The Laryngoscope
Lippincott Williams & Wilkins, Inc.

Lateral Pharyngoplasty: A New Treatment for Obstructive Sleep Apnea Hypopnea Syndrome

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Objective: **The role of the lateral pharyngeal muscular walls in the pathogenesis of obstructive sleep apnea hypopnea syndrome (OSAHS) is crucial. My objective is to describe a surgical procedure for splinting the lateral pharyngeal walls in patients with OSAHS and report the initial results.** ***Study Design:*** **Prospective randomized pilot study performed in an academic tertiary center as part of a doctoral thesis.** ***Patients and Methods:*** **Ten adults with OSAHS, originally selected for treatment with uvulopalatopharyngoplasty, underwent the lateral pharyngoplasty procedure, which consists of a microdissection of the superior pharyngeal constrictor muscle within the tonsillar fossa, sectioning of this muscle, and suturing of the created laterally based flap of that muscle to the same-side palatoglossus muscle. In addition, a palatopharyngeal Z-plasty is performed to prevent retropalatal collapse.** ***Results:*** **Lateral pharyngoplasty improved sleep and daytime symptoms related to OSAHS. After at least 6 months of follow-up, the median apnea-hypopnea index decreased from 41.2 to 9.5 ($P = .009$) and the median total sleep time spent in rapid eye movement (REM) sleep and in stages 3 plus 4 non-REM sleep increased from 14.1% to 22.1% ($P = .059$) and from 5.3% to 16.3% ($P = .037$), respectively. Also, there was statistically significant improvement in snoring, daytime sleepiness, and overall impact of the disease on quality of life. Swallowing disturbances lasted a median of 14.5 days after the surgeries, and all patients returned to normal subjective swallowing function in the follow-up.** ***Conclusions:*** **Lateral pharyngoplasty is a safe and straightforward approach to lateral pharyngeal muscles and has produced appreciable benefits for OSAHS patients.** ***Key Words:*** **Obstructive sleep apnea, lateral pharyngoplasty, pharyngeal muscular walls.**
Laryngoscope, **113:1961–1968, 2003**

INTRODUCTION

The pathogenesis of upper airway occlusions in obstructive sleep apnea hypopnea syndrome (OSAHS) is not fully understood. Structural narrowing of the pharyngeal airway increases the inspiratory pressure within this region and is considered to be a contributing factor to the syndrome. Apneas and hypopneas occur when the inspiratory transpharyngeal pressure exceeds the pharyngeal dilating muscle action.[1] Most of the surgeries already proposed for treating OSAHS aim to decrease the inspiratory upper airway pressure by enlarging the pharyngeal lumen with removal of soft tissues or stretching of its surrounding structures.[2] However, if enlargement of the pharynx were the key step for treating this disorder, we would find upper airway differences between OSAHS patients and anthropometric matched control subjects without OSAHS. Furthermore, we should be able to establish a relationship between airway dimensions and polysomnographic parameters that are considered to be hallmarks of the severity of the disease. Unfortunately, some studies contradict this current concept of lack of pharyngeal space that causes the OSAHS in adults.[3–6] In fact, some authors have experimentally proved that the upper airway collapsibility can be reduced without changing its cross-sectional areas but rather by increasing its longitudinal tension.[7]

The importance of the lateral pharyngeal muscular walls (LPW) in the pathogenesis of OSAHS has been demonstrated in a series of articles with imaging examinations.[8–10] Except for controlling the patients for body mass index (BMI) and neck circumference, LPW narrowing appears to be the sole independent oropharyngeal finding that poses a risk factor for OSAHS in men.[11] Also, it has been recently shown that there is not a specific, effective surgical procedure for treating the usual lateral to medial pharyngeal wall collapse present in OSAHS patients during the Müller maneuver.[12]

The purpose of this article is to describe the first surgical technique specifically designed to splint the LPW in patients with OSAHS and report its initial results. I called this technique lateral pharyngoplasty.

PATIENTS AND METHODS

In 1999, as a doctoral thesis project at the University of São Paulo Medical School, I proposed the lateral pharyngoplasty as a treatment for patients with OSAHS. Since then, we have been conducting a randomized controlled trial study with patients originally selected for uvulopalatopharyngoplasty who are subse-

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Editor's Note: This Manuscript was accepted for publication June 9, 2003.

