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LITERATURE REVIEW

Adenotonsillectomy for obstructive sleep apnea in obese children: A meta-analysis

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OBJECTIVE: The purpose of this study was to determine the effectiveness of adenotonsillectomy (T&A) for treating obstructive sleep apnea (OSA) in obese children.

DATA SOURCES: PubMed and Ovid databases.

REVIEW METHODS: A meta-analysis of studies that reported sleep parameters in obese children with OSA before and after T&A. Data were analyzed using the random effects model. Statistical significance was $P \leq 0.05$.

RESULTS: Data from four studies that included 110 children were analyzed. The mean sample size was 27.5 (range, 18-33). The mean body mass index *z* score was 2.81. The mean pre- and postoperative apnea-hypopnea index (AHI) was 29.4 (range, 22.2-34.3) and 10.3 (range, 6.0-12.2), respectively. The weighted mean difference between pre- and postoperative AHI was a significant reduction of 18.3 events per hour (95% confidence interval [CI], 11.2-25.5). The mean pre- and postoperative oxygen saturation nadir was 78.4 percent (range, 73.9%-81.1%) and 85.7 percent (range, 83.6%-89.9%), respectively. The weighted mean difference was a significant increase of the oxygen saturation nadir of 6.3 percent (95% CI, 3.9-8.7). Forty-nine percent of children had a postoperative AHI <5, 25 percent of children had a postoperative AHI <2, and 12 percent of children had a postoperative AHI <1.

CONCLUSIONS: T&A improves but does not resolve OSA in the majority of obese children. The efficacy and role of additional therapeutic options require more study. The high incidence of obesity in children makes this a public health priority.

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The prevalence of obesity in children has increased dramatically over the past 2 decades. Recent estimates suggest that 33 percent of children in the United States are overweight, and 17 percent are obese.[1] There is also evidence that the increasing prevalence of obesity is a global phenomenon.[2] The long-term health effects of obesity are well established, and obese children are much more likely to become obese adults.[3]

Obese children are at an increased risk for obstructive sleep apnea (OSA).[4,5] Previous studies have shown that up to 27 percent of asymptomatic obese children have moderate to severe OSA, and 25 percent to 40 percent of obese patients with sleep symptoms have OSA.[6,7] OSA in children is associated with a decreased quality of life as well as behavioral, neurocognitive, cardiovascular, metabolic, endocrine, and psychiatric complications.[8]

The etiology of OSA in obese children is thought to be secondary to a dysfunction in neuromotor control leading to airway collapse.[6] Adipose tissue deposited around the pharynx or subcutaneous tissue of the neck leads to compression of the pharynx and a reduction in its cross-sectional area.[9-11] Adenotonsillar tissue further narrows the airway, and clinical findings of lymphoid tissue hypertrophy are correlated with objective findings of obstructive apnea.[12]

Adenotonsillectomy (T&A) is the treatment of choice for OSA in children and has been shown to improve respiratory sleep parameters and quality of life in patients with OSA.[13] However, obesity increases the risk of persistent OSA after T&A.[14] Studies evaluating postoperative outcomes in obese children have been limited by a small sample size or a lack of objective data from polysomnography. The goal of the present study is two-fold: (1) to summarize and analyze the available data on the effectiveness of T&A for the treatment of OSA in obese children and (2) to highlight the likely direction of future research in this important field.

METHODS

Literature Search

A systematic literature search was performed using PubMed and Ovid databases for articles published between January 1, 1998, and January 1 2008. Search terms included key words "obesity" OR "obese" OR "overweight" AND "tonsillectomy" OR "adenotonsillectomy" OR "adenoidectomy". We performed additional searches using medical subject headings and the following key word terms: "outcome," "polysomnography," "sleep apnea," "apnea," and "children."

