (2) PAS: the posterior airway space measured between the posterior pharyngeal wall and the dorsum of the tongue on a line joining gonion to the supamandible (maximal concavity on the anterior surface of the mandible with normal dimensions of 11 ± 2 mm); and (3) PNS-P: distance from the posterior nasal spine to the tip of the palate. This gives a measure of the length of the soft palate and has a normal range of 34 ± 6 mm.

In addition to the three routinely measured parameters (PNS-P, PAS, MP-H), the width (thickness) of the soft palate was also recorded (W), corresponding to the maximal anteroposterior distance of the soft palate.

**Computed Tomography**

The purpose of CT scanning13 was to measure the luminal area of the airway at the level of the nasopharynx, oropharynx, and hypopharynx. Scans were performed using a CT scanner (CE, 12,000; CGR; Buc, France) with a 3.4-s scan time. Patients were closely observed to ensure that they remained awake throughout the procedure and did not swallow during imaging. Scanning was performed during quiet breathing. The subjects were placed in the supine position on the scanning table with the neck placed in a neutral position midway between flexion and extension. A lateral scout view of the neck was obtained that enabled measurement of the length of the soft palate in the supine position using the cursor of the CT scanner. Two sections were obtained at the levels of the nasopharynx and the maxillary sinuses in order to detect any nasal occlusion or other anatomical abnormality at these levels. Slices, 5 mm thick, were imaged every 10 mm, each giving 10 mGy of radiation on the skin surface. Axial cuts were then taken at each 10-mm plane from 10 mm above (–10) to 40 mm below (+40) the hard palate (level 0). Care was taken to ensure that all cuts were perpendicular to the airway lumen to allow accurate assessment of the cross-sectional area (CSA).

For measurements of CSA, an integral software program was used to determine the contours of the pharyngeal lumen. In the cephalic regions of the UA, only the velopharyngeal airway posterior to the soft palate was measured. Air in the oral cavity anterior to the soft palate was ignored. The minimal airway CSA was determined also at the levels of the nasopharynx, the oropharynx, and the hypopharynx. The size of the base of tongue was estimated as described by Larsson et al.14 by the measurement of the interhyoglossal distance and the genioglossal width at the intersection with the hyoglossus. The window settings were standardized for all the patients.

**Polysomnography**

Continuous recordings were taken of the EEG with electrode positions C3/A2, C4/A1, Cz/A1, of the International 10-20 Electrode Placement System, eye movements, chin electromyogram, and ECG with modified V2 lead. Respiration was monitored with uncalibrated inductance respiratory photoplethysmography. Airflow was measured by the sum of buccal and nasal thermistor signals, and oxygen saturation was measured with an oximeter (Biox-Ohmeda 3700; Ohmeda; Liberty Corner, NJ).

The polysomnogram was scored manually according to standard criteria.15 Episodes of apnea were defined as complete cessation of airflow for >10 s and hypopnea as a >50% decrease in oronasal airflow lasting for at least 10 s. Apnea/hypopnea events were classified as central, obstructive, or mixed according to the absence or presence of breathing efforts.

**Statistical Analysis**

An analysis of variance was used to evaluate the changes in UA measurements and in polysomnography before and after surgery. An analysis of subgroups was used to compare good responders and nonresponders to UPPP (Mann-Whitney U test). A successful surgical outcome was defined by an AHI inferior to 10/h or a reduction in AHI of at least 50% in cases in which the preoperative AHI was inferior to 10/h.

**Results**

**Patient Symptoms After UPPP and Complications**

Ten patients (50%), of whom 2 were good responders and 8 were nonresponders, reported snor-
ing persisting after UPPP. Three of 20 patients (15%) reported respiratory pauses during sleep and 7 others (35%) reported persistent daytime sleepiness. There were no major complications seen as a result of the surgery. Minor complications included nasal speech, nasal regurgitation (n=4, 20%) of which all regressed after 2 months, dehiscence at sutures (n=1), sensory symptoms of the pharynx (n=4, 20%), and minor perioperative hemorrhage (n=1).

Effect of UPPP on Sleep and Respiratory Disturbances

The results of the surgery were mediocre with 7 good responders (35%) and 13 nonresponders (65%) defined by polysomnographic criteria (Table 1). The AHI for the population as a whole was not changed by the surgery (14±13 vs 18±16/h) despite the stability of the BMI (26±4 vs 26±4 kg/m²). In the case of the nonresponders, the AHI worsened, being initially 14.5±14 and subsequently 25±15/h after surgery (p=0.003). For the whole group, the sleep structure was modified with a significant reduction in stages 1 and 2 (75±10% vs 69±11%, p≤0.04) in favor of stages 3 and 4 (4±5% vs 7±5%, p≤0.01). However, the number of microarousals was unchanged for the group as a whole; the tendency for microarousals to be reduced in the good responders was offset by a significant increase in microarousals in the nonresponders (112±38 vs 175±104, p≤0.02). The quality of sleep, while being marginally improved in patients in whom surgery was successful, was globally altered in patients with failure of surgery with a reduced total sleep time (445±88 vs 376±41 min, p≤0.03) and a significant increase in the number of changes of sleep stage (312±85 vs 464±245, p≤0.008).

