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Original Article

Bimaxillary expansion therapy for pediatric sleep-disordered breathing

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ABSTRACT

Introduction: The aim of this retrospective study was to evaluate the results of bimaxillary expansion as a treatment option for pediatric sleep-disordered breathing.
Methods: Forty-five children, aged 3–14 years, with sleep-disordered breathing underwent bimaxillary expansion. They were subjected to baseline clinical evaluations, cephalometric X-rays, and polygraphic sleep studies. Three to six months after bimaxillary expansion, posttreatment sleep studies were performed. Data were analyzed with nonparametric Wilcoxon signed-rank test, and Spearman's correlations were performed to correlate cephalometric facial structures to the effectiveness of treatment.
Results: The majority of the children ($n = 30$) showed improvement in their sleep scores and symptoms after bimaxillary expansion. The initial severity of the obstructive sleep apnea (OSA) indicated by the apnea–hypopnea index (AHI) was a much better predictor of positive results. However, in the "mild OSA" group, patients with smaller MP–SN or counterclockwise mandibular growth, worsened with bimaxillary expansion, while patients with clockwise mandibular growth showed greater improvement; in the "severe OSA" group, patients who initially had shorter mandibular base lengths showed lesser AHI improvements.
Conclusions: Bimaxillary expansion can be a treatment option for improving respiratory parameters in children with sleep-disordered breathing. This study also suggests that retrognathia in an anterior growth rotation pattern may not respond to efforts of bimaxillary expansion.

1. Introduction

Obstructive sleep apnea syndrome (OSAS) is the most common sleep-disordered breathing (SDB) abnormality. It is characterized by the abnormal collapse of the upper airway pharyngeal muscles during sleep resulting in sleep disruption [1–3]. In both children and adults, this abnormal collapsibility has been related to the state of sleep itself and intrinsic and extrinsic governing factors. During sleep, the pharyngeal muscle tone and reflex responses are modified, rendering the airway more collapsible. The upper airway has an intrinsic collapsibility that may be characterized by the "critical closing pressure," but extrinsic factors may lead to increased collapsibility. The three external factors that are firmly established to affect the upper airway space are the nasal cavity, and the retropalatal and the retroglossal upper airway space.

Obstructions that affect the mode of breathing can affect the width, length, and height of the maxillomandibular complex with a subsequent impact on the nasal cavity, and retropalatal and retroglossal upper airway space, as shown in both children and experimental animals [4–8]. Craniofacial alterations of a narrowed maxilla, altered tongue position, and narrowed dentition have been described in numerous studies [9–13]. Concomitant changes in the lower jaw have been cited as an altered mandibular posture and clockwise rotation. Similar to the narrowing of the maxilla, narrowing of the mandibular width has been shown [14,15]. Maxillary narrowing has often been described and treated using rapid maxillary expansion (RME), but the mandibular narrowing is seldom mentioned and thus not treated.

RME was first suggested as a therapy for adult OSA in 1998, to address maxillary width alterations [16]. It was based on the previous description of efficacy of RME on other medical conditions such as enuresis, diaphoresis, allergies, and asthma as early as 1974 [17]. In children, the peak onset of symptoms occurs between two and eight years of age [3]. The long-term health risks of untreated sleep apnea in the pediatric population parallel the same risks as the adult population. The emphasis in effectively treating this disease is on early detection and early treatment. In 2004, Pirelli et al. [18]

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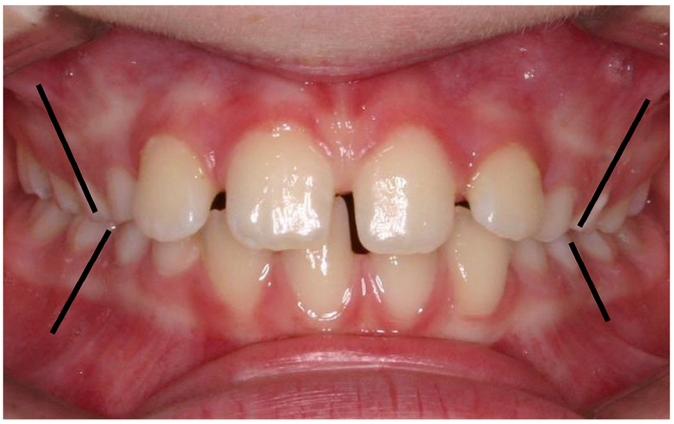

Fig. 1. Depiction of inward lingual tipping of the upper and lower dentition. Legend: Black lines indicate the lingual inclination of the upper and lower teeth.

