Snoring and Obstructive Sleep Apnea in Children

A 6-Month Follow-up Study

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Background: Snoring children may present symptoms suggestive of obstructive sleep apnea syndrome (OSAS). Different and controversial methods to establish the diagnosis and to choose the treatment modalities have been proposed.

Objectives: To study children with symptoms raising the suspicion of OSAS with overnight polysomnography (PSG). To evaluate the efficacy of adenotonsillectomy as treatment of pediatric OSAS and to elucidate the natural history of OSAS and primary snoring.

Design: A controlled, prospective, nonrandomized clinical trial.

Setting: Academic medical center.

Subjects: Fifty-eight snoring but otherwise healthy children aged 3 to 10 years with symptoms suggestive of OSAS underwent PSG twice, 6 months apart. Thirty healthy children served as controls.

Results: Twenty-seven children had OSAS with an obstructive apnea/hypopnea index greater than 1, while 31 had primary snoring. There were statistical differences in the symptoms and signs among the 3 study groups. Adenotonsillectomy was curative in the 21 children with OSAS who were operated on. Obstructive apneas and hypopneas in the healthy, nonsnoring children were almost nonexistent in this study.

Conclusions: Half of the children or fewer with symptoms suggestive of OSAS actually had the condition. Clinical symptoms may raise the suspicion, but it is not possible to establish the diagnosis without PSG. Because snoring and obstructive symptoms may resolve over time, a normal PSG finding may help the clinician decide on an observation period. Adenotonsillectomy is curative in most cases of pediatric OSAS. Obstructive symptoms may continue after adenoidectomy alone.


MORE THAN 10% of preschool-aged children snore regularly.¹ However, obstructive sleep apnea syndrome (OSAS), a condition related to snoring with possible serious complications if untreated,² is less common. In a recent study, the prevalence of OSAS was estimated at about 3%.³ There is no doubt about the seriousness of pediatric OSAS, but no universally accepted criteria have been developed for its diagnosis and treatment. Much lower apnea indexes and shorter apneas than those considered acceptable for adults² have been recognized as indicators of OSAS in children.³,⁶ However, the natural history of mild OSAS and primary snoring (PS), ie, snoring with obstructive symptoms but nonpathologic polysomnography (PSG) findings, is not well known.⁷ The most common cause for snoring and OSAS in normal children is adenotonsillar hypertrophy.³

This follow-up study was designed to study children with symptoms suggestive of OSAS, the presumptive cause of the symptoms being adenotonsillar hypertrophy. These signs and symptoms were related to the overnight polysomnographic (PSG) findings. The effect of adenotonsillectomy as a treatment of the syndrome was evaluated in a repeated study 6 months later in the cases with a PSG finding indicative of OSAS. The children with PS who underwent no therapeutic interventions also received a follow-up examination 6 months later.

RESULTS

FIRST VISIT

Twenty-seven of the 58 children had pathologic OAHIs higher than 1 (OSAS group), while the remaining 31 children had nonpathologic PSG recordings (PS group). The main general findings for both groups and the control group are given in Table 1.
SUBJECTS, METHODS, AND MATERIALS

We reviewed all referrals from primary health care sites to the Department of Otorhinolaryngology in the Oulu University Hospital for adenoidectomy, tonsillectomy, or both, for children aged 3 to 10 years from 1994 to 1997. Parents of children with reported snoring, apneas, or difficult nighttime breathing completed a detailed questionnaire regarding their child’s nighttime and daytime symptoms. Children with known upper airway anomalies, an underlying disease predisposing to upper airway obstruction, asthma, or perennial allergy were excluded at this point.

Based on the questionnaire responses, the children with obstructive symptoms underwent an ear, nose, and throat evaluation. Where recognized, upper airway anomalies and abnormal facial morphologic characteristics led to exclusion. After the patient history had been thoroughly updated and the questionnaire responses checked, 78 children (43 boys; mean age, 5.7 years; range, 2.4-10.5 years) fulfilled the inclusion criteria (ie, had symptoms suggestive of OSAS, were regular snorers, and/or had apneas during sleep, the presumable cause for the symptoms being adenotonsillar hypertrophy). If a child had a previous adenoidectomy, this did not lead to exclusion.

The children were examined twice, 6 months apart. Fifty-eight children (31 boys; mean age, 5.7 years) participated in the entire follow-up study. Fifteen children did not participate in the second part of the study; in 4 cases, there was a protocol violation, and in 1 case, technical problems. Thirty nonsnoring, healthy children (17 boys; mean age, 7.1 years) were also examined for comparison and to establish normative values for overnight PSG.

More children in the OSAS group than in the PS and control groups had had a previous adenoidectomy. The average palatine tonsillar size was also greater in the OSAS group, with 93% (25/27) having enlarged tonsils, while 68% (20/31) had them in the PS group and only 3% (1/30) in the control group.

SYMPTOMS

On the symptom scale, the children with OSAS had a median value of 12 (range, 4-14), those with PS, 9 (range, 2-14), and the controls, 1 (0-7). The mean OSA score was 3.1 for the OSAS group, 2.1 for the PS group (P = .03 for difference from OSAS group), and −3.7 for the control group (P < .01 for difference from OSAS group). The constancy of snoring and the frequency of nights when the parents had detected apneas differed between the OSAS and PS groups (Table 1).

Nighttime and daytime mouth breathing and odd sleeping positions were significantly less common in the control group than in the OSAS and PS groups. Restless sleep was significantly more typical of the OSAS than PS group, 54% vs 22% (P = .03), being nonexistent in the control group (P < .01). Excessive daytime somnolence, the hallmark for OSAS in adults, was rare, and only 6 children (23%) in the OSAS group suffered from it daily.

POLYSOMNOGRAPHY

The main polysomnographic results of the OSAS, PS, and control groups are shown in Table 2.

Inclusion of apneas shorter than 10 seconds into the OAH1 would not have changed the categorization. Of all obstructive events lasting longer than 10 seconds, hypopneas accounted for 76.6% in the OSAS and 98.7% in the PS groups. Short obstructive events were much less common than those lasting longer than 10 seconds in both groups. Central apneas did not differ among the 3 groups. There were no significant differences in apneic events between the PS group and the control group. Four children, 3 of them in the OSAS group, had 1 central apnea each lasting longer than 20 seconds with desaturation below 90%.

The 4% and 10% desaturation episodes per hour indexes, which indicate events with 4% and 10% drops in the oxygen saturation level, were manually checked. Both indexes were higher for the OSAS than PS group (Table 2). Both groups had higher 4% indexes than the control group (Table 2). The average width of the saturation distribution, which shows the variation in oxygen saturation between the highest and lowest percentiles, was greater in the OSAS than the PS group (P = .05), while there was no significant difference between the PS and control groups. Periods of cardiac arousals were more frequent in
midline; and grade 4 tonsils have a maximum distance of 4 mm between them. Tonsils graded 3 and 4 were considered enlarged in this study.

POLYSOMNOGRAPHY

Polygraphsographic monitoring was done in the hospital under the surveillance of a trained nurse. The children were accompanied by a parent through the night.

A 6-channel computerized PSG was used, developed by the Department of Clinical Neurophysiology, with leads for an oronasal thermistor, a thoracoabdominal strain gauge, pulse oximeter, body-position sensor, leg electromyograph, and static charge-sensitive bed. Although the analysis program included a possibility of automatic analysis of the nighttime events, all recordings were manually checked by the same clinical neurophysiologist (U.T.).

In this study, the apneas lasting for 5 to 10 seconds and those lasting longer than 10 seconds were counted separately. An obstructive apneic episode was defined as a total cessation of oronasal airflow, as detected by the thermistor, in the presence of continuous breathing efforts, as revealed by the thoracoabdominal strain gauge or the static charge-sensitive bed, and hypopnea as a reduction of 50% or more in the airflow signal, as proposed by Gould et al.11 A central apnea was defined as a cessation of the airflow in the absence of breathing efforts. Central apneas followed by a deep sigh were disregarded. Mixed apneas and hypopneas starting with a central component and continuing with an obstructive one were classified into the obstructive apnea category.

In this study, an obstructive apnea/hypopnea index (OAHI) greater than 1, including apneas/hypopneas lasting for 10 seconds or more, was considered abnormal and used as the criterion for OSAS. Central apneas were not included in this index. Since there was no electroencephalographic or electro-oculographic tracing, arousals could not be quantified reliably. To detect periods of increased upper airway respiratory resistance outside of clinically significant apneas and hypopneas, an indirect method was used. The PSG recordings were manually checked for periodic hypopneas with a less than 50% decrease in the oronasal signal amplitude linked with a pulse increase at the termination of hypopneas. These respirotonically induced pulse increases evidently indicate arousals,12 and they may be linked to increased upper airway resistance syndrome. According to Noda et al.,13 these pulse increases are cardiac arousals.

TREATMENT PROTOCOL

The children with OAHI of 2 or higher underwent tonsillectomy (and adenoidectomy, if not previously performed) shortly after the first visit. The children with OAHI s lower than 2 were followed up without intervention. Two children with OAHI s above 2 were included in this group (2.6 and 2.7, respectively) because, owing to ongoing speech therapy, the phoniatrician suggested that surgical therapy should be avoided.

STATISTICAL ANALYSIS

The data were processed using the SPSS for Windows software (version 6.1; SPSS Inc, Chicago, Ill). The t test for 2 independent samples and paired samples was applied for the normally distributed data. The nonparametric Mann-Whitney U test was applied for the nonnormally distributed data. The Mantel-Haenszel $y^2$ test was applied to the ordinal variables. The correlation coefficients were counted with the Pearson 2-tailed test.

the OSAS group ($P = .01$), while there was also a significant difference between the PS and the control groups ($P < .01$). The correlation coefficient between OAHI and apneas starting with a central component and continuing with an obstructive one was counted as the criterion for OSAS. Central apneas were not included in this index. Since there was no electroencephalographic or electro-oculographic tracing, arousals could not be quantified reliably. To detect periods of increased upper airway respiratory resistance outside of clinically significant apneas and hypopneas, an indirect method was used. The PSG recordings were manually checked for periodic hypopneas with a less than 50% decrease in the oronasal signal amplitude linked with a pulse increase at the termination of hypopneas. These respirotonically induced pulse increases evidently indicate arousals, and they may be linked to increased upper airway resistance syndrome. According to Noda et al., these pulse increases are cardiac arousals.

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FOLLOW-UP VISIT

Surgical Group

Twenty-one children with OAHI greater than 2 underwent adenotonsillectomy shortly after the first visit. The results are shown in Table 3; 73% of the children operated on (16/21) had had previous adenotomectomies, which had not resolved the obstructive symptoms, or the symptoms had begun after the adenotomectomy. At the operation evaluated for this study, the epipharynx was checked, and none of the children appeared to have substantial regrowth of adenoidal tissue.

Clinically, all children benefited from the operation. All the symptoms of 16 children disappeared. Five children continued to have some minor symptoms. None had any need for additional treatment. On the symptom scale, the median score for the children operated on dropped from 12 (range, 7-15) to 1 (range, 0-6). The mean OSA score had changed significantly from 3.4 to -3.1 ($P < .01$). The only child with a postoperative OSA score greater than 0 was a boy, the only one to still snore on most nights. The snoring was thought to be lighter, however, and he was more alert in the daytime. The other 4 children continued to snore irregularly; 1 of them reported still having laborious breathing at night.

There was a distinct improvement in the PSG results after the operation (Table 3). All indexes except those for central apneas were significantly improved. The mean OAHI dropped from 6.9 to 0.3 ($P < .01$). The remaining obstructive events were also improved, as were all hypopnea indexes. Two children still had pathologic OAHI s postoperatively: the boy mentioned above had a score of 5.1 (8.9 at first measurement) and a girl had 1.0 (11.8 at first measurement). All the other children had OAHI s of less than 1. The periods of cardiac arousals decreased significantly, but did not disappear.

Conservative Group

According to the responses to the follow-up questionnaire, 6 children had developed more symptoms, and their mean (SD; range) OSA scores had risen from 1.2 (2.3; −3.1 to 3.3) to 2.4 (2.4; −2.4 to 4.0), though they all had normal PSG findings (OAHI = 0). Fifteen children had unchanged symptoms, while 16 reported decreased symptoms. The mean indexes in this group had not changed significantly over the follow-up period (Table 3). Two children had abnormal OAHI s at the second visit: 1 was a boy with unchanged OAHI 2.6. For 1 child, the OAHI increased from 0.5 to 1.0.

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As the number of adenoidectomies and tonsillectomies performed declines because of the increased use of antimicrobial therapy, the risk for obstructive sleep disorders may increase in the pediatric population.14 Children who snore and have symptoms suggestive of OSAS are frequently encountered in clinical practice.

The natural history of mild OSAS is not well known, though it may be rather uneventful, as also suggested by our results. It is not known, either, how high apnea indexes and a long duration of OSAS are capable of producing complications.

The pathologic apnea index for children is reported to be greater than 15 when real obstructive apneas lasting for at least 2 to 3 normal breath cycles are counted into...
the index. Obstructive apneas seem to be rare in normal, nonsnoring children, which is confirmed by the results of this study, where only occasional short obstructive hypopneas occurred in the control group. Hypopneas accounted for most obstructive events in this study. In children, obstructive events often consist of periods of prolonged hypoventilation instead of clear-cut apneas, which are rather a culmination of the hypopnea syndrome. In this study, OAHIs higher than 1 were considered abnormal, when obstructive and mixed apneas and hypopneas lasting for 10 seconds or more were included in the index. The criterion of 10 seconds for apneas, which is probably too high for children because children have a smaller respiratory capacity and is also quite expensive. Simpler montages suitable for home use have been shown to be sensitive and yield reliable information of the nighttime events. The PSG device used in this study meets most of the minimum requirements of the consensus statement of the American Thoracic Society (1996). In this study separate arousals could not be quantified owing to a lack of electroencephalographic channels, while periods of cardiac arousals were quantified from the PSG recordings. The intervals of periodic hypopneas with a less than 50% decrease in oronasal signal amplitude linked to pulse increase at the termination of the hypopneas were manually checked. These respiratorily induced pulse increases evidently indicate arousals, and may be linked to increased upper airway resistance. Video recording, which helps detect most arousals was not used on all patients, but the nurse on duty reported in writing both all interventions with reattachments of sensors and waking periods. The role of arousals in pediatric OSAS remains unclear, and arousals are probably not an important mechanism in the termination of respiratory events. Hence, electroencephalographic criteria are not essential for determining the clinical severity of OSAS in the pediatric population. In our study, none of the children with PS showed excessive daytime somnolence, and only 23% in the OSAS group did so, supporting the assumption that interruption of sleep is not a major problem for these children.

Central apneas were excluded from the index, whether or not they were accompanied by a desaturation of greater than 10%. Central apneas occur in children without obstructive symptoms, and in this study they were seen equally often in children with OSAS and PS and in controls. That pediatric OSAS is equally common in both sexes is supported by the results of this study. The age distribution of the studied children was quite even. All the children participating in this study had symptoms of various degrees, causing a suspicion of OSAS. The symptom score for OSAS developed by Brouilette and coworkers did not discriminate very well between the children with OSAS and those with PS, as has been noted earlier. Though only 1 child in the OSAS group had an OSA score lower than 3.5, children with PS had OSA scores higher than 3.5, which are considered by Brouilette et al diagnostic for OSAS. The

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**Table 3. Polysomnography Results of the Children Operated On (OAHÍ ≥2) and Not Operated On**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Subjects Operated On (n = 21)</th>
<th>Subjects Not Operated On (n = 37)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Visit 1</td>
<td>P‡</td>
</tr>
<tr>
<td>Age, y</td>
<td>5.6 (2.1) (2.4-10.5)</td>
<td>...</td>
</tr>
<tr>
<td>Earlier adenoidectomy, %</td>
<td>76 (16/21)</td>
<td>...</td>
</tr>
<tr>
<td>OSA score</td>
<td>3.4 (1.3) (-0.3-4.0)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Apnea index</td>
<td>7.5 (4.4) (2.1-16.8)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Obstructive apnea/hypopnea index</td>
<td>6.9 (4.1) (2.0-14.6)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Obstructive apnea index</td>
<td>1.4 (1.2) (0.1-4.2)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Obstructive hypopnea index</td>
<td>5.5 (3.9) (0.4-14.5)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Central apnea index</td>
<td>0.7 (1.3) (0-5.8)</td>
<td>.08</td>
</tr>
<tr>
<td>Obstructive apnea index</td>
<td>1.6 (1.6) (0.5-7.7)</td>
<td>.001</td>
</tr>
<tr>
<td>Short central apnea index</td>
<td>1.6 (1.1) (0.3-5.0)</td>
<td>.23</td>
</tr>
<tr>
<td>4% Oxygen desaturation events per hour (ODI4)</td>
<td>5.0 (7.4) (0-29.6)</td>
<td>.04</td>
</tr>
<tr>
<td>10% Oxygen desaturation events per hour (ODI10)</td>
<td>0.2 (0.4) (0-1.4)</td>
<td>.02</td>
</tr>
<tr>
<td>Saturation distribution, %</td>
<td>3.4 (1.9) (0.8-10.2)</td>
<td>.19</td>
</tr>
<tr>
<td>Increased respiratory resistance, min/h</td>
<td>1.4 (1.3) (0-2.46)</td>
<td>.001</td>
</tr>
<tr>
<td>Total sleeping time, h</td>
<td>8.7 (0.8) (7.0-10.0)</td>
<td>.21</td>
</tr>
</tbody>
</table>

*OAHÍ indicates obstructive apnea/hypopnea index; ellipses, not applicable; and ODI, oxygen desaturation index. Unless otherwise indicated, data are mean (SD) (range).
†Two children with pathologic OAHÍs are included in the group not operated on because of contraindications for surgery.
‡Difference before and after surgery.
§Difference between those operated on and those not at the first visit.
wide range of OSA scores in both groups confirms the difficulty of establishing a diagnosis on a clinical basis, and in many study series, only half or fewer of the children with suspected OSAS are proved to have it by the PSG criteria. Most of the daytime and nighttime symptoms reported as typical for children with OSAS, ie, morning headaches, bed wetting, profuse sweating, sleepwalking, nightmares, irritability, concentration difficulties, and frequent nighttime awakenings, were quite similar among the OSAS and PS groups, while most of these symptoms were significantly less common in the control group. Only the habitual snoring, apneas, and restless sleep detected by the parents were significantly more common in the OSAS than the PS group.

Tonsillectomy and adenoidectomy are effective in the treatment of pediatric OSAS, especially when there is no underlying predisposition. This fact was confirmed by the present results. All children who were operated on improved in terms of symptoms and PSG results. Only 1 boy had clearly pathologic OAHI postoperatively, with some but notably milder obstructive symptoms. There was also a decline in mild hypopnea-induced cardiac arousals, but no total disappearance. Could this result indicate that these children had a predisposition for obstructive sleep disorders? Why do some children snore, while some do not, with apparently similar facial morphologic characteristics and pharyngeal status? It has been proposed that primary snoring, upper airway resistance syndrome, and OSAS constitute a continuum. Most children in the conservative group had periods of obstructed breathing causing cardiac arousals equally or more often than the children with OSAS postoperatively. Is there a similar tendency toward obstructed breathing in these children, but some difference in the upper airway regulatory reflexes? Deficit in the central ventilatory drive is probably not a major factor in the pathogenesis of OSAS.

Normal PSG findings can help the clinician justify an observation period for the snoring child, since the stability of the PSG findings in the conservative group seems to indicate that, during a short interval, primary snorers do not have a tendency to develop OA, supporting the earlier findings. The finding that snoring may be restricted to a period of childhood, and that almost half of the children in the conservative group showed spontaneous regression of the obstructive symptoms over the follow-up period, further supports the idea of an observation period.

A history of previous adenoidectomy in snoring children has been shown to be a risk factor for OSAS. In our study, most of the children with OSAS had had earlier adenoidectomies, which indicates that adenoidectomy alone is not always effective in the treatment of pediatric OSAS. Nor did adenoidectomy seem to have reconstituted nighttime nasal breathing in these children, which further indicates that oropharyngeal factors are dominant in producing obstructive symptoms.

Half or fewer children with symptoms suggestive of OSAS actually have the syndrome. Clinical symptoms may raise this suspicion, but the diagnosis cannot be established without PSG. A normal PSG finding may help the clinician decide on an observation period, since snoring and obstructive symptoms may resolve over time. Adenotonsillectomy is, in most cases, a curative treatment of pediatric OSAS, while adenoidectomy alone does not seem to be enough.

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