Malocclusion in Pediatric Obstructive Sleep Apnea Refractory to Adenotonsillectomy Referred for Sleep Study

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Abstract

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Introduction: The sequelae of untreated pediatric Obstructive Sleep Apnea (OSA) are serious, however, its treatment is not always evident. The first line treatment of pediatric OSA is adenotonsillectomy, but this is not successful in all. It is plausible that underlying dentofacial characteristics are associated with those who have refractory OSA despite adenotonsillectomy. Should certain dentofacial findings be prevalent in those with refractory OSA, this may represent a point of early identification and intervention for OSA.

Objective: The aims of this study were to describe dentofacial characteristics (overjet, overbite, posterior crossbite, facial convexity) in children referred to a tertiary pediatric sleep center for OSA. Amongst those subjects, comparisons were made between groups based on surgical status (naïve vs adenotonsillectomy), severity of OSA (none, mild, moderate, severe), and with general population control data available from the third National Health and Nutrition Examination Survey (NHANES III).

Methods: Subjects were recruited from Seattle Children's Hospital Sleep Center at Overlake. Subject profile photo, intraoral examination, and dental history questionnaire were recorded. Intraoral examination consisted of Angle classification, overjet, overbite, and posterior crossbite presence. The obstructive Apnea-Hypopnea Index (oAHI) was obtained from the sleep study results. Univariate analysis was used to evaluate how oAHI severity varies with overjet, overbite, posterior crossbite, and facial convexity based on exposure to prior adenotonsillectomy.

Results: 95 subjects were enrolled in this study. 26 subjects had prior adenotosillectomy surgery and 69 subjects were surgically naïve. 21 subjects were diagnosed with no OSA, 54 subjects diagnosed with mild OSA, and 20 subjects were diagnosed with moderate to severe OSA. No statistically significant differences in dentofacial characteristics were found between the subjects who had prior adenotonsillectomy compared to the surgically naïve group. Facial convexity of the Moderate to Severe OSA group showed a statistically significant difference (P=0.02) when compared to the No OSA group however this may be caused by the confounding variable of obesity. All other dentofacial characteristics

exhibited no statistically significant difference between the OSA severity groups. Comparison with the NHANES III population data showed a significant increase in posterior crossbite in 8-11 years old patients with Mild and Moderate to Severe OSA.

Conclusion: There were no significant differences in the dentofacial characteristics between the children who had prior adonetosillectomy and those who were surgically naïve. There were some dentofacial differences between the children in different OSA severity groups (facial convexity and posterior crossbite), however due to the small sample size and confounding variables these findings may be spurious.

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Introduction

Background

Obstructive Sleep Apnea (OSA) is defined by a recurrent occlusion of the upper airway during sleep. This dynamic imbalance between airway patency and collapse during sleep leads to recurrent airway blockage (partial or complete) resulting in gas exchange abnormalities and/or cortical arousals, which has been linked to numerous negative health effects. OSA is one of the conditions that fall under the umbrella term Sleep Disordered Breathing (SDB). Other conditions that are included in SDB are Primary snoring, Sleep-related hypoxemia, and Central sleep apnea syndromes.

The science of obstructive sleep apnea (OSA) was first described when Gastaut et al. coined the term in 1966 in France.[1] Initially, this condition was thought to be associated with obesity, as it was known as the "Pickwickian Syndrome" derived from Charles Dicken's book, *The Pickwick Papers*.[2] With the discovery by Gastaut, sleep research shifted its focus from treating the patient's obesity to understanding the patient's compromised airway.

In 1970, Stanford University established its first sleep research center led by Dr. William C. Dement. In the following years, Stanford's Sleep Center acquired prominent sleep researchers such as Drs. Mary Carskadon, Christian Guilleminault and Vincent Zarcone. The Sleep Center had the ability to conduct full polysomnography and multiple sleep tests under the guidance of sleep specialists.[3] In the next five years, multiple sleep centers were established including Montefiore Medical Center (New York), Ohio State

University, Baylor University, University of Cincinnati, and University of Pittsburgh that all contributing to the progress in OSA research.

