Effect of Treating Obstructive Sleep Apnea by Tonsillectomy and/or Adenoidectomy on Obesity in Children

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Background: Obstructive sleep apnea is common in obese children who have enlarged tonsils and adenoids.

Objective: To determine if treatment of obstructive sleep apnea by tonsillectomy and/or adenoidectomy will result in normalization of an obese child’s weight, as it does in underweight children, and as it does with other signs and symptoms.

Design: Retrospective cohort study. We recorded weight and height changes after tonsillectomy and/or adenoidectomy and compared changes of the obese and morbidly obese patients with those of the other patients.

Setting: A tertiary care inner-city hospital.

Participants: Children (n = 45) who underwent tonsillectomy and/or adenoidectomy for obstructive sleep apnea in 1994-1995; their mean (±SD) age was 4.9 ± 2.4 years at operation.

Results: At the time of surgery, 25 children were of normal weight; 3, underweight; 7, obese; and 10, morbidly obese. Postoperatively, 31 children (69%), including 10 of the 17 who were obese or morbidly obese, had substantial weight gain: the z score ± SD for weight of the entire group increased from 1.37 ± 2.49 to 2 ± 2.27 (P < .001). The mean z score ± SD for height increased from 0.03 ± 1.08 to 0.58 ± 0.94 (P < .001). The body mass index (BMI or Quetelet index): calculated as weight in kilograms divided by the square of the height in meters increased in 28 patients (62%) (P = .004).

Conclusion: Treating obstructive sleep apnea by tonsillectomy and/or adenoidectomy is associated with increased gain in height, weight, and body mass index in most children, including the obese and morbidly obese.


Editor’s Note: Now here’s one to think about. Is it better to have obstructive sleep apnea or morbid obesity? Might initial treatment of the morbid obesity solve both problems? Would that it was this easy.

Catherine D. DeAngelis, MD

RESULTS

It has been shown that obstructive sleep apnea (OSA) may cause poor growth in children, which can be severe enough to present as failure to thrive. Growth may improve and failure to thrive may resolve following tonsillectomy and/or adenoidectomy.12

Obstructive sleep apnea occurs frequently in obese children who have enlarged tonsils and/or adenoids, and complete or partial improvement in OSA following tonsillectomy and/or adenoidectomy in this group of patients has been documented.13-16 Obesity, which is difficult to treat, contributes to this condition.

Disturbed sleep and sleep deprivation with subsequent daytime hypersomnia and decreased activities are also known effects of OSA in children.14 We thought that this decreased energy expenditure might cause obesity, and therefore we hypothesized that treatment of OSA by surgery might improve sleep quality, decrease hypersomnia, increase activity, and lead to weight loss.

Of the 45 patients, 13 (29%) were girls and 32 (71%) were boys. Most of the children were African American or of Caribbean descent. Their mean (±SD) age at the time of surgery was 4.9 ± 2.4 years (age range, 1.4-10.25 years). The mean (±SD) follow-up period after surgery was 15 ± 8.5 months (range, 6-36 months). All of the patients were evaluated at the pediatric sleep apnea clinic and had a preoperative clinical diagnosis of OSA, with enlarged
PATIENTS AND METHODS

We recorded the heights and weights, at the time of operation and postoperatively, of the children who underwent tonsillectomy and/or adenoidectomy for OSA from 1994 to 1995. The study was performed at the Children's Medical Center of the State University of New York, Health Science Center at Brooklyn, which includes 2 large urban hospitals, the Kings County Hospital Center and the University Hospital of Brooklyn. We obtained the information by reviewing the medical records retrospectively and by inviting patients to come in for evaluation when the information was incomplete.

Forty-five children who underwent tonsillectomy and/or adenoidectomy in the study were evaluated postoperatively in our center, either in the sleep apnea clinic or in the general pediatric clinic. Follow-up measurements were taken 6 to 12 months postoperatively from 32 patients, and 2 to 3 years postoperatively from 13 patients. We also questioned the parents about apnea, disturbed sleep, and daytime activities of the children from the postoperative perspective.

