Sphincter Pharyngoplasty as a Treatment of Velopharyngeal Incompetence in Young People*

A Prospective Evaluation of Effects on Sleep Structure and Sleep Respiratory Disturbances

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Background: Sphincter pharyngoplasty (SP) appears to be the more “physiologic” surgical technique to treat velopharyngeal incompetence (VPI). This procedure creates a dynamic sphincter of variable diameter and keeps the flexibility of the soft palate. SP also induces velopharyngeal size reduction, mainly in the transverse diameter, which may cause upper airway (UA) occlusions during sleep.

Aim: To prospectively evaluate the effects of SP by a modified Orticochea procedure on sleep structure and sleep respiratory disturbances.

Methods: Polysomnographic studies before and after surgery in 17 consecutive patients treated by a modified Orticochea procedure SP for VPI.

Results: For the whole group, SP did not induce significant impairment of apnea-hypopnea index or nocturnal oxygen saturation. Slow-wave sleep (SWS) was significantly reduced after surgery (25 ± 9% of total sleep time [TST] vs 28 ± 9% of TST before SP [p = 0.04]). Following surgery, there was a trend for an increase in the microarousal index (p = 0.09) and more specifically in respiratory-related microarousals.

Conclusion: SP, although creating a clinically obvious reduction of velopharyngeal diameter, generally did not lead to the occurrence of an obstructive sleep apnea syndrome. However, we found a significant reduction of SWS quantity and a trend toward an increase in the number of cortical microarousals. These findings suggest that the reduction of UA diameter associated with the surgical technique leads to increases in respiratory effort sufficient to induce sleep fragmentation and SWS reduction, even in the absence of apneas or hypopneas.

(CHEST 2004; 125:864–871)

Key words: cleft palate; dynamic sphincter pharyngoplasty; sleep apnea; velopharyngeal insufficiency

Abbreviations: AHI = apnea-hypopnea index; IQR = interquartile range; NS = not significant; OSAS = obstructive sleep apnea syndrome; SP = sphincter pharyngoplasty; SWS = slow-wave sleep; TST = total sleep time; UA = upper airway; VPI = velopharyngeal Incompetence

Cleft palate represents a highly prevalent craniofacial malformation occurring in one case for every 800 births.¹ In cleft palates, congenital short palates, or in cases of inefficient cleft palate surgery, the soft palate is unable to act as a competent muscular sphincter controlling the air column between the oral and the nasal cavities. The patients then exhibit velopharyngeal insufficiency and open rhinolalia.²

Surgical techniques available to correct velopharyngeal incompetence (VPI) are posterior pharyngeal wall augmentation (obtained for example by Teflon injections),³ posterior pharyngeal flaps, or

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Manuscript received March 20, 2003; revision accepted September 15, 2003.

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sphincter pharyngoplasty (SP). The common goal of these surgical techniques is to create a permanent partial obstruction of the velopharyngeal space in order to correct hypernasal speech. Teflon injections have received attention owing to their relative simplicity. Unfortunately, long-term evolution is usually associated with local chronic inflammatory response, which eventually leads to pharyngeal mucosa lesions and Teflon extrusion. This technique is no longer used as a treatment of VPI. Posterior pharyngeal flaps are created by suturing a superior, or more rarely an inferior posterior, pharyngeal wall flap to the free side of the soft palate. Chronic complications include hyponasality, nasal obstruction, and lateral-port stenosis. SP, also called lateral pharyngoplasty, is a standard surgical treatment of VPI. It appears to be a more "physiologic" solution, as it intends to preserve the circumferential nature of the muscles and their contribution to velopharyngeal closure. This procedure creates a dynamic sphincter of variable diameter and preserves the flexibility of the soft palate. Finally, SP has the highest rate of resolution of VPI with the lowest risk of complication. The data reviewed above explain why a modified Orticochea procedure SP (see "Materials and Methods" for description) is the surgical treatment of choice for VPI in our center.