In 1993, Young et al. published the first major epidemiologic study in the US that showed OSA affecting more people than previously estimated. They identified the prevalence of OSA to be far more common with 2% and 4% of women and men being affected, respectively.[4] Young concluded that obesity was a modifiable risk factor, and there might be other risk factors contributing to OSA. The turning point in OSA research came in 2000, when four separate articles uncovered the health effects related to OSA.[5] These articles made association between hypertension and OSA in well-developed researches. These articles helped shed light on the risk of OSA, and since then, OSA and sleep disordered breathing research have dramatically increased.

Risks and Significance

In the pediatric population, the prevalence of OSA is 1% - 4%.[6] The sequelae of untreated OSA are associated with serious physical and psychosocial consequences on the child's development. These morbidities include measurable impairments to growth, cardiovascular and metabolic health, behavior, cognition, academic performance, mood, and urinary continence.[7,8,9,10,11,12] Risk factors for pediatric OSA include adenotonsillar hypertrophy, anatomic causes of narrowed upper airway, craniofacial malformations, obesity, and genetic syndromes particularly those leading to hypotonia.[13,14,15,16,17,18]

Pediatric patients with untreated OSA have serious risk of developing significant systemic health problems; yet, the pathophysiology of this disease remains uncertain.

OSA in children may result from complex factors including inflammation of the lymphoid tissues as well as altered neuro-musculature in the child's airway. One of the risk factors that is interesting for orthodontists is the altered craniofacial development, as this topic applies directly to orthodontists.

Children with OSA frequently exhibit chronic mouth breathing, which in turn is thought to influence changes in craniofacial growth.[19] Numerous research studies support this theory, as mouth-breathers often exhibit changes in facial development by increased facial height, decreased maxillary width, and retrognathic mandible. [20,21,22,23,24,25] Furthermore, commonly reported craniofacial characteristics in children with OSA are narrow maxilla, long faces, and retrognathic mandible.[35,26] In 2013, Flores-Mir et al. conducted a systematic review and meta-analysis assessing the craniofacial morphological characteristics associated with non-syndromic pediatric patients with diagnosed OSA.[27] Flores-Mir et al. concluded that pediatric patients with OSA had steeper mandibular plane angle, retrusive chin, vertical grown pattern, and tendency for class II malocclusion, and when these craniofacial morphologies are identified as well as suspicion of breathing disorder, dentists should refer the child to an otolaryngologist.[27] Katyal et al. in 2013 also performed a systematic review and meta-analysis to evaluate the association between craniofacial disharmony and pediatric sleep-disordered breathing, and found that children with OSA showed a weighted mean difference in ANB angle of 1.64° when compared with the controls.[28] However, an ANB angle increase of less than 2° in children with OSA could be considered as having marginal clinical significance, and they conclude that, "evidence for a direct causal relationship between craniofacial structure and

pediatric sleep-disordered breathing is unsupported by this meta-analysis."[28] Both Flores-Mir et al. and Katyal et al. conclude that further research is pertinent to understand the factors that are important contributors to the etiology and pathophysiology of pediatric OSA.

Diagnosis

The practice guideline for 2017 published by the American Academy of Sleep Medicine (AASM) recommends the use of polysomnogram (PSG), also known as a sleep study, to diagnose OSA.[29] PSG is an overnight monitoring system which uses electric channels to evaluate the patient's physiological functions. These functions include the recording of patient's brain activities, body movements, respiratory efforts, oxygen saturation, and cardiac variables (ECG), all while under the close supervision of the sleep technician. Further detailed settings for the channel can be found in the 2007 AASM manual.[79] From the PSG, Apnea/hypopnea index (AHI) is defined by the total number of apneas and hypopneas averaged per hour of sleep. While apneas are defined as 10 seconds in adults, the pediatric criteria are much shorter. Obstructive apneas and hypopneas are scored if the child experiences duration of at least 2 breaths, even if they are less than 10 seconds in duration.[30] Children have lower functional residual capacity because of their faster respiratory rate; hence, OSA in children may lead to faster oxygen desaturation. Consequently, children are more prone to suffer physiologic consequences from brief apneic episodes.[35] Pediatric apnea is defined when peak signal excursions drop by ≥ 90% of pre-event baseline and the episodes meet the duration and respiratory effort criteria. [35]

