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# Attention-deficit/hyperactivity disorder with obstructive sleep apnea: A treatment outcome study

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**Abstract**

*Background:* Children diagnosed with attention-deficit/hyperactivity disorder (ADHD), based on Diagnostic and Statistical Manual of Mental Disorders, Fourth edition (DSM-IV) criteria, may also have obstructive sleep apnea (OSA), but it is unclear whether treating OSA has similar results as methylphenidate (MPH), a commonly used treatment for ADHD.
*Methods:* This study enrolled 66 school-age children, referred for and diagnosed with ADHD, and 20 healthy controls. Polysomnography (PSG) performed after ADHD diagnosis showed the presence of mild OSA. After otolaryngological evaluation, parents and referring physicians of the children could select treatment of ADHD with MPH, treatment of OSA with adenotonsillectomy or no treatment. Systematic follow-up was performed six months after initiation of treatment, or diagnosis if no treatment. All children had pre- and post-clinical interviews; pediatric, neurologic, psychiatric and neurocognitive evaluation; PSG; ADHD rating scale, child behavior checklist (CBCL) filled out by parents and teacher; test of variables of attention (TOVA); and the quality of life in children with obstructive sleep disorder questionnaire (OSA-18).
*Results:* ADHD children had an apnea-hypopnea index (AHI) $> 1 < 5$ event/hour; 27 were treated with MPH, 25 had adenotonsillectomy, and 14 had no treatment. The surgical and MPH groups improved more than the non-treatment group. When comparing MPH to post-surgery, the PSG and questionnaire sleep variables, some daytime symptoms (including attention span) and TOVA subscales (impulse control, response time and total ADHD score) improved more in the surgical group than the MPH group. The surgical group had an ADHD total score of $21.16 \pm 7.13$ on the ADHD rating scale (ADHD-RS) post-surgery compared to $31.52 \pm 7.01$ pre-surgery ($p = 0.0001$), and the inattention and hyperactivity subscales were also significantly lower ($p = 0.0001$). Finally, the results were significantly different between surgically and MPH-treated groups (ADHD-RS $p = 0.007$). The surgical group also had a TOVA ADHD score lower than $-1.8$ and close to those obtained in normal controls.
*Conclusion:* A low AHI score of $> 1$ considered abnormal is detrimental to children with ADHD. Recognition and surgical treatment of underlying mild sleep-disordered breathing (SDB) in children with ADHD may prevent unnecessary long-term MPH usage and the potential side effects associated with drug intake.


*Keywords:* Attention-deficit/hyperactivity disorder; Methylphenidate; Adenotonsillectomy; Obstructive sleep apnea; Polysomnography; Outcome

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## 1. Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a behavioral abnormality commonly seen in children and adolescents. Its main symptoms include inattention, hyperactivity, and impulsivity [1]. According to the Diagnostic and Statistical Manual of Mental Disorders, Fourth edition (DSM-IV), the prevalence of ADHD in the United States is estimated at 3–7% [1,2] and at 8.4–11.7% in Taiwan [3]. The different incidence rates are probably based on the nature of the population sampled. Until now, ADHD has only been diagnosed according to the symptomatic criteria as it is an etiologically heterogeneous disorder that can be caused by biological, psychological, or social conditions [4]. In fact, over 50% of children with ADHD meet the criteria for two comorbid conditions [5]. However, the existing research does not clarify the etiology of ADHD or the mechanism responsible for the comorbidity of ADHD with other conditions. Several previously published neurophysiological studies show that children with ADHD often exhibit abnormal nocturnal sleep [6–13]. In their questionnaire study, Kaplan et al. found that parents of children with hyperactivity considered their children to have many sleep problems [9]. Corkum et al. reported that 25–50% of children and adolescents with ADHD had sleep problems [10]. Simonds and Parraga also reported that ADHD children displayed significantly more snoring, head-banging restlessness, and problems with nighttime awakening [14]. Recently, various types of sleep disorders [15–21] [e.g., obstructive sleep apnea syndrome (OSAS), periodic limb movement disorder (PLMD), enuresis, delayed sleep phase syndrome] have been particularly associated with ADHD children, and the presence of hyperactivity, inattention and learning problems has been noted with OSA, restless legs syndrome (RLS), and periodic limb movements (PLMs) [17–19,22–25]. Until now, we did not know if a sleep disorder was a cause of ADHD or a comorbid disorder [26]. However, Naseem et al.'s case report [27] suggested that sleep disorders, especially sleep apnea, may be one of the underlying causes of ADHD. O'Brien et al. [28] used a questionnaire and showed that sleep disturbance was more common in patients with ADHD symptoms ($n = 77$) than in normal control patients ($n = 53$). Chervin [19] and O'Brien [20] also reported that children with mild symptoms of ADHD showed a high prevalence of snoring and sleep problems. We have previously reported that nearly 56% of Taiwanese children, successively seen in a specialized university clinic and diagnosed with ADHD based on International Classification of Sleep Disorders (ICSD) criteria [29] presented an elevated apnea–hypopnea index (AHI > 1) at systematic polysomnography (PSG), as found in studies performed in the United States[30]. However, Sangal et al. [31] recently performed PSG as part of a recruitment effort for a trial of atomoxetine and methylphenidate (MPH) for treatment of ADHD children. After exclusion of sleep disorder symptoms and selection of a "respiratory disturbance index" (based only on apnea and hypopnea, i.e., an AHI) cut-off point of 5 events/hour, the authors concluded that OSA is not a common finding or etiological factor in ADHD. Indeed, some researchers believe that children who have an AHI between 1 and 5 have no pathology and do not need treatment. This position argues against the data from Naseem et al. [27], O'Brien et al. [20,28] and Chervin et al. [19,32] Based on previous reports [20,21] [26,30], there is no significant lineal correlation between the severity of AHI, ADHD symptoms, neurocognitive function impairment and performance deficits.