SP also induces velopharyngeal size reduction, mainly in the transverse diameter. Such upper airway (UA) narrowing during the daytime may cause UA occlusions during sleep. Several cases of obstructive sleep apnea after pharyngoplasty have been reported. The majority of these studies are poorly documented case reports without polysomnography, or retrospective studies. In addition, two prospective studies have evaluated posterior pharyngeal flap, another surgical technique that potentially results in a less physiologic reconstruction of the pharyngeal sphincter than SP. Therefore, the aim of the present study was to prospectively evaluate the effects of SP by a modified Orticochea procedure on sleep structure and sleep respiratory disturbances.

**Patients and Methods**

**Patients**

Patients were recruited among cleft palate subjects with a diagnosis of residual VPI after appropriate speech therapy conducted by a trained orthophonist. VPI was accepted when residual speech difficulties corresponded to stage 2B or 3 of the Borrel-Maisonny classification. The patients were then offered SP.

Seventeen consecutive patients (10 male) with a mean age of 14 ± 8 years and a mean body mass index of 17.7 ± 3.4 (± SD) were prospectively studied using polysomnography before and after SP (median delay after surgery, 164 days; range, 88 to 624 days). All of the patients were available for follow-up after surgery.

**Polysomnography Before and After SP**

Continuous overnight polysomnographic recordings of EEG (with electrode positions placement system 10–20), electrooculogram, chin electromyogram, and ECG were obtained. Tracings were scored manually, according to standard criteria for sleep stages and cortical microarousals. Microarousals ending respiratory events were specifically scored as respiratory-related microarousals.

Oronasal thermistors were used to assess airflow. Respiratory effort was evaluated by monitoring thoracic and abdominal movements. Oxygen saturation was measured with a BiOx-Ohmeda 3700 oximeter (Ohmeda; Louisville, CO).

Apneic events were classified as central, obstructive, or mixed, depending on the absence or presence of breathing efforts. Episodes of apnea were defined as complete cessation of airflow for ≥5 s. Hypopnea was defined as a >50% or >30% decrease in oronasal airflow or amplitude of the thoracic and abdominal signals, respectively, lasting for at least 5 s and associated with an arousal and/or a desaturation of 4%.

Normal children do not exhibit any obstructive apneas, and the apnea-hypopnea index (AHI) is classically <3 per hour of sleep. In our study, each child was his own control subject, and the main outcome measure we considered was the AHI variation between before and after surgery. In the corresponding range of age, the cut-off values for apneas, hypopneas, and AHI were those that apply for children.

**Surgical Technique**

The surgical technique used was a modified Orticochea procedure SP. The new pharyngeal sphincter is obtained in moving the posterior tonsil pillars with their enclosed palatopharyngeus muscle from the lateral pharyngeal walls to the midsection of the posterior pharyngeal wall (Fig 1). Our team has modified the technique by inserting the palatopharyngeus muscles higher into the posterior pharyngeal wall and by using a Z-plasty suture.

**Statistical Analysis**

Polysomnographic results before and after surgery were compared using nonparametric (Wilcoxon) or parametric (paired t test) tests depending on the normal distribution of the data. Distribution normality was assessed by an Omnibus test. Linear regression was performed using Spearman correlation. All results are shown as mean ± SD; p < 0.05 was considered significant.

**Results**

**Patients**

Patients were concerned about their speech problems and complained about them. One patient reported daytime sleepiness before surgery. At time of the postsurgical evaluation, three patients mentioned snoring, one of them with an increased AHI from 14.9 to 24/h of sleep.