Pediatric hypopnea is defined when peak signal excursions drop by ≥ 30% of pre-event baseline for the duration of 2 breaths in association with either ≥ 3% oxygen desaturation or an arousal. [35]

Treatment Options

According to the clinical practice guidelines from the American Academy of Pediatrics (AAP), adenotonsillectomy (T&A) is considered the first-line therapy for children with OSA; thus, it is the most common treatment rendered in the US.[31] Current literature shows resolution occurring in about 50% to 75% of the children when the underlying cause is adenotonsillar hypertrophy. [32,33,34,35] Residual OSA in remains high in these children and additional treatment strategies are needed to manage these patients.

For those children who have OSA after T&A procedure, OSA management is complicated and it is often accomplished by several treatment approaches. Most often, a continuous positive airway pressure (CPAP) therapy is prescribed indefinitely. Yet, it is associated with its own set of risks and complications. Roberts et al. in 2016 reported that, "Pressure to the midface from compliant nasal CPAP use may alter normal facial growth." That is, pressure exerted by the CPAP on the midface acts as a restrictive mechanism during the time of rapid facial growth causing altered growth.[36] Additionally, compliance is crucial for OSA treatment success, but worsens risk of midface retrusion. Generally, older children are more tolerant of CPAP.[37],[38] Younger children and those with behavioral problems may have difficulty adhering to the treatment.[39] Other treatment ranges from weight loss strategies to surgical approaches. Yet, according to AAP, "Most adjunctive

measures in the treatment of childhood OSA have not been prospectively evaluated." Perhaps the etiology of the problem might be the patient's craniofacial characters. It is ineffective and inefficient to treat children with OSA using CPAP if the underlying cause is morphological, such as a constricted maxilla. Furthermore, there hasn't been any research conducted to study the dentofacial characteristics of children with OSA who have undergone T&A surgery.

If OSA is caused by dentofacial problems, it is possible that an orthodontist may be the most appropriate to address these issues as they are the specialist of craniofacial growth and development. Orthodontists routinely administer oral appliances such as rapid palatal expander (RPE) and mandibular repositioners to correct jaw discrepancies. Systematic review and meta-analysis performed by Camacho et al., which included 17 studies, examined the effects of RPE to treat OSA and have reported improvements in post-RPE apnea-hypopnea index (AHI) from 8.9 ± 7.0/hr to 2.7 ± 3.3/hr (70% reduction), but only studied in the short-term.[40] Similarly, Huynh et al. in 2015 conducted a systematic review on the effectiveness of orthodontic treatments, and found orthopedic mandibular advancement and/or RPE may have the potential to manage and improve pediatric obstructive sleep apnea.[41] However, critical limitations were present as total number of studies included were low and the sample sizes were small; thus, Huynh concludes, "Orthodontic treatment of pediatric OSA guidelines cannot be extrapolated and generalized from this systematic review and meta-analysis."

Caution must be taken when recommending orthodontics to treat OSA. The literature between OSA and dentofacial characteristics is complicated and remains

uncertain. Children who snore and are diagnosed with OSA often exhibit mouth breathing. This chronic mouth breathing is usually associated with tonsillar hypertrophy.[42] Furthermore, from this constant mouth breathing habit, postural changes may affect dentofacial growth and increase the risk of developing dolichofacial morphology. [43,44,45,46] The directionality and causality of the relationship between the airway and dentofacial development is still not clear.