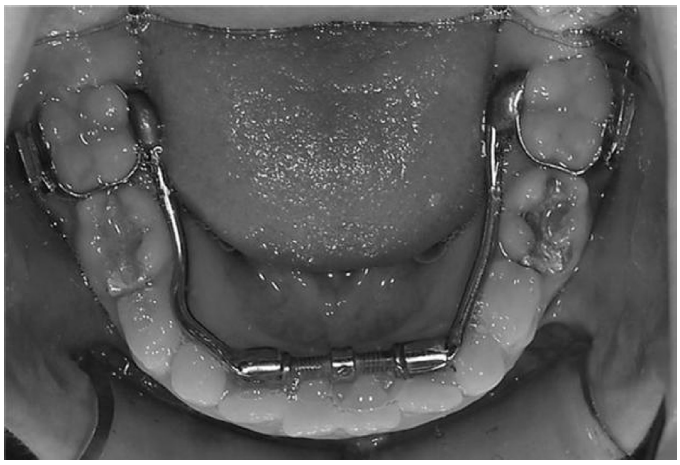

Fig. 2. Lower expander anchored to the permanent lower first molars.

published the positive impact of RME on a group of OSA children with narrow maxillas and no adenotonsillar hypertrophy, with a recent follow-up study demonstrating treatment stability after a 12-year follow-up [19]. Other RME published studies also describe the variable effects of maxillary expansion in treating children with narrow palates and/or retrognathic mandibles [18,20–23].

Skeletal maxillary expansion using RME can be achieved by applying tension across the intermaxillary or midpalatal synchondrosis/suture. The maxilla can be enlarged in width, while this suture is patent in growing children. Unlike the maxilla, the mandible as one piece of endochondral bone without sutures is not subject to skeletal width expansion, but instead it will only yield dentoalveolar expansion. The skeletal widths (or archforms) of the jaws can be quantified with radiography, but it can also be assessed visually on intraoral examination by the lingual inclination of the maxillary and mandibular dentition (Fig. 1), and on dental casts by the reduced intermolar distance [24].

The aim of this retrospective study differs from all previous maxillary expansion work by assessing the effect of bimaxillary expansion (BE) treatment on 45 children with varying facial types or jaw morphologies who were diagnosed with OSA. Because OSA children often present with variable growth disturbances involving both maxilla and mandible, BE was used as a strategy to address the bimaxillary width distortions that can result from abnormal breathing. The effectiveness of BE as a treatment option for pediatric SDB was first described a decade ago [25], and it refers to skeletal width expansion in the upper jaw and dentoalveolar width expansion in the lower jaw.

2. Materials and methods

2.1. Patient selection

The data were obtained retrospectively from one orthodontic office from children who were diagnosed with OSA and treated with BE between 2001 and 2011. The inclusion criteria were as follows: (1) diagnosis of SDB before the initiation of any orthodontic treatment, (2) treatment with BE without any cotherapy, and (3) completeness of data in the chart (including polysomnography (PSG) reports and lateral cephalograms). The exclusion criteria were as follows: (1) any concurrent soft-tissue surgery (adenoidectomy, tonsillectomy, and turbinectomy), (2) initial and posttreatment PSG studies performed under the control of different certified specialists and sleep laboratories, and (3) absence of syndromic craniofacial anomalies. Forty-five patients' charts satisfied the inclusion and exclusion criteria, and these charts were analyzed.

With a mean age of 7.58 ± 2.82 (range 3–14 years), 45 children (32 boys and 13 girls) reported symptoms of SDB and had undergone a type 1 PSG. Five children with associated neurological syndromes not clinically affecting their overall facial growth (Tourette's syndrome, autism and fetal alcohol syndrome, and cerebral palsy) were also included in this retrospective review. These data were rendered anonymous as requested by the institutional review board (IRB). No sample size calculation was performed, as this is the first study to assess the effect of BE on SDB in children.

