Adenotonsillectomy for Obstructive Sleep Apnea in Obese Children

Effects on Respiratory Parameters and Clinical Outcome

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Objective: To assess the efficacy of adenotonsillar surgery on respiratory sleep parameters and avoiding continuous positive airway pressure (CPAP) treatment in morbidly obese children with obstructive sleep apnea syndrome (OSAS).

Design: Retrospective.

Setting: Tertiary referral institution.

Patients: Children aged 2 to 18 years, with a body mass index (BMI) at or higher than the 95th percentile (adjusted for age and sex), undergoing adenotonsillar surgery for OSAS.

Interventions: Adenotonsillectomy.

Main Outcome Measures: Preoperative and postoperative respiratory disturbance index, oxygen saturation nadir, overall severity of OSAS (mild, moderate, or severe) and candidacy for CPAP treatment were assessed and compared. Variables such as age, severity of disease, adenotonsillar size, and BMI $z$ scores were compared between responders and nonresponders to surgical treatment.

Results: A total of 19 patients with full preoperative and postoperative data for evaluation were identified. The median (SD) age was 78 months (53.3 months). The median (SD) BMI $z$ score was 2.84 (0.94). Eighteen patients (95%) had OSAS preoperatively to warrant CPAP treatment. Surgery reduced the overall median (SD) respiratory disturbance index from 20.7 (24.5) to 7.3 (14.9) ($P<.001$) and improved the median (SD) oxygen saturation nadir from 77.5% (16.3%) to 88.5 (13.1%) ($P<.01$). A total of 7 patients (37%) were cured by surgery. Ten patients (53%) had postoperative disease of sufficient severity to require CPAP. Surgery obviated the need for further treatment in only 8 (44%) of the 18 patients with preoperative disease warranting CPAP. No differences were identified between responders and nonresponders to surgical treatment.

Conclusions: Adenotonsillar surgery improves sleep respiratory parameters in morbidly obese children with OSAS. Most patients have residual OSAS requiring further treatment.

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Obstructive sleep apnea syndrome (OSAS) is an increasingly recognized disease entity in childhood, affecting 2% of the pediatric population. Most cases result from adenotonsillar hypertrophy and are thus amenable to surgical cure. It now represents the most common indication for pediatric adenotonsillectomy. In Western populations, childhood morbid obesity has risen dramatically over the past 2 decades. This increase has significant implications for the otolaryngologist because OSAS is reported to occur in 37% to 46% of morbidly obese children. This suggests that morbid obesity has a major role in the pathogenesis of OSAS in this subgroup. Notwithstanding this, adenotonsillar hypertrophy may also be present in these patients. The relative contribution each of these entities—morbid obesity and adenotonsillar hypertrophy—to resultant OSAS in these children is not clear. The obvious implication is that adenotonsillectomy will be increasingly performed on morbidly obese children.

To our knowledge, there are only a handful of publications in the literature addressing the effect of surgery on OSAS in morbidly obese children, and none have specifically addressed the need for further treatment after surgery. The aim of this study is to assess adenotonsillecto-
tomy in morbidly obese children with OSAS with respect to the effect of surgery on respiratory parameters during sleep and avoidance of further treatment.

**METHODS**

We reviewed the medical charts of all morbidly obese children (defined as those having a body mass index [BMI] calculated as weight in kilograms divided by height in meters squared] at or greater than the 95th percentile adjusted for age [2-18 years] and sex), who underwent adenotonsillectomy for OSAS over a 36-month period. Only patients with preoperative overnight sleep studies available for review that included polysomnography (PSG) or nocturnal sleep oximetry, and more than 3 months of postoperative follow-up, including PSG where appropriate, were included.

Demographic data recorded included age, sex, height, weight, and BMI. A BMI z score was calculated for each patient to facilitate comparative analysis. Tonsil size was graded on a 4-point scale: Grade 1 tonsils were those lying lateral to the tonsillar fauces; grade 2, at the level of the fauces; grade 3, medial to the fauces but not to midline; and grade 4, touching at the midline. Adenoidal grading was on a 4-point scale: 0, absent; 1, minimal adenoidal tissue; 2, moderate hypertrophy; and 3, obstructing the choanae, as assessed at the time of operation.