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quently assigned into two groups: in the first group, we performed the uvulopalatopharyngoplasty and, in the second group, we performed the lateral pharyngoplasty. The subjects included in this article are the first 10 consecutive patients from the second group who completed our protocol. The study was approved by the ethics committee of our institution, and each patient signed a consent form that outlined the objectives of the research and experimental risks.

We included in this study individuals who habitually snored and who were over 18 years of age with an apnea-hypopnea index (AHI, number of apneic plus hypopneic events per hour of sleep) greater than 10 (to avoid misdiagnosis of OSAHS) who failed to tolerate or refused therapy with continuous positive airway pressure (CPAP). All subjects failed to improve from OSAHS with positional therapy, and clinical measurements for weight loss were also ineffective. The patients were selected for pharyngeal surgery on the basis of the presence of "excessive" soft tissue structures within the oropharynx on physical examination, with the mouth opened maximally and the tongue remaining in the mouth in a relaxed position. In addition, the inclusion criteria included at least two of the following three findings: 1) lateral peritonsillar narrowing (defined as impingement by the peritonsillar tissues of greater than 25% of the pharyngeal space, i.e., the internal distance of the mandible measured at the midcranial-caudal level of the tonsillar fossa); 2) tonsillar enlargement (defined as lateral impingement by the tonsils of greater than 50% of the distance between left and right posterior tonsillar pillars), and 3) a low-lying soft palate (avoiding the visualization of the posterior pharyngeal wall). In addition to these findings, the subjects had to present, on fiberoptic pharyngoscopy, narrowing or collapse in the retropalatal region without narrowing in the hypopharynx, both at rest and during the Müller maneuver.

We excluded from this study patients over 130 kg (computed tomography table limit; an exam that we used for measuring airway dimensions, which data are not included in this article and morbidly obese patients. Other exclusionary criteria were uncontrolled hypothyroidism or hypothyroidism that has been under control for less than 1 year and gross maxillary or mandibular deformities.

Clinical Evaluation

The sample comprised 8 men and 2 women, with a median age of 50 (range 43–59) years. They were operated on from September 1999 to July 2000. Each patient was evaluated preoperatively and at least 6 months after the surgery. At these periods, we assessed the BMI (the weight in kilograms divided by the square of the height in meters), the neck circumference (measured at the level of the thyrohyoid membrane, in centimeters), the Epworth sleepiness scale scores, and the results from a doctor-administered questionnaire with scales ranging from 1 (irrelevant) to 10 (severely affected/debilitating) to evaluate the following variables: snoring, daytime sleepiness, morning headaches, and the overall impact of the disease on quality of life. A similar questionnaire was used to assess the postoperative pain, and we recorded the number of days it took the patients to return to normal nourishing after the surgeries.

Polysomnography

All subjects underwent overnight polysomnography in a sleep center in a standard fashion with a test time of 7 to 8 hours both preoperatively and at least 6 months after the surgeries. The parameters monitored included electroencephalogram (C4-A1, C3-A2, O2-A1 of the international 10–20 electrode placement system), right and left electroculograms, chin and tibial electromyograms, electrocardiogram, oronasal airflow (with thermistors), thoracic-abdominal movements (noncalibrated inductive plethysmography), oxygen saturation (pulse oximetry), recording of body position, and snoring intensity. The polysomnograms were scored according to standard criteria[13] and analyzed by physicians trained in polysomnography reading who were unaware of the type of surgery performed in each case. The definitions of apneas and hypopneas used were those latest recommended by the American Academy of Sleep Medicine[14]: apnea, absence of airflow for at least 10s; hypopnea, reduction (for at least 10 seconds) by more than 50% of the basal ventilatory value or a reduction of 50% or less that is associated with a decrease in oxyhemoglobin saturation above 3% or with an arousal. In addition to obstructive and mixed apneas, the few central apneas present were summed to obtain the total number of apneas. The variables evaluated included AHI, the apnea index (AI, number of apneic events per hour of sleep), the oxyhemoglobin desaturation nadir (LSAT), the percentage of total sleep time spent in stages 3 plus 4 nonrapid eye movement (NREM) sleep and the percentage of total sleep time spent in rapid eye movement (REM) sleep.

Surgical Procedure

All the procedures were performed by the same surgeon, with patients under general anesthesia, using a McIvor mouth gag, both grooved and nongrooved, to give adequate exposure. Patients were orally intubated and had their heads extended. I used 4.0 Vicryl for all sutures.