**Polysomnographic Data Before and After Surgery**

SP did not systematically induce respiratory disturbances impairment, as evidenced by the absence
of postoperative changes in AHI (6.5±6.0/h before surgery vs 7.9±5.9/h after surgery, p = not significant [NS]) or nocturnal oxygen saturation (Table 1). This was true even for the subgroup of subjects demonstrating the highest AHI before surgery (Fig 2). With a so-small difference in AHI before and after surgery (1.4/h), >100 subjects should have been included to find a statistical difference. Even in the presence of such statistical difference an increase of <2.0/h appears as not clinically relevant. Only two patients (12%) demonstrated a clinically significant increase in AHI (4.8/h before surgery vs 15.2/h after surgery; and 6.3/h before vs 12.3/h after surgery, respectively).

Slow-wave sleep (SWS) was significantly reduced after surgery (25±9% of total sleep time [TST]) vs 28±9% before SP (p = 0.04) [Table 2, Fig 3]. In addition, there was a trend to an increase in the microarousal index (p = 0.09) and more specifically in respiratory-related microarousals. Moreover, after surgery, a negative correlation was found between SWS quantity and the number of respiratory-related microarousals (r = −0.50, p = 0.04) [Fig 4].

Discussion

To our knowledge, this is the first study to prospectively compare polysomnographic data before and after dynamic SP. We were able to demonstrate that, although it leads to a substantial reduction in the velopharyngeal diameter, SP by a modified Orticochea procedure did not lead to the occurrence of more apneas or hypopneas after surgery than before surgery. However, following pharyngoplasty we had indirect evidence of UA obstructive events, since we demonstrated reduction in SWS related to the microarousal index. These findings suggest that the reduction in UA diameter associated with the surgical technique leads to increases in respiratory effort sufficient to induce sleep fragmentation and SWS reduction even in the absence of apneas or hypopneas.

Sleep Apnea Syndrome as a Complication of Surgical Treatment of VPI

Obstructive sleep apnea syndrome (OSAS) has been reported as a complication of VPI surgery in the short-term as well as long-term follow-up of patients3,5,11–15 (Table 3). The prevalence of sleep

Table 1—Sleep Respiratory Disturbances Before and After SP*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Before Surgery</th>
<th>After Surgery</th>
<th>p Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index</td>
<td>17.7±3.4</td>
<td>17.2±3.5</td>
<td>NS</td>
</tr>
<tr>
<td>Apnæs index, per hour of sleep</td>
<td>0.7±0.5</td>
<td>0.7±1.0</td>
<td>NS</td>
</tr>
<tr>
<td>Hypopnea index, per hour of sleep</td>
<td>5.8±5.9</td>
<td>7.2±5.3</td>
<td>NS</td>
</tr>
<tr>
<td>AHI, per hour of sleep</td>
<td>6.5±6.0</td>
<td>7.9±5.9</td>
<td>NS</td>
</tr>
<tr>
<td>AHI &gt; 3/h of sleep, No. (%)</td>
<td>14 (82)</td>
<td>13 (76)</td>
<td>NS</td>
</tr>
<tr>
<td>Mean nocturnal SaO₂, %</td>
<td>96.4±2.0</td>
<td>96.4±0.9</td>
<td>NS</td>
</tr>
<tr>
<td>Minimal nocturnal SaO₂, %</td>
<td>91.6±3.5</td>
<td>92.8±2.3</td>
<td>NS</td>
</tr>
<tr>
<td>T &lt; 90% of SaO₂, % of TST</td>
<td>0.34±0.71</td>
<td>0.01±0.034</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD unless otherwise indicated; SaO₂ = arterial oxygen saturation; T < 90% of SaO₂ = time spent below 90% oxygen saturation (% of TST).
†Paired t test or Wilcoxon signed-rank test probability.
breathing disorders is probably highly variable depending on the surgical technique and other factors. For example, the incidence of OSAS increased in children concurrently exhibiting craniofacial malformations. In such preexisting unfavorable anatomic situations, UA edema induced by the surgery then led to severe acute UA obstructions.