Preoperative and postoperative overnight sleep study data were recorded.

Overnight sleep studies included full overnight PSG or overnight sleep oximetry. The PSG was performed by measuring the following standard neurophysiologic and respiratory signals (Comumedics Sleep Pty Ltd, E-series, Abbotsford, Australia): electroencephalogram with central, anterior, and occipital leads; electro-oculograms; submental and diaphragmatic electromyogram using external electrodes; and heart rate and rhythm recorded by electrocardiogram. Airflow was recorded using a nasobuccal thermistor. Nasal pressure was measured using a nasal cannula connected to a pressure transducer. Chest wall excursion was measured by respiratory inductive plethysmography (Respitrace PT, model 105-042-01; NIMS Inc, Miami Beach, Fla). Oxygen saturation was measured by pulse oximetry set at a 2-second averaging time (Nellcor, Hayward, Calif). Body position was noted, and digital videotapes were taken. The following PSG results were recorded: respiratory disturbance index (RDI), defined as the sum of obstructive apneas and hypopneas per hour of sleep, and the oxygen saturation nadir, the lowest percentage of oxygen saturation recorded during sleep study. An RDI of more than 5 and less than 10 was considered mild OSAS; at least 10 but less than 20, moderate OSAS; and 20 or higher, severe disease. All sleep studies were evaluated by a pediatric sleep physician (A.W.). The presence of preoperative disease of sufficient severity to warrant treatment with continuous positive airway pressure (CPAP) was evaluated and assigned by the pediatric sleep physician.

The follow-up evaluation for each patient was assessed with regard to polysomnographic parameters recorded during the overnight sleep study, and the outcome with regard to the need for CPAP therapy was noted.

Paired data were analyzed using 2-tailed paired t test. Other statistical analysis was performed using the Mann-Whitney test and Fisher exact test. A P value of less than .05 was considered significant.

![Graph showing BMI vs age](image-url)
A total of 26 morbidly obese patients were identified who underwent adenotonsillar surgery as the primary procedure over a 36-month period; of these, 20 had sleep-study–documented OSAS. Of these 20, 19 had preoperative and postoperative data available for analysis and were included in this study. All patients had a full preoperative respiratory and sleep physician evaluation, and none had a comorbid diagnosis of significant pulmonary disease. The cohort included 2 patients with named syndromes: 1 with Albright osteodystrophy and 1 with Down syndrome.

Fourteen patients were boys, and 5 were girls. The Figure represents the demographical data for the patient cohort, plotting age against BMI on sex-specific percentile charts. The median age (SD) was 78 (53.3) months (range, 24-212 months). The median (SD) BMI z score was 2.84 (0.94) (range, 2.02-5.28). Median tonsillar and adenoidal size were grades 3 and 2, respectively.

All 19 patients had overnight sleep studies preoperatively, either full overnight PSG (n=18) or overnight sleep oximetry alone (n=1). Median (SD) preoperative RDI was 22.0 (33.5) (range, 7.6-129.2). The median (SD) oxygen saturation nadir measured during sleep was 75.0% (15.9%) (range, 22%-92%). Preoperative disease severity was judged to be severe in 10 patients (53%), moderate in 8 (42%), and mild in 1 (5%). Based on the preoperative severity of disease, 18 patients (95%) were considered candidates for CPAP.

Seventeen patients had undergone adenotonsillectomy, 1 (an older patient with bulky uvular tissue) also had undergone a modified uvulopalatopharyngoplasty, and another had undergone only tonsillectomy, in the absence of adenoidal tissue for resection. All were clinically assessed no sooner than 6 weeks postoperatively. One patient had full resolution of all symptoms, including snoring. Although a follow-up sleep study had been planned, the patient’s parent refused to participate because full symptomatic resolution, including that of snoring, had occurred postoperatively. In view of the absence of snoring and associated preoperative symptoms, this patient was considered cured. The remaining 18 patients had full overnight PSG at a mean 13-week interval after surgery (range, 8-30 weeks). Paired preoperative and posttreatment PSG data were available for these patients. The median (SD) postoperative RDI was 7.3 (14.9) (range, 0.57-7.7). A 2-tailed paired t test confirmed the difference between preoperative and postoperative RDIs to be highly significant (Table 1). Similarly, the difference between preoperative and postoperative oxygen saturation nadirs was also significant (Table 1).