In 2011, Huynh et al. studied the OSA symptoms and dentofacial morphology and concluded that OSA symptoms are associated with craniofacial features, which are, "dolichofacial, high mandibular plane angle, narrow palate, and severe crowding in the maxilla and the mandible."[47] In 2017 Pliska et al. investigated the association between OSA and craniofacial characteristics. His team examined dentofacial indications for orthodontic intervention in a cohort of children referred to a tertiary care center.[48] They evaluated cross-sectional data of 90 consecutive children aged 5 to 10 years old and reported no greater prevalence of malocclusion in children referred for sleep study.[48]

To determine if orthodontics is a valid treatment for children with OSA, we must first establish if OSA is related to certain dentofacial characteristics. There is also no literature available that ascertains the severity of OSA with respect to the dentofacial characteristics. Characterizing these dentofacial characteristics which are associated with OSA would help healthcare providers in both medicine and dentistry to routinely assess for those features, ideally leading to earlier identification and treatment for those who are at risk for OSA. Furthermore, if no association is discovered, administrating adjunctive orthodontic treatment for OSA without proper indication must be re-evaluated.

Objectives

The objectives of this study were to characterize the dentofacial characteristics, (Angle classification, overjet, overbite, and facial profile) in children who were referred to a tertiary Sleep center for suspected OSA. It is hypothesized that childhood OSA may be associated with certain dentofacial problems when the underlying cause is not adenotonsillar hypertrophy. To test this hypothesis, comparisons were made between the groups of children referred for a sleep study who had undergone T&A and those who didn't. In addition, dentofacial features of children with different severities of OSA were compared. Finally, the craniofacial characteristics of our study population will be compared to those of the population national norms as reported in the Third National Health and Nutrition Examination Survey (NHANES III).

The specific aims for this project were:

1. To compare the prevalence of overjet, overbite, crossbite, and facial profile in pediatric subjects diagnosed with OSA who had undergone adenotonsillectomy (T&A) to those who are surgically naïve.
2. To characterize four dentofacial features (overjet, overbite, crossbite, and facial convexity) in pediatric subjects amongst the groups diagnosed with no OSA, mild, moderate, and severe OSA.

3. To compare the prevalence of overjet, overbite, and posterior crossbite between national norms to pediatric subjects diagnosed with OSA who had undergone adenotonsillectomy (T&A).

Materials and Methods

The study used a prospective, convenience sample of pediatric subjects undergoing polysmnnography (PSG) at the Seattle Children's Hospital Sleep Center at Overlake during the months of January 2019 to November 2019. Subjects included were 8 to 18 years old who did not have previous orthodontic treatment for their OSA. The lower age limit was chosen because children 8 years or older typically have completed eruption of the maxillary and mandibular central incisors.[49] Other inclusion criteria were the subjects' completion of the Sleep Study at the Seattle Children's Hospital Sleep Center, and the willingness to participate in the study. Exclusion criteria included children with congenital or acquired craniofacial syndromes. These craniofacial syndromes include, but are not limited to, Down Syndrome, Cleft lip and palate, Apert Syndrome, Crouzon Syndrome, Pfeiffer syndrome, Pierre Robins sequence, Treacher Collins syndrome, etc. Children with previous orthodontic treatment to correct their airway were also excluded. Finally, participants were excluded at the time of recruitment if they were enrolled in other research conducted by the Seattle Children's Hospital. This was decided to prevent subject burnout from multiple studies.

The Medical record of potential subjects was pre-screened by the investigator for the inclusion/ exclusion criteria listed above and families were approached at the time of the PSG. Consent from guardians and assent from minors was obtained. The study protocol was conducted immediately prior to the PSG set-up. This study was approved by the Human Subjects Division of the University of Washington by the Institutional Review Board.

Questionnaire

A questionnaire on the subject's dental history was completed by parent/guardian. Questions included the following:

1. Does your child see a dentist at least once/year?
2. Has your child been recommended for orthodontic (braces) treatment?
3. Was it specifically for his/her breathing?