The lateral pharyngoplasty initiates with a bilateral tonsillectomy. If that had been previously performed, I proposed to remove the tonsillar fossa mucosa until I could identify the palatoglossus and palatopharyngeus muscles (Fig. 1). Next, with the operative microscope fitted with a 300 mm object lens and using $\times 10$ and $\times 16$ magnifications, I undermined and elevated the superior pharyngeal constrictor (SPC) muscle within the tonsillar fossa as far as its glossopharyngeal part (Fig. 2). Because of our initial uncertainty regarding the postoperative swallowing function, in the first four cases, I limited this dissection inferiorly to a region immediately cranial to the tongue origin of the SPC muscle. In the next cases, however, the full proposed dissection was accomplished. Once detached from the lateropharyngeal space, I sectioned the SPC muscle in a cranial to caudal direction, resulting in two muscle flaps: one medially based flap that was not further manipulated and one laterally based flap that was sutured anteriorly to the same-side palatoglossus muscle with three separate stitches (Fig. 3). Then, I made a half thickness incision

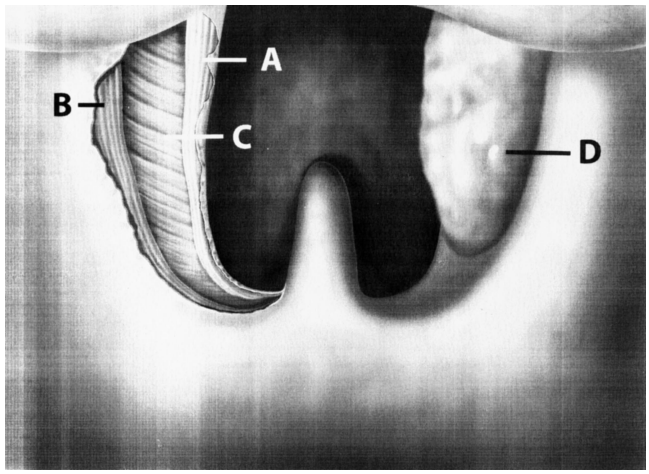

Fig. 1. Operative view after left tonsillectomy. **(A)** palatopharyngeus muscle, **(B)** palatoglossus muscle, **(C)** superior pharyngeal constrictor muscle and **(D)** right tonsil.

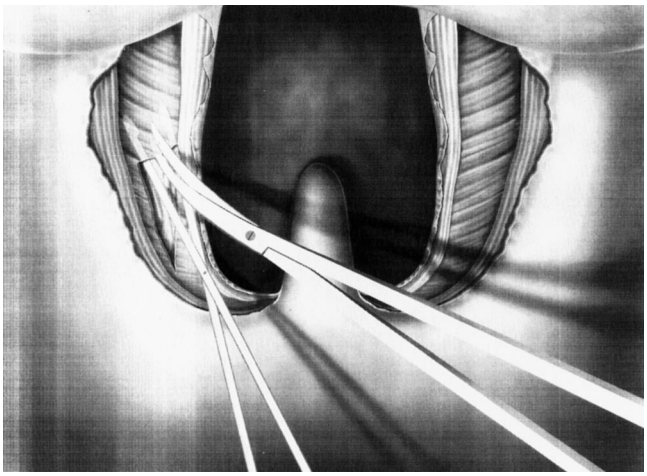

Fig. 2. Elevation and section of the left superior pharyngeal constrictor muscle.

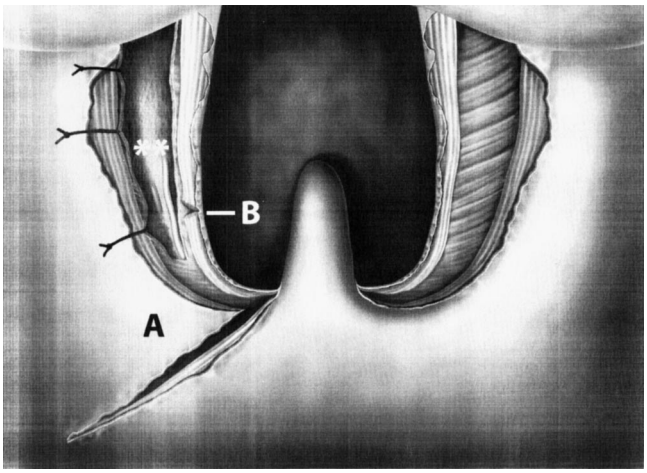

Fig. 4. **(A)** palatine flap, **(B)** section of the palatopharyngeus muscle, (**) external palatine vein (right) and ascending palatine artery (left).

over the oral face of the soft palate, straight from the lateral base of the uvula extending laterally and superiorly to a point approximately 0.5 cm proximal to the soft palate lateral margin and within a height corresponding to that reached by the lateral superior traction (in a 45 degree direction) of the upper part of the palatopharyngeus muscle, creating, then, a palatine laterally based flap (Fig. 4). Next, I made a transverse subtotal section of the palatopharyngeus muscle in its superior part, creating a superior and an inferior flap (Fig. 4). This superior flap and the palatine flap were sutured in a Z-plasty fashion, and they covered the superior part of the tonsillar fossa, as others have described[15] (Fig. 5). The inferior part of the tonsillar fossa was then closed by suturing the inferior palatopharyngeus muscle flap to the anterior pillar. Every step was then repeated on the opposite side and, finally, the distal one third of the uvula was removed and sutured for hemostasis (Fig. 6). The total volume of the tissues removed (tonsils plus part of the uvula) was assessed using Archimed's principle (the immersion of the tissues displaces a volume of liquid that equals the volume of the tissues).

Postoperative Period

Extubation was delayed until the patients were fully awake. However, I prefer to keep patients supported with an artificial oropharyngeal airway, such as a large Guedel cannula (Fig. 7), within the initial minutes after extubation to guarantee upper airway opening. The patients stay monitored in the recovery room for 2 to 4 hours before going to the ward. Hospital stay usually lasts 2 days, and discharge criteria include pain control and adequate oral intake of fluids. None of the subjects had to be fed by tubes. Antibiotic coverage is used for 7 days (amoxicillin, 500 mg orally 3 times a day or cephalexin, 500 mg orally 4 times a day), and steroids are used during hospitalization (hydrocortisone, 200 mg intravenously every 8 hours) followed by nonsteroidal antiinflammatory drugs (sodium diclofenac, 50 mg orally 3 times daily for 5 days). Despite the interference that nonsteroidal antiinflammatory drugs produce in platelet function, we have not seen bleeding with the routine use of these drugs after uvulopalatopharyngoplasties, perhaps because of the local hemostasis provided by the surgical sutures. In addition, we use painkillers (dypiron, 500 mg orally every 6 hours and, if needed, tramadol hydrochloride, 75 mg orally every 8 hours) and topical anesthetics (benzocaine 0.4%, 4 sprays in the throat up to 4 times daily), usually for 10 days after the procedures. The patients also receive

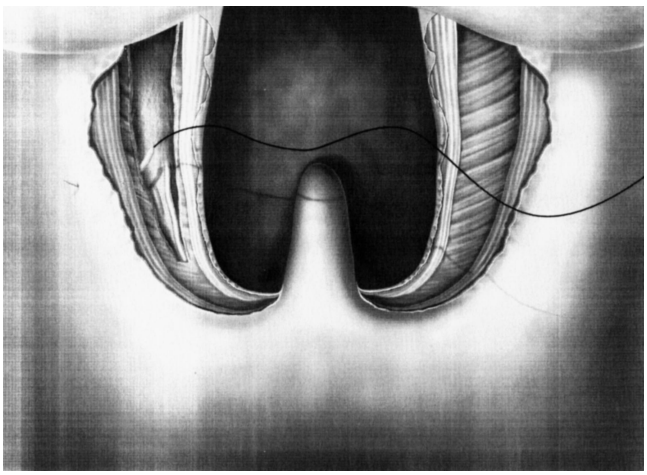

Fig. 3. Anterior suture of the superior pharyngeal constrictor muscle (lateral flap) to the palatoglossus muscle.

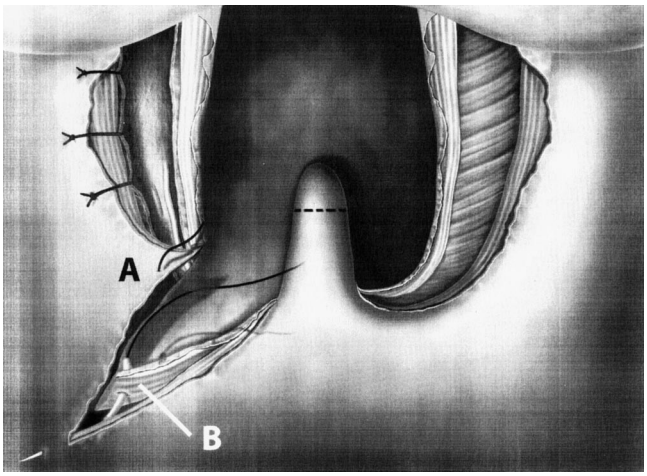

Fig. 5. Z-plasty covering the superior part of the tonsillar fossa. **(A)** palatine flap, **(B)** upper part of the palatopharyngeus muscle. Incision to remove part of the uvula (dashed line).

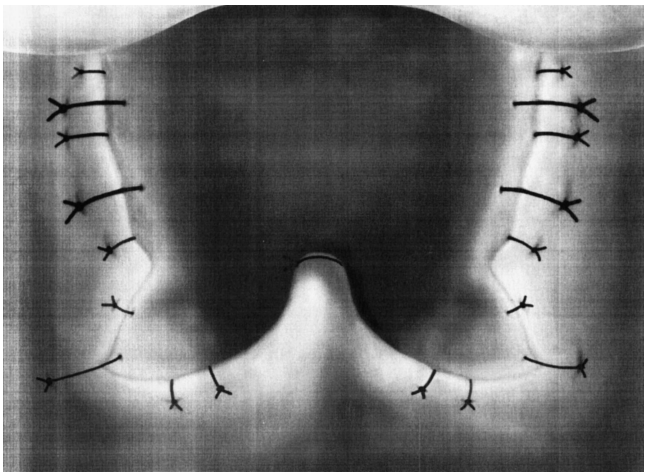

Fig. 6. Final aspect of the lateral pharyngoplasty.

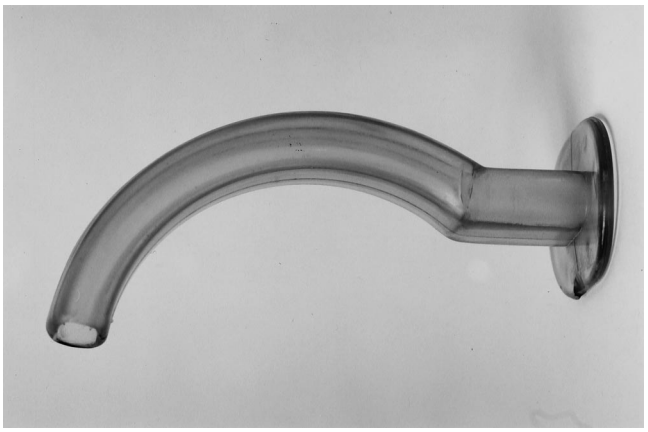

Fig. 7. Guedel-type oropharyngeal airway.

gastric acid antisecretory agents (omeprazole, 20 mg orally daily for 14 days).

Nasal Obstruction

Patients with daily nasal obstruction and anatomic features such as deviated septum and turbinate hypertrophy which did not improve with medical therapy were also treated with nasal surgery. In this reported group, three patients underwent septoplasty at the same time of lateral pharyngoplasty.

Statistical Analyses

The nonparametric Wilcoxon test was used to compare pre- and postoperative data because of the small number of patients in this report and also because not every variable studied has a normal distribution. In the Results, values for the medians are followed by the respective quartile ranges. A P value less than .05 was considered significant.

RESULTS

All 10 patients had their tonsils present at the time of the lateral pharyngoplasty. The pre- and postoperative weight, BMI, and neck circumference are shown in Table I. The follow-up period ranged from 6 to 12 (median 8) months. There was a statistically significant reduction in the BMI but not in the neck circumference after the surgeries. The median Epworth sleepiness scale value significantly improved from 13 (12, quartile range) to 5 (4) (P = .005), as well as the self-reported symptoms of snoring (median decreased from 10 to 3.5, P = .005), daytime sleepiness (median decreased from 5 to 1, P = .011), and overall impact of the disease on quality of life (median decreased from 5.5 to 1.5, P = .018). Also, we noted a tendency toward improvement in morning headaches after the surgeries (median value in the questionnaire was 1 both pre- and postoperative, but the quartile range decreased from 5 to 2, P = .068).

TABLE I.
Pre- and Postoperative Anthropometric Data and Diet Delay.

Patient	Follow-up (months)	Weight (kg)		BMI (kg/m^2)		Neck Circumference (cm)		Diet Delay (days)†
		Pre	Post	Pre	Post	Pre	Post	
1	8	95	89	31.7	29.7	46.0	44.0	12
2	8	82	82	29.4	29.4	44.0	44.0	8
3	12	69	65	28.0	26.4	42.0	41.0	12
4	6	79	75	28.3	26.9	41.0	41.0	25
5	6	84	74	30.9	27.2	40.0	40.0	10
6	9	78	72	30.5	28.1	42.0	41.0	10
7	7	70	72	27.7	28.5	39.0	39.0	17
8	10	87	87	28.1	28.1	43.5	44.5	20
9	8	93	89	30.4	29.1	45.0	45.0	20
10	8	96	81	33.2	28.0	53.0	46.5	70
Mean	8.2	83.3	78.6	29.8	28.1	43.6	42.6	20.4
Median	8	83	78	29.9	28.1	42.8	42.5	14.5
Quartile range	1.5	15	15	2.8	1.9	4.0	3.5	10
P value	–	0.017*		0.017*		0.138		–

* Postoperative significant difference ($P < .05$).
† Time required for patients to return to full preoperative diet after the surgeries.
BMI = body mass index.

On the 10-point scale, the patients usually reported moderate pain postoperatively, giving a median value of 4.5 (4.0). After 10 days after the procedures, analgesics were usually no longer required. The median time it took for patients to return to normal nourishing was 14.5 (10) days (Table I). After the pain was gone, swallowing dysfunction was responsible for this delay, especially for solid, dry foods. We detected no alterations in tongue mobility, and the full recovery time of the pharyngeal swallow varied greatly in this group (ranging from 8–70 days). Also, many patients avoided solid foods postoperatively, reporting being afraid of loosing their stitches.

The polysomnographic data are shown in Table II. The group showed a statistically significant improvement in the AHI, in the AI, and in the percentage of total sleep time spent in stages 3 plus 4 NREM sleep. Also, we noted a tendency toward an increase in the total REM sleep ($P = .059$) and no significant changes in the LSAT ($P = .359$). Only one case showed a worsening of the AHI (patient 4). In this case, we performed a limited dissection of the SPC muscle, and the patient reported no clinical benefit after the procedure. In the follow-up, he developed clear symptoms of a major depressive episode and was sent to psychiatric management. The median tissue volume removed in the surgeries was 5.0 (2.9) mL, and the bulk of it corresponded to the tonsil volume.

Extubation occurred uneventfully in all cases, and we did not observe immediate or delayed complications such as bleeding, abscesses, nasopharyngeal stenosis, and alterations in speech. Wound dehiscence typically occurred in the region of the caudal stitch of the tonsillar fossa some days after the surgeries and did not demand any special attention. Patient 8 referred to some episodes of mild oronasal reflux of liquids during swallowing that resolved after 6 weeks after the surgery. There was no case of permanent palatal incompetence in this series. Patient 9 reported mild taste loss for chocolate, which recovered completely after 6 months after the procedure.

DISCUSSION

We call the respiratory sleep disturbances in adults with OSAHS obstructive and not peripheral events. This is a very adequate terminology because this avoids the misconception of considering this disease the single result of anatomical abnormalities. In fact, pharyngeal airflow resistance is higher in patients with OSAHS than in weight-matched control subjects without OSAHS even though there is no difference in pharyngeal cross-sectional areas between them, suggesting the existence of a functional impairment of the pharynx.[3] Also, it has been demonstrated in rabbits that the upper airway patency depends on neuromuscular mechanisms.[16] In addition, several authors were unable to find correlations between polysomnographic parameters of patients with OSAHS and their pharyngeal cross-sectional areas and volumes.[4–6] It seems that, to keep the upper airway opened, pharyngeal space means very little without control.

The goal of a surgical treatment for OSAHS is to reduce the upper airway collapsibility. The pharynx of these patients is more collapsible, both structurally[17] and functionally.[8] The surgical techniques for treating OSAHS, apart from tracheotomy, reduce upper airway narrowing by either removing soft tissues from the pharyngeal lumen or mobilizing the surrounding bones to enlarge the airway.[2] Although current surgeries are designed to modify either the retropalatal or retroglossal region of the pharyngeal airway, there are no universally accepted and validated clinical methods for defining the specific site of obstruction within the pharynx.[2] Actually, because of the complex pharyngeal innervation, it seems unlikely that

TABLE II.
Pre- and Postoperative Polysomnographic Data.

	AHI		AI		LSAT (%)		Stages III + IV (%)		REM sleep (%)	
Patient	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	41.9	9.3	7.9	3.2	68.0	78.0	0.0	23.2	10.7	3.8
2	29.9	15.6	6.5	3.6	88.0	77.0	14.8	26.6	15.7	19.4
3	43.6	38.7	30.4	23.0	58.0	41.5	6.8	9.5	6.6	25.8
4	17.1	26.2	7.2	0.6	81.0	79.0	17.6	7.3	16.4	12.5
5	63.9	8.5	60.3	8.0	63.0	82.0	0.0	18.8	11.9	19.9
6	78.7	8.7	37.7	0.6	81.0	91.0	3.7	15.3	18.4	14.1
7	33.8	9.6	3.2	3.1	86.0	79.0	16.0	28.9	15.4	24.2
8	40.4	8.3	8.6	2.9	76.0	83.0	0.4	9.6	19.1	26.6
9	14.1	0.4	3.5	0.0	82.0	90.0	14.9	3.4	12.8	25.2
10	94.5	26.8	58.5	2.7	50.0	77.0	0.0	17.2	9.8	24.7
Mean	45.8	15.2	22.4	4.8	73.3	77.8	7.4	16.0	13.7	19.6
Median	41.2	9.5	8.3	3.0	78.5	79.0	5.3	16.3	14.1	22.1
Quartile range	34.0	17.7	31.2	3.0	19.0	6.0	14.9	13.7	5.7	11.1
P value	.009*		.005*		.359		.037*		.059	

* Postoperative significant difference ($P < .05$).
AHI = apnea-hypopnea index; AI = apnea index; LSAT = oxyhemoglobin desaturation nadir; REM = rapid eye movement.

OSAHS is caused by a focal functional pharyngeal impairment. This impression is corroborated by imaging examination findings.[18–20] Therefore, new surgeries for OSAHS should address the whole collapsible pharynx.

The LPW in OSAHS patients are more distensible and collapsible than normal when pressured by the airflow.[8] Also, being thicker than normal, they are the predominant anatomic factor causing airway narrowing in apneic subjects.[9] Interestingly, when one progressively increases the nasal CPAP pressure in normal subjects, one can observe enlargement of the airway, mainly in its lateral dimensions, with reduction of lateral pharyngeal muscular wall thickness and, perhaps more interestingly, with minimal changes in the soft palate and tongue.[10] In fact, the genioglossus muscle abnormalities found in OSAHS patients are likely a consequence and not a cause of this disease because both structural and functional changes of that muscle in apneic subjects are corrected by CPAP.[21] However, the cause of the OSAHS, which is not known, is not corrected by CPAP. Therefore, it is a valid assumption that treating retroglossal collapsibility does not necessarily mean treating the tongue, but rather dealing with the lateral pharyngeal muscular retroglossal wall. It seems that problems in this region are likely to increase airway inspiratory pressures and, therefore, affect the stability of the tongue. In addition, changing the pharyngeal muscular wall properties is more effective in reducing upper airway collapsibility than anterior tongue displacement.[7]

How can surgery change the pharyngeal wall properties? One way is through tensioning the pharyngeal walls through repositioning the surrounding bone. Recently, it has been shown that the maxillary and mandibular advancement osteotomy gives support to the lateral pharyngeal walls through the constrictor muscles and that this could explain the success of this treatment despite the lack of relationship between airway lumen changes with this surgery and objective polysomnographic outcome.[22] In fact, considering the stretch needed to splint the pharyngeal walls, this concept might also explain the existence of a significant relationship between clinical outcome and the amount of bone advancement in that surgery.[22] Another way of changing pharyngeal structural properties is by approaching its lateral muscular walls. To my knowledge, the lateral pharyngoplasty is the first surgical technique that directly manipulates the superior constrictor pharyngeal muscle to splint the upper airway in apneic subjects.

As other authors have pointed out,[23] randomized controlled trials are the gold standard of clinical research. Because uvulopalatopharyngoplasty is the most common surgical procedure for treating OSAHS in adults, it seems reasonable that this surgery is included in controlled trials designed to study new surgical treatments for OSAHS. This is the reason why lateral pharyngoplasty was performed in a group of patients selected for uvulopalatopharyngoplasty according to standard criteria.[2]

As a rule, patients lost weight immediately after lateral pharyngoplasty, and they did not regain it completely in the follow-up (Table I). It is known that obesity, in some persons, may be a result of the respiratory sleep disorder instead of a contributing cause.[24] Currently, we have been trying to clarify whether this weight-loss effect of the surgery occurs specifically in obese persons (BMI over 30) or not. Interestingly, there was not a noticeable change in the neck circumference in this group after weight loss, and this might suggest that fat deposition within parapharyngeal spaces does not play a major role in collapsing the airway.[9]