The anthropometric measurements were performed at the Children's Medical Center with standard medical equipment. The percentiles, percent of medians, and $z$ scores of the heights and weights for age of all patients were obtained by using the Epi Info program (Version 6.04b, Centers for Disease Control and Prevention, Atlanta, Ga). The program is based on the data obtained from the National Center for Health Statistics. Patients who weighed more than 120% of their ideal body weight were considered obese and those who weighed more than 150% were considered morbidly obese.17 Ideal body weight was defined as the weight at the 50th percentile for the age at which the child's height is at the 50th percentile.17 Children with weights for age that fell below the fifth percentile were considered underweight.

The $z$ score for height and weight, which represents the variance from the population median in units of the population SD of the height or weight at a given age, is obtained by using the following equation:

$$z = \frac{\text{Actual Height or Weight} - \text{Median Height or Weight for Age}}{\text{SD}}$$

and was used to assess changes in height or weight for age over time.18 We considered postoperative increase or decrease in the $z$ score of more than 0.2 as a significant change. Body mass indices (BMI or Quetelet index), calculated as weight in kilograms divided by the square of the height in meters: weight (kg)/height (m$^2$), were used as an indirect measure of adiposity in these patients preoperatively and postoperatively.19 The percentiles for these indices were obtained using the standardized curves for white children and adolescents.19 To eliminate the effect of the natural increase or decrease in BMIs over time, which is known to occur in children,19 the difference between the patient's BMIs and the population median (BMI score = patient's BMI − median BMI for age) was used to assess changes of BMIs over time. A change in the BMI score of more than 0.2 was arbitrarily considered significant.

Daytime nap polysomnography (PSG) was performed on 20 patients. The children went to sleep spontaneously after sleep deprivation the night before the study. The sleep state was confirmed by the technician who performed the PSG. The nasal/oral airflow, the heart rate, chest and abdominal wall movements, and pulse oximetry were recorded simultaneously. The respiratory disturbance index (number of apneas and hypopneas per hour of sleep) was calculated, and a respiratory disturbance index of 5 episodes or more per hour was considered diagnostic of OSA. Echocardiograms were performed on 23 children.

Microsoft Excel (Version 97, Microsoft Corporation, Redmond, Wa) was used for statistical analysis. The paired $t$ test was used to compare $z$ scores and BMI scores before and after surgery. A 1-way analysis of variance was used to compare the $z$ score changes over time between the different weight groups.

tonsils and adenoids seen on physical examination or by radiography.

Nap PSG, which was performed on 20 of 45 children, was diagnostic of OSA; the median ($±$SD) sleep time was 49 ± 19 minutes (range, 40-104 minutes), the median respiratory disturbance index ($±$SD) was 13.8 ± 21.4 episodes per hour (range, 5-69.5 episodes per hour). Oxygen desaturation was found in 9 patients; the lowest desaturation ranged between 59% and 86%. Echocardiograms revealed signs of pulmonary hypertension in 12 of the 23 patients.

All children underwent tonsillectomy and adenoidectomy except for 2 who only had an adenoidectomy and 1 who only had a tonsillectomy, based on assessment of airway compromise by the ears, nose, and throat surgeon.

The children's weights at the time of the surgery were distributed into 4 categories (Table 1); 17 (38%) were categorized as obese or morbidly obese. Postoperatively, there was an increase in weight percentiles in 31 patients (69%) (Figure 1 and Figure 2). The mean ($±$SD) $z$ score for weight of the whole group increased from 1.37 ± 2.49 to 2 ± 2.27 ($P = .001$). No change in the $z$ score for weight was noted in 5 patients (11%), and the $z$ score for weight of 9 patients (20%) decreased. A change in $z$ score does not always indicate a change in category; a child's $z$ score could increase or decrease but remain in the same category. All 3 underweight patients gained enough weight to attain normal percentiles. Of the 25 patients of normal weight, 7 became obese and the rest remained normal. Of the 7 obese patients, 3 became morbidly obese and the others remained obese. Of the 10 morbidly obese, 6 actually increased their $z$ scores and became even more obese, 1 remained the same, and 3 decreased their $z$ scores but remained morbidly obese (Table 1). A total of 24 (53%) of the children were obese or morbidly obese on follow-up (vs 38% preoperatively).

