Premiere Publications from
The Triological Society

Read all three of our prestigious publications, each offering high-quality content to keep you informed with the latest developments in the field.

The Laryngoscope
FOUNDED IN 1896
Editor-in-Chief: Michael G. Stewart, MD, MPH
The leading source for information in head and neck disorders.
Laryngoscope.com

Laryngoscope Investigative Otolaryngology
Open Access
Editor-in-Chief: D. Bradley Welling, MD, PhD, FACS
Rapid dissemination of the science and practice of otolaryngology-head and neck surgery.
InvestigativeOto.com

ENTtoday
A publication of the Triological Society
Editor-in-Chief: Alexander Chiu, MD
Must-have timely information that Otolaryngologist-head and neck surgeons can use in daily practice.
Entttoday.org

WILEY
Persistent Respiratory Effort After Adenotonsillectomy in Children With Sleep-Disordered Breathing

Jean-Benoit Martinot, MD; N. Nam Le-Dong, MD, PhD; Stéphane Denison; Hervé Jean-Pierre Guénard, MD, PhD; Jean-Christian Borel, PhD; Philip E. Silkoff, MD; Jean-Louis Pepin, MD, PhD; David Gozal, MD, MBA, FCCP

Objectives: Adenotonsillectomy (AT) markedly improves but does not necessarily normalize polysomnographic findings in children with adenotonsillar hypertrophy and related sleep-disordered breathing (SDB). Adenotonsillectomy efficacy should be evaluated by follow-up polysomnography (PSG), but this method may underestimate persistent respiratory effort (RE). Mandibular movement (MMas) monitoring is an innovative measurement that readily identifies RE during upper airway obstruction. We hypothesized that MMas indices would decrease in parallel of PSG indices and that children with persistent RE more reliably could be identified with MMas.

Methods: Twenty-five children (3–12 years of age) with SDB were enrolled in this individual prospective-cohort study. Polysomnography was supplemented with a midsagittal movement magnetic sensor that measured MMas during each respiratory cycle before and > 3 months after AT.

Results: Adenotonsillectomy significantly improved PSG indices, except for RE-related arousals (RERA). Mandibular movement index changes after AT significantly were correlated with corresponding decreases in sleep apnea–hypopnea index (AHI) and O2 desaturation index (ODI) (Spearman’s rho = 0.978 and 0.922, respectively), whereas changes in MMas duration significantly were associated with both RERA duration (rho = 0.475, P = 0.017) and index (rho = 0.564, P = 0.003). Conditional multivariate analysis showed that both AHI and RERA significantly contributed to the variance of MMas index after AT (P = 0.0003 and 0.0005, respectively), whereas MMas duration consistently was related to the duration of RERA regardless of AT.

Conclusion: Adenotonsillectomy significantly reduced AHI. However, persistent RERA were apparent in a significant proportion of children, and this was reflected by the remaining abnormal MMas pattern. Follow-up of children after AT can be recommended and readily achieved by monitoring MMas to identify persistent RE.

Key Words: Adenotonsillectomy, respiratory effort, mandibular movements, obstructive sleep apnea.

Level of Evidence: 4.

INTRODUCTION

Adenotonsillar hypertrophy is the main contributing factor for the presence of obstructive sleep-disordered breathing (SDB) in children.1,2 Consequently, surgical adenotonsillectomy (AT) currently is recommended as the first-line treatment for children with SDB.1,2 However, the efficacy of AT has been questioned, with several studies identifying the presence of residual obstructive SDB after surgery in a significant proportion of surgically treated children,3–5 as well as being accompanied by suboptimal improvements in attentiveness and executive function after AT.6,7 These observations have raised concerns about the appropriate identification of candidates for AT,8 as well as identification of those children in whom postoperative follow-up polysomnography (PSG) might be indicated.9

Although PSG remains the gold standard for diagnosing persistent respiratory effort (RE), it is a labor-intensive and onerous procedure that would not be practically implementable in the follow-up of children after AT. The main signal for detecting persistent respiratory effort is flow limitation, but a high quality of nasal pressure recording is more difficult to achieve in children than in adults. We have previously demonstrated that abnormal patterns of mandibular movements (MMas) readily can identify RE during sleep in children with adenotonsillar hypertrophy and obstructive SDB.10,11 We hypothesized that MMas indices for RE would decrease in parallel with classical PSG indices, and that children with persistent respiratory effort more reliably could be identified with MMas. Accordingly, we explored the effects of AT on the MMas patterns in a group of children with obstructive SDB to determine the

Additional supporting information may be found in the online version of this article.

From the CHU UCL Namur (J.-M.M., S.D.); the RespiSom Private Research Medical Center (N.N.L.-D.); Namur, Belgium; the University Bordeaux (H.J.P.-D.), Bordeaux, France; the University Grenoble Alpes (J.-C.B., J.-L.P.); CHU de Grenoble, Laboratoire EFCR, Pôle THORAX et VAISSEMAUX (J.-L.P.), Grenoble, France; the Temple University (P.E.S.), Philadelphia, Pennsylvania; and the Department of Pediatrics, Pritzker School of Medicine, Biological Sciences Division, University of Chicago (D.G.), Chicago, Illinois, U.S.A.

Editor’s Note: This Manuscript was accepted for publication July 6, 2017.

The authors have no funding, financial relationships, or conflicts of interest to disclose.

Send correspondence to Jean-Benoit Martinot, MD, Centre du Sommeil et de la Vigilance, CHU UCL Namur Site Ste Elisabeth, 15, Place Louise Godin, 5000 Namur, Belgium. E-mail: martinot.j@scarlet.be

DOI: 10.1002/lary.26830
postsurgical changes in MMas and the relationship with persisting RE as assessed with conventional PSG.

MATERIALS AND METHODS

Subjects

The study was performed according to the Declaration of Helsinki, approved by the Medical Ethics Committee of the Clinique et Maternité Sainte Elisabeth Namur Belgium (B166201215073), and parents or legal caretakers provided written informed consent prior to study commencement. All enrolled children were confirmed to have SDB based on in-lab polysomnography. Adenotonsillectomy was performed if the sleep apnea–hypopnea index (AHI) was > 5/hour total sleep time (TST) or between 2 and 5/hour TST in the presence of morbidities involving the cardiovascular system, altered somatic growth, evidence of excessive daytime sleepiness, or the presence of cognitive or attention deficits. None of the children exhibited evidence of midface or mandibular hypoplasia, or was known to suffer from neuromuscular or genetic abnormalities. Potential concurrent atopy and clinical asthma were inquired. The presence of residual symptoms after AT was recorded, and laboratory-based PSG was performed in all subjects 3 to 6 months after surgery.

Polysomnography Recording

Routine laboratory-based PSG was recorded with a Dream Medatec device (Brussels, Belgium). The parameters monitored included electroencephalogram (EEG) (Fz-A+, Cz-A+, Pz-A+); right and left electrooculogram; submental electromyography (EMG); tibial EMG; chest and abdominal wall motion by respiratory inductance plethysmography (SleepSense S.L.P. Inc, St. Charles, Illinois, U.S.A.); nasal and oral flows, respectively, with a pressure transducer and a thermistor; and O2 saturation by digital oximetry displaying a pulse wave form (Nonin, Nonin Medical, Plymouth, Minnesota, U.S.A.).

MMas

A midsagittal MMas magnetic sensor (Brizzy Nomics, Liege, Belgium) measured the distance in mm between two parallel, coupled, resonant circuits placed on the forehead and on the chin (Fig. 1). The transmitter generated a pulsed magnetic wave of low energy, at 10 Hz. The change in the magnetic field...
recorded at the receiver is inversely related to the cube of the distance between the chin and forehead probe. The probes were connected to an electronic module, and a measure of the distance was computed before transmission to the PSG. The resolution of the measurement was 0.1 mm. The mandibular positions during each respiratory cycle were recorded in the breathing frequency band of 0.25 to 0.6 Hz. The signal was interpreted as follows: the more negative the value of the signal became during inspiration, the lower the mandible position and the greater the mouth opening.

**Patterns of MMas**

Our previously published findings in children indicate that an exaggerated amplitude of MMas (≥ 0.3 mm from peak to peak) over at least two respiratory cycles constitutes a reliable marker of RE.⁹,¹⁰ Such periods of RE often terminate with a cortical arousal: in this case, MMas firstly comprises a period of at least 0.3 mm, and then a sudden very large MMas (amplitude > 1 mm) disrupting the previous breathing frequency and the envelope around the previous peak-to-peak mandibular displacement.

**Data Analysis and Scoring**

All respiratory events on the PSG were scored separately by two trained observers who were blinded to the MMas according to the current American Academy of Sleep Medicine rules for the scoring of sleep and associated events.¹³ Detection of RE was based on the monitoring of out-of-phase thoracoabdominal excursions, with an uncalibrated respiratory inductance plethysmography and/or on the flattening of the inspiratory portion of the nasal pressure wave form often associated with snoring. Visual analysis of the pulse transit time signal (inspiratory increase in pulse transit time between two adjacent cardiac beats) also allowed the observers to relate the events to RE.¹⁴⁻¹⁶ When this period of RE ended with a cortical arousal and lasted at least two breaths, it was denoted as a respiratory effort-related arousal (RERA). MMas were all scored by a European Sleep Research Society-certified sleep specialist trained in the identification of abnormal MMas patterns and blinded to any other findings from PSG (Fig. 1) (Supporting Fig. S1).

**Statistical Methods**

Data were analyzed using R programming language (R Foundation for Statistical Computing, Vienna, Austria; http://www.R-project.org). Spearman’s rank-order correlation analysis was used for evaluating the relationship between the variables. Hourly index and cumulated durations of respiratory events during PSG and MMas recording were analyzed by linear mixed models,¹⁷ with best-fit distributions using the gamlss package. A Yeo-Johnson transformation¹⁸ was applied for normalizing those data when necessary. This modelling approach enabled capturing of the natural scale and the distribution mode of data, as well as mixed, random effects and multivariate analysis. Statistical inference was based on 95% confidence interval (CI), and null hypothesis testing was set at statistical significance level of 5%.

**RESULTS**

Twenty-five consecutive children with SDB and adenotonsillar hypertrophy consecutively were recruited and had PSG evaluations performed before and after AT, with a time interval of 3.24 to 24 months. Taken as covariate in the mixed model, the time interval did not show any significant effect on the PSG and MMas outcomes (Supporting Table SII). The clinical characteristics and the main polysomnographic outcomes are reported in Tables I and II. The distributions of the results can be found in the Supporting Figures S2a,b and the Supporting Table SII.

**Effects of Adenotonsillectomy on MMas and Polysomnography Indices**

The effects of AT on PSG measures and MMas indices are shown in Figure 2 and Supporting Table SIII. Most of indices were significantly improved, including: AHI (21 of 25 children, P < 0.001), respiratory disorder index (RDI) (21 of 25, P < 0.001), O₂ desaturation index (ODI) (18 of 25, P < 0.001), and cortical arousal index (CAI) (21 of 25, P < 0.001). Adenotonsillectomy also favorably impacted on MMas (significantly reduced in 22 of 25 children, P < 0.001). The concomitant changes in AHI and MMI as distributions according to the severity of SDB, as estimated by AHI < 1, AHI > 1 but < 5, and AHI > 5/hour TST, are shown in Figures 3a and 3b. As presented in Table III, the reduction of MMas index was strongly correlated to the corresponding improvements in PSG indices (Spearman's

---

**TABLE I.**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Before-AT (n = 25)</th>
<th>After-AT (n = 25)</th>
<th>Absolute Change</th>
<th>Relative Change (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (F/M)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (yr)</td>
<td>5.9 (1.7 to 13.0)</td>
<td>7.0 (2.1 to 14.4)</td>
<td>9 (3.7 to 22.6)⁶ (months)</td>
<td>16.4 (4.6 to 42.8)⁶%</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>16.6 (14.5 to 31.7)</td>
<td>0.52 (0.69 to 3.42)⁶</td>
<td>3.4 (–6.0 to 21.2)⁶%</td>
<td></td>
</tr>
<tr>
<td>Obesity (n,%)⁷</td>
<td>6 (24%)</td>
<td>6 (24%)</td>
<td>0</td>
<td>0 %</td>
</tr>
<tr>
<td>Allergy (n,%)</td>
<td>14 (58%)</td>
<td>5 (22%)</td>
<td>–9</td>
<td>–64.3%</td>
</tr>
<tr>
<td>Witnessed apnea (n,%)</td>
<td>24 (96%)</td>
<td>3 (12%)</td>
<td>–21</td>
<td>–87.5%</td>
</tr>
<tr>
<td>Witnessed snoring (n,%)</td>
<td>23 (92%)</td>
<td>9 (36%)</td>
<td>–14</td>
<td>–60.9%</td>
</tr>
<tr>
<td>Behavior problems (n,%)</td>
<td>16 (67%)</td>
<td>4 (17%)</td>
<td>–12</td>
<td>–75.0%</td>
</tr>
</tbody>
</table>

*Median (95% CI).
†Obesity is defined by a BMI >95th centile on the childhood growth curve by World Health Organization.
Relative change (%) is determined as 100(after-before)/before.
BMI = body mass index; CI = confidence interval; F = female; M = male.
rho coefficients ranging between 0.92 and 0.98), except for RERA. The presence of elevated BMI appeared to limit the beneficial effects of AT on MMas index (rho = 0.46, P = 0.02).

RE-related arousals index did not significantly change following AT (P = 0.229). Only nine children showed an improvement in RERA, whereas 15 children showed increased RERA. The effects of obesity and underlying atopy/asthma status on the risk for residual RE examined by Wilcoxon’s test emerged as negative (Supporting Fig. S3).

A multivariate-conditional analysis (Supporting Table SIV) showed that, after AT, the changes in both AHI and RERA index contributed significantly to the variance of MMas index improvements (P = 0.0003 and 0.0005, respectively).

### TABLE II.

**PSG- and MMas-Based Respiratory Events in the Children (n = 25) Before and After AT.**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Before-AT (n = 25)</th>
<th>After-AT (n = 25)</th>
<th>Absolute Change</th>
<th>Relative Change (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PSG and MMas Index</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TST (minutes)</td>
<td>482 (296.2 to 533.6)</td>
<td>490 (338.0 to 550.6)</td>
<td>26 (-105.2 to 221.2)</td>
<td>5.4 (-21.2 to 78.9)</td>
</tr>
<tr>
<td>Arousal index (n/h)</td>
<td>15.1 (7.1 to 33.4)</td>
<td>9.4 (3.6 to 14.9)</td>
<td>-7.17 (-25.18 to 1.87)</td>
<td>-49.8 (-83.4 to 15.3)</td>
</tr>
<tr>
<td>AHI (n/h)</td>
<td>11.3 (2.8 to 31.3)</td>
<td>4.0 (0.5 to 9.3)</td>
<td>-7.48 (-27.75 to 1.70)</td>
<td>-62.6 (-96.4 to 33.7)</td>
</tr>
<tr>
<td>RERA index (n/h)</td>
<td>1.2 (0.0 to 5.7)</td>
<td>1.9 (0.3 to 5.3)</td>
<td>0.54 (-4.22 to 4.07)</td>
<td>57.5 (-88.3 to 388.5)</td>
</tr>
<tr>
<td>RDI (n/h)</td>
<td>12.5 (4.3 to 31.8)</td>
<td>5.6 (1.8 to 11.84)</td>
<td>-6.70 (-27.54 to 1.529)</td>
<td>-53.7 (-93.2 to 35.5)</td>
</tr>
<tr>
<td>ODI (n/h)</td>
<td>1.7 (0.5 to 54.3)</td>
<td>1.2 (0.2 to 6.5)</td>
<td>-3.25 (-52.76 to 1.21)</td>
<td>-77.6 (-98.1 to 350.9)</td>
</tr>
<tr>
<td>MMias (n/h)</td>
<td>12.9 (4.4 to 26.5)</td>
<td>7.5 (2.4 to 12.6)</td>
<td>-5.45 (-19.28 to 3.15)</td>
<td>-41.7 (-67.2 to 60.8)</td>
</tr>
<tr>
<td><strong>Cumulated Duration</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dtAH (minutes)</td>
<td>28.1 (4.3 to 178.8)</td>
<td>8.8 (0.8 to 26.4)</td>
<td>-23.06 (-165.65 to 4.34)</td>
<td>-73.9 (-97.4 to 106.2)</td>
</tr>
<tr>
<td>dtRERA (minutes)</td>
<td>78.8 (0.4 to 287.1)</td>
<td>77.9 (2.5 to 193.2)</td>
<td>-5.27 (-253.89 to 124.44)</td>
<td>-6.7 (-97.4 to 243.9)</td>
</tr>
<tr>
<td>dtRD (minutes)</td>
<td>128.9 (24.9 to 374.3)</td>
<td>97.2 (12.5 to 201.0)</td>
<td>-37.38 (-293.62 to 142.47)</td>
<td>-24.6 (-91.9 to 516.8)</td>
</tr>
<tr>
<td>dtMMas (minutes)</td>
<td>249.9 (115.2 to 471.2)</td>
<td>177.9 (26.0 to 387.9)</td>
<td>-33.44 (-364.13 to 173.38)</td>
<td>-24.2 (-92.4 to 100)</td>
</tr>
</tbody>
</table>

**Note:** all parameters are presented as Median and (95% CI). Relative change (%) is determined as 100*(after–before)/before.

AHI = apnea–hypopnea index; CAR = cortical arousal; CI = confidence interval; dt = cumulated duration; min = minutes; MMias = mandibular movement (type MMas) index; ODI = O₂ desaturation index; PSG = polysomnographic; RD = respiratory disorder; RDI = respiratory disorder index; RERA = respiratory effort-related arousal index; TST = total sleep time.

**Fig. 2.** Change after AT in PSG and MMas hourly index (A) and cumulated durations (B) of sleep-disordered breathing parameters. The circle and error bars represent marginal effect of the models that evaluate the change of PSG and MMas indices and duration under AT. Detailed information of these models could be found in the Supporting Tables SII and SIII.

AHI = apnea–hypopnea index; AT = adenotonsillectomy; CAR = cortical arousal index; dt = cumulated duration (in minute); MMAs = mandibular movement; MMias = mandibular movement (type MMas) index; ODI = O₂ desaturation index; PSG = polysomnographic; RD = respiratory disorder; RDI = respiratory disorder index; RERA = respiratory effort-related arousal; TST = total sleep time; (*) = significant: statistical test against a null-hypothesis indicating that AT showed no effect on the target variable; NS = nonsignificant.
Effect of Adenotonsillectomy on the Cumulated Durations of MMas and Polysomnography Respiratory Events

To further examine the relationship between the changes in MMas and the incongruence of the changes in AHI and RERA PSG-indices, we evaluated the cumulative duration of these events using TST.

The effect of AT on duration of PSG and MMas events is presented in Figure 3 and Supporting Table 3. The variance in MMas duration could not be modelled by the effect of AT alone, and a multivariate model was required that took into account the partial effects of both AH and RERA durations (Supporting Table SV).

The model suggested that RERA and AH significantly might contribute to the change of MMas duration by a conditional effect: at baseline, both AH and RERA durations significantly participate in the variance of MMas duration \( (P = 0.03 \text{ and } 0.005, \text{ respectively}) \); after AT, MMas duration was not reduced significantly by the decrease in AH duration \( (P = 0.98) \) but was proportional to RERA duration \( (P = 0.008) \) (Supporting Fig. S4).

Taken together, the univariate and multivariate analyses showed that, although AT significantly did improve the duration of AHI events, the changes in MMas duration consistently were related to RERA.

Changes in MMas duration only were correlated to the corresponding changes in the duration of RERA, RERA index, and RDI (Table III), suggesting that AT only improved AHI and MMas index but did not improve the duration of the residual post-AT events.

**DISCUSSION**

This study shows that MMas recordings can identify dynamic changes in SDB-related measures after AT, and that MMas monitoring can detect persistent RE after AT. Indeed, the index of the typical MMas observed in presence of obstructive sleep apnea or hypopnea (MMas) decreased to the same extent as AHI when the latter was assessed by PSG. Furthermore, the cumulative duration of MMas closely correlated to the duration of sustained RE events that were terminated by CAr (RERAs). Current findings corroborate our previous studies whereby sleep MMas emerged as a reliable marker for RE in the presence of increased upper airway resistance in children.\(^{10} \)

Indeed, when MMas events are restricted to the time in which either obstructive apnea or hypopnea occur, AHI and MMas are strongly correlated. However, even when AHI normalizes following AT, prolonged periods of RE (some of them lasting several minutes) still are identified and end in cortical arousal. Thus, strong correlations emerged between MMas and RERA for both their respective indices and durations after AT. These observations further reinforce the concept that MMas can reliably detect RE both during apnea and hypopnea, as well as when only respiratory effort is present and terminates in arousal, that is, RERA. Of note, the TST duration following AT (on average increased by 26 minutes) did not impact either the index or the duration of the covariables.

The classification of AHI and MMas according to the level of disease severity showed that, when post-AT AHI remained elevated, similar findings were detected by MMSas. Respiratory effort characteristics, when assessed by mandibular displacement monitoring during obstructive sleep apnea, hypopnea, or periods of flow limitations, typically are accompanied by mouth opening and resolve with abrupt mouth closure on arousal. This is easily identified by a sharp and large movement of the mandible, disrupting its previous displacement and resolve time occurred in 16 children and usually was terminated by cortical arousals.\(^{19,20} \) These events suggest the presence of residual increased upper airway resistance. Upper airway collapsibility is lower in children compared to adults,\(^{21} \) and in this population is expected that the spectrum of residual events depict a predominance of RERAs instead of apneas or hypopneas. This underscores the need for reliable tools to monitor respiratory effort in full polysomnography and cardiopulmonary sleep studies in the pediatric population.

The clinical significance and consequences of persistent upper airway resistance in sleeping children after AT remain uncertain and have not been thoroughly investigated, probably because these events are underrecognized in a majority of sleep studies.\(^{4,22,23} \) Notwithstanding, the same considerations that prompted referral for AT should still hold, albeit to a lesser degree, when evidence of residual SDB and RE persists after AT. Indeed, AT, the first-line treatment of childhood OSA, did not exhibit the anticipated improvements in cognitive performance and behavioral abnormalities in the context of the only randomized controlled trial for AT, the Childhood Adenotonsillectomy Trial (CHAT).\(^{6} \) However, we should also note that more recent subanalyses from this trial revealed some selective improvements across specific functional categories, including

---

**TABLE III.**

Relationship Between the Changes in MMas Duration and Other PSG Variables Under Adenotonsillectomy.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Spearman’s rho</th>
<th>95% CI</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ODI</td>
<td>0.92</td>
<td>0.83 to 0.96</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>AHI</td>
<td>0.98</td>
<td>0.95 to 0.99</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>RERA index</td>
<td>0.02</td>
<td>−0.4 to 0.41</td>
<td>0.9332 (NS)</td>
</tr>
<tr>
<td>RDI</td>
<td>0.98</td>
<td>0.96 to 0.99</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>CAr index</td>
<td>0.92</td>
<td>0.82 to 0.96</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>ODI</td>
<td>0.20</td>
<td>−0.21 to 0.55</td>
<td>0.334 (NS)</td>
</tr>
<tr>
<td>AHI</td>
<td>0.25</td>
<td>−0.16 to 0.59</td>
<td>0.226 (NS)</td>
</tr>
<tr>
<td>RERA index</td>
<td>0.56</td>
<td>0.22 to 0.78</td>
<td>0.003</td>
</tr>
<tr>
<td>RDI</td>
<td>0.29</td>
<td>−0.11 to 0.62</td>
<td>0.155 (NS)</td>
</tr>
<tr>
<td>CAr index</td>
<td>0.35</td>
<td>−0.05 to 0.66</td>
<td>0.083 (NS)</td>
</tr>
<tr>
<td>AH duration</td>
<td>0.20</td>
<td>−0.21 to 0.55</td>
<td>0.328 (NS)</td>
</tr>
<tr>
<td>MMas duration</td>
<td>0.47</td>
<td>0.09 to 0.73</td>
<td>0.017</td>
</tr>
<tr>
<td>RD duration</td>
<td>0.46</td>
<td>0.09 to 0.73</td>
<td>0.019</td>
</tr>
</tbody>
</table>

AHI = apnea–hypopnea index; CAr = cortical arousal; CI = confidence interval; MMas = mandibular movement (type MMas) index; NS = not significant; ODI = \( \text{O}_2 \) desaturation index; PSG = polysomnographic; RD = respiratory disorder; RDI = respiratory disorder index; RERA = respiratory effort-related arousal index.
Persistent RE as a cause of sleep disruption has been suspected as a risk factor for aggressive behavior, learning disabilities, daytime somnolence, and attention deficit/hyperactivity. Prolonged durations of RE have been documented in children with SDB, but it remains uncertain if AT, even when it normalizes AHI, can decrease RERAs. Therefore, it is possible that the relatively disappointing neurobehavioral outcomes associated with the CHAT study may be partly explained by the presence of persistent RE and unrecognized sleep fragmentation. Accordingly, studies urgently are needed to examine the consequences of persistent respiratory abnormalities after AT, even if quality-of-life improvements are apparent after the surgical intervention. To this effect, the ability to record persistent RE with a simple device, such as a MMas magnetometer that is suitable for domiciliary use, potentially would be attractive for monitoring children after AT and merits further exploration. We also will remark that parentally reported snoring was not consistently indicative of persistent RE detected as RERA, a finding that reinforces the need for objective measurements to be performed after AT. Of note, this assertion is true for both pre-and postoperative PSGs when considering the current tools routinely used to estimate RE. Our data and other recent studies demonstrate that there is room for improvement in this specific domain by using new measurements such as MMas or innovative analytical approaches in the context of thoracoabdominal asynchrony.

Fig. 3. Changes under adenotonsillectomy of AHI (A) and MMas (B) distributions according to sleep-disordered breathing severity. AHI = apnea-hypopnea index; MMas = mandibular movement (type MMas) index.
Study Limitations

It has been reported that approximately 50% of RE periods in children do not terminate in a cortical arousal, and their cessation may occur in the presence of subcortical or autonomic arousals and potentially lead to sympathetic activation.\textsuperscript{35–37} In the present study, we considered only cortical EEG arousals to detect the previous period of RE, and this approach could have underestimated the degree of persistent RE. Moreover, we did not use end-tidal PCO2 monitoring to support detection of sustained RE leading to obstructed alveolar hypoventilation and RERA. Instead, increased RE was identified in the presence of a prolonged sequence of breaths characterized by a plateau on the inspiratory portion of the nasal pressure and at least one of the following associated signs: an out of phase in thoracoabdominal excursions, an inspiratory pulse transit time increase up 15 ms between adjacent cardiac beats, or snoring. We therefore cannot comment as to whether addition of end-tidal carbon dioxide monitoring would have further increased the evidence for residual increased RE.

Our study was not powered to explore a potential relationship of MMs with the clinical phenotype of pediatric SDB; therefore, no quality-of-life questionnaires or neuropsychological testing were incorporated to the protocol. Future studies, including these endpoints, with regard to MMs as a method to assess RE are required. Of note, REM sleep stage is prone to modify the behavior of the mandible by decreasing the tonic activity of the submental muscles. However, the high resolution (one-tenth mm) of the technique enables detection of very small respiratory displacements of the mandible, which limits the possibility of not recognizing RE events.

CONCLUSION

In conclusion, AT significantly reduces AHI, CARI, and ODI, and the MMs index decreases to the same extent as PSG-based AHI. However, the number of RERA episodes did not normalize, which also was reflected by the presence of persistently abnormal MMs pattern. Follow-up of children after AT surgery using objective assessments is recommended, and home-based single-channel MMs monitoring should enable convenient and reliable identification of persistent RE.

Acknowledgment

J-B.M., N.N.L-D., J-L.P., and D.G. designed the study. J-B.M., S.D., and J-C.B. collected and verified the data. J-B.M., N.N.L-D., J-C.B., H-J.F.G., and P.E.S. analyzed the data and interpreted the results. J-B.M., N.N.L-D., H-J.F.G., J-C.B., P.E.S., and J-L.P. wrote the article. J-B.M. and N.N.L-D. created the illustration. J-L.P., P.E.S., H-J.F.G., and D.G. corrected and approved the final version of article. All the authors read and approved the final version of article.

The authors wish to thank Dr. Nathalie Coumans, Ms. Valérie Cuthbert, and Ms. Sandrine Lamy for their secretarial assistance and support.

BIBLIOGRAPHY