2.2. Orthodontic procedures

Fixed bimaxillary screw-activated expansion devices were anchored on the dentition. The upper appliances were the RME type for skeletal expansion, and the lower appliances were placed for dental uprighting and tooth expansion. For patients in the permanent dentition group, the upper appliance was attached to the first bicuspids and the first molars, while the lower appliance was attached to the lower first molars (Fig. 2). For patients in the mixed dentition group, if the upper primary second molars were present, then the appliance was attached to these two teeth on the upper jaw (Fig. 3). If the lower first molars had not erupted, then the lower appliance was attached to the lower second primary molars.

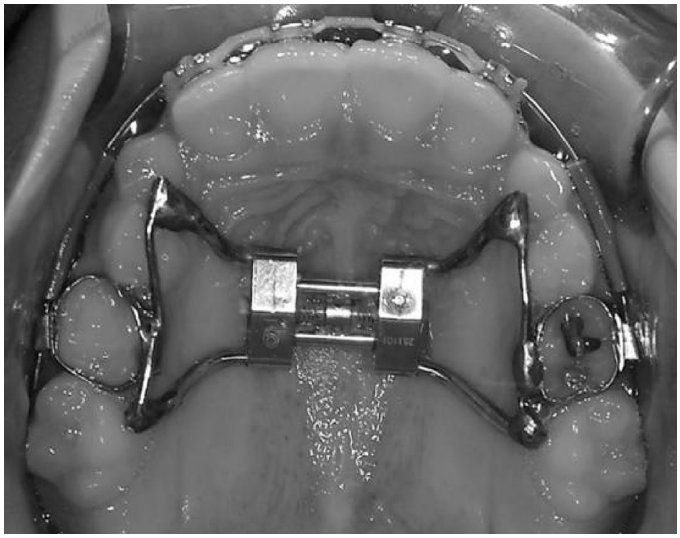

Fig. 3. Design of upper expander in the mixed dentition.

Table 1
Polysomnography Results of All Patients ($n = 45$).

	Baseline (T0)	Post-BE (3 months)	*p*-value
Total Sleep Time (min)	469.40 (204.5–554.0)	456.20 (93.2–583.4)	**0.05**
Sleep Efficiency (%)	89.85 (64.9–97.5)	89.20 (40.9–98.3)	0.35
Sleep-Onset Latency (min)	15.40 (0.0–67.0)	16.60 (1.9–95.6)	0.73
AHI	7.60 (0.1–32.5)	6.67 (0.0–30.8)	**0.05**
SPO_2 Mean Desaturation (%)	97.50 (94.5–99.5)	97.20 (92.3–99.4)	0.32

Data presented as median (min–max). Wilcoxon signed-rank tests were performed.

Parents were instructed to turn/activate both the upper and lower appliance once per day if the patient was <13 years of age. If the patient was older than 13 years, the parents were instructed to turn the upper appliance twice per day, and the lower appliance once daily for a predetermined number of days. The minimum amount of expansion generated was 6 mm and the largest amount of expansion created was 10 mm, as measured at the expansion screw.

2.3. PSG reports

Initial and final full sleep studies (level 1 PSG) rendered anonymous had been performed by certified specialists and were scored using the "recommended" criteria established by the American Academy of Sleep Medicine (AASM) guidelines [26] for sleep and wakefulness and the respiratory variables: apnea, hypopnea, lowest oxygen saturation, and calculation of an apnea–hypopnea index (AHI) based on the "recommended" criteria.

2.4. Cephalometry

Lateral cephalograms were available in 41 of the 45 patients. One of the patients was wheelchair bound (cerebral palsy child) and was unable to fit inside the cephalostat (Planmeca ProMax), while three others had cephalograms from other offices, taken on different machines. These were not included to avoid magnification errors that can result from different machines in different settings. The remaining 41 cephalograms were analyzed using the Bjork [27] and Harvold analyses (1974) [28]. Cephalometric measures included maxilla and mandible positions, occlusal and mandibular plane inclination, incisor position, and hyoid bone position. Dolphin Imaging software™ (Patterson Technology, Chatsworth, CA, USA) was used to digitize and trace the cephalograms.