The answer options were either "yes", "no" or "I don't know". "Yes" answers were recorded as a positive response. Answers of "I don't know", or "no" were considered as a negative response. Next, the subject's habits were evaluated by asking the following question:

Does your child have any habits? (Circle all that apply)

(1) Thumb-sucking, (2) nail-biting, (3) Mouth Breathing, (4) Teeth grinding.

Clinical Examination and Photos

Intraoral and extraoral evaluations and photos were recorded prior to the sleep study. All subjects were evaluated by the same examiner (K.K.). The examiner was blinded to the results of the questionnaires at the time of examination and photographs. The intraoral and extraoral evaluation forms were used to standardize the clinical examination. A profile photo was taken with a digital camera. Examinations were separated into 1. Profile and 2. Intraoral exams.

Profile Exam

The facial profile was evaluated using the photo taken by the examiner. Subjects were positioned to capture their right profile photo. The pitch of the face was positioned so it is horizontal to the floor.[50] Facial convexity or concavity was evaluated by assessing the curvature of subject's glabella, subnasale, and pogonion. Curvature greater than 10° from glabella-subnasale to subnasale-pogonion, was recorded as convex, and curvature less than 0° was recorded as concave. Facial Profile was recorded as straight/balanced, convex, or concave.

Intraoral Exam

Intraoral findings were recorded through clinical examination of each subject using a standardized examination form (figure 1). In the AP dimension, subject's Angle classification and overjet were measured. Molar and canine Angle classifications were recorded on both right and left sides, using the subject's permanent 1st molar and subject's primary or permanent canines. Angle classification was determined by using the

relationship between subject's mesiobuccal cusp of the maxillary permanent 1st molar and the buccal groove of the mandibular permanent 1st molar. Half-cusp, and full-cusp and beyond were recorded as the full cusp classification (i.e. End-On class II recorded as class II). For the primary dentition, maxillary and mandibular primary second molars were used to determine mesial-step (MS), flush terminal plane (FTP), or distal-step (DS) by evaluating the distal surface relationships between the two primary molars. Measurements at or above half cusps were recorded as a step classification (MS or DS). The Angle classification of molars and canines were combined into a global Angle classification. This was done by choosing the most frequently diagnosed classification on the right and left molars and canines. When the classifications were split evenly between two different classifications, the presence of teeth were investigated to determine if the subject had early loss of tooth. From there, appropriate classification was assigned.

Overjet was recorded by the average distance between the two upper central incisors and the two lower central incisors. When the subject exhibited single anterior crossbite, the tooth that was in crossbite was recorded as well as the overjet of the contralateral tooth.

Vertical dimension measured subject's overbite or openbite of both permanent and primary dentition. Overbite was measured by the averaged amount of the two central incisors overlap to the two lower central incisors in maximum intercuspation. Openbite was evaluated similarly, and the distance between the incisal edges of the maxillary and

mandibular central incisors were measured. Measurement discrepancies between the left and right central incisors were averaged. Subjects who exhibit lack of incisor overlap only on one tooth was noted as mixed subjects.

Transverse dimension was evaluated for the presence of posterior crossbites greater than or equal to cusp-to-cusp in the primary or permanent posterior dentition. Crossbites were noted only if two or more adjacent teeth were involved; one of which had to be a permanent molar. Maxillary posterior teeth could be either palatal or completely buccal in relation to the mandibular posterior teeth.

Intraoral Examination Form		
1. Molar class	Permanent R: I / II / III / Missing L: I / II / III / Missing	Primary R: MS / FTP / DS / Missing L: MS / FTP / DS / Missing
2. Canine class	Permanent R: I / II / III / Missing L: I / II / III / Missing	Primary R: I / II / III / Missing L: I / II / III / Missing
3. Overjet	_____mm [ ] negative overjet	
4. Overbite	_____% overlap (_____mm) [ ] open bite	
5. Crossbite	Posterior: None / Present	

Figure 1: Intraoral Examination Form

<u>Sleep Study / Polysomnography (PSG)</u>

All subjects underwent PSG testing at the Seattle Children's Hospital Sleep Center at Overlake. The facility is accredited by the American Academy of Sleep Medicine. PSGs were scored in guidance to the American Academy of Sleep Medicine. The breakdown of

the scores is available in the Appendix 1. For this study, the obstructive Apnea-Hypopnea Index (oAHI) was used to evaluate the child's severity of OSA.

Subject's age, weight, height, and tonsil sizes were obtained from the PSG report. Age, weight, and height were used to calculate the subject's BMI Z-score. Z-score of the BMI was used in lieu of BMI, as it predicts the level of obesity in the pediatric population using external reference, such as national data, rather than internal reference. Must et al. illustrated that when internal reference is used, "exactly 5% of every sample would be above the 95th percentile, and the specific BMI cut point designating the 95th percentile would not be the same across samples. In most research applications, either BMI z-scores or BMI-for-age percentiles can be used to determine cut points and classify weight status of children and adolescents."[51]

Statistical Analysis

The subjects were initially classified into two distinct groups: subjects with OSA who had previous T&A vs subjects with OSA who are naïve to T&A. These subjects were then categorized based on the variables of malocclusion (overjet, overbite, crossbite, and facial profile). For these categorical variables, Pearson chi-square test and Fischer exact test were used, and each variables of malocclusion were tested for statistical significance that was determined using $p < 0.05$ for significance.

Next, the study sample was categorized based on the OSA diagnosis. Subjects grouped in No OSA were used as a control to compare the dentofacial variables (overjet,

overbite, crossbite, and facial profile) against the Mild OSA and Moderate/Severe OSA groups. Pearson chi-square test and Fischer exact test were used, and each variable was tested for statistical significance (using $p < 0.05$ for significance).

Finally, NHANES III data reported two separate age groups, the 8-11 year old group and the 12-17 year old group. To analyze the results comparatively, Post-T&A OSA group was further divided into two groups by the same age range. NHANES III data were then used to compare the dental variables (overjet, overbite, and crossbite) against the 8-11 year old and 12-17 year old Post-T&A OSA groups. Pearson chi-square test was used, and each variable was tested for statistical significance (using $p < 0.05$ for significance).

Results

A total of 103 subjects were considered for recruitment from the sleep clinic at Seattle Children's from February 2019 to October 2019. Two subjects declined to enroll in the study, and six subjects were not approached because they were recruited for a separate research study that same night. The sleep clinic manager deemed that it was excessive for the subject to be enrolled in two research efforts. The final enrollment was 95 subjects. Treatments rendered by the sleep physicians were not available in the sleep study results since subsequent appointments after the sleep study were not accessible by the primary researcher. Figure 2 illustrates the subject flowchart of the study population.

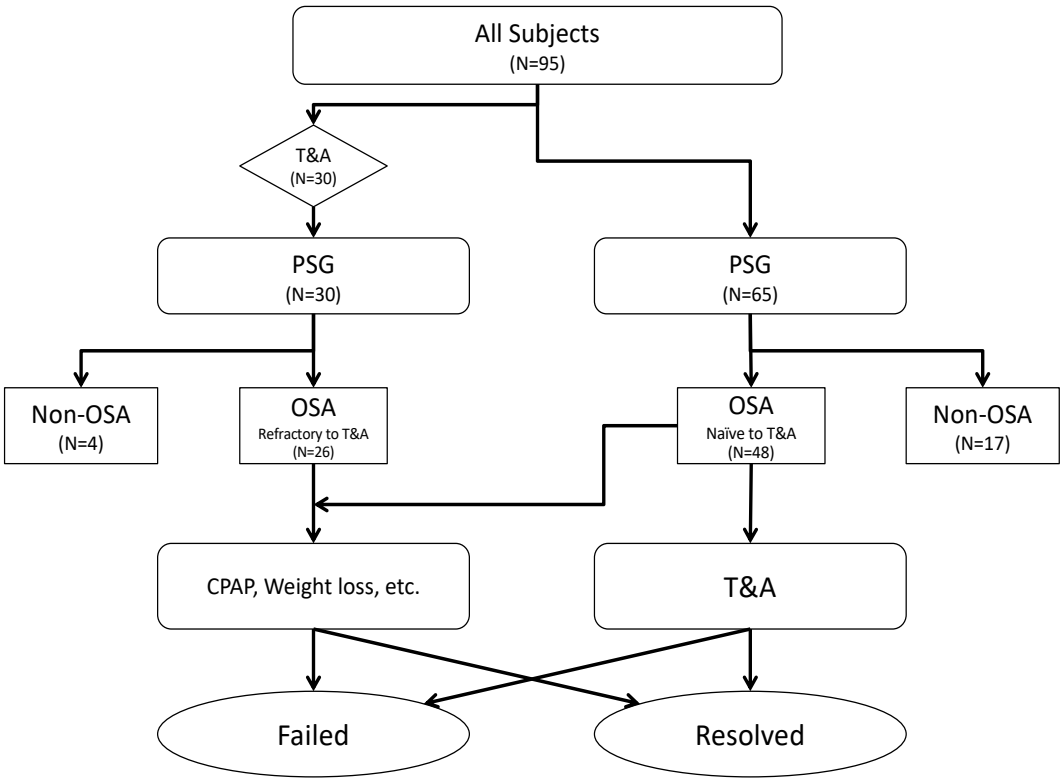

Figure 2: Overall Subject Flowchart. Each subject underwent intraoral and extraoral examinations at the time of the Polysomnography (PSG). Subjects were categorized into Non-OSA, OSA Refractory to T&A Surgery (Refractory to T&A), or OSA Naïve to T&A Surgery (Naïve to T&A).

The average age of the subjects was 11.9 years (SD = 2.9 years; range = 8-17 years). There were 62 males and 33 females in the sample. There was a similar distribution of males and females among all OSA groups. Demographic characteristics are presented in Table 1.

	Total (N=95)	**No OSA (N=21)**	**Mild OSA (N=54)**	**Moderate to severe OSA (N=20)**
Male: n (%)	62 (65.3%)	13 (61.9%)	35 (64.8%)	14 (70.0%)
Female: n (%)	33 (34.7%)	8 (38.1%)	19 (35.2%)	6 (30.0%)

Age: mediasn (IQR)	11 (9.0, 13.0)	10 (9.0, 12.0)	12 (9.0, 14.0)	12 (11.0, 13.0)
BMI Z-Score: median (IQR)	1.0 (-0.04, 2.1)	0.4 (-0.3, 1.4)	0.6 (-0.5, 1.8)	2.2 (2.0, 2.6)
oAHI: median (IQR)	2.0 (1.0, 4.7)	0.7 (0.3, 0.8)	2.0 (1.5, 3.5)	8.8 (7.2, 15.1)
Mallampati: median (IQR)	2 (1.0, 2.0)	2.0 (1.0, 2.0)	2.0 (1.0, 2.0)	2.0 (1.0, 2.1)
T&A Status				
Surgically Naïve: n	65	17	34	14
Post-T&A: n	30	4	20	6

Table 1: Study Demographics. IQR= inter quartile range

Surgically Naïve vs Post-T&A Comparison

There was no statistical difference in the measured dentofacial characteristics between the surgically naïve subjects diagnosed with OSA and those subjects with prior exposure to T&A surgery. As expected, the tonsil size between the surgically naïve group to the post-T&A group exhibited statistically significant difference. The distribution of baseline covariates and the dentofacial characteristics between the two groups are available in Appendix 2 and 3, respectively.

Tonsil sizes exhibited statistically significant difference (p=<0.001) as the Post-T&A group had undergone surgery to remove these structures. Interestingly, there were four subjects post-T&A surgery who exhibited tonsil size grading of 2+, and 1 subject exhibited tonsil size grading of 1+. 21 subjects (80.8%) had no tonsils reported by the sleep technician.

The prevalence of crossbite in both groups exhibited no difference as the surgically naïve subjects and the Post-T&A groups exhibited crossbites in 20.8% and 23.1% of the population, respectively. There was a small trend of increased concavity of the facial profile of the Post-T&A subjects. When comparing the two groups, the surgically naïve group had 20.8% of subjects with balanced/straight facial profile and 14.6% of subjects with a concave facial profile.

Angle classification, overjet, and overbite did not show significant differences between the Surgically Naïve and the Post-T&A groups. When comparing the distribution of overjet between the two groups, the majority of the subjects were in the increased (> 2mm) overjet value with no statistically significant difference between the subject with and without exposure to T&A surgery ($p = 0.25$). For overbite, the difference between the two distributions also showed no significant difference ($p = 0.17$). The surgically naïve group and the Post-T&A groups had 0 mm overbite frequencies of 8.3% and 23.1%, respectively. However, the difference in the number of subjects was only 2 subjects between the two groups. There was one subject in the surgically naïve group that had an overbite greater than 7mm. Angle classifications had similar distribution with no statistically significant difference ($p = 0.50$).

OSA Severity Comparison

There was a statistically significant difference in the facial profile of the Moderate to Severe OSA group when compared to the No OSA Group ($p=0.02$). All other dentofacial characteristics exhibited no significant difference between the No OSA, Mild

OSA, and Moderate to Severe OSA groups. The distribution of dentofacial characteristics between the three groups is available in Appendix 4.

The facial profile of the Moderate to Severe OSA group had a roughly even distribution between all three categories with 30% of the subjects having concave facial profile, and 35% each for subjects with convex and balanced/straight facial profiles. In comparison, the majority of the subjects who were diagnosed with no OSA and mild OSA exhibited a greater prevalence of a convex facial profile. All the subjects who exhibited a facial concavity were diagnosed with some level of OSA.

Intraorally, Angle classification, overjet, overbite, and posterior crossbites all did not exhibit significant difference when the Mild OSA and Moderate to Severe OSA groups were separately compared to No OSA group.

NHANES Comparison

We compared the Post-T&A OSA group to the NHANES III data for crossbite, overjet, and overbite. There were statistically significant differences when comparing the NHANES data to the Post-T&A OSA groups in the number of posterior crossbites in the 8-11 year old group, the amount of overjet in both the 8-11 year old and 12-17 year old groups, and the amount of overbite in both the 8-11 year old and 12-17 year old groups. Posterior crossbite in the 12-17 year old Post-T&A group showed no statistically significant difference ($p=0.76$) when compared to the NHANES III data. The distributions of the dentofacial features in both age groups of the NHANES III and the Post-T&A OSA groups are available in Appendix 5.

In the NHANES III data, the percentage of posterior crossbites in the 8-11 year old group was 7.1% and the 12-17 year old group was 8.8%. In comparison, the Post-T&A OSA group in this study had 38.5% of subjects exhibiting a posterior crossbite in the 8-11 year old group. This was statistically significant ($p = <0.001$). On the contrary, the 12-17 year old group exhibited a lower prevalence of posterior crossbite (7.7% when compared to the NHANES III group); however, there was only one subject in this Post-T&A OSA group.

NHANES III data report an overbite distribution around the ideal measurement of 0 – 2 mm with 45.0% of the 12 – 17 year old population in this category, and with 34.7% in the moderate (3 – 4mm) overbite group. In our study, we found a similar trend. In the 12 – 17 year-olds, the majority had an overbite of 0-2mm (38.1%) with the second highest prevalence being in the moderate overbite category (3-4mm). We did find a tendency toward a greater percentage of open bite 16.7% in our study versus 3.5% in the NHANES data ($p=0.01$).

Questionnaire

All 95 questionnaires were completed by the accompanying parent or guardian. 94 patients reported going to the dental office for an examination in the past 6 months, and only one subject denied going in for dental checkup in the past year.

All habits except bruxism shared similar ratios among different OSA groups. