Differences of Craniofacial Characteristics in Oral Breathing and Pediatric Obstructive Sleep Apnea

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Background: Oral breathing (OB) was considered associated with specific craniofacial structures and same for pediatric obstructive sleep apnea (OSA). This study aimed to investigate the differences of craniofacial structures between OB and OSA.

Methods: In this retrospective study, 317 children under age 18 years were recruited and divided into OB group, OSA group, and control group. OSA group (15 boys, 4 girls) were referred from qualified sleep center and diagnosed as pediatric OSA with full-night polysomnography. OB group (10 boys, 10 girls) were mostly referral from pediatric or ENT department, some of whom undertook polysomnography and were not OSA. Control group consisted of orthodontic patients within the same period. Lateral cephalograms were obtained in all groups and their parameters were compared with Chinese normal values and each other.

Results: R-PNS of OB group (18.04 ± 2.49 mm) was greater than OSA group (14.27 ± 4.36 mm) and even control group (16.22 ± 3.91 mm) ($P < 0.01$). U1-NA was also the greatest in OB group (7.15 ± 2.92 mm), followed by OSA group (4.88 ± 2.66 mm), while control group was the smallest (5.71 ± 2.94 mm) ($P < 0.05$). In addition, OB group presented the smallest adenoids and tonsils among three groups. Bony nasopharynx development, mandibular length and growth direction of mandible of OB group were all better than OSA group.

Conclusion: Despite of oral breathing, anatomical morphology (well-developed dentoalveolar structures; mild adenotonsillar hypertrophy) might protect children from developing OSA.

Key Words: Adenoid hypertrophy, cephalometric, craniofacial structure, oral breathing, pediatric sleep apnea

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Oral breathing (OB) and obstructive sleep apnea (OSA) are common sleep disordered breathing (SDB) in children. Both of them have similar pathological causes as adenotonsillar hypertrophy resulting in similar dento-maxillofacial deformities such as mandibular retrognathia. However, few studies comparing oral breathing children and pediatric OSA have been carried out and findings of craniofacial deformities caused by oral breathing and OSA were inconsistent. A meta-analysis[1] focusing on lateral cephalograms of children (0–18 years of age) with OSA and primary snoring elucidated an increased ANB angle and decreased sagittal parameters of upper airway than controls. Nevertheless, an increased ANB angle of less than 2 degrees could merely be marginal clinical significance. Other dentofacial deformities associated with SDB were increased anterior facial height, protrusive upper lip, increased mandibular plane angle, and high-arched palate.[2–4] And oral breathing was reported to affect upper and lower teeth, maxilla and mandible, as well as facial profile.[5–13] Thus, are these inconsistent findings due to the sample differences among regions, races and age of studies or differences of craniofacial characteristics in oral breathing children and pediatric OSA?

On the other hand, it is controversial that craniofacial deformity may have an impact on children's growth and development. In clinical practice, pediatric OSA is often associated with multiple craniofacial syndromes and the modified craniofacial morphology occurred before symptoms like snoring.[14,15] However, Arens et al did not find a primary skeletal difference between OSA and control group in maxilla and mandible width, length or volume.[16] Some scholars thought that referral bias could explain some of the differences in data between sleep and dental clinics, that patients with retrusive mandible tent to be referred to dental clinic.[3] Considering this point, comparing to general population, there are more children with mandibular retrognathia in department of orthodontics, especially in Asia. From nonsyndrome children seeking for orthodontic treatment, we aimed to investigate that though with mandible retrusion, why some children persisted oral breathing and even develop to OSA, while others had normal breathing pattern.

This study intended to conduct a retrospective cross-sectional study to compare differences of craniofacial characteristics in oral breathing children and pediatric OSA. The null hypothesis was that there were no differences between 2 groups.

MATERIALS AND METHODS

Subjects

This study was approved by committee of PKUSSIRB (No. 201947093). Since it is a retrospective study, it would be impractical to obtain informed consent. But lateral cephalograms are routinely taken in orthodontic treatment and individuals and their legal guardians were informed the scientific use of their x-ray.