Clinical correlation of PSG parameters to postoperative disease stratification was assessed (Table 2). Seven of the cohort were cured following surgical intervention. Of the remaining 12 patients, the disease stratification of 5 patients improved, either converting from severe to moderate OSAS (n=3) or severe to mild OSAS (n=2). The disease severity of 6 patients was on follow-up PSG, and 1 patient’s OSAS was up-staged from moderate to severe. Ten patients (53%) were found to have residual disease of sufficient severity to warrant further treatment in the form of CPAP therapy. This was tolerated well in all patients. Of the 19 patients who underwent surgery, 18 (95%) had initial disease of sufficient severity to warrant CPAP therapy. Of this group (n=18), 10 (56%) ultimately required this treatment, with 8 (44%) improving after surgery to a degree that CPAP was not indicated, either by cure or by mild residual disease.

Comparison of the group that received postoperative CPAP therapy and the group that did not require further treatment was performed (Table 3). No significant difference was identified between the 2 groups with regard to age, BMI z score, tonsil or adenoid size, preoperative RDI, or preoperative oxygen saturation nadir.

### Table 1. Effect of Adenotonsillar Surgery on Sleep Study Parameters*

<table>
<thead>
<tr>
<th>Sleep Study Variable</th>
<th>Preop</th>
<th>Postop</th>
<th>P Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>RDI</td>
<td>20.7 (24.5)</td>
<td>7.3 (14.9)</td>
<td>.001</td>
</tr>
<tr>
<td>OSN</td>
<td>77.5 (16.3)</td>
<td>88.5 (13.1)</td>
<td>.007</td>
</tr>
</tbody>
</table>

*Data are presented as median (SD).
†Two-tailed paired t test.

### Table 2. Effect of Surgery on Clinical Outcomes

<table>
<thead>
<tr>
<th>Clinical Outcome</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Was the patient cured?</td>
<td>7 (37)</td>
<td>12 (63)</td>
</tr>
<tr>
<td>If there is residual disease, is the patient improved?</td>
<td>5 (42)</td>
<td>7 (56)</td>
</tr>
<tr>
<td>Was CPAP therapy given postoperatively?</td>
<td>10 (53)</td>
<td>9 (47)</td>
</tr>
<tr>
<td>Did surgery avoid the need for CPAP?</td>
<td>8 (44)</td>
<td>10 (56)</td>
</tr>
</tbody>
</table>

### Table 3. Comparison of Group Requiring CPAP vs Group Needing No Further Treatment*

<table>
<thead>
<tr>
<th>Variable</th>
<th>10 Patients Who Received CPAP Therapy</th>
<th>9 Patients Who Received No CPAP Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mo</td>
<td>94.5 (65.6)</td>
<td>76.0 (39.2)</td>
</tr>
<tr>
<td>BMI z score</td>
<td>2.81 (0.7)</td>
<td>2.84 (1.2)</td>
</tr>
<tr>
<td>RDI</td>
<td>27.2 (30.2)</td>
<td>15.1 (38.2)</td>
</tr>
<tr>
<td>OSN, %</td>
<td>77.5 (18.7)</td>
<td>71.0 (12.6)</td>
</tr>
</tbody>
</table>