Additional Treatments Required After UPPP

Treatment supplementary to UPPP was required in nine patients (45%). Continuous positive airway pressure (CPAP) was instituted in four patients and refused by two patients. Positional treatment was initiated in two patients. One patient received both CPAP and maxillofacial surgery.

Anatomic Modifications of the UA Induced by Surgery

Minimal Oropharyngeal CSA—Size, Site, and Relation to Outcome: There was no significant increase in the minimal CSA of the oropharynx as a result of surgery for the patients as a whole (79±36 vs 94±68 mm²) (Table 2). In patients in whom surgery was successful, there was a trend toward an increase in the size of the minimal CSA (74±24 vs 133±82 mm², p=0.09) that was not found in patients in whom surgery was unsuccessful. In addition, the change in size of the oropharynx was correlated with the change in AHI (r=-0.54, p<0.02, Fig 1). The minimal CSAs of both nasopharynx and hypopharynx remained unchanged.

The site of maximal UA narrowing before UPPP was located in most patients at 10 mm (10 patients) and 20 mm (seven patients) below the hard palate. After UPPP, there was a displacement superiorly of the site of maximal narrowing (Fig 2) to the level of the hard palate (eight patients) and 10 mm below it (seven patients). This upstream displacement of the site of pharyngeal narrowing as a result of surgery was seen in both good responders and nonresponders.

Measurements of the dimensions of the UA using CT slices every 10 mm confirmed the existence of an enlargement in the oropharyngeal airway in good

### Table 1—Sleep and Anthropometric Characteristics Before and After UPPP*

<table>
<thead>
<tr>
<th>Overall Group (n=20)</th>
<th>Good Responders (n=7)</th>
<th>Nonresponders (n=13)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before After p Value</td>
<td>Before After p Value</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>26±4 NS 26±4</td>
<td>25±2 NS 25±3</td>
</tr>
<tr>
<td>AHI, events/h</td>
<td>14±13 18±16</td>
<td>13±12 ≤0.01 4.5±6.5</td>
</tr>
<tr>
<td>TST, min</td>
<td>428±78 NS 391±41</td>
<td>393±32 NS 421±21</td>
</tr>
<tr>
<td>Stage 1-2 sleep, % TST</td>
<td>75±10 ≤0.04 69±11</td>
<td>75±6 ≤0.07 69±14</td>
</tr>
<tr>
<td>Stage 3-4 sleep, % TST</td>
<td>4±5 ≤0.01 7±5</td>
<td>5±5 ≤0.06 8±7</td>
</tr>
<tr>
<td>REM sleep, % TST</td>
<td>17.1±7.0 NS 19.1±7.4</td>
<td>19.2±2.8 NS 18.7±6</td>
</tr>
<tr>
<td>Microarousals, No.</td>
<td>114±36 NS 141±100</td>
<td>117±37 NS 68±22</td>
</tr>
<tr>
<td>Sleep stage changes, No.</td>
<td>333±90 NS 385±236</td>
<td>349±110 NS 215±79</td>
</tr>
<tr>
<td>Mean SaO₂ %</td>
<td>94±2 ≤0.05 95±2</td>
<td>94±2 NS 95±1</td>
</tr>
<tr>
<td>Minimal SaO₂ %</td>
<td>83±11 NS 86±6</td>
<td>86±5 NS 88±5</td>
</tr>
</tbody>
</table>

*TST=total sleep time; REM=rapid eye movement; SaO₂=arterial oxygen saturation; NS=not significant. Microarousals were scored according to the rules of the American Sleep Disorders Association (ASDA).27
Correlation between AHI before-after UPPP and changes in minimal CSA at the oropharyngeal level after surgery. The changes in the oropharynx after UPPP were correlated with the changes in AHI (r = -0.54, p<0.02). In good responders, an increase in minimal CSA was often noted (dashed area). This was not found in most nonresponders.
group. Moreover Miljeteig and colleagues\textsuperscript{16} showed that for their patients, this subjective improvement was not accompanied by an objective decrease in the number of snoring episodes or a significant reduction in intensity of snoring. This discrepancy between objective and subjective assessments of surgical outcome may be explained by the fact that UPPP modifies the snoring frequency making it less noticeable to the spouse.

Our study is one of the few to report details of the microstructure of sleep before and after UPPP. As expected, improvement in sleep architecture occurred only in the good responders and consisted of a reduction in the number of microarousals and sleep stage changes and an increase in the amount of slow-wave sleep. In support of this observation, Boudewyns et al.\textsuperscript{17} in a retrospective study of 10 nonapneic snorers, demonstrated an improvement in the arousal index after UPPP. Conversely, the number of microarousals and the total sleep time worsened after surgery in nonresponders explaining a persistent daytime sleepiness.

The incidence of complications in our study is similar to that reported in the literature.\textsuperscript{18,19} There were no major complications. It was not judged necessary to treat the patients with CPAP prior to surgery as their OSA was mild to moderate rather than severe.

**Additional Treatments Required After UPPP**

Only 7 of 20 patients responded favorably to the surgical procedure and some of them actually got worse. As a consequence, 45\% of the patients in our study required additional treatment after UPPP. This is similar to the findings of Larsson and colleagues\textsuperscript{20} in whose study 21 of the 48 patients followed up at 46 months went on to have additional treatment. In that study, 11 patients received CPAP, 2 underwent phase I maxillofacial surgery, and 1 patient had nasopharyngeal intubation. One patient considered to have severe apnea refused all further treatment. Among the more mild OSA sufferers, three were not given further treatment, and six were advised to lose weight. The high proportion of patients requiring further treatment after UPPP...
Figure 3. Measurements of the dimensions of the UA using CT slices every 10 mm in good and nonresponders before and after surgery. Note an enlargement in the oropharyngeal airway in good responders. This is not seen in the nonresponders. There is a significant difference (p<0.02) in the size of the oropharyngeal area before and after UPPP at the level of 20 mm below the soft palate in good responders. Level 0=hard palate; asterisk=significant difference.

Figure 4. Measurements of the dimensions of the UA using CT slices every 10 mm in good and nonresponders after surgery. Note an enlargement in the oropharyngeal airway in good responders. This is not seen in the nonresponders. There is a significant difference (p<0.04) in the size of the oropharyngeal area at the level of 10 mm below the soft palate between good and nonresponders. Level 0=hard palate; asterisk=significant difference.

Anatomic Modification of the UA Induced by UPPP

Minimal Oropharyngeal Area: There was no change in the minimal CSA of the oropharynx for the population as a whole as a result of UPPP (79±36 vs 94±68 mm²). This is in agreement with Polo and colleagues who used CT to examine the UA preoperatively and postoperatively. In contrast, Shepard and Thawley reported an increase in the minimal oropharyngeal CSA that was not, however, accompanied by a change in its mean value. The conflicting findings of these studies might be expected in relation to their small sample sizes.

Minimal Pharyngeal CSA and UPPP Outcome: In the present study, the changes in the oropharyngeal diameter were correlated with the change in AHI (r = −0.54, p<0.02), signaling the importance of the change in oropharyngeal caliber in determining surgical outcome. There was no significant alteration in the hypopharyngeal diameter. In the literature, there is only one study (to our knowledge) looking at the caliber of the UA using imaging before and after surgery. The authors concluded that a significant reduction in UA CSA occurred after UPPP in nonresponders at the level of the hard palate. Additionally, surgical success seemed to be linked to an increase in the CSA of the UA at the level of the oropharynx.

If the hypothesis is correct regarding the predominant role of the oropharynx, then the site of UA collapse during sleep in patients who failed to respond to UPPP ought to occur at this level. In effect, this has been found to be the case in several studies.
comprising small numbers of patients evaluating the site of airway collapse postoperatively during sleep. In 84% of 43 patients, the collapse of the UA remained after UPPP at the level of the oropharynx (Table 3).

Abnormal increase in pharyngeal resistance during sleep may also play a part in the response to surgery. Change in pharyngeal resistance could occur secondary to changes in pharyngeal compliance or the activity or efficacy of pharyngeal muscular contraction. Thus, although the role of reducing oropharyngeal narrowing is clear in ensuring a good surgical outcome, an abnormal level of pharyngeal resistance may persist even after UA enlargement.

**Soft Palate Length and Morphology—Soft Palate Length:** The soft palate is important in causing collapse of the pharyngeal airway during sleep. In our study, there was a large reduction in PNS-P after UPPP (40±6 vs 29±5; p=0.0006). The greater reduction in soft palate length seen in the good responders (39%) compared with the nonresponders (22%) would not seem to be enough to explain the different surgical outcomes. Although Kimmelman and colleagues claimed that more extensive resection of the palate increased the success rate of UPPP, other authors argued that this resulted in greater morbidity in the absence of increased efficacy. In the study of Kimmelman et al, it should be noted that only the apnea index was measured preoperatively and postoperatively, and hypopneas were not taken into account.