Table II discloses the objective effects of lateral pharyngoplasty on sleep. The median AHI decreased from 41.2 (34.0) to 9.5 (17.7). Sixty percent of the patients had a reduction of the preoperative AHI over 50%, with a postoperative AHI of less than 20. Although useful, this so-called success definition[25] does not take into account relevant facets of this disease. Today, it is recognized that, because of the individual variability of the AHI, other clinical and polysomnographic parameters must be evaluated in these patients.[26] Also, the AHI benchmark of 20, above which AHI is associated with increased mortality, was actually set for the AI,[27] and data relating the enlarged definition of hypopneas used here[14] with mortality is still lacking. It should be pointed out that, among the four initial cases of lateral pharyngoplasty, three were failures according to the AHI criterion. In these cases, I accomplished a limited dissection of the SPC muscle because, initially, postoperative effects on swallowing function were not known. The sleep architecture improved for the entire group, as did the Epworth sleepiness scale values and self-reported snoring, daytime sleepiness, and quality of life, with a tendency toward improvement of morning headaches. Therefore, patients received, statistical, objective, and subjective improvement after lateral pharyngoplasty.

Because I cut the mylopharyngeal and glossopharyngeal parts of the SPC muscle, swallowing difficulties, as expected, were the most troublesome effects of this procedure. During the recovery period, patients complained that some foods got stuck in their throats, causing cough and discomfort; this was eased by neck extension during swallowing. Patient 10 took the longest to return to his full preoperative diet (70 days, very far from the group median). Interestingly, he graded his postoperative pain as irrelevant (grade 1), and he had the largest preoperative AHI in the group. Whether this delay was related to preexisting neurologic problems in his lateral pharyngeal wall or to an aggressive surgical approach remains to be shown. Occasionally, during follow-up, some patients reported the need to drink fluids so as to swallow particularly dry foods, such as popcorn. This eventually disappeared after a few months. Permanent velopharyngeal incompetence, a possible complication in palatal surgeries, did not occur in this group and, unlike uvulopalatopharyngoplasty,[28] even temporary oronasal reflux seemed unusual with lateral pharyngoplasty. I attribute this to the minimum tissue resection in this technique and to the postoperative control of possible scarring factors such as infections.

Verse et al.[29] reported excellent cure rates after removal of markedly enlarged tonsils in adults with OSAHS. In their series, the volume of tissue removed ranged from 10 to 29 (mean 17.3) mL. In our group, this

did not seem to influence the outcome we had because our patients had smaller tonsils, and the mean tissue volume we removed was 5.2 (range 1.8–10.7) mL.

Upper airway closure during sleep is not related to SPC muscle activation.[30] Therefore, in theory, paralyzing or simply cutting the SPC muscle would not make much difference in the sleep respiratory pattern of OSAHS patients. This is why, after the cranial to caudal section of this muscle, I sutured its lateral portion to the same-side palatoglossus muscle. Because the origins of the SPC muscle in this region are more lateral (mandibular mylohyoid line) and more posterior (side of the base of the tongue) than the palatoglossus muscle, those sutures intend to provide lateral and anterior support to the oropharynx and tongue. The associated palatopharyngeal Z-plasty[15] also provides palatine and oropharyngeal support through orienting soft tissue retractions in the lateral port areas, a principle already discussed in the literature.[31]

Finally, I must stress the importance of the use of the operative microscope when dissecting the SPC muscle: there are numerous vessels within the tonsillar fossa, and the view with the microscope makes the procedure safer regarding bleeding control and also prevents accidental injuries to cranial nerves IX and X. Because of the potentially severe vagal reflexes in this part of the surgery, it is mandatory to warn the anesthesiologist of this risk.

CONCLUSION

A surgical option for treating OSAHS is the changing of the pharyngeal muscular wall properties to splint the entire collapsible pharynx. A straightforward approach to the lateral pharyngeal wall may be preferred to a stretch effect caused through bone repositioning. Lateral pharyngoplasty repositions pharyngeal muscles, giving support to the lateral pharyngeal wall and producing subjective and objective improvement in sleep in OSAHS patients.

Acknowledgments

The author thanks Maria Lucia Oliveira de Souza Formigoni, PhD, and Gilberto Guanaes Simões Formigoni, MD, for statistical analysis of the data and José Falcetti and Rodrigo Ricieri Tonan, from Artes Médicas do Instituto de Psiquiatria da Faculdade de Medicina da Universidade de São Paulo for the illustrations.

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