Subjects were selected from patients who visited Dr. Gao and Dr. Gong in Department of Orthodontics, Peking University School and Hospital of Stomatology from June 2016 to June 2018. Patients met the diagnostic criteria of pediatric OSA according to the AASM

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scoring manual were classified as OSA group. Patients with history and symptoms of oral breathing but did not meet the OSA diagnostic criteria were classified as OB group. And the control group came from orthodontic patients within the same period.

A total of 317 subjects (133 boys, 184 girls), with age range from 5 to 16 years, were included in the study. The general information of three groups is shown in Supplementary Digital Content, Table 1, http://links.lww.com/SCS/B760 and the specific classification criteria for three groups were as follows:

OSA group: Referral patients diagnosed with pediatric OSA after full-night polysomnography (PSG) at 2 qualified children's sleep laboratories in Beijing. There were 19 patients in total (15 boys, 4 girls), with average age of 8.84 ± 2.57 years, BMI of $17.19 \pm 4.55\,kg/m^2$ and apnea hypopnea index (AHI) of 6.48 ± 2.37 events/h.

OB group: Patients with obvious oral breathing habits and referred for myofunctional therapy, some of them were confirmed by PSG that were not OSA. There were 20 patients in total (10 boys, 10 girls), with average age of 13.15 ± 2.64 years, BMI of $18.40 \pm 3.05\,kg/m^2$.

Nonsnoring control group: The following patients were excluded-older than 18 years; with chief complaints of congenital syndrome, cleft lip and palate, orthognathic surgery; and persisting habits of oral breathing. There were 278 patients in total (108 boys, 170 girls), with average age of 12.17 ± 2.55 years, BMI of $18.32 \pm 3.50\,kg/m^2$.

Polysomnography

Single-night polysomnography was performed in the sleep center at 2 qualified children's sleep laboratories in Beijing. Each record was scored by a certified sleep disorder technician and verified by a researcher. The evaluation consisted of a number of parameters including EEG, EOG, EMG, ECG, recordings of nasal and oral airflow, snoring index, sleep position, etc. AHI was used to assess the severity of sleep apnea. According to the AASM diagnostic and coding manual, children with AHI over 5 events/h or obstructive apnea index over 1 event/h were diagnosed as pediatric OSA.

Diagnosis of Oral Breathing

It was observed in clinic that the child could not breathe with lips closed, and nasal flaring or actions of breaking free with doctor's help to close lips. A change in posture was often present and parents always noticed snoring or heavy breathing of their children. Some children had visited ENT clinic because of adenotonsillar hypertrophy. Three children were recommended to take PSG at qualified sleep center and their AHI did not meet diagnostic criteria of OSA.

Cephalometric Analysis

Cephalograms were routinely taken by OC-100 (Instrumentarium Imaging Company, Finland) at clinic. With the use of orbital pointers and mechanical earplugs, patients' Frankfort planes were adjusted parallel to the horizontal plane. Patients were asked to bite at intercuspal occlusion, and breathe smoothly without swallowing. If images of the upper airway and surrounding tissues were blurred as a result of swallowing or other movements, they would be excluded from the study.

One researcher rearranged the lateral cephalograms to cover patients' identity and shuffled the sequence. All measurements were done by the other researcher and data were recorded by another researcher.

Measurements of craniofacial structures, the upper airway and adenotonsillar size were carried out on self-developed system. After

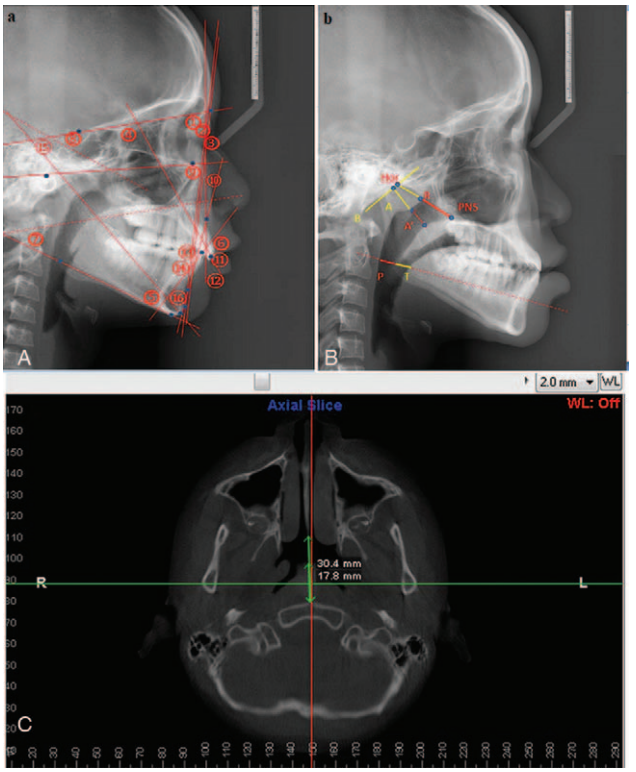

FIGURE 1. A. Cephalometric illustration. ① SNA; ② SNB; ③ ANB; ④ U1-SN; ⑤ L1-MP; ⑥ U1-L1; ⑦ SN-MP; ⑧ MP-FH; ⑨ FH/NP; ⑩ NA/PA; ⑪ U1-NA; ⑫ U1/NA; ⑬ L1-NB; ⑭ L1/NB; ⑮ *Y*-axis; ⑯ Pg-NB. B. Upper airway, adenotonsillar measurements. B line: The tangent of extracranial occipital slope; A line: Perpendicular distance from the most protrusive point of adenoid to B line; A′ line: Width of the airway space along the A line; Hor-PNS: Distance between Hor point (the point locatedat the intersection between the greater wing and the body of the sphenoid bone) and PNS (posterior nasal spine); R-PNS: Distance between R point (the point located at the intersection between posterior pharyngeal wall and PNS-Horline) and PNS; T line: Width of tonsillar along the Go-B line (Go is most posterior inferior point on angle of mandible and B is most concave point on mandibular symphysis); P line: Width of the airway space along the Go-B line. C. Verification of adenoid measurement on 3D images.

landmark identification, data were generated by the system and calculated automatically (Supplementary Digital Content, Table 2, http://links.lww.com/SCS/B760, Fig. 1A). The parameters of craniofacial structures were compared with published normal values of Chinese in mixed or permanent dentition.

Measurements of upper airway and adenotonsillar size are summarized in Supplementary Digital Content, Table 2, http://links.lww.com/SCS/B760, Figure 1B.

Computed Tomography Verification

This study used cephalometrics to measure adenotonsillar size and the morphology of dentoalveolar and craniofacial structures. However, whether a three-dimensional anatomy could be correctly observed in a two-dimensional assessment, we chose a few patients to verify its feasibility. Thirty children undertook computed tomography assessment due to impacted tooth or temporomandibular joint disease and we found out that adenoids were mainly distributed in the nasopharyngeal fornix, which meant the diameter on cephalograms could reflect its three-dimensional size (Fig. 1C).

Intra-Class Correlation

Fifty cephalograms were selected 2 weeks later and measured for the second time to test intra-reliability. Intra-class correlation (ICC) of dentoalveolar, upper airway, and adenotonsillar parameters were between 0.88 and 0.94.

Statistical Analysis

The statistical analysis was performed on SPSS 22.0 (IBM Corp, Armonk, NY). Nonparametric Chi-square test was used to analyze number of subjects and gender. Normal distribution of continuous variables was verified by Shapiro-Wilk test and normally distributed parameters were summarized as means and standard deviations. If variances were homogeneous, one-way ANOVA was applied to compare inter-group differences and post hoc analysis of Bonferroni was used for multiple comparisons. If not, Kruskal–Wallis test and nonparametric Kolmogorov–Smirnov test were used for comparison. Statistical significance was considered when $P < 0.05$.

RESULTS

The General Information of OB Group, OSA Group, and Control Group

As Supplementary Digital Content, Table 1, http://links.lww.com/SCS/B760 shows, children in OSA group were younger, therefore their height and weight were smaller than the other 2 groups. But there was no significant difference in BMI among 3 groups. There was no statistical difference between OB and control groups.

Craniofacial Characteristics of OB Group and OSA Group

As Supplementary Digital Content, Table 3, http://links.lww.com/SCS/B760 shows, OSA group presented a very classical characteristic, which were decreased Hor-PNS, SNB, and FH-NP and increased ANB, NA/PA, MP/SN, and MP/FH.

On the other hand, OB group had quite similar parameters with control group, except that R-PNS and U1-NA not only did not decrease but became larger. Many indicators of OB group were contrary to OSA group, such as Hor-PNS, R-PNS, NA/PA, and U1-NA.

Comparisons of Adenotonsillar Size and Upper Airway Dimension

OSA group presented obvious tonsillar and adenotonsillar hypertrophy, while both anatomies were smaller in OB group. There was also a certain degree of adenotonsillar enlargement in control group. A' was also opposite in OSA and OB group (Supplementary Digital Content, Table 3, http://links.lww.com/SCS/B760).

DISCUSSION

The Effect of Adenotonsillar Hypertrophy on the Development of Pharyngeal Cavity in Children

Adenoid and tonsillar could be observed in most children but not all of whom were associated with craniofacial deformities. Hypertrophy of the adenoids and tonsillars was currently considered main contribution to SDB.[17–20] However, as a component of pharyngeal lymphoid ring, adenoids and tonsils have an effect on children's immunity and grow rapidly at certain age. Their growth pattern is consistent with other lymphoid tissue and they could be found largest relative to the surrounding anatomy in late childhood and graduate atrophy in the later years.[21] It is overgrowth or delayed involution that causes hypoventilation in the upper airway.

In our observation, there was no significant difference of adenoid among three groups, the main discrepancy was tonsillar which should be due to age difference. A cephalometric study of 300 subjects (50% males, 50% females; age range 6–20 years) in Japan suggested that adenoid atrophy started at lower grade of primary school (8.1 ± 0.7 year of age) until adulthood; while tonsillar atrophy began from higher grade of primary school (10.3 ± 0.8 year of age) and junior high school (13.6 ± 0.9 year of age).

At present, most study evaluated adenotonsillar hypertrophy subjectively,[22] but some researchers suggested only objective evaluation was predictive of OSA severity.[23] In addition, researches of adenoids and tonsillars were mostly investigated separately and there were few studies focusing on both anatomies on the same individual and even rarer reporting remaining upper airway size.[18]

Craniofacial Characteristics of Oral Breathing and Pediatric OSA

In our study, patients of OSA group were younger than the other 2 groups and children in the latter groups were undergoing puberty. Nevertheless, it was obvious to observe mandibular retrognathia (smaller SNB and FH-NP, greater ANB and NA/PA) in OSA group, which was partially derived from skeletal pattern in mixed dentition, but the overdevelop in vertical dimension (increased MP/SN and MP/FH) of OSA group indicated the impact of abnormal breathing on craniofacial development. Rossi et al carried out an observational case-control study and found that, in contrast to 19 to 57 years of age, only in 5 to 12 years and 13 to 18 years could an association of oral breathing and retruded mandible be observed, which seemed to be more severe until adolescence.[5] It was considered that some children tried to stretch their neck to relieve the pressure within upper airway.[24,25] Such posture could affect muscular balance resulting in abnormal maxillofacial development, especially during puberty.[26–30]

Previous to our study, out hypothesis was that differences of 3 groups might be gradient, with OB group in the middle of the other 2 groups. However, in this study, OB group and OSA group presented the opposite results. Compared to control group, OSA group had smaller bony nasopharynx (Hor-PNS and R-PNS) and upper incisor inclination (U1-NA), while OB group had larger structures. Adenotonsillar (T line, A+T line) were larger in OSA group and smaller in OB group, leading to the narrowest nasopharynx (A') in OSA group, followed by control group, and then OB group. There appeared to be an anatomical protective factor in the OB group to prevent its progression to OSA, which might derive from family inheritance or growth and development stages. It was considered in extensive literature that there was an association between oral breathing and mandibular retrognathia (Angle class II convex facial profile) especially in 8 to 10 years.[5–13,31–36] During puberty of 10 to 12 years, growth of skeletal structures helped to relieve respiratory disorders, especially in nonobese children, though fat deposition during adolescence could make some children gain weight.[37]