*Data are presented as median (SD); P values from the Mann-Whitney test were nonsignificant.
This study confirms the overall positive effect of surgery on respiratory parameters during sleep, namely, RDI and oxygen saturation nadir. Mitchell and Kelly recently reported similar findings in a prospective study of 30 morbidly obese children with a diagnosis of OSAS proved by polysomnography. Other reports have also observed a beneficial result following surgery, but only Weit et al had PSG documentation of disease and resolution in a pediatric population that was morbidly obese. Mitchell and Kelly also noted the high prevalence of residual disease in their study population, with 36% of children having moderate or severe disease. Although the current study was retrospective and had a smaller cohort, a higher prevalence of significant residual disease was identified (56%). One of the difficulties that exists in relation to pediatric OSAS and the use of PSG as the gold standard diagnostic tool is that the correlation between the magnitude of RDI and oxygen saturation nadir abnormalities measured during PSG and resultant adverse sequelae of OSAS has not been established. It follows that improving RDI and oxygen saturation scores is of uncertain clinical benefit. The additive risk of adverse outcomes with increasing disease stratifications, from mild to severe, also remains ill-defined. Therefore, it also remains unclear whether regression of disease severity without a cure, as seen in 26% (5 of 19) of this study population, has any significance on the outcome of the disease if left untreated.

Notwithstanding this, few would argue the justification of treating pediatric OSAS when diagnosed. Most often, adenotonsillectomy represents the first line of treatment for pediatric OSAS, with CPAP reserved for patients in whom surgery was not successful or those in whom surgery is contraindicated. Therapy with CPAP is a low-risk, well-tolerated treatment option in children with OSAS. However, it is not without attendant problems. It requires expensive equipment, parental training, and patient habitation. In addition, it must be used for an unspecified, prolonged period of time and entails multiple hospital admissions for dose titration and disease surveillance, further adding to the cost burden. As an alternative, the option of a one-time surgical procedure—adenotonsillectomy—is indeed an attractive option. In the study group, on the basis of preoperative disease severity, 18 patients were candidates for CPAP. Subjecting this cohort to surgery obviated the need for further treatment in only 8 (44%) of 18 patients. Ten patients (56%) had surgery that improved individual respiratory sleep parameters overall and even disease stratification in some but did not alter the outcome in terms of avoiding CPAP. To the best of our knowledge, this is the first report to highlight the failure of surgery to avoid CPAP in morbidly obese children with significant OSAS.

Nonetheless, in the combined results of Mitchell and Kelly and the results presented herein with regard to the effect of surgery on disease categories, 57% of children had either a cure or residual mild disease following surgery. Surgery is effective in some morbidly obese children and not in others. Possible variables that may influence the efficacy of surgery include age, BMI z score, severity of preoperative disease, and size of the adenotonsillar tissue. None of these preoperative factors were significantly different between the group that received postoperative CPAP and the group that received no further treatment in the current study, although the small numbers preclude any firm conclusions being drawn. In the absence of preoperative variables predictive of a positive surgical outcome, surgery may well be offered to all morbidly obese children with significant OSAS in the knowledge that this approach will avoid the need for CPAP in half of them. However, this requires justification by risk- and cost-benefit analyses to confirm primarily the safety and secondarily the economic validity of this approach. The former is of particular concern considering the higher risk of postoperative respiratory complications in children undergoing adenotonsillectomy for OSAS. The risks specific to the morbidly obese subgroup have not been clarified.

Adenotonsillectomy for OSAS in morbidly obese children improves RDI and oxygen saturation nadir. Fewer than half of these children are cured by surgery. Similarly, only 44% of those patients with preoperative OSAS severe enough to warrant CPAP are ultimately spared this intervention by undergoing surgery. No preoperative variables predictive of responders and nonresponders to surgery have been identified. Further studies evaluating the risk-benefit and cost-benefit equations of performing adenotonsillectomy in morbidly obese children will clarify the appropriateness of offering surgery to all patients in this group as first-line treatment.

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Author Contributions: All authors had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Shine, Lannigan, Coates, and Wilson. Acquisition of data: Shine and Wilson. Analysis and interpretation of data: Shine, Lannigan, and Coates. Drafting of the manuscript: Shine and Lannigan. Critical revision of the manuscript for important intellectual content: Shine, Lannigan, Coates, and Wilson. Statistical analysis: Shine. Administrative, technical, and material support: Shine, Lannigan, and Wilson. Study supervision: Lannigan and Coates.

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REFERENCES