Inclusion criteria were as follows: (1) children age 0 to 18 years old; (2) obese children, body mass index (BMI) greater than the 95th percentile; (3) surgery limited to ton-

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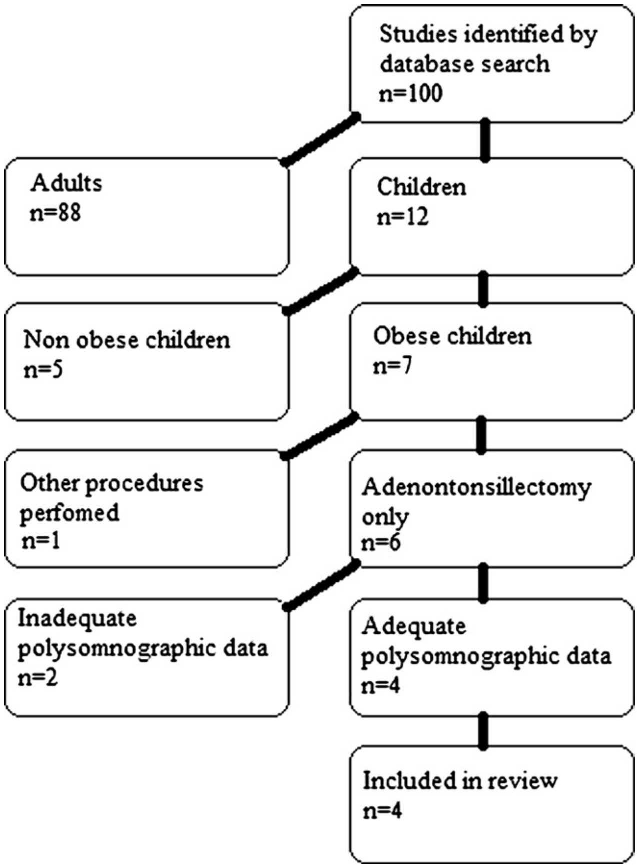

Figure 1 A flowchart for the review and selection of included studies.

sillectomy and adenoidectomy; and (4) polysomnography was performed before and after adenotonsillectomy. Studies that included children with neurologic or craniofacial disorders were excluded from the review. Language other than English was not an exclusion criterion. Article titles and abstracts were reviewed by the two authors independently to determine eligibility using a prepared form. The results were compared and discrepancies resolved by referring back to the original articles.

One hundred studies were identified in the literature search, 12 of which were relevant to this study. Four studies were identified that presented adequate data and met inclusion criteria[15-18] (Fig 1).

Statistical Analysis

The primary outcome measure was the apnea-hypopnea index (AHI) and/or the respiratory disturbance index (RDI), which were considered equivalent. Pre- and postoperative overnight oxygen saturation nadir was used as a secondary outcome measure. The BMI *z* score was calculated from raw data using the Centers for Disease Control and Prevention National Center for Health Statistics growth charts.[19] Data were analyzed by using the random effects model, and statistical analysis was performed using Comprehensive Meta Analysis 2.0 (Biostat, Englewood, NJ). Raw pre- and postoperative polysomnographic data, when available, were analyzed, and cure rates were calculated using the definition of cure as a postoperative AHI <5, <2, and <1. The pooled mean was calculated by adding the mean value for each group and dividing it by the number of groups. The weighted mean difference was calculated by using a random effects model by assigning studies a weight based on sample size and precision of the results. Data are presented as the difference in means between preoperative and postoperative AHI with 95% confidence interval (CI). I^2 is a measure of heterogeneity that describes the percentage of total variation across studies that is caused by heterogeneity rather than chance.[20] Values for I^2 of 25% suggest low, 50% moderate, and 75% high heterogeneity. Publication bias was evaluated by review of a funnel plot and calculation of the Egger's intercept to determine the degree of funnel plot asymmetry.[21] A P value ≤ 0.05 was considered significant.

RESULTS

Individual patient data were available from three of the four studies. Three studies reported pre- and postoperative RDI and one study reported AHI as the primary outcome measure. Three studies were prospective and one retrospective. Two prospective studies included a control group of nonobese children.

The data from 110 children were analyzed (Table 1). The mean sample size was 27.5 children (range, 18-33), and the pooled mean patient age was 8.4 years (range, 7.3-9.3). The pooled mean preoperative BMI was 29.7 (range, 28.3-32.1), and the mean preoperative BMI *z* score was 2.81 (99.75th

Table 1
A summary of nonrespiratory information from the included studies

Study	Study design	Sample size	Mean age (y)	Mean BMI	Mean BMI *z* score	Mean time from T&A to PSG (mo)
Mitchell and Kelly,[15] 2004	Prospective	30	9.3	28.6	2.43	5.6
Shine et al,[16] 2006	Retrospective	18	7.8	32.1	3.20	3.0
O'Brien et al,[17] 2006	Prospective	29	9.0	NR	2.60	NR
Mitchell and Kelly,[18] 2007	Prospective	33	7.3	28.3	3.14	5.7

NR, not reported; *PSG*, polysomnography; *y*, years; *mo*, months.