**Occurrence of OSAS During the Perioperative Period**

Data from the literature are summarized in Table 3. Symptoms of sleep apnea commonly occur early after VPI surgery. The reported prevalence of OSAS during the days immediately following surgery varied from 7.2 to 90% (Table 3). However, numerous studies were not prospectively designed and/or were limited to clinical assessments without polysomnography. Only two studies\(^{14-15}\) have addressed the evolution of polysomnographic parameters before and after pharyngeal flaps. In the study by Orr et al,\(^{14}\) all but 1 of the 10 patients demonstrated a marked increase in AHI 3 days after surgery (preoperative AHI, 0.7/h; vs postoperative AHI, 21.7/h). Sirois et al\(^{15}\) studied 40 children before and after pharyngeal flap surgery. Fourteen patients (35%) acquired sleep apnea syndrome postoperatively. Preoperative polysomnography had no predictive value in separating subjects with low or high risk of

Figure 2. AHI before and after SP. SP did not induce impairment in AHI even in the highest preoperative AHI subgroup. The line drawn through the middle of the box is the median, and the top and bottom lines are the 25th and 75th percentiles, respectively (the box represents the middle 50% of the data). The length of the box is thus the interquartile range (IQR). The upper adjacent value is the largest observation that is less than or equal to the 75th percentile plus 1.5 times IQR. The lower adjacent value is the smallest observation that is greater than or equal to the 25th percentile minus 1.5 times IQR.

### Table 2—SP Effects on Sleep Parameters*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Before Surgery</th>
<th>After Surgery</th>
<th>p Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>TST, min (n = 17)</td>
<td>414 ± 39</td>
<td>396 ± 64</td>
<td>NS</td>
</tr>
<tr>
<td>Stage 1–2, % of TST (n = 17)</td>
<td>51 ± 10</td>
<td>54 ± 11</td>
<td>NS</td>
</tr>
<tr>
<td>SWS, % of TST (n = 17)</td>
<td>28 ± 9</td>
<td>25 ± 9</td>
<td>0.04</td>
</tr>
<tr>
<td>Rapid eye movement, % of TST (n = 17)</td>
<td>21 ± 4</td>
<td>21 ± 5</td>
<td>NS</td>
</tr>
<tr>
<td>Total microarousals index, per hour of sleep (n = 12)</td>
<td>9.3 ± 7.0</td>
<td>12.8 ± 7.3</td>
<td>0.09</td>
</tr>
<tr>
<td>Respiratory microarousals index, per hour of sleep (n = 12)</td>
<td>2.8 ± 4.6</td>
<td>5.8 ± 6.4</td>
<td>0.12</td>
</tr>
<tr>
<td>Nonrespiratory microarousals index, per hour of sleep (n = 12)</td>
<td>6.5 ± 4.2</td>
<td>7.0 ± 2.8</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD.
†Paired t test or Wilcoxon signed-rank test probability.
postoperative OSAS. The high prevalence of sleep apnea during the acute postsurgical phase might explain some of the complications occurring after this surgery. Kravath et al were the first to report a death 4 weeks after velopharyngeal flap surgery. Of the 219 children studied by Valnicek et al, 36 had early complications (20 with airway obstruction) in the first 24 h; 3 patients required

![Figure 3](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20385/)

**Figure 3.** SWS before and after SP. SWS was significantly reduced after surgery (25 ± 9% of TST vs 28 ± 9% before SP) [p = 0.04]. See Figure 2 legend for descriptions.

![Figure 4](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20385/)

**Figure 4.** Correlation between SWS and respiratory-related microarousals index after SP (n = 17). After surgery, a negative correlation was found between SWS quantity and the residual number of respiratory-related microarousals (r = −0.50, p = 0.04). *Spearman correlation test probability.
 reintubation, and there was one death. Wells et al reported that 50% of patients required reintubation in the early postoperative period after posterior pharyngeal flap surgery. A systematic monitoring by nocturnal oximetry might be justified in these children to prevent life-threatening complications.19,20

**Long-term Persistence of OSAS After VPI Surgery**

Seven studies, summarized in Table 3 and Figure 5,13,14,15 have assessed both short-term and long-term incidence of sleep breathing disorders after VPI surgery. The prevalence of OSAS several months after surgery varies from 1 to 20%. In most patients, UA obstruction resolved after a few months (3 to 6 months), suggesting that resolution of UA edema following the acute postoperative period leads to a decrease in the number of respiratory events.

**Prevalence of OSAS After VPI Surgery Depends on the Surgical Technique Used**

There are only two retrospective studies6,10 comparing the prevalence of OSAS after pharyngeal flaps or SP; the results of the two studies are in agreement, showing a higher prevalence of OSAS after pharyngeal flaps (Table 4). Conversely, as in the present study, SP did not lead to long-term postsurgical OSAS.

**Does SP Induce Subtle Respiratory Events During Sleep?**

Our prospective data and previous studies6,10 demonstrate that SP is not associated with an impairment of sleep respiratory disturbances. Thus, although narrower than before surgery, the UA seems to be stiff enough to avoid complete UA collapse. However, UA size reduction could be associated with more subtle respiratory events than apneas or hypopneas. Detection of these subtle events, such as flow limitation episodes or respiratory effort-related arousals, requires sensitive diagnostic tools. Neither previous studies nor the present protocol used nasal pressure coupled with esophageal pressure or pulse transit time, which would allow identification of such respiratory events.21 Our study is the first to monitor sleep architecture and microarousals before and after surgery. We found a significant decrease in SWS and a trend toward an increase in microarousals after surgery. We hypothesize that in spite of an absence of OSAS, there was an increase in subtle respiratory events after surgery that were undetected by the diagnostic tools that we used. Such events are subtle but significant enough to slightly impair sleep quality. Further studies are needed to test this hypothesis and to objectively assess daytime sleepiness and cognitive function before and after surgery.

**Conclusions and Guidelines for Further Evaluation of SP**

**Management of Potential Sleep Problems in Patients Referred for SP**

Short-term studies available in the literature6,11,14,15 suggest that transient sleep apnea syndrome can occur in the days or weeks immediately following the different surgical procedures proposed to treat VPI. A systematic monitoring by nocturnal oximetry might be justified during the first postoperative days to prevent life-threatening complica-
tions. SP is the reference surgical technique for VPI, as the number of patients acquiring long-term sleep respiratory disturbances appears significantly lower than after pharyngeal flaps.

As our study does not demonstrate a high prevalence of OSAS 3 to 6 months after surgery, we do not recommend systematic polysomnography before and 6 months after surgery in asymptomatic patients. Patients with associated craniofacial dysmorphia or symptoms related to sleep apnea should however be evaluated.

Research Agenda

Further studies using new diagnostic tools (ie, nasal pressure and pulse transit time) are needed to detect subtle respiratory events associated with sleep fragmentation, which could result from postoperative reduction of UA size. It could be interesting to study these patients after years of evaluation to confirm the stability of these results.

ACKNOWLEDGMENT: The authors thank Dr. Sandrine Lau-nois for reading the manuscript and helpful criticism.

REFERENCES


Table 4—Prevalence of OSAS After Pharyngeal Flap and After SP: Clinical and/or Polysomnographic Assessment*

<table>
<thead>
<tr>
<th>Source</th>
<th>Surgical Technique</th>
<th>Subjects, No.</th>
<th>Polysomnography</th>
<th>Prevalence of OSAS, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pensler and Reich10</td>
<td>PF 75</td>
<td>No</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>de Serres et al6</td>
<td>SP 10</td>
<td>No</td>
<td>0</td>
<td></td>
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<tr>
<td></td>
<td>PF 18</td>
<td>Yes</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SP 16</td>
<td>Yes</td>
<td>0</td>
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</table>

*See Table 3 for expansion of abbreviation.