### 1.1. Protocol goal

The aim of our study was to determine the best treatment for children who have ADHD with mild OSA. Our data may in addition shed some light on the relationship between mild OSA and ADHD. Of course, for such study purposes, the best protocol would be a randomized double-blind adenotonsillectomy versus a stimulant-treatment study. Considering the lack of data ethically justifying this study, which involves general anesthesia and surgery, we report on a clinical study that may justify such a step and may also provide valid data. As for the treatment of ADHD, there are different proposed treatments for children with ADHD, but stimulant medication, particularly MPH [33–36], is the most common choice for reduction of ADHD symptoms. Therefore, we used the effective dosage of MPH for treatment of ADHD in our study. Moreover, the most commonly recommended treatment for OSAS in children is adenotonsillectomy [22,37]. Chervin et al. suggested that if habitual snoring was effectively treated, ADHD symptoms in 25% of all children with ADHD could be eliminated [17]. Other studies have shown that adenotonsillectomy was also effective in improving behavioral symptoms linked to ADHD, with AHI that is low but considered abnormal (AHI > 1), and even in isolated chronic snoring with AHI < 1 [38–40]. We designed a prospective study that would involve three ADHD groups of children and one healthy control group.

### 1.2. Pre-set inclusion–exclusion criteria

Prior to the onset of the study, the following inclusion and exclusion criteria were set based on current knowledge of ADHD in children.

*1.2.1. Inclusion criteria*

Subjects must have ADHD according to DSM-IV criteria [1]. In addition, in the second step, children must have an AHI > 1 and must be aged 6–12 years; also, subjects must be of normal intelligence, defined as showing no evidence of significant general intellectual deficit and achieving a score of 70 or more when Wechsler Intelligence Scale for Children, 3rd Edition (WISC-III) IQ testing [41] was administered. To be considered for possible OSA treatment, subjects must have a pediatric otolaryngologist evaluation to confirm adenotonsil hypertrophy. Subjects and parents must have been judged by an investigator to be reliable and able to keep appointments for clinic visits, all tests and PSGs and must show prospect of having good treatment compliance.

*1.2.2. Exclusion criteria*

Subjects with PSG diagnosis of periodic limb movement index (PLMI) > 5 and those with a history of seizure disorder, systemic disease, other physical disease, or major psychiatry disease (such as pervasive developmental disorder, bipolar disorder, major depression, anxiety disorder, psychosis, or substance abuse history) were excluded after examinations by pediatrician and child psychiatrists. As structured psychotherapy is frequently offered to ADHD patients, such treatment approach was banned during the six-month trial; however, educative psychotherapy offered in school to any student was permitted.

A healthy control group recruited from the same community as the ADHD children would also be submitted to the same tests.

Comparison of ADHD children who had mild OSA and had accepted adenotonsillectomy to the other groups would be performed: six-month post-treatment data analysis would be done to understand the relationship between OSA and ADHD. Then, comparison of the post-surgery results to those obtained with MPH treatment, and to those of normal controls, would be tabulated.

## 2. Methods

From January 2002 to December 2004 the study enrolled 125 successively seen school-age (6–12 years) children who were referred to the child psychiatry clinic of a university medical center in the Taipei area of Taiwan. The children were referred for behavioral problems suggestive of ADHD. Simultaneously, 27 children without any complaint of ADHD were recruited from the community to serve in the control group. All control children were evaluated at the "child developmental evaluation center" of the hospital. The same specialists who saw the children with suspected ADHD and mild OSA performed physical, neurological, mental evaluations, and all tests.

The protocol involved two steps:

*A. Identification of ADHD children responding to entry criteria*

Initial screening based on DSM-IV criteria [1] was performed by two experienced psychiatrists to diagnose ADHD following the standard structured interview with the schedule for Affective Disorder and Schizophrenia for School-Age Children, Adolescent Chinese version (K-SADS-E) [42]. This evaluation affirmed the clinical diagnosis and excluded children who had comorbid pervasive developmental disorder, or had a history of bipolar disorder, psychosis, anxiety disorder, seizure disorder, substance abuse history or mental retardation. The ADHD-Rating scale [43] was performed at the same time. The ADHD-Rating scale-parent [43] is a structured interview by the investigator based on the information from the parent and child. Each of the items has a 4-point response scale, and each item explores one of the 18 criteria outlined in the DSM-IV to diagnose ADHD. Higher scores indicate greater severity of ADHD.

Other physical and neurological examinations were performed by a pediatrician and neurologist to rule out central nervous system or other physical diseases. Once the diagnosis of ADHD without known comorbid conditions had been established, all the eligible children and parents signed informed assents and consents. All children were kept drug-free for a duration based on the half-life of administered medication and in any case for at least one week before the series of tests began.

The following investigations were then performed on patients and controls:

(1) Comprehensive neuropsychological tests (test of variables of attention (TOVA) [44] and WISC-III IQ test [41]) were administered by clinical child psychologists. All tests were performed in a specifically designated testing room ("attention testing room") to decrease testing condition variability. The TOVA-Visual test (TOVA-V) [44] is a 22.6-min neuropsychological test for the evaluation of attention, impulse control, and ADHD. It is a computerized continuous performance test, not influenced by culture and, therefore, results can be compared in different parts of the world. It includes a target stimulus and a non-target stimulus. A computer recorded all reaction times, omissions, commissions, response times, and response time variability and detectability. *Omissions* were scored when the target stimulus appeared but the participant did not react by pressing the button; the score represents the participant's level of inattention. *Commissions* were scored when the target stimulus did not appear but the participant reacted by pressing the button; it represents the participant's inability to control impulse. *Response Time*

is the time in milliseconds used to respond to each trial, and *Response Time Variability* is based on the deviation from the mean time to give a correct response. *Detectability* ($D'$) is an evaluation of response sensitivity (ratio of hit frequency to frequency of false response), and it is interpreted as a measure of perceptual sensitivity. An ADHD score is determined from the total subscores.

(2) Parents completed questionnaires (the child behavior checklist (CBCL) [45] and the quality of life in children with obstructive sleep disorder questionnaire (OSA-18) [46]).

The CBCL, [45] given to parents and teachers, evaluates a child's social and behavior competence. The test contains eight subscales: depression/anxiety, thought/obsessive, somatic complaint, social withdrawal, hyperactivity, aggressive behavior, delinquency, internalizing behavior, and externalizing behavior.

The OSA-18 [46] is a reliable instrument that is easy to administer and has shown validity for detecting change and evaluating quality of life after adenotonsillectomy in children with OSAS (Chinese version). This instrument, which contains an 18-item quality of life survey, was filled out by parents on a 7-point scale from "none" to "all of the time". The items cover sleep disturbance, physical suffering, emotional distress, daytime problems, caregiver concerns and total quality of life.

(3) Teachers' questionnaires were distributed and collected back from the school.

(4) An otolaryngologic evaluation was performed by an ear, nose, and throat (ENT) specialist. Clinical evaluation of tonsils and adenoids were performed. There was a visual evaluation of tonsil size based on a scale derived from Friedman et al. [47] Adenoid enlargement was evaluated visually with naso-fibroscopy and lateral cephalometric X-ray, looking also at cranio-facial skeleton, as was presence of a deviated nasal septum, presence of allergic rhinitis and presence of sinus infection. Positive history of asthma was also systematically documented.

(5) Finally, after signing a new consent form, as children were required to sleep in the hospital, nocturnal PSG was performed. All children were free of drugs for the test for at least seven days (no drug was prescribed during the entire work-up). A family member was present on the premises during the nocturnal recording. Video recording assessed behavior. All measurements were collected on a computerized sleep system (Ultrasom, Nicolet USA). The following variables were systematically monitored: electroencephalogram (EEG) (C3/A2, C4/A1, O1/A2), electro-oculogram (EOG) (right and left eyes), chin and leg electromyogram (EMG), and electrocardiogram (ECG) modified V2 lead. Respiration was monitored by nasal and mouth thermistors, piezzo electric thoracic and abdominal bands, neck microphone, pulse oximetry, and body position sensor.

Scoring of PSGs was performed by an individual blind to the condition of the child (patient versus control). Calculation of sleep–wake variables and AHI was based on international criteria [48–51].

OSA was defined as absence of airflow, and apnea was defined as a decrease in airflow from 80% to 100% on thermistor channels with continued chest wall and abdominal wall movement for a duration longer than two breaths [49–51]. Hypopneas were defined as a decrease in nasal flow between 30% and 80% from baseline with a corresponding decrease in $SpO_2$ of 3% and/or arousal. Events were subdivided into central, obstructive and mixed. The AHI was defined as the number of apneas and hypopneas per hour of total sleep time. An AHI $>1$ event/h [29] was considered elevated. The definition of periodic leg movements (PLMs) [29] was at least four movements of 0.5–5 s in duration and between 5 and 90 s apart. A periodic limb movement index (PLMI) $>5$ per hour is considered abnormal in children[29]. PLMs associated with breathing events were not scored; only those independent of apnea/hypopnea were considered.

### *B. Treatment study*

Children who met the criteria of the study were enlisted. Parents and referring physicians not involved in the study were presented with results of diagnostic investigation and were faced with three different alternative treatment approaches based on the obtained results: (1) treatment with MPH at effective dosage [52,53] decided by their own psychiatrist in the ADHD clinic, who was not involved in the study (private specialists had the liberty to increase MPH treatment if they thought this increase would be useful); (2) treatment of mild OSA with systematic adenotonsillectomy performed by the same otolaryngologist, and (3) if parents and referring physicians opted for a wait-and-see attitude for the following six months, regular follow-up and evaluation was planned with re-evaluation at six months (no treatment group).

At six-month follow-up, the same variables as those obtained at entry were obtained again (clinical evaluation, ENT evaluation, TOVA, CBCL parent–teacher, OSA-18 and nocturnal PSG).

Pre- and post-treatment evaluations had to be completed within one month of individual initial contact with the clinic.

### 2.1. Statistical analysis

The data collected from this study were analyzed with the statistical software package SPSS, Version 10. Variables are presented as means ± standard deviation (SD) and frequency. Based on normality of distribution, the following tests were applied chi-square, paired *t*-test, and analysis of variance (ANOVA) for comparison of the outcome of TOVA data, CBCL data, ADHD-RS, PSG and OSA-18 quality of life survey data. If ANOVA was significant, post hoc analysis for paired comparison applying the conservative Fisher's least significant difference (LSD) criteria was used for between-group comparisons. Paired *t*-test was used for pre-post within-group data comparison. The Fisher exact test was used when small groups were compared.

## 3. Results

Sixty-nine out of 125 (55.2%) children with ADHD complaint had AHI > 1 events/h and met the other ADHD entry criteria. Sixty-six terminated the study, three children in the wait-and-see attitude group dropped out of the study and did not have a follow-up at six months. After contacting the parents and teachers in the same school district as potential ADHD cases, 27 age- and gender-related children (6 to 12 years) were recruited from the surrounding schools in the community, but only 20 children completed pre- and post-six-month tests, with seven children dropping out either during initial testing or follow-up evaluation. Table 1 presents the demographic data of the 86 subjects who completed the pre- and post-six-month tests. Twenty-seven children with ADHD were treated with MPH, 25 had adenotonsillectomy, and 14 had no treatment but received regular follow-up for a total of 66 ADHD children with AHI > 1 and 20 healthy controls. All children had an AHI < 5, indicating mild SDB. The mean dosage of MPH in the MPH group was 0.878 ± 0.21 mg/kg/day (28.974 mg ± 7.4/day) and was based on the Taiwanese dosage of MPH (26.7 mg ± 18.8/ day for 5 to 18 years ADHD)[53].

As can be seen (Table 1), no significant demographic difference existed between the groups, particularly for age, gender distribution, body mass index (BMI), subtype of ADHD, ADHD-RS (severity) and AHI between the three ADHD with mild OSA groups. The mean IQs of these four groups are 90.50 ± 12.21 (surgical group), 94.67 ± 18.94 (MPH group), 93.25 ± 8.18 (non-treatment group) and 101.60 ± 17.44 (control group). By comparing the groups who had ADHD with mild OSA to the healthy control group based on the ADHD-RS (mean = 30.17 to 32.62), the severity of the ADHD with mild OSA groups was considered mild to moderate.

The results of the ENT evaluation are presented in Table 2. No significant difference between groups could be demonstrated for presence of allergic rhinitis, deviated septum, asthma, and sinusitis, but there was a significant difference in tonsil size; more children had a tonsil size of 1+ (i.e., small) in the MPH group, and there were more children with enlarged adenoids in the surgical treatment group.

Table 1
Demographic data of children involved in the treatment study (ADHD children with 1 < AHI ⩽5 – $n = 66$; and normal controls $n = 20$)

| ($N = 86$) | ($N = 25$) surgical Tx. | ($N = 27$) MPH Tx. | ($N = 14$) non-Tx. | ($N = 20$) control | $p$ |
|---|---|---|---|---|---|
| Age, M (SD) | 8.08(1.28) | 8.19(1.73) | 8.07(2.30) | 8.85(2.13) | 0.433[a] |
| Gender *F* (%) | | | | | |
| Male | 23(92%) | 24(88.9%) | 12(85.7%) | 16(80%) | 0.56[a] |
| Female | 2(8%) | 3(11.1%) | 2(14.3%) | 4(20%) | |
| Subtype *F* (%) | | | | | |
| Inatt | 12(48%) | 9(33.3%) | 8(57.1%) | | |
| Hyper. | 2(8%) | 2(7.4%) | 2(14.3%) | 0 | 0.434[b] |
| Mix | 11(44%) | 16(59.3%) | 4(28.6%) | | |
| Comobidity | | | | | |
| LD, *F* (%) | 6(24%) | 5(18.5%) | 4(28.6%) | 0 | 0.753[b] |
| TIC, *F* (%) | 5(20%) | 4(14.8%) | 0(0) | 0 | 0.212[b] |
| ODD, *F* (%) | 7(28%) | 7(25.9%) | 4(28.6%) | 0 | 0.979[b] |
| CD, *F* (%) | 2(8%) | 0(0) | 0(0) | 0 | 0.184[b] |
| Enuresis, *F* (%) | 3(15%) | 3(13%) | 3(21.4%) | 0 | 0.789[b] |
| BMI (kg/m$^2$), M (SD) | 18.51(1.28) | 19.25(3.66) | 18.18(4.02) | 18.84(3.66) | 0.665[a] |
| AHI (event/hour), M (SD) | 3.32(1.11) | 2.24(1.44) | 2.56(1.46) | 0.41(0.40) | 0.478[b] |

The sample size is small, Fisher exact test was used Inattentive, inattentive type; Hyper., hyperactive-impulsive type; Mix, combined type; LD, learning disorder; ODD, oppositional defiant disorder; CD, conduct disorder; BMI, body mass index; M, mean; SD, standard deviation; surgical Tx., adenotonsillectomy treatment; MPH Tx., methylphenidate treatment.

[a] $p$ value relate to comparison of four groups with ANOVA (surgical Tx, MPH Tx, non-Tx. and healthy control group).

[b] $p$ value relate to comparison of three groups with ANOVA (surgical Tx., MPH Tx. and non-Tx. group).

Table 2
Size of tonsils and adenoids between groups of children in "treatment" study

| ($N = 86$) | ($N = 25$) surgical Tx. $F$ (%). | ($N = 27$) MPH Tx. $F$ (%) | ($N = 14$) non-Tx. $F$ (%) | $p$ (ANOVA) | ($N = 20$) control |
|---|---|---|---|---|---|
| Tonsils (%) | | | | | |
| **1**+ | 3(12%) | 12(44.4%) | 3(21.4%) | 0.038* | 13(65%) |
| **2**+ | 11(44%) | 9(33.3%) | 5(35.7%) | | 7(35%) |
| **3**+ | 9(36%) | 5(18.5%) | 5(35.77%) | | 0 |
| **4**+ | 2(8%) | 1(3.7%) | 1(7.14%) | | 0 |
| Adenoids enlargement (%) | | | | | |
| No | 5(20%) | 9(33.3%) | 6(42.9%) | 0.016* | 18(90%) |
| Yes | 20(80%) | 18(66.6%) | 8(57.1%) | | 2(10%) |
| Nasal septum deviation (%) | | | | | |
| No | 23(92%) | 25(92.6%) | 14(100%) | 0.639 | 20(100%) |
| Yes | 2(8%) | 2(7.4%) | 0(0%) | | 0 |
| Allergic rhinitis (%) | | | | | |
| No | 17(68%) | 17(61.5%) | 8(57.1%) | 0.688 | 16(80%) |
| Yes | 8(32%) | 10(38.5%) | 6(42.9%) | | 4(20%) |
| Asthma (%) | | | | | |
| No | 23(92%) | 24(88.5%) | 12(85.7%) | 0.781 | 20(100%) |
| Yes | 2(8%) | 3(11.5%) | 2(14.3%) | | 0 |
| Sinusitis (%) | | | | | |
| No | 24(96%) | 24(88.5%) | 14(100%) | 0.313 | 20(100%) |
| Yes | 1(4%) | 3(11.5%) | 0(0%) | | 0 |

Size of tonsils, visually scored: **1**+, endophytic tonsils, anterior and posterior pillars visible. **2**+, tonsils extend to tonsillar pillars. **3**+, tonsils extend beyond tonsillar pillars, approximating uvula. **4**+, tonsils touching each other in the midline. As can be seen, 44% of the surgical group and 22.2% of the methylphenidate group had tonsil sizes scored 3 or 4. Adenoid enlargement was evaluated with naso-fibroscopy and lateral cephalometric X-ray skull lateral view. The $p$ value relates to comparison of three groups (Surgical Tx., MPH Tx. and non-Tx. group) with ANOVA.

When PSG variables were analyzed (Table 3), none of the sleep variables were significantly different between the three groups of ADHD with mild OSA. Comparison of pre-post surgery condition for the adenotonsillectomy subgroup showed a significant difference in AHI ($p = 0.001$), apnea index (AI) ($p = 0.005$), snoring index (SI, $p = 0.04$), rapid eye movement (REM) percent ($p = 0.028$), total sleep time ($p = 0.047$), and slow wave sleep percent ($p = 0.05$), supporting a beneficial effect of the surgery and indicating a successful response to surgery (see Table 3); BMI and PLM index stayed unchanged over conditions. No statistically significant difference was noted when comparing PSG variables between the post-surgery condition and the results of the healthy group. As expected, no improvement in AHI or SI was noted in the MPH or non-treatment groups after six months. Table 4 presents the results of the OSA-18 quality of life scale. Comparing the results of the surgical and non-treatment groups, the post hoc comparison test with LSD demonstrates the presence of a significant difference in the sleep disturbance subscale ($p = 0.048$). A similar finding was noted when comparing surgical and MPH groups: there was a significant difference in the sleep disturbance subscale ($p = 0.042$). Although other subscales did not reach statistical difference, an overall trend toward greater improvement in the surgical group than the other groups was noted. When a within-group analysis was performed, several subscale scores and the total score were significantly better post-surgery than pre-surgery in the surgical treatment subgroup, and no statistical significance existed between the post-surgical and healthy group variables. The ANOVA performed on the six-month follow-up CBCL score did not show a significant difference between the improved surgical group and the non-treatment group or the surgical and MPH groups, but the comparison within each group (pre-post data) shows that the adenotonsillectomy subgroup had a significantly lower score (i.e., indication of improvement) compared to baseline (Table 5). The ANOVA with post hoc multiple comparison (LSD) test shows that the ADHD-RS total ($p = 0.002$) and inattentive ($p = 0.007$) scores were significantly different between surgical and MPH treatment. The mean ADHD total score was 21.16 for the surgically treated group, clearly lower than 25, a score often considered as a cut-off point for ADHD; it was also lower in the MPH group at 24.71 but just below threshold, while it was still above threshold in the non-treated group (see Table 5).

Comparison within groups indicates that total, inattentive and hyperactivity subscores were significantly improved between pre- and post-treatment conditions in both surgical and MPH groups, while there was no significant change in the untreated group.

It is interesting to note that even if both surgically and MPH treated children improved scores at CBCL

Table 3
Comparison of the outcome of PSG variables: (pre and post 6 months)

| ($N = 86$) | ($N = 25$) surgical Tx. M (SD) | ($N = 27$) MPH Tx. M (SD) | ($N = 14$) non-Tx. M (SD) | $p$ (ANOVA) | ($N = 20$) control M (SD) |
|---|---|---|---|---|---|
| AHI(/h) | | | | | |
| Pre | 3.32(1.11) | 2.24(1.44) | 2.56(1.46) | $0.003^{++}$ | 0.41(0.40) |
| Post | 0.89(0.63)*** | 2.50(2.95) | 2.31(2.19) | | 0.37(0.40) |
| AI(/h) | | | | | |
| Pre | 1.77(0.60) | 0.98(0.96) | 1.36(0.99) | 0.147 | 0.35(0.51) |
| Post | 0.22(0.26)** | 0.42(0.54) | 1.36(0.78) | | 0.30(0.25) |
| SI(/h) | | | | | |
| Pre | 33.89(42.02) | 38.83(41.36) | 12.54(15.82) | 0.866 | 8.50(4.19) |
| Post | 20.83(34.18)* | 35.92(45.60) | 15.82(13.49) | | 13.00(5.15) |
| DI(/h) | | | | | |
| Pre | 0.64(1.24) | 0.35(0.49) | 0.76(1.86) | 0.608 | 0.05(0.14) |
| Post | 0.50(0.68) | 0.55(0.74) | 0.98(1.89) | | 0.03(0.09) |
| Mean $S_aO_2$ | | | | | |
| Pre | 96.32(2.17) | 97.17(1.43) | 97.50(1.32) | 0.354 | 97.44(1.34) |
| Post | 97.32(1.15) | 97.03(1.52) | 95.6(1.68) | | 97.62(0.68) |
| Awake (%) | | | | | |
| Pre | 4.95(3.91) | 8.11(8.25) | 8.25(12.31) | 0.980 | 5.17(4.12) |
| Post | 4.74(5.16) | 8.38(8.38) | 7.75(7.65) | | 6.04(5.06) |
| REM (%) | | | | | |
| Pre | 10.17(5.52) | 10.43(5.64) | 13.52(6.12) | 0.371 | 13.18(4.78) |
| Post | 13.52(6.12)* | 12.71(8.16) | 12.18(3.98) | | 15.84(3.78) |
| Stage 1 (%) | | | | | |
| Pre | 14.73(8.06) | 11.43(7.04) | 12.28(6.25) | 0.482 | 11.21(7.87) |
| Post | 8.77(5.65) | 9.26(4.19) | 7.70(4.17) | | 6.36(3.02) |
| Stage 2 (%) | | | | | |
| Pre | 37.48(10.32) | 40.43(16.06) | 38.72(14.45) | 0.592 | 42.95(8.82) |
| Post | 43.42(6.60) | 43.63(12.87) | 43.40(8.15) | | 43.82(8.93) |
| SWS (%) | | | | | |
| Pre | 26.32(5.80) | 28.78(15.12) | 30.98(9.19) | 0.968 | 29.26(8.14) |
| Post | 31.56(10.70)* | 27.29(9.52) | 27.20(9.24) | | 28.92(9.65) |
| Total sleep time (min) | | | | | |
| Pre | 390.19(25.83) | 372.00(35.89) | 372.08(41.34) | 0.768 | 391.76(24.66) |
| Post | 408.27(25.21)* | 371.33(30.28) | 373.00(28.56) | | 399.70(21.66) |
| Efficiency (%) | | | | | |
| Pre | 91.49(5.27) | 87.37(9.71) | 86.18(12.13) | 0.726 | 92.01(5.66) |
| Post | 92.30(6.69) | 87.52(8.07) | 89.73(7.88) | | 91.58(5.51) |
| Sleep latency (min) | | | | | |
| Pre | 22.52(25.05) | 18.53(16.06) | 22.00(10.87) | 0.310 | 11.63(12.52) |
| Post | 16.16(15.36) | 18.30(8.99) | 19.67(5.49) | | 10.30(5.63) |
| Arousal counts | | | | | |
| Pre | 19.30(11.34) | 23.0(10.05) | 22.0(5.06) | 0.831 | 21.16(5.98) |
| Post | 18.20(18.86) | 20.0(18.38) | 21.05(8.34) | | 20.25(7.50) |
| PLMI(/h) | | | | | |
| Pre | 1.11(4.32) | 1.56(3.05) | 0.72(0.72) | 0.700 | 0.40(1.08) |
| Post | 1.14(2.03) | 0.34(0.70) | 0.25(0.79) | | 0.28(0.63) |
| BMI | | | | | |
| Pre | 18.51(1.28) | 19.25(3.66) | 18.18(4.02) | 0.810 | 18.84(3.66) |
| Post | 17.81(5.36) | 17.12(3.23) | 19.22(2.7) | | 18.90(1.20) |

SI, snoring index; SWS, slow wave sleep; min, minute; DI, desaturation index; number of desaturation of 4% or more per hour of sleep. $^{+}p < 0.05$, $^{++}p < 0.01$, ANOVA test with post hoc comparison of improvement (pre/post) between the 3 groups (surgical Tx., MPH Tx. and non-Tx. group): comparison of improvement between the surgical and non-Tx. group and between the surgical and MPH group showed no significant differences except in AHI with $p = 0.0001$ and $p = 0.001$, respectively.

* $p < 0.05$.

** $p < 0.01$.

*** $p < 0.001$ (pair $t$-test) pre/post comparison within group.

Table 4
Comparison of outcome of OSA-18 quality of life survey: comparison of pre and post 6 months data

| ($N = 86$) | ($N = 25$) Surgical Tx. | | ($N = 27$) MPH Tx. | | ($N = 14$) non-Tx. | | ($N = 20$) control | |
|---|---|---|---|---|---|---|---|---|
| | M (SD) | Change (mean) | M (SD) | Change (mean) | M (SD) | Change (mean) | M (SD) | Change (mean) |
| Sleep disturbance | | | | | | | | |
| Pre | 13.50(4.50) | −4.32[a,b] | 13.75(4.86) | 0.37[a] | 10.50(2.12) | 1.5[b] | 8.63(2.79) | 1.04 |
| Post | 9.18[***](4.23) | | 14.13(6.64) | | 12.00(1.41) | | 9.67(1.15) | |
| Physical symptoms | | | | | | | | |
| Pre | 14.27(5.07) | −3.63 | 12.25(6.71) | 0.00 | 13.67(4.73) | −2.66 | 10.74(3.28) | 0.33 |
| Post | 10.64[*](4.28) | | 12.25(4.33) | | 11.00(2.65) | | 11.07(3.79) | |
| Emotional distress | | | | | | | | |
| Pre | 11.09(4.01) | −1.32 | 12.75(4.46) | −0.25 | 10.00(4.36) | −0.67 | 7.63(3.62) | 1.87 |
| Post | 9.77[*](4.76) | | 12.50(3.30) | | 9.33(1.53) | | 9.50(3.56) | |
| Daytime problems | | | | | | | | |
| Pre | 12.14(2.53) | −2.46 | 11.75(2.60) | −0.37 | 9.68(4.32) | −0.01 | 7.16(3.53) | 0.84 |
| Post | 9.68[**](4.32) | | 11.38(3.7) | | 9.67(3.06) | | 8.00(5.66) | |
| Caregiver concerns | | | | | | | | |
| Pre | 17.91(4.96) | −6.05 | 15.38(4.44) | −1.5 | 9.67(4.03) | 1.00 | 8.11(4.32) | 1.89 |
| Post | 11.86[**](6.84) | | 13.88(6.10) | | 10.67(2.08) | | 10.00(4.24) | |
| Quality of life | | | | | | | | |
| Pre | 5.00(1.53) | 1.11 | 4.83(0.75) | 0.7 | 5.33(2.52) | −0.33 | 7.00(1.75) | 0.00 |
| Post | 6.11[*](1.84) | | 5.33(1.63) | | 5.00(0.02) | | 7.00(0.10) | |

ANOVA test with post hoc comparison of improvement (pre/post) between 2 groups. The lower the subscale score, the better the score is, except for the score of quality of life, which is converse. "Change" indicated comparison of score "six months after treatment" compared to baseline. The negative values indicate greater improvement, except, again, with the subscale of quality of life.

[a] shows significant difference between surgical and MPH groups.

[b] Significant difference between surgical and non-treatment groups.

[*] $p < 0.05$.

[**] $p < 0.01$.

[***] $p < 0.001$ (pair $t$-test) pre/post comparison within group.

and ADHD-RS scales, none went back to scores similar to those obtained from the normal control group as shown by ANOVA, including all four groups post-treatment.

Analysis of TOVA results after normalization of the raw data are presented in Table 6. The ADHD scores were abnormal for all subgroups at entry (<−1.8). An ANOVA with post hoc test for paired comparison (LSD) was performed to compare the improvement (pre/post) between the surgical groups and the non-treatment group. It was significantly different for the ADHD score ($p = 0.045$). The within-group comparison indicated that the adenotonsillectomy subgroup improved post-surgery in nearly all variables, and the lowest ADHD score was seen in this subgroup post-surgery. Comparing the results of surgical and MPH groups, there were significant differences in Response Time ($p = 0.021$) and ADHD score ($p = 0.017$). It also showed that other subscales (see the change in Omission, Response Time and Response Time variability data in Table 6) did not improve in the MPH group: the ADHD score was still low (−2.10). This indicated the presence of neurocognitive deficits in the children who had ADHD with OSA with appropriate MPH treatment for ADHD.

## 4. Discussion

Our study is an outcome investigation addressing the important question of treatment for ADHD children with mild OSA. There is a controversy on the AHI limit for pathology. A low AHI > 1 has not always been recognized as pathological, but the ICSD-2005 recognizes that this score is pathological when children have an AHI > 1 associated with clinical symptoms which improve with a decrease in AHI. Chronic heavy snoring has itself been shown to have a detrimental impact on children. Guilleminault et al. [39,40], using nasal cannula and pressure transducer and esophageal pressure to investigate heavy snorers with low AHI, have shown that these children have SDB with an abnormal respiratory disturbance index (RDI). This study did not use esophageal pressure monitoring and RDI could not be calculated, but all children had an AHI > 1. ADHD is a syndrome defined clinically, and there is good evidence that several sleep disorders may lead to a clinical presentation similar to ADHD [13,16–26], but sleep disorders may be very much ignored by caretakers. Our children did not present with severe OSA, considering their AHI scores, but all of them had an abnormal score even if their OSA was unknown by their parents. There is

Table 5
Comparison the outcome of parents' CBCL and ADHD-RS data: comparison of pre- and post-6-months of data

| ($N = 86$) CBCL | ($N = 25$) surgical Tx. | | ($N = 27$) MPH Tx. | | ($N = 14$) non-Tx. | | $P$ (ANOVA) | ($N = 20$) Control | |
|---|---|---|---|---|---|---|---|---|---|
| | M (SD) | Change (mean) | M (SD) | Change (mean) | M (SD) | Change (mean) | | M (SD) | Change (mean) |
| Depression/anxiety | | | | | | | | | |
| Pre | 61.67(12.46) | −3.96 | 62.13(9.50) | −2.13 | 68.33(7.51) | −6.33 | 0.79 | 54.56(8.21) | −2.23 |
| Post | 57.71(11.11) | | 60.00(10.62) | | 62.00(10.00) | | | 52.33(8.97) | |
| Thought/obsessive | | | | | | | | | |
| Pre | 61.21(10.10) | −5.33 | 62.06(8.18) | −2.62 | 68.33(4.04) | −4.33 | 0.99 | 54.44(7.7) | −3.77 |
| Post | 55.88(10.02)* | | 59.44(9.48) | | 64.00(9.40) | | | 50.67(8.68) | |
| Somatic complaint | | | | | | | | | |
| Pre | 56.33(11.69) | −4.37 | 54.33(7.04) | −1.8 | 66.33(13.05) | −3.00 | 0.54 | 54.00(12.28) | −4.31 |
| Post | 51.96(10.57)* | | 52.53(7.87) | | 63.33(7.87) | | | 49.69(9.29) | |
| Social withdraw | | | | | | | | | |
| Pre | 62.96(12.95) | −4.63 | 65.06(11.46) | −5.06 | 59.38(11.21) | −1.75 | 0.53 | 52.44(12.28) | −2.44 |
| Post | 58.33(14.41)* | | 60.00(13.37) | | 57.63(10.41) | | | 50.00(9.17) | |
| Hyperactive | | | | | | | | | |
| Pre | 65.42(13.69) | −3.04 | 70.93(7.69) | −4.18 | 69.50(8.11) | −3.62 | 0.21 | 50.56(11.22) | 2.44 |
| Post | 62.38(13.09) | | 66.75(11.00) | | 65.88(14.58) | | | 53.00(10.39) | |
| Aggressive behavior | | | | | | | | | |
| Pre | 63.29(13.32) | −3.37 | 66.000(9.58) | −1.31 | 63.13(11.58) | −3.5 | 0.93 | 50.00(6.01) | 0.67 |
| Post | 59.92(12.07) | | 64.69(13.55) | | 59.63(11.31) | | | 50.67(9.61) | |
| Delinquent | | | | | | | | | |
| Pre | 65.71(14.17) | −5.54 | 65.00(9.18) | −0.81 | 59.75(8.86) | −2.12 | 0.43 | 49.81(8.56) | 4.52 |
| Post | 60.17(15.17)* | | 64.19(12.49) | | 57.63(10.54) | | | 54.33(6.35) | |
| Internalizing behavior | | | | | | | | | |
| Pre | 61.71(11.09)* | −5.67 | 61.94(7.81) | −3.56 | 64.75(10.35) | −2.12 | 0.92 | 54.63(7.95) | −4.62 |
| Post | 56.04(11.57) | | 58.38(10.19) | | 62.63(9.01) | | | 50.01(10.14) | |
| Externalizing behavior | | | | | | | | | |
| Pre | 64.71(12.17) | −3.17 | 67.75(6.02) | −2.44 | 66.00(7.71) | −3.87 | 0.68 | 50.19(8.14) | 1.81 |
| Post | 61.54(12.37) | | 65.31(11.39) | | 62.13(9.42) | | | 52.00(8.89) | |
| ADHD-RS total score | | | | | | | | | |
| Pre | 31.52(7.01) | −10.31[d,e] | 32.62(7.31) | −8.91[d] | 30.17(6.98) | −3.09[e] | **0.04** | 10.48(5.66) | −1.05 |
| Post | 21.16(7.13)[d,e] | | 24.71(8.45)[d] | | 27.08(6.61)[e] | | | 9.43(4.92) | |
| ADHD-RS Inatt. subscore | | | | | | | | | |
| Pre | 18.20(2.7) | −5.6[a,b] | 17.90(3.62) | −4.33[a] | 17.42(3.23) | −1.59[b] | **0.01** | 7.38(3.28) | 0.43 |
| Post | 12.60(3.12)*** | | 13.57(5.07)** | | 15.83(2.59) | | | 6..90(2.84) | |
| ADHD-RS hyper. subscore | | | | | | | | | |
| Pre | 13.32(5.42) | −5.06[c] | 14.71(5.50) | −3.57 | 12.75(5.88) | −1.50[c] | 0.52 | 3.10(3.11) | 1.08 |
| Post | 8.56(4.42)*** | | 11.14(5.55) | | 11.25(4.97)[c] | | | 2.43(2.42) | |

[a] Surgical and MPH groups are significantly different, $p = .002$.
[b] Surgical & non-Tx. groups are significantly different, $p = .0001$.
[c] Surgical and non-Tx. groups are significantly different, $p = .011$.
[d] Surgical and MPH groups are significantly different, $p = .007$.
[e] Surgical and non-Tx. groups are significantly different, $p = .001$.
* $p < 0.05$.
** $p < 0.01$, $^{+}p < 0.006$, $^{++}p < 0.002$ (pair $t$-test) pre/post comparison within group. ANOVA test with post hoc comparison of improvement (pre/post) between 2 groups (between surgical group and non-Tx. group or between surgical group and MPH group): The lower the CBCL score, the better the score is. The ADHD-RS scale: higher scores indicate greater severity in ADHD, (score > 25 suggestive of ADHD in boys and >22 in girls). "Change" indicates comparison of the score "six months aftertreatment" with baseline. The negative values indicate better improvement.
*** $p < 0.001$.
**** $p < 0.0001$.

good evidence that sleep disorders lead to daytime disturbances closely mimicking ADHD [17–19]. Chervin et al. [54] also showed that snoring and other symptoms of SDB are strong risk factors for the future emergence or exacerbation of hyperactive behavior in his four-year prospective cohort study. Our children underwent an extensive evaluation performed by specialists dealing with ADHD on a routine basis. They underwent a

Table 6
Comparison of the outcome of TOVA data: comparison of pre- and post-6 months of data

| ($N = 86$) | ($N = 25$) surgical Tx. | | ($N = 27$) MPH Tx. | | ($N = 14$) non-Tx. | | ($N = 20$) control | |
|---|---|---|---|---|---|---|---|---|
| | M (SD) | Change (mean) | M (SD) | Change (mean) | M (SD) | Change (mean) | M (SD) | Change (mean) |
| Omission | | | | | | | | |
| Pre | −0.94(1.58) | 0.69 | −1.16(1.23) | −0.43 | −0.28(0.65) | −0.61 | −0.05(0.79) | −0.4 |
| Post | −0.25(1.12)* | | −1.59(1.62) | | −0.89(0.55) | | −0.45(1.04) | |
| Commission | | | | | | | | |
| Pre | 0.19(1.37) | 0.38 | −0.29(1.22) | 0.11 | −0.06(1.26) | 0.56 | 0.63(0.52) | 0.00 |
| Post | 0.57(0.86) | | −0.18(1.17) | | 0.70(0.79) | | 0.63(0.18) | |
| RT | | | | | | | | |
| Pre | −0.83(1.22) | 0.51[a] | −0.56(0.98) | −0.33[a] | −0.99(1.06) | 0.03 | 0.05(0.64) | −0.15 |
| Post | −0.32(0.94)* | | −0.89(1.01) | | −0.96(0.98) | | −0.20(0.78) | |
| RT variability | | | | | | | | |
| Pre | −0.61(1.26) | 0.47 | −0.85(1.23) | −0.2 | −1.50(1.43) | 1.34 | 0.57(0.61) | −0.69 |
| Post | −0.14(1.14) | | −1.09(1.22) | | −0.16(1.11) | | −0.12(1.00) | |
| $D'$ | | | | | | | | |
| Pre | −0.59(0.89) | 0.5 | −0.84(0.50) | −0.1 | −0.58(0.70) | −0.04 | 0.03(0.80) | −0.05 |
| Post | −0.09(1.06) | | −0.94(0.75) | | −0.62(0.67) | | −0.02(0.51) | |
| ADHD score | | | | | | | | |
| Pre | −2.08(2.14) | 2.07[a,b] | −2.69(2.20) | 0.59[a] | −2.75(2.13) | 0.94[b] | 0.66(1.38) | −1.04 |
| Post | −0.01(2.06)** | | −2.10(1.71) | | −1.81(2.43) | | −0.38(1.57) | |

RT: Response time. D′: detectability. ***$p < 0.001$ (pair $t$-test) pre/post comparison within group. ANOVA test with post hoc comparison of the improvement (pre/post) between the 2 groups. The TOVA results, compared to normal same-gender, same-age, and average IQ group, are reported as standard deviations. The more negative the standard deviations, the greater the problem. Normal range is −1.00 to +1.00. The ADHD score of −1.80 or less (more negative) is suggestive of ADHD. "Change": results showed positive value, indicating greater improvement.

[a] Indicates significant difference between surgical and MPH groups.

[b] Indicates significant difference between the surgical and non-treatment groups.

* $p < 0.05$.

** $p < 0.01$.

multidisciplinary evaluation as well as a systematic and rigorous clinical testing, with testing that may not always be offered before the label of ADHD may be applied in a clinical setting. All children underwent PSG and neurocognitive tests to avoid biases for the study; these tests were scored blind to each patient's condition. The results of the ADHD rating scale show that children who received MPH not only had an effective dose but also a beneficial effect, as the scale was <25 after six months of treatment, indicating symptomatic improvement.

The question of what the best treatment recommendation is in these cases has been an unresolved issue [38,55]. As indicated in the introduction, considering the amount of information available, a randomized surgery/MPH treatment with a placebo group was deemed unethical at this stage. Our protocol was thus a clinical study, offering parents of ADHD children and their physicians a choice of treatments based on results of a thorough investigation, including a systematic PSG. Results indicated that parents' selection allowed a good representation of children in both MPH and adenotonsillectomy groups. The non-treatment group was smaller but still allowed for statistical analyses. Parents who opted for the non-treatment approach displayed an overall trend to score the different items of the OSA-18 quality of life survey with better scores than parents of children in other groups. This parental view may explain the wait-and-see attitude and also help us to understand the parental approach. Although the study is a non-randomized open trial, from Table 1 we can see that the base data obtained between these three groups did not reach any statistical significant difference. Symptom-wise, based on the ADHD-RS and CBCL score, this group of children who had ADHD with mild OSA was found to be within mild to moderate ADHD severity.

The ANOVA performed for comparing the two groups of surgical and MPH treatment shows in all cases here better results in children who were submitted to surgery than in those who received MPH treatment. Comparison of the behavioral scales obtained post-surgery and MPH treatments show a significant difference for total ADHD score and inattention subscore, but not for the CBCL subscales which have much greater standard deviation. This greater variability of response from parents and teacher is an interesting point as this greater variability was also found in the same group of children on another questionnaire not shown here, the disruptive behavior rating scale, while the ADHD-RS,

filled out by two independent child psychiatrists, had more internal consistency.

The effect on PSG variables, as expected, was much more important post-surgery. There was not only an effect on AHI, total sleep time and snoring index, but also on the OSA-18 quality of life survey. Daytime drowsiness, poor attention span and sleep disruption were still reported with MPH treatment despite its stimulant effect. The difference between post-surgery and MPH results were also noted with TOVA: with MPH, mild improvement was noted but subscales (Omission, Response Time and Response Time Variability) and total ADHD score did not reach significance in opposition to post-surgery condition. The TOVA results are very much in line with the results from the ADHD-RS total score. The results strongly support the need to treat OSA first when identified in the presence of an ADHD clinical presentation.

The true relationship between OSA and ADHD is still unclear [26]. So far, there is no study to prove whether it is a correlation or comorbidity. In our study, as we compared the surgical group to the non-treatment group (i.e., with treatment of the factor "OSA"), the improvement of AHI in the surgical group and symptoms related to it could be obviously observed. Questionnaire response indicates that caregivers also observed improvement in patients' sleep disorders and daytime behavioral problems (OSA-18). After treating this "OSA" factor alone, the ADHD symptoms in the surgical group were also improved more than the non-treatment based on the ADHD-RS and CBCL. In addition, the neurocognitive test TOVA showed improvement in attention and response time. The TOVA ADHD score ($<-1.8$) after surgery was close to that measured for the normal control group. TOVA has been previously used in children with OSA (diagnosed based on clinical presentation and not on PSG) prior to and after adenotonsillectomy, [56] and significant changes at this test post-surgery were noted also in these OSA children, confirming the neurocognitive impact of OSA. The non-treatment group, independent of AHI or other sleep disturbances or ADHD symptoms, had no observed clinical improvement after half a year. Even if the CBCL shows improvement at six months, the ADHD-RS scores of these untreated children ($27.08 \pm 6.61$) are still in the ADHD diagnosis range and far from those noted on controls.

In summary, when comparing our groups with only one factor (OSA) modified, it is clear that improvement of the sleep disorder and ADHD symptoms changed in parallel. A word of caution, however, is warranted: despite significant improvement pre- and post-surgery, a difference is still seen between the scores of the improved ADHD subjects and the scores of CBCL and ADHD-RS obtained for normal controls (and this is even more evident with the other treatment approaches). Several explanations for this finding include the fact that our study was not double-blind and that scales are always subjective. Despite these limitations, our results clearly indicate the impact of a low AHI not only on sleep variables but also on neurocognitive functions and behavioral symptoms on mild to moderate ADHD. More importantly, the results emphasize the need to search for symptoms associated with OSA in ADHD and to perform a PSG, looking for a low AHI score of $> 1$ considered abnormal, as treatment of OSA will alleviate long-term prescription of a drug with potential side effects.

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