Sleep Characteristics Following Adenotonsillectomy in Children With Obstructive Sleep Apnea Syndrome*

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Objective: **To compare the effect of adenotonsillectomy on rapid eye movement (REM)- and non-REM–related respiratory and sleep architecture characteristics in children with obstructive sleep apnea syndrome (OSAS).**

Study design: **This prospective study evaluated 36 children (median age, 6.9 years; range, 1.8 to 12.6 years) with OSAS using polysomnography before and a few months after adenotonsillectomy. Primary outcomes included the number of obstructive apnea and hypopnea and arousals per hour of sleep.**

Results: **At 4.6 months (range, 1 to 16 months) after adenotonsillectomy, there was a significant improvement of all respiratory parameters. The median respiratory disturbance index (RDI) decreased from 4.1/h (range, 0 to 85/h) to 0.9/h (range, 0 to 13/h) after adenotonsillectomy ($p < 0.0001$). The median non-REM RDI decreased from 3.0/h (range, 0 to 89/h) to 0.4/h (range, 0 to 13/h) [$p < 0.001$] as compared with REM RDI, which decreased from 7.8/h (range, 0 to 69/h) to 2.3/h (range, 0 to 54/h) after adenotonsillectomy ($p < 0.01$). Median arousal index decreased following adenotonsillectomy from 17.5/h (range, 7 to 57/h) to 14.0/h (range, 6 to 47/h) [$p < 0.03$].**

Conclusions: **Adenotonsillectomy resulted in a greater improvement in non-REM RDI as compared with REM-RDI, and a decrease in the number of arousals.**

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Key words: adenotonsillectomy; arousal; rapid eye movement sleep; sleep apnea

Abbreviations: ASDA = American Sleep Disorders Association; DI = desaturation index; OSAS = obstructive sleep apnea syndrome; RDI = respiratory disturbance index; REM = rapid eye movement; Sao_2 = arterial oxygen saturation

Obstructive sleep apnea syndrome (OSAS) is a disorder of breathing during sleep characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction that disrupts normal sleep patterns and normal ventilation during sleep. The prevalence of OSAS in children is estimated at 1 to 2%.[1,2] The most common cause of OSAS in children is adenotonsillar hypertrophy; therefore, adenotonsillectomy is the treatment of choice.

Previous reports[3–5] on changes in sleep characteristics after adenotonsillectomy in children with OSAS emphasized the changes in clinical outcome measurements and in oxygen saturation. Initial reports[6,7] including polysomnography indicated a 75 to 100% cure of OSAS following adenotonsillectomy. Nieminen et al[8] reported a 95% cure rate for a group of 21 children after adenotonsillectomy. In a more detailed prospective study, Suen and coworkers[9] showed that children with OSAS had a lower respiratory disturbance index (RDI) after surgery. All 26 children showed improvement, although 4 children still had an RDI > 5/h after surgery. They have shown that children with OSAS had a higher arousal index that improved following adenotonsillectomy.

The two main possible causes of the complications of OSAS in adults are sleep-associated gas exchange abnormalities and sleep fragmentation. Sleep fragmentation is not well defined in children. Goh et al[10]

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showed that OSAS in children is not associated with changes in sleep architecture, although they may present a higher number of arousals during sleep.

The purpose of the present study was to assess the changes in the respiratory and sleep architecture parameters before and after adenotonsillectomy. As OSAS in children is mostly related to rapid eye movement (REM),[10] we compared the effect of treatment on REM- and non-REM–related respiratory disturbance.

Materials and Methods

Patients

Children aged 1 to 13 years who were referred to the Sleep-Wake Disorder Unit for evaluation of possible OSAS were recruited. Children were referred from the pediatric pulmonology and pediatric otolaryngology outpatient clinics of the Soroka University Medical Center, the only major hospital for the Negev area, covering a population of approximately 750,000. Inclusion criteria included children with a history consistent with sleep-disordered breathing who had an RDI > 1/h, whose parents agreed to return for a follow-up study a few months after adenotonsillectomy. Children with other chronic medical illnesses such as asthma, genetic disorders, or facial anomalies were excluded. The institutional ethics committee approved the study protocol, and the parents of all children gave written informed consent for their children to participate in the study.

The diagnostic evaluation included a detailed clinical history by a standard questionnaire,[11] physical examination by a pediatric pulmonologist and a pediatric otolaryngologist, and nocturnal polysomnography. The indication for adenotonsillectomy was made by the otolaryngologist based on the history, physical examination, and polysomnographic results.