**Soft Palate Position and Relationship With Minimal CSA:** There was an increase in the thickness of the soft palate in anteroposterior dimension

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**Table 3—Evaluation During the Wake Period of UA Narrowing (Top) and During Sleep of UA Collapse (Bottom) After UPPP**

<table>
<thead>
<tr>
<th>First Author, yr</th>
<th>Methods of UA Evaluation</th>
<th>No. of Patients</th>
<th>Predominant OP Narrowing After UPPP (No. of Patients)</th>
<th>OP Collapse After UPPP (No. of Patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shepard and Thawley, 1989</td>
<td>Pharyngeal CT scan</td>
<td>23</td>
<td>20/23</td>
<td>...</td>
</tr>
<tr>
<td>Polo et al, 1989</td>
<td>Pharyngeal CT scan</td>
<td>4</td>
<td>4/4</td>
<td>...</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>27</td>
<td>24/27 (89%)</td>
<td>...</td>
</tr>
<tr>
<td>Shepard and Thawley, 1990</td>
<td>Measurements of UA pressure</td>
<td>6</td>
<td>...</td>
<td>4/6</td>
</tr>
<tr>
<td>Hudgel et al, 1991</td>
<td>Measurements of UA pressure</td>
<td>11</td>
<td>...</td>
<td>11/11</td>
</tr>
<tr>
<td>Metes et al, 1991</td>
<td>Measurements of UA pressure</td>
<td>8</td>
<td>...</td>
<td>6/8</td>
</tr>
<tr>
<td>Skatvedt, 1992</td>
<td>Measurements of UA pressure</td>
<td>7</td>
<td>...</td>
<td>7/7</td>
</tr>
<tr>
<td>Woodson and Wooten, 1994</td>
<td>Manometry-videoendoscopy</td>
<td>11</td>
<td>...</td>
<td>8/11</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>43</td>
<td>...</td>
<td>36/43 (84%)</td>
</tr>
</tbody>
</table>

*OP=oropharyngeal. During the wake period, OP was the predominant site of UA narrowing (89% of the patients). During sleep in patients with failed UPPP, pharyngeal collapse occurred at oropharyngeal level in 84% of the cases.
was bulged palate after surgery. The occurrence of the CSA oropharyngeal retropalatal region was also found by Shepard and Thawley. In our study, the increase in the thickness of the palate occurred in a similar fashion in good responders and nonresponders. The complex relationship between the palate and the posterior pharyngeal wall and the tongue would appear to determine the surgical outcome. If the palate tended to have its long axis parallel to the posterior pharyngeal wall and the end resting on the base of the tongue, there was an enlargement of the pharyngeal lumen, and UPPP was successful despite the widening of the soft palate. In contrast, if the palate bulged out into the pharyngeal lumen reducing the retropalatal space, UPPP failed (Fig 5).

Welch et al24 have very recently used MRI in five patients to assess UA anatomic changes after UPPP. These data demonstrated that the airway decreased by 28±27% in the remaining retropalatal region due to an increasing soft palate width of 25±37%. These preliminary data are in accordance with our study and show the necessity of designing surgical techniques to enlarge the retropalatal area. Transpalatal advancement pharyngoplasty25 has been proposed to enlarge the velopharynx and retropalatal segment by excising the posterior hard palate and advancing the soft palate anteriorly into the defect. Woodson26 has suggested that such a procedure increases retropalatal size and decreases collapsibility in the immediate postoperative period. Randomized studies are needed to compare different surgical techniques in terms of long-term outcome and UA modification.

**CONCLUSION**

Only 7 of our 20 patients (35%) responded favorably to the surgical procedure, and some of them actually got worse. UPPP was effective in eliminating snoring in only half of them. In nonresponders, the number of microarousals and the total sleep time worsened after surgery. The change in minimal oropharyngeal CSA was correlated with surgical outcome with good responders showing an enlargement of the oropharynx. The persistence of oropharyngeal narrowing after UPPP was linked to an increase in the thickness of the soft palate that occurred in both good responders and nonresponders. The orientation of the palate after surgery was critical in surgical outcome in that UPPP was more likely to fail in patients in whom the residual palate bulged out into the pharyngeal lumen, thus reducing the CSA of the airway. This complex change in airway morphology as a result of UPPP may explain why there are no strict criteria that can be used to predict outcome after such surgery.

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