Many scholars have realized that craniofacial morphology have conversely formed protection of sleep breathing. Some patients with enlarged adenoid and tonsillar did not suffer from SDB, however, for some children after adenotonsillectomy, postoperative conditions worsened rather than improved, suggesting that abnormal craniofacial structure could be one of the causes of SDB.[38–40] By the analysis of multiple linear regression, it was suggested that as children grow, adenotonsillar size contributed less to SDB and skeletal morphology became more pronounced in puberty.[41] Due to genetically-determined facial skeleton morphology in east Asia,

study of craniofacial deformity and SDB has additional significance.[42,43]

Strengths and Limitations

Not all children in OB and control group fulfilled PSG. Polysomnography, particularly full-night PSG performed in sleep laboratories was difficult to carry out in pediatric population.[44] Therefore plenty of studies selected symptoms, signs or questionnaires instead of PSG. Still, PSG is the gold standard for diagnosis of SDB and can accurately define the attributes of patients. We made our effort to consult the judgment with qualified sleep center.

The control group came from orthodontic patients, the advantage of which was that the deformity of the background population could offset the referral bias to a certain extent, while the limitation was that congenital skeletal deformities might reduce the positive rate of the study.

Patients of OSA group was younger than the other 2 groups. They have not reached puberty and have not been affected by rapid skeletal growth, which may further highlight the differences between oral breathing and OSA but may not affect the hypothesis of craniofacial protection for sleep breathing. It would be more convincing to continue observation of OSA group till adolescence to test above mentioned hypothesis but it would be against patients' beneficence.

Lastly, as with most pediatric studies, a larger sample size is needed.

CONCLUSIONS

With the assessment of lateral cephalograms of 317 oral breathing, pediatric OSA and control subjects, OSA patients were found present with significantly retrusive and vertically overdeveloped mandible. While craniofacial structures of oral breathing patients were not transition from OSA to the control. They had somewhat smaller adenoids and tonsillars and broader bony nasopharynx, which seemed to be a morphological protection factor.

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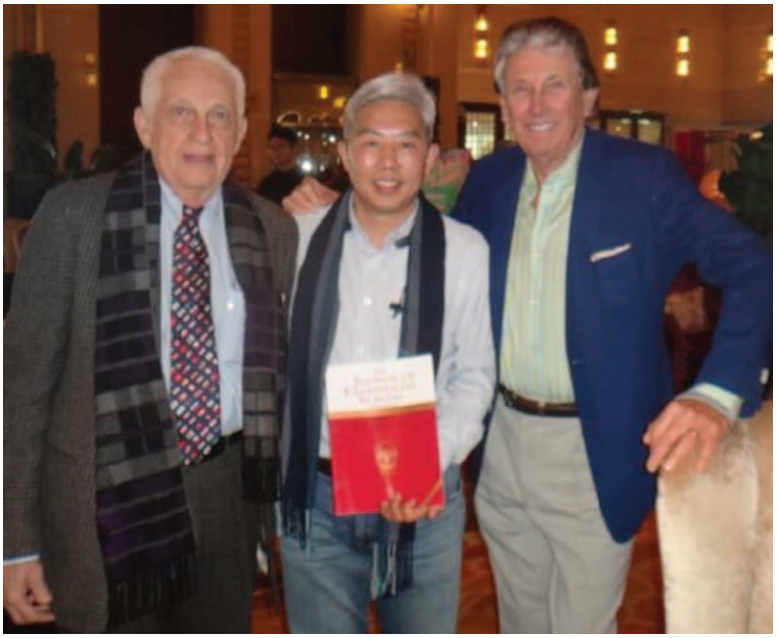

Dr Salyer with the editor and the president of the international society Dr Mu.