The criteria for abnormal polysomnography findings in children is not well defined. Marcus et al[12] suggested an obstructive apnea index of > 1/h, while Suen et al[9] recommended to use an RDI > 5/h, when RDI includes apneas and hypopneas per hour of sleep.

Polysomnographic Evaluation

All children were studied before and 3 to 10 months after adenotonsillectomy. Polysomnography was done using a computerized, commercially available, sleep monitoring system (Somno-Star Model 4100; SensorMedics; Yorba Linda) and streamed through to an optical disk for later analysis. Polysomnography was performed as previously described.[13]

Scoring

Scoring was done by a trained technician and reviewed by a trained polysomnographer (A.T.), and a report was sent to the referring pediatric otolaryngologist (A.L.). For the purpose of this study, one of the investigators (A.B.) re-scored all the overnight polysomnograms in a blind and randomized fashion without knowing if the scoring was done for a preadenotonsillectomy or postadenotonsillectomy polysomnographic study.

Sleep/wake and sleep stages were scored according to Rechtschaffen and Kales criteria.[14] Arousals and awakenings were scored twice: first, using American Sleep Disorders Association (ASDA) criteria[15] relating mainly to events of ≥ 3 s and then according to the ASDA task force recommendations, with appropriate modifications for children.[16] According to these definitions, arousals were defined by the presence of any of the following: (1) a period of ≥ 1.5 s of α frequency EEG activity with augmentation of the submental electromyogram; (2) EEG K complex or desynchronization of EEG, if clearly associated with leg movements or apnea; and (3) sleep stage shift, if clearly associated with leg movement or apnea.[16] Arousals were classified as respiratory-induced (when terminating an apneic or hypopneic event) or spontaneous arousals, when not related to a respiratory event or leg movement. Technician-induced arousals (recorded while the night technician had to enter the patient's room) were identified using the technician's log and were excluded. Awakenings were defined as the presence of > 15 s waking EEG after sleep onset with augmentation of the submental electromyogram. The arousal index was calculated as the number of arousals per hour of sleep. The number of shifts to lighter sleep stages and frequency analysis of consecutive epochs were analyzed in order to improve the definition of sleep continuity, as previously described.[17,18]

Obstructive apneic and hypopneic events were scored according to the recommended pediatric criteria of the American Thoracic Society.[19] Obstructive apnea was defined as paradoxical breathing for at least two respiratory cycles with complete cessation of nasal airflow (airflow reduction of at least 80%). A hypopnea was scored when the paradoxical breathing was accompanied by a reduction of at least 50% in airflow, resulting in either an arousal or in oxygen desaturation of at least 4%. The RDI was calculated as the number of respiratory events (apnea/hypopnea) per hour of sleep. The oxygen saturation variables included mean and nadir arterial oxygen saturation (Sao_2) and the desaturation index (DI), the number of desaturation events (Sao_2 decrease of at least 4%) per hour of sleep.

Data analysis

Data analysis was performed using SPSS. All data were tested for normal distribution (Kolmogorov-Smirnov test) and are presented as median (range). The Wilcoxon signed-rank test was used to compare the preadenotonsillectomy and postadenotonsillectomy data (Table 1, Fig 1). A Spearman correlation test was performed to determine the correlation between arousal index and RDI, RDI and DI, both before and after adenotonsillectomy. The frequency analysis of consecutive epochs is presented as the median value and analyzed by Mann-Whitney *U* test. An α level of 0.05 was used for statistical significance. The null hypothesis was rejected at the 5% level.

In order to assess the relationship between arousal index and OSAS severity, we also retrospectively analyzed the change of

Table 1—*Respiratory Characteristics Before and After Adenotonsillectomy

Characteristics	Before	After	p Value†
RDI, events/h			
Total	4.1 (0–85)	0.9 (0–13)	< 0.0001
Non-REM	3.0 (0–89)	0.4 (0–13)	< 0.0001
REM	7.8 (0–69)	2.3 (0–54)	< 0.01
DI, events/h	0.95 (0–43)	0.5 (0–3.3)	0.07
Mean Sao_2, %	97 (88–99)	98 (95–99)	< 0.03
Nadir Sao_2, %	91 (73–95)	92 (84–98)	0.07

*Data are presented as median (range); n = 36.
†Two-sided tests.

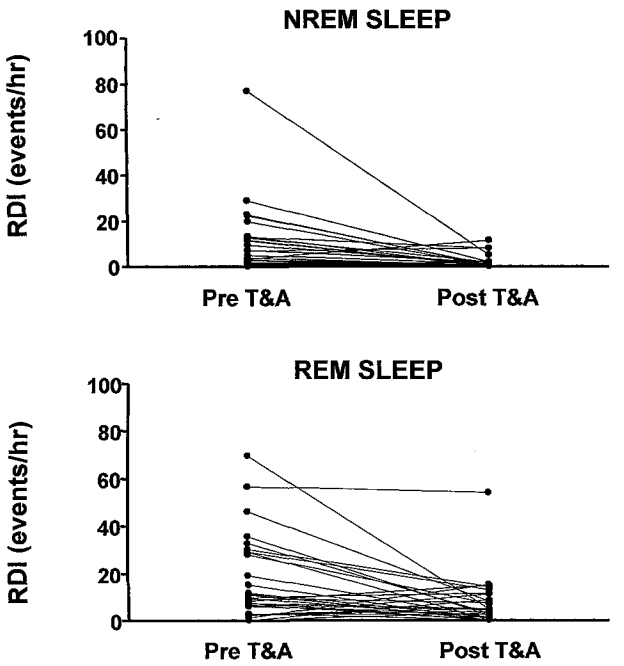

FIGURE 1. Individual data of RDI before and after adenotonsillectomy (T&A) in non-REM (NREM) sleep (*top*) and in REM sleep (*bottom*). Note that after adenotonsillectomy there were many fewer events during non-REM sleep, and there were still children with residual abnormally high RDI in REM sleep.

arousal index for three different groups. We arbitrarily grouped the patients according to their RDI: mild OSA, RDI < 3/h; moderate OSA, RDI between 3/h and 10/h; and severe OSA, RDI > 10/h. Because of the small number of patients in the subgroups, the statistical power in the subgroups was low. We realize that in such cases the interpretation of negative results should be done with caution.

RESULTS

Patients

Seventy children were recruited consecutively, of whom 56 underwent adenotonsillectomy. Thirty-six children, 25 boys and 11 girls, completed both the preadenotonsillectomy and postadenotonsillectomy polysomnographic studies, and constitute the study group. Their median age at initial evaluation was 6.7 years (range, 1.8 to 12.6 years). The duration from the evaluation until adenotonsillectomy was 2.4 months (range, 0.4 to 19 months), and from adenotonsillectomy to the second polysomnographic study was 3.7 months (range, 1 to 16 months). The clinical characteristics and median RDI of the 20 children who did not complete the postadenotonsillectomy polysomnographic study was not significantly different from the 36 children who comprised the study group. The main reason for not completing the postadenotonsillectomy polysomnographic study was the refusal of the parents to repeat the study, months after surgery, when their children were much improved.

Respiratory Characteristics

All respiratory parameters improved significantly after adenotonsillectomy (Table 1). Median RDI for the whole group decreased from 4.1/h (range, 0 to 85/h) to 0.9/h (range, 0 to 13/h) [$p < 0.0001$]. All SaO_2 variables (DI, mean and nadir SaO_2) also improved after surgery (Table 1). There was a linear correlation between RDI and DI, both before and after adenotonsillectomy ($R = 0.53$, $p < 0.001$, and $R = 0.33$, $p < 0.05$, respectively).

Before adenotonsillectomy, the RDIs during REM and non-REM sleep were equal in 22% of the patients, while in 50% of the patients RDI during REM was higher than in non-REM sleep, and in 28% of the patients RDI during non-REM was higher than in REM sleep. Following adenotonsillectomy, in 36% of the patients, RDI during REM was equal to non-REM sleep; in 53% of the patients, RDI during REM was higher compared to non-REM sleep; and in only 11% of the children, RDI during non-REM was higher than in REM sleep. Thus, after adenotonsillectomy, there were many fewer respiratory obstructive events during non-REM sleep (Table 1, Fig 1). However, there were still children with residual abnormally high RDIs, mainly due to REM-related events (Fig 1). In 4 of 36 children (11%), the RDI after adenotonsillectomy was > 5/h. One of them, a 9-year-old boy, is receiving nasal continuous positive airway pressure therapy.

Sleep Architecture

There was no significant change in the cumulative or the median consecutive sleep stages duration after adenotonsillectomy (Table 2). Median consecutive slow-wave sleep analysis was the only parameter that significantly improved after adenotonsillectomy ($p < 0.04$). For the entire group, the arousal index decreased significantly after adenotonsillectomy, from 17.5/h (range, 7 to 57/h) to 14.0/h (range, 6 to 47/h) [$p < 0.03$]. When ASDA criteria[16] for arousal were used (scoring only events with $\geq$ 3-s duration), the trend was similar, but with a smaller number of arousals both before and after adenotonsillectomy (Tables 2, 3). Table 3 describes the arousal index of children with different obstructive sleep apnea severities, before and after adenotonsillectomy. The number of arousals per hour of sleep increased significantly with obstructive sleep apnea severity. The reduction in the arousal index after adenotonsillectomy was significant only in children with severe obstructive sleep apnea (RDI > 10/h).

Table 2—*Sleep Architecture Before and After Adenotonsillectomy

Parameters	Before	After	p Value†
Sleep stages, cumulative, %			
Stage I	0.7 (0–25)	0.8 (0–16)	0.3
Stage II	55 (31–80)	54 (28–74)	0.6
Slow-wave sleep	26 (11–50)	29 (13–58)	0.17
REM	16 (2–34)	15 (4–36)	0.49
Sleep stage: frequency analysis of consecutive epochs			
Stage I, epochs	1.5 (0 ± 16)	1 (0 ± 16)	0.5
Stage II, epochs	17.5 (6 ± 48)	13.5 (6 ± 48)	0.6
Slow-wave sleep, epochs	24 (2 ± 81)	24.7 (7 ± 65)	< 0.04
REM, epochs	13.5 (2 ± 50)	11.5 (6 ± 47)	0.86
Arousal (≥ 1.5 s) index,‡ events/h	17.4 (7 ± 57)	17.1 ± 9.9	< 0.03
Arousal (≥ 3 s) index,§ events/h	16.8 (9 ± 64)	13.1 (6 ± 31)	< 0.01
DownSh, events	18.5 (8 ± 39)	19 (7 ± 30)	1.0

*Data are presented as median (range) or median ± SD; n = 36. Downsh = sleep stage downward shift.
†Two-sided test.
‡From Bar et al.[13]
§From Marcus et al.[12]

Discussion

The results of the present study confirm previous findings that indicate that several months following adenotonsillectomy the breathing difficulties during sleep significantly improve. In addition, our data indicate that the improvement is mainly a result of the reduction in the non-REM sleep obstructive events. Following treatment, the arousal index of children with severe OSAS decreased, but remained high.

Respiratory Characteristics

The respiratory parameters (RDI and Sao_2 variables) improved after adenotonsillectomy, as previously described.[3–5,9] In contrast to adults, the majority of children with OSAS have a higher RDI level during REM sleep than in non-REM sleep.[10] The adenotonsillectomy resulted in an "asymmetrical" reduction in obstructive respiratory events; namely, the non-REM RDI responded better than the REM RDI to the surgical treatment (Fig 1). As seen in Table 1, RDI improved significantly, both in REM and in non-REM sleep; however, after adenotonsillectomy, median non-REM RDI was as low as 0.4/h, while in REM it remained as high as 2.3/h. The pathophysiology of OSAS in children is not well defined, but is related to a combination of anatomic narrowing and neuromuscular function.[20] Pathophysiologic factors include structural factors, the hypertrophied adenoids and tonsils, role of upper airway neuromotor tone,[21] and genetic factors.[22] Our finding that surgical treatment resolved the anatomic component associated with non-REM–RDI recovery may indicate that in non-REM OSAS, the anatomic component is more dominant than in REM-OSAS, a speculation that as yet is unproven.

The indications for adenotonsillectomy in children are not well defined. In the present study, the decision for surgery was made by the otolaryngologist, based on clinical decision and the polysomnographic results. For the purpose of the present study, we analyzed the data using a empirical polysomnography severity classification. As seen in Table 3, 14 of our patients had symptoms of OSAS with hypertrophied adenoids and tonsils, with low RDI values as < 3/h. In some of them, there was evidence of intermittent hypoxemia during REM sleep. There are not enough data in the pediatric literature to conclude what is the minimal RDI that can be used as guideline for the need for surgical treatment. If polysomnography is used as the main guideline, the suggestion of Suen et al[9] of using an RDI > 5/h should be further validated. According to our data, the indication for adenotonsillectomy based only on polysomnography is justified only when RDI is ≥ 10/h; however, other factors are usually taken into consideration, such as morbidity, growth, behavior, and quality of life measures. The lack of a comparison group without intervention is a relative limitation of this study, although it may present an ethical problem.

Table 3—*RDI and Arousal Index in the Three Severity Groups Before and After Adenotonsillectomy

Groups	Patients, No.	RDI			Arousal (≥ 1.5 s) Index, Events/h		
		Before	After	p Value†	Before	After (Mograss Modification‡)	p Value†
Mild	14	1.5 (0–3)	0.5 (0–2)	0.09	14.6 (7–37)	13.1 (6–44)	0.6
Moderate	9	5.3 (3–10)	1.25 (0–13)	0.12	14.5 (7–37)	14 (10–47)	1.0
Severe	13	14.9 (10–85)	2 (0–13)§	0.0002	31.2 (12–57)	15.3 (6–40)§	0.003

*Data are presented as median (range).
†Two-sided test.
‡From Mograss et al.[16]
§$p < 0.001$.

Sleep Architecture

OSAS in children is well known to be associated with an "abnormally disturbed sleep"[23] and "restless sleep."[11] Stradling and colleagues[4] used a computerized video movement analysis system to show fewer nocturnal movements after tonsillectomy than before. Using ASDA criteria,[15] the arousal index of children with severe OSAS was reported as 11/h, as compared to 5/h in the control group.[10] There is no comprehensive definition or characterization of arousals and sleep fragmentation in children. Guilleminault and colleagues[24] suggested considering a diagnosis of sleep fragmentation in children who present with > 11 arousals of ≥ 3 s per hour of sleep, or if parents at home reported more than three long (> 15 min) awakenings, at least 4 nights per week, in children older than 2 years. Mograss and colleagues[16] proposed a simple systematic classification of movement arousals in children, which was used in the present study as well as in our previous publications.[13,18,25] The reliance on EEG as the only indicator of arousals may be misleading by underestimating the clinical severity of sleep-disordered breathing in infants and children,[26] as well as underestimating the severity of sleep disruption, as reported in adult patients.[27] This may be the reason why Goh and colleagues[10] found a relatively low arousal index in children with OSAS. Using the Mograss modified definitions,[16] the arousal index in nine healthy control children previously studied in our own sleep laboratory[18] was 13.3/h. Looking at the data of Mograss et al,[16] in their patients with severe OSAS there were between 21.4 and 35.9 respiratory and spontaneous arousals per hour, consistent with our finding that the arousal index correlated with OSAS severity.

Our data are in accordance with previous findings.[5,9–11] OSAS in children may cause a higher number of arousals during sleep without altering the duration of various stages. The significantly increased arousal index in severe OSAS was not associated with any other abnormality in the sleep architecture. In a smaller group of children with OSAS, we previously did find significantly more slow-wave sleep (sleep stages 3 and 4) after adenotonsillectomy.[13]

A possible limitation of the present study is the "first-night effect," which may affect the sleep architecture of individuals during their first night of the polysomnographic evaluation; however, in a previous study[18] we showed that the only significant change between two consecutive nocturnal polysomnography studies in eight children with atopic dermatitis (mean age, 6 years) was REM latency in minutes and percentage of cumulative REM sleep. Thus, with each patient used as his own control, we do not think the first-night effect affects our interpretation of the data. Another limitation of this study is the fact the decision for adenotonsillectomy was based not only on polysomnography, and thus some of the patients were treated even with low severity of sleep-disordered breathing based on polysomnography; however, we had the opportunity to follow up polysomnographic characteristics after adenotonsillectomy even in mild cases. The fact that only 36 of the 56 children who underwent adenotonsillectomy completed the follow-up study represents a possible bias: it may be that more children who did not fully improve came for a follow-up study. However, the purpose of the present study was to assess the changes in polysomnographic data before and after adenotonsillectomy; therefore, we are confident that this did not affect the interpretation of the data.

In summary, our results indicate that after adenotonsillectomy in children with OSAS, respiratory parameters improve significantly in most children, especially in non-REM sleep (median non-REM RDI decreased from 3.0/h [range, 0 to 89/h] to 0.4/h [range, 0 to 13/h]). However, although REM-related obstructive disturbance also significantly improves (from 7.8/h [range, 0 to 69/h] to 2.3/h [range, 0 to 54/h]) after adenotonsillectomy, it remains slightly abnormal (median RDI > 2/h). The only parameter of sleep architecture that significantly changes following adenotonsillectomy is the number of arousals per hour of sleep (from 17.5/h [range, 7 to 57/h] to 14.0/h [range, 6 to 47/h]). There is a need for further studies to elucidate the role of arousals and sleep disruption, as well as of the respiratory events during sleep (especially during REM sleep), on the long-term outcome of children with OSAS.

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