There was no significant difference in preoperative to postoperative $z$ score changes in weight between the obese or morbidly obese patients and the other patients ($P = .17$).

The mean ($±$SD) $z$ score for height of these patients increased from 0.03 ± 1.08 to 0.58 ± 0.94 ($P < .001$). The percentile changes can be seen in Figure 3 and
There was also a significant positive correlation between the changes in \( z \) scores of the height and the weight \((r = 0.45, P < .001)\).

Postoperative BMI score changes are given in Table 2, and the percentile graphs are shown in Figure 5 and Figure 6. The mean (±SD) BMI score increased from 3.8 ± 6.4 to 4.7 ± 6.7 \((P = .004)\).

Of the 32 patients who had measurements 6 to 12 months after surgery, the mean (±SD) \( z \) score for weight increased from 1.7 ± 2.7 to 2.3 ± 2.5 \((P = .002)\). The mean (±SD) \( z \) score for height increased from 0.0 ± 1.1 to 0.4 ± 0.9 \((P = .03)\), and the mean (±SD) BMI score increased from 1.8 ± 5 to 2.9 ± 5.6 \((P = .02)\).

Following the surgery, 9 of the 10 morbidly obese patients were reevaluated in the apnea clinic. Eight parents reported resolution of the apnea and improvement of the quality of sleep of their children, except for persistent snoring without apnea in 2 patients. One patient’s parent reported persistence of apnea and disturbed sleep.

Our results are similar to previous studies that found that the treatment of OSA with tonsillectomy and/or adenoidectomy can result in an increase in height and weight percentiles for those with low scores.\(^1\)-\(^12\) We did not confirm our hypothesis that treatment of OSA will reduce obesity in either the obese or the morbidly obese children. To the contrary, we found that the treatment of OSA can make them even more obese, with the BMI increase.
ing in 65% of the obese and morbidly obese children. Studies in obese adults with OSA who were treated with chronic nasal continuous airway pressure found that they maintained their weight during treatment.21

To determine whether the length of time before follow-up would have an effect on our results, a separate analysis of the children was done that compared those seen 6 to 12 months after surgery with those seen after 2 to 3 years, which still revealed a significant increase in z scores for heights and weights in both groups.

Several mechanisms have been found to cause poor growth in children with OSA: (1) increased energy expenditure from the increased work of breathing during sleep,1 (2) poor appetite and difficulty swallowing,4 (3) stress and catabolism,3 and (4) decreased or abnormal growth hormone secretion secondary to disturbed sleep architecture.2,22 Perhaps these or other factors counterbalance the effects of the presumed sedentary lifestyle in obese children with OSA and make them less obese and shorter than they would have been without OSA. As a result, the treatment of OSA allows them to reach their biologically “predestined” height and weight.

We reviewed the history of the obese patients to determine if they did not lose weight because they did not respond to surgical therapy and if they continued to have apnea due to the obesity alone and found that this was not the case. Of the 9 morbidly obese patients on whom follow-up information could be obtained, 8 reported improvement in the symptoms of OSA. However, it is possible that we might have missed a few patients who still had OSA postoperatively. Suen et al23 found that 1 of 6 children whose mothers reported postoperative cure of their children’s symptoms still had apnea when PSG was repeated.

We found that 17 (38%) of the children in the group with OSA were obese or morbidly obese prior to therapy, which increased to 24 (53%) postoperatively, higher than the 2 of 22 and 5 of 50 reported previously by Brouillette et al10 and Guilleminault et al.11 This is a remarkable finding for a disease known to cause failure to thrive. A high incidence (37%-66%) of OSA in obese children has been reported,13-15 which suggests that OSA may also be an effect of obesity rather than a cause.

Obesity can cause OSA by narrowing the upper airway, as a result of fat deposition in the pharyngeal wall24-25 and external compression from superficially located fat in the subcutaneous area in the neck.26 Additionally, obesity reduces the size of the upper airway indirectly through a reflex mechanism secondary to a decrease in lung volume in obese patients.27 Enlarged tonsils and adenoids further compromise the airway.

Table 2. Postoperative BMI Score Changes

<table>
<thead>
<tr>
<th>Weight Category at Operation</th>
<th>No. of Children (%)</th>
<th>Postoperative BMI* Score Changes†</th>
<th>Increased</th>
<th>Decreased</th>
<th>No Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight</td>
<td>3 (6.7)</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Normal weight</td>
<td>25 (55.6)</td>
<td>16</td>
<td>9</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Obese</td>
<td>7 (15.5)</td>
<td>4</td>
<td>3</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Morbidity obese</td>
<td>10 (22.2)</td>
<td>7</td>
<td>3</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Total, No. (%)‡</td>
<td>45 (100)</td>
<td>28 (62)</td>
<td>15 (33.3)</td>
<td>2 (4.4)</td>
<td></td>
</tr>
</tbody>
</table>

* BMI indicates body mass index.
† Values in parentheses are percentages, rounded to the nearest tenth.
‡ Postoperatively, the mean BMI score of the whole group increased significantly (P = .004).

Figure 4. Height of each girl at the time of surgery and at the time of follow-up. (The percentile lines are drawn from published data.20)

Figure 5. Body mass index of each boy at the time of surgery and at the time of follow-up. (The percentile lines are drawn from published data.19)

Figure 6. Body mass index of each girl at the time of surgery and at the time of follow-up. (The percentile lines are drawn from published data.19)

Table 2. Postoperative BMI Score Changes

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Various treatments of OSA in obese children have been described. In a group of morbidly obese children with OSA, Kudoh and Sanai found that tonsillectomy and/or adenoidectomy and partial weight reduction resolved the OSA even if the patients remained morbidly obese. Willi et al. found sleep apnea in 5 of 6 morbidly obese children, and weight loss led to resolution of the apnea. Harris and Allen found that children with Prader-Willi syndrome had OSA. The severity of OSA was related to their weight, and the OSA diminished following weight reduction. In adults, the severity of OSA was also found to be proportionate to a patient's weight, and weight reduction resolved the OSA

Nap PSG, which was used to diagnose OSA in 20 children, might underestimate the severity of OSA in these children. Marcus et al. found that nap PSG underestimates the severity of OSA in children as compared with overnight PSG. However, OSA detected by nap PSG was always confirmed by overnight PSG, so that this factor does not affect our study.

Whether the increase in obesity we observed following treatment by tonsillectomy and/or adenoidectomy constitutes a substantial health risk in some cases awaits longer-term follow-up of such patients. Some health risks, such as the development of diabetes, coronary artery disease, and hypertension, are increased because of obesity itself, others, such as hyperventilation, cor pulmonale, heart failure, and death, could develop as a consequence of the recurrence of sleep apnea as a further consequence of the increasing obesity even in the absence of obstructing tonsils and adenoids. It is important to pursue ways to effectively treat obesity in children and its resultant OSA and thus eliminate this problem.

In summary, treatment of OSA by tonsillectomy and/or adenoidectomy in obese and morbidly obese children leads to clinical improvement of the OSA, but will not help with weight reduction and might even exacerbate the obesity. When obesity complicates OSA, we think that attention should be paid to reducing weight by such measures as exercise, diet, and behavioral therapy, in addition to treatment of the OSA.

Accepted July 20, 1998.

We thank Peter Homel, PhD, from the Scientific Academic Computing Center, State University of New York at Brooklyn, NY, for assistance with the statistical analysis.

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REFERENCES