Table 2
A summary of data from included studies

Study	Mean preoperative AHI	Mean postoperative AHI	Mean preoperative SaO_2 nadir (%)	Mean postoperative SaO_2 nadir (%)	Criteria for cure	Percent success
Mitchell and Kelly,[15] 2004	30.0	11.6	79.8	84.4	RDI <5	46
Shine et al,[16] 2006	34.3	12.2	73.9	84.8	RDI <5	39
O'Brien et al,[17] 2006	22.2	11.3	81.1	89.9	RDI <5	45
Mitchell and Kelly,[18] 2007	31.1	6.0	78.9	83.6	AHI <2	24

SaO_2, arterial oxygen saturation.
Percent success = percentage of children cured of OSA based on polysomnographic criteria set by the author.

percentile). The time from T&A to postoperative polysomnography was available in three studies, and the pooled mean was 4.8 months (range, 3-5.7).

Pooled mean pre- and postoperative AHI was 29.4 (range, 22.2-34.3) and 10.3 (range, 6.0-12.2), respectively (Table 2). All studies showed a mean improvement in the AHI value after T&A with a range of 10.9 to 25.1 (Fig 2). The weighted mean decrease in AHI after T&A was 18.3 events per hour (95% CI, 11.2-25.5; $P < 0.00001$). The value of I^2 was 44 percent, indicating a moderate degree of heterogeneity between the studies.

The mean pre- and postoperative oxygen saturation nadir was available for all four studies and was 78.4 percent (range, 73.9%-81.1%) and 85.7 percent (range, 83.6%-89.9%), respectively (Table 2). The weighted mean increase in oxygen saturation nadir was 6.3 percent (95% CI, 3.9-8.7; $P < 0.00001$) (Fig 3). The value of I^2 was 1.1 percent, indicating low heterogeneity and a low degree of variation across studies.

Three studies defined success as a postoperative AHI less than 5, and one study defined success as an AHI less than or equal to 2. Pooled mean percent success using the study criteria was 38.5 percent (range, 24%-46%). Raw pre- and postoperative polysomnographic data were available for 81 children. Forty-nine percent of children had a postoperative AHI less than 5, 25 percent had an AHI less than 2, and 12 percent had an AHI less than 1 (Table 3).

Publication bias was evaluated by a visual review of a funnel plot in which standard error is plotted against the difference in means. Publication bias was considered unlikely for both AHI and oxygen saturation nadir from the graphical review. Egger's intercept was 2.3 ($P = 0.51$) for AHI and 1.39 ($P = 0.57$) for oxygen saturation nadir, suggesting a low likelihood of publication bias.

DISCUSSION

This study shows that T&A significantly reduces the severity of OSA in obese children. However, T&A is rarely curative, and 88 percent of obese children have evidence of persistent OSA when using the definition for OSA as an AHI greater than one.[22,23] Similarly, the oxygen saturation nadir for obese children in this study significantly improved after T&A but did not reach the mean nadir of 94.6 percent reported in healthy children during sleep.[23]

A recent meta-analysis of studies evaluating T&A for OSA in normal-weight children reported a mean reduction in AHI of 13.9, a mean postoperative AHI of 2.4, and a cure rate of 83 percent.[24] The meta-analysis did not include a recent study of 79 normal-weight children undergoing T&A for OSA that showed a mean change in AHI from 27.5 to 3.5.[25] The present study demonstrates that T&A in obese children also leads to a reduction in AHI but is less likely to result in a cure. This may be partially because of a higher preoperative AHI in obese compared with normal-weight children because severe OSA is less likely to be cured by T&A independent of obesity.[25] O'Brien et al[17] reported no increased risk of persistent OSA in obese children after

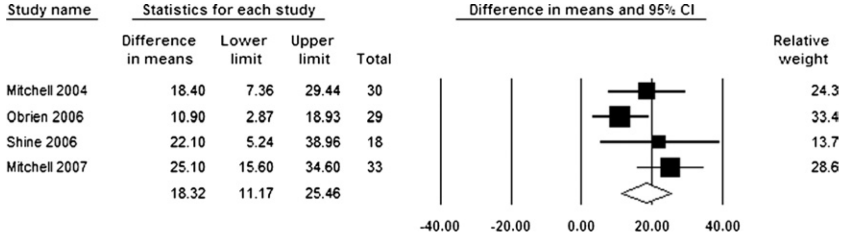

Figure 2 A forest plot of mean pre- and postoperative AHI. The location of the black squares represents the mean change from pre- to postoperative AHI. The size of the square is proportional to the weight of the study, and the horizontal bars represent the 95 percent CI for mean change in AHI. Heterogeneity measured by I^2 = 44 percent.

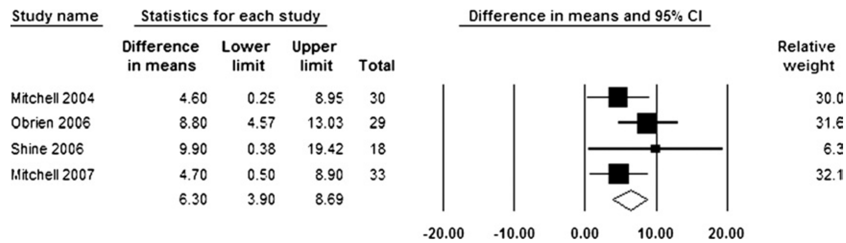

Figure 3 A forest plot of mean pre- and postoperative oxygen saturation nadir. The location of the black squares represents the mean improvement between pre- and postoperative oxygen saturation nadir. The size of the square is proportional to the weight of the study, and the horizontal bars represent the 95 percent CI for mean change in oxygen saturation nadir. Heterogeneity measured by $I^2 = 1.1$ percent.

controlling for preoperative disease severity. However, Mitchell and Kelly[18] reported that increased BMI and preoperative AHI were risk factors for persistent OSA after T&A. The mechanism of OSA in obese children is likely to be caused by multilevel obstruction, similar to that of obese adults, and therefore less amenable to cure via T&A in the majority of children.

Several studies were excluded because of a lack of pre- and postoperative polysomnography. Full-night polysomnography is the most accurate and the only objective method of diagnosing OSA in children.[26] Caregivers cannot reliably differentiate primary snoring from OSA using symptom scores.[27] However, the limited availability and expense of polysomnography precludes the full evaluation of many children before surgical intervention. Our results suggest that postoperative polysomnography should be performed routinely in obese children after T&A for OSA because the majority of obese children have persistent disease and may require long-term management of their sleep disorder.

Polysomnographic data are one of several outcome measures for obstructive sleep apnea in children. Hypoxia during sleep is associated with adverse effects on behavior and cognition in children.[8,28] Thus, quality of life, behavior, and health care utilization are additional outcome measures used in evaluating therapy for obstructive sleep apnea. Improvements in both quality of life and behavior after adenotonsillectomy for OSA in children have been shown.[29,30] Very few studies have looked specifically at quality of life or behavioral outcome measures for OSA in obese children after adenotonsillectomy. Future research is clearly needed in this field.

Table 3
The outcome of T&A based on a variety of definitions for pediatric OSA (n = 81)

Definition of pediatric OSA	Number of children preoperative	Number of children postoperative	Percentage of children who normalized
AHI ≥5	75	38	49
AHI ≥2	81	61	25
AHI ≥1	81	71	12

The treatment of residual OSA in obese children requires consideration of the level of airway obstruction. The relief of nasal obstruction may obviate the need for therapy in children with residual mild OSA.[31,32] CPAP should be considered in obese children with persistent OSA after T&A. However, there are only a few studies that have looked at the efficacy of CPAP in obese children. Shine et al[16] treated 10 children with persistent OSA successfully with CPAP. The authors noted no significant difference in age, BMI *z* score, or preoperative RDI between patients requiring postoperative CPAP and those who did not. Waters et al[33] reported the use of CPAP in 80 children under 15 years old, six of whom were obese. Sixty-five children had previously undergone T&A, and five had a tonsillectomy with a limited adenoidectomy or an adenoidectomy alone. Sixty-three children used CPAP successfully, and RDI decreased from 27.3 ± 20.2 to 2.6 ± 2.7 (mean ± SD) including some obese children. Massa et al[34] reviewed the use of nasal CPAP in 66 children age 0 to 19 years with moderate to severe OSA in whom surgical treatment was not possible. Most had craniofacial or neuromuscular disorders, and four were obese. CPAP was technically successful in 74 percent of patients. Of these patients, 86 percent established long-term CPAP therapy, and 4 percent derived no benefit from CPAP. There is, therefore, some evidence that CPAP is effective in obese children with residual obstruction after T&A, and this field also requires further studies.

Weight loss is the primary therapy for adults with OSA.[35,36] Few studies have looked at the role of weight loss for children with OSA. Willi et al[37] evaluated the efficacy of a low-calorie diet in six morbidly obese adolescents. The average weight loss was 18.7 kg in 20 weeks, and the mean apnea index decreased from 14.1 to 1.6. Davis et al[38] studied 100 children who participated in supervised daily exercise for 10 to 15 weeks and found that regular vigorous exercise improved snoring in obese children despite no change in BMI *z* score. Kalra et al[39] noted a decrease in mean AHI from 9.1 to 0.65 in 10 obese adolescents after bariatric surgery. Thus, weight loss may be the most effective therapy in resolving pediatric OSA in obese children but is limited by compliance issues.

One of the arguments for treating OSA in obese children is to improve their quality of life so they are able to exercise more and reduce their weight. However, perhaps surprisingly, two articles describe an increase in BMI after surgical therapy.[40,41] Other studies have shown an insignificant change in BMI after surgery for OSA in children.[15,42] Even though patients undergoing T&A show improvement in the respiratory parameters of OSA, the adverse effects of obesity are likely to persist. This is especially important in light of evidence that the BMI may actually increase after T&A for pediatric OSA in obese children, and the increase may lead to worsening of respiratory parameters over time.

Uvulopalatopharyngoplasty (UPPP) has been successfully performed in adults with OSA, but there are limited data available on this procedure in children. Three studies have been published on neurologically impaired children undergoing UPPP,[43-45] and the procedure has shown to be efficacious in this population. The role of UPPP in obese children is unknown, and further study is required to determine the indications for further surgical interventions in obese children after T&A.

The strengths of this study include a sample size larger than any prior study evaluating the outcome of T&A in obese children. The study also reflects current trends in the diagnosis of OSA in children using more conservative AHI values. Finally, the study provides insight regarding the prognosis for obese children with OSA after T&A.

This study is subject to limitations including heterogeneity in surgical technique, patient population, and the format of the outcome data. The effect of heterogeneity is reduced by the random effects model but cannot be eliminated. Publication bias is difficult to detect when few studies with small sample sizes are reviewed. Additionally, there is a patient selection bias because children referred for consideration of surgical therapy may have more severe disease. Finally, it is unknown why T&A is effective in curing some obese children with OSA and not others. It is equally unknown whether the degree of obesity correlates to preoperative OSA severity or to persistent postoperative OSA. Identifying variables that predispose children to persistent OSA would allow for effective postoperative planning.

The aim of this meta-analysis is to define changes in polysomnographic values after T&A in obese children. The combined data and 95 percent confidence intervals quantify the impact of surgery and increase the external validity such that these results may be predictive of patients with similar preoperative characteristics.

One potential conflict is the senior author's (RM) involvement in two of the studies included in the meta-analysis. However, the other two studies were performed in a different country, and a low-to-moderate degree of heterogeneity was noted between all four studies. The presence of similar outcomes despite different methods and patient populations reinforces that the results are unlikely to reflect bias.

CONCLUSIONS

Sleep improves but rarely normalizes after T&A in obese children with OSA. Up to 88% of obese children have persistent OSA after T&A. The efficacy and role of additional therapeutic options requires more study. The high incidence of obesity in children makes this a public health priority.

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AUTHOR CONTRIBUTIONS

Dary J. Costa, data collection, data analysis, writer; **Ron Mitchell**, study design, data analysis, writer.

FINANCIAL DISCLOSURE

None.

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