2.5. Statistics

Data normality was analyzed with Shapiro–Wilk tests. Nonnormally distributed PSG data are presented as median (minimum–maximum). Normally distributed cephalometric data are presented as mean ± standard error of the mean (SEM). Nonparametric Wilcoxon signed-rank test was applied for within-group comparisons (night preexpansion vs. night postexpansion) for all sleep and breathing variables. The one-sample *t*-test was applied for between-group comparisons (measured cephalometry vs. normative values) for all cephalometric measurements.

Z-score was calculated in order to normalize the values in comparison to the normative values, where $Z\text{-score} = (X - \text{Norm})/\text{SD of norm}$. It is estimated that 95% of the population is within the normative range of $Z\text{-score} = 0 \pm 2$. Spearman's correlations were calculated to compare sleep and breathing variables to the cephalometric measurements. For this, raw data were used for the cephalometric measurements (not *Z*-scores), whereas sleep and breathing raw data or AHI difference between preexpansion and postexpansion nights were used.

Statistical analyses were performed using SPSS (version 17, SPSS Inc., Chicago, IL, USA). *P*-values were considered significant at ≤0.05.

3. Results

3.1. Sleep and breathing variables

PSG variables are presented in Tables 1 and 2. Overall, total sleep time and AHI significantly decreased after BE (Wilcoxon, $p = 0.05$; see Table 1). Among all 45 patients, the AHI decreased in the majority of patients (30/45) following BE, though as a group it was a small but statistically significant improvement. The AHI increased in a third (15/45) of the children.

3.2. Cephalometry

The analyzed cephalometric data are presented in Table 3. *T*-tests and *Z*-scores showed that, although still within the normative range, skeletal measures showed an increased ramus height (Ar–Go), a shorter mandibular base length (Go–Pg), shorter total face length (N–Gn), and increased anteroposterior relationship between the maxilla and mandible (ANB; $p \leq 0.02$). An increased posterior cranial base length (S–Ar), decreased mandibular plane inclination (MP–SN angle), decreased maxillary plane inclination (SN–PP angle), mandibular retrusion [decreased (Sella-Nasion-B point) angle and decreased SNPg (Sella-Nasion-Pogonion) (*t*-test, $p \leq 0.03$)], and an increased horizontal jaw difference between the maxilla and the mandible (ANPg (*t*-test, $p \leq 0.01$)) were also observed. Dental findings included an increased interincisal angle (U1-L1) ($p < 0.001$), increased overjet, and increased overbite (*t*-test, $p \leq 0.01$).

3.3. AHI severity

The data were stratified according to severity of the pretreatment SDB (see Table 2). According to their AHI severity category, the severe group (AHI > 10) significantly decreased its median (min–max) AHI of 22.0 (10.4–32.5) to 10.3 (3.7–25.3) following treatment (Wilcoxon, $p = 0.001$). Fifteen patients showed improvement and only one patient worsened. In the moderate group (AHI ≥ 5 and ≤ 10) of 17 patients, although there was improvement in the AHI values following BE in 12 patients, this change was not statistically significant. Five patients in this category had an increased AHI after BE. The mild group showed a significant increase in the AHI in nine patients (Wilcoxon, $p = 0.03$), while the AHI decreased only in three patients.

3.4. AHI severity and cephalometric measures

Within the mild group, 83% had bimaxillary retrusion. Among the nine mild patients whose AHI increased after the treatment, 56% had bimaxillary retrusion and 11% had maxillary protrusion (increased SNA). Correlations between AHI and cephalometric measures showed that patients in the mild group with a normal mandibular plane inclination showed more AHI improvement after BE (MP–SN, Spearman's rho –0.584, $p = 0.02$; Figs 4 and 5). The mandibular plane inclination (MP–SN) is used as a measure of mandibular and lower facial growth direction. Patients with a smaller MP–SN, or counterclockwise/anterior mandibular growth (retrognathia), worsened with BE, whereas patients with clockwise mandibular growth showed greater im-

Table 2
Polysomnography results by OSA severity group ($n = 45$).

	Mild AHI < 5 ($n = 12$)			Moderate AHI between 5 and 10 ($n = 17$)			Severe AHI > 10 ($n = 16$)		
	Pre-BE	Post-BE	*p*-value	Pre-BE	Post-BE	*p*-value	Pre-BE	Post-BE	*p*-value
Total Sleep Time (min)	450.5 (204.5–515.5)	448.2 (372.0–519.5)	0.75	468.4 (368.1–554.0)	446.0 (93.2–539.0)	0.06	498.5 (322.5–543.4)	468.5 (224.0–583.4)	0.12
Sleep Efficiency (%)	89.9 (76.5–95.9)	87.8 (71.1–93.8)	0.94	90.0 (72.5–97.4)	88.6 (49.0–98.3)	0.16	89.5 (64.9–97.5)	90.1 (40.9–96.6)	0.90
Sleep-Onset Latency (min)	20.0 (0.0–43.0)	16.3 (1.9–47.5)	0.88	13.2 (0.0–44.6)	18.8 (2.9–49.8)	0.57	13.9 (1.7–67.0)	14.5 (2.2–95.6)	0.93
AHI	2.9 (0.1–0.4)	6.1 (0.0–30.8)	**0.03**	7.1 (5.2–10.0)	6.1 (0.0–20.2)	0.25	22.0 (10.4–32.5)	10.3 (3.7–25.3)	**0.001**
SPO_2 Mean Desaturation (%)	97.6 (96.2–99.5)	97.7 (96.2–99.4)	0.80	97.8 (94.9–99.4)	97.2 (96.0–98.0)	0.17	97.2 (94.5–98.9)	97.2 (92.3–99.3)	0.38

Data presented as median (min–max). Wilcoxon signed-rank tests were performed.

Table 3
Overall cephalometric values ($n = 41$).

Value	Mean ± SD	Norm	*p*-value
Saddle Angle SN–Ar (degrees)	122.29 ± 0.91	124	
Gonial/Jaw Angle Ar–Go–Me (degrees)	127.10 ± 1.11	129.7	**≤0.02**
Anterior Cranial Base SN (mm)	66.66 ± 1.17	68.8	
Posterior Cranial Base S–Ar (mm)	29.44 ± 0.60	28.5	**≤0.03**
Ramus Height Ar–Go (mm)	40.17 ± 0.95	33.9	
Mand Base Length Go–Pg (mm)	58.39 ± 1.10	73	**≤0.02**
Upper Face height N–ANS (mm)	45.32 ± 0.84	50	
Lower Face Height ANS–Gn (mm)	58.88 ± 0.96	65	
Total Face height N–Gn (mm)	103.68 ± 1.66	115	**≤0.02**
Mandibular Plane Inclination MP–SN (degrees)	31.93 ± 0.87	36.3	**≤0.03**
CranioMxBase/SN–PP (degrees)	8.22 ± 0.62	7.3	**≤0.03**
PP–OP (degrees)	8.88 ± 0.63	10	
Mand Plan to Occ Plane (degrees)	13.88 ± 0.58	13.5	
PP–MP (degrees)	23.34 ± 0.77	25	
SNA (degrees)	80.98 ± 0.59	82	
SNB (degrees)	76.78 ± 0.60	80.9	**≤0.03**
SNPg (degrees)	77.22 ± 0.62	80	**≤0.03**
ANB (degrees)	3.63 ± 0.36	1.6	**≤0.02**
U1-PP (degrees)	106.73 ± 1.51	110	
SN–Ba (degrees)	127.61 ± 0.88	131	
Overjet (mm)	4.00 ± 0.29	2.5	**≤0.01**
U1-L1 (degrees)	139.44 ± 1.82	130	**<0.001**
ANPg (degrees)	3.29 ± 0.36	2	**≤0.01**
Overbite (mm)	3.66 ± 0.32	2.5	**≤0.01**
Midface Length Co–A (mm)	78.05 ± 1.25	86.5	
Mx Length Co–ANS (mm)	80.55 ± 1.19	90	
Md Unit Co–Pog (mm)	96.30 ± 1.52	113	
Harvold CoPog–CoANS (mm)	15.27 ± 0.79	20	

provement. However, in the severe group, those with an initially shorter mandibular base length (Go–Pg) showed lesser AHI improvements (Spearman's rho –0.489, trend $p = 0.06$; Figs 6 and 7).

4. Discussion

The study results suggest that in a pediatric population BE improves the upper airway collapsibility (measured by AHI) and improves sleep by increasing the total sleep time. The expansion

Fig. 4. AHI change (*y*-axis) and mandibular plane (MP–SN, *x*-axis) of individual patients according to the AHI severity groups.

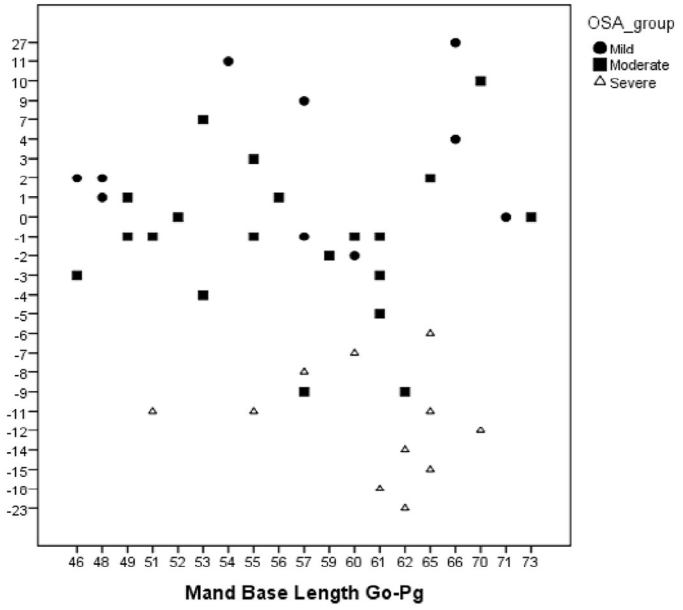

Fig. 5. AHI change (y-axis) and mandibular base length Go–Pg (x-axis) of individual patients according to AHI severity groups.

had no appreciable effect on sleep-state distribution, as previously described in prior expansion work [29]. AHI was most improved in patients with a mandibular length within the normative values for age and sex. BE, in the transverse spatial plane, was not enough to improve AHI in those with a shorter mandibular length (shortened anteroposterior spatial plane).

Original work by Harvold and Vargervik demonstrated that the archform and teeth positions are adaptive responses to both an altered breathing state and changes in the tonicity of the orofacial musculature [4–6]. BE was used because of the dental compensations in both the maxilla and mandible that result from a narrowed maxilla. The dentition tips inward toward the tongue, which creates a narrowed intraoral space. Aside from the reported enlargement of the nasal cavity with RME, BE increases the intraoral volume, dental arch circumference, and dental arch width. Lower dentition posterior dental uprighting by tipping the teeth outward allowed more transverse upper jaw skeletal expansion across the midpalatal suture. This was a necessity to maintain the dental occlusion.

From this work, BE should be considered as a therapeutic option for pediatric OSA treatment. RME has already been shown to effectively treat this disorder in some children. However, also treating the mandibular narrowing in conjunction with the maxillary narrowing can potentially yield better results in that patients other than those with crossbite malocclusions or dental crowding are also benefitted. This suggests that the narrowing of both archforms should be treated, and that the maxillomandibular lingual inward tooth inclination should be evaluated.

As shown in the PSG data, not all patients will respond favorably to BE. Most patients reported subjective improvement in clinical symptoms, which was not necessarily reflected in a decrease in the AHI on the sleep study. Those who had a positive response to therapy did show residual OSA on posttreatment PSGs. OSA was not eliminated in any of the BE patients.

Five of the children in this study had the following neurologic deficits: cerebral palsy (one), Tourette's syndrome (one), autism (two), and fetal alcohol syndrome (one). Most of these syndromes do not directly affect the oral facial muscle tone during wakefulness. Five patients had a positive response to BE and a decreased number of arousals and an improvement in clinical symptoms.

Similar to the many other reports of pediatric OSA treatment, multifactorial, multidisciplinary, and multiple treatments are necessary to fully address the disorder in children.

4.1. Facial morphology/cephalometry

None of the children in this study were preselected based on any type of facial morphology or cephalometric measure, and BE was used for all craniofacial types. Certain cephalometric markers to as-

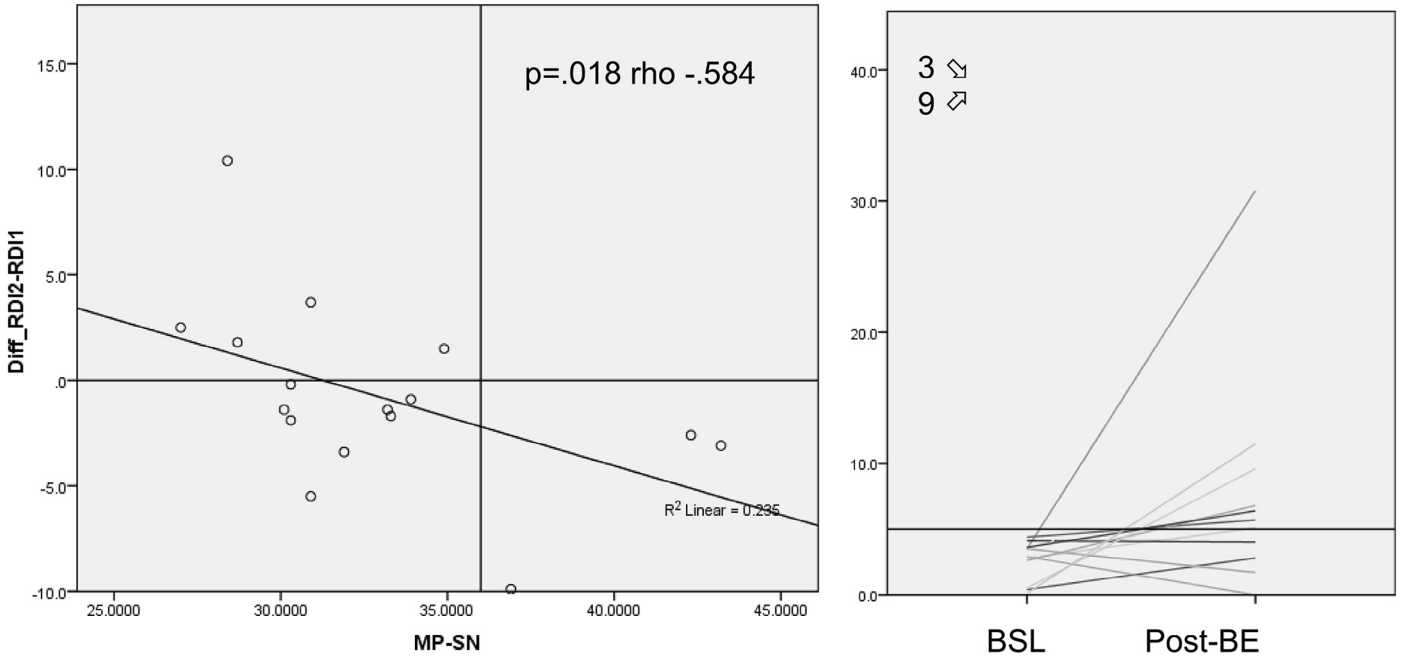

Fig. 6. Correlation between the AHI difference between prebimaxillary expansion and postbimaxillary expansion nights, and mandibular plane inclination (MP–SN) in the mild OSA group.

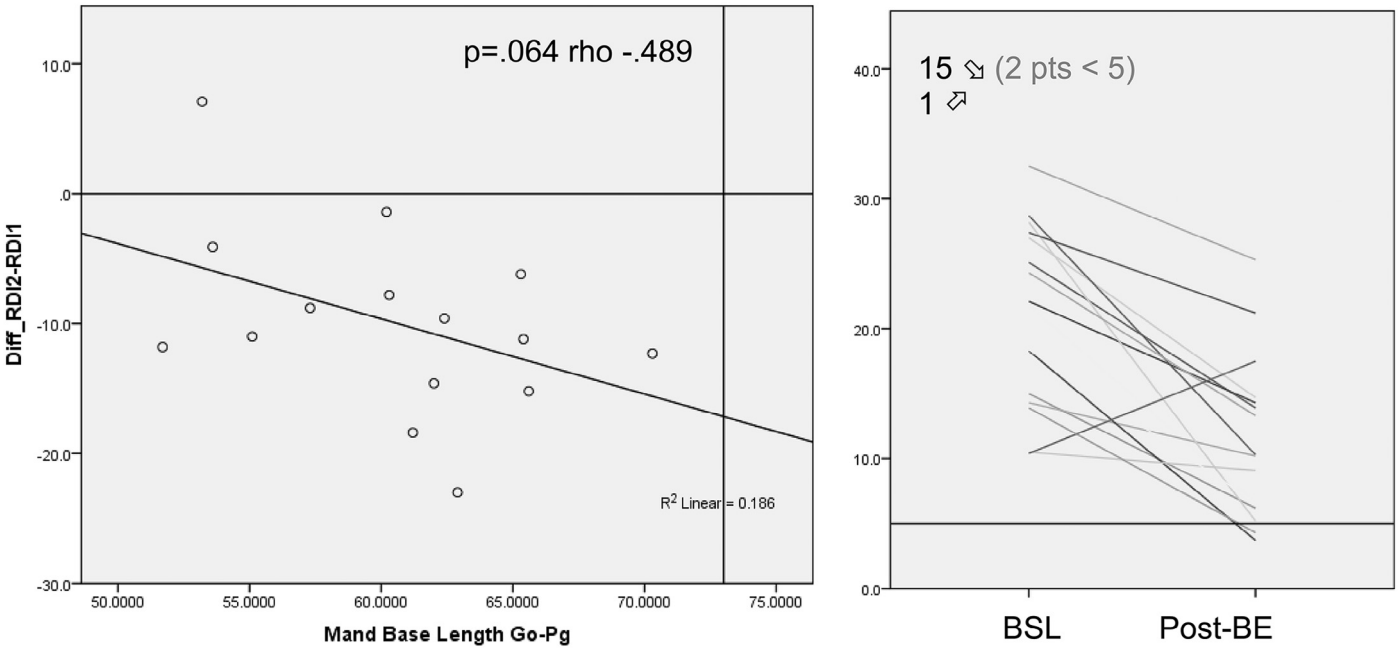

Fig. 7. Correlation between the AHI difference between prebimaxillary expansion and postbimaxillary expansion nights, and mandibular base length (Go–Pg) in the severe OSA group.

sociate facial type with at-risk populations for OSAS have been described [7,29–32] as mandibular retrognathia, narrow maxilla, high mandibular plane angle, and increased lower facial height. By contrast, our study demonstrated OSAS in patients with decreased mandibular plane inclination and decreased total facial height (decreased lower facial height and decreased upper facial height), suggesting that a different morphotype at risk of pediatric OSA may be identified.

Because of the wide cross section of patients included here, it is not surprising that the data show that there was not a specific cephalometric marker among the sample size at entry. Similar to other work, our study corroborates an increased ANB and a shortened mandibular plane length in pediatric SDB populations [30–32].

4.2. Limitations

Despite data collection at only one orthodontic office with BE treatment and cephalometric measurements performed by the same operator, there was no control or observation group for comparison; hence, these results should be interpreted with caution. The number of children was limited in part due to the strict criteria of inclusion and exclusion. The wide cross section of patients compounded with the small sample size did not allow for specific cephalometric markers. Although our data showed improvements throughout the age ranges, the small sample size did not support stratification by age to demonstrate the importance of early interventions in young children.

5. Conclusions

BE reduced the number of arousals and improved collapsibility (as measured by AHI) and increased the total sleep time in 66% ($n = 30$) of our pediatric SDB population. The effectiveness was shown to be dependent on the severity of the disease, with the more severe group showing improved outcomes with BE. As shown here, not all children will benefit from BE in the treatment of OSA. There were no significant associations with any cephalometric reference to ascribe a facial morphology that would benefit from BE therapy, but rather BE may not be an effective therapy for children with an anterior jaw growth pattern. OSA can present in children with an anterior jaw growth pattern with mandibular retrusion and a decreased facial height. Screening for airway problems should be performed at an early age with collaborations of pediatric dentists and orthodontists to improve the diagnosis and treatment of pediatric OSA.

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Conflict of interest

The authors had no conflicts of interest to declare in relation to this article.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2016.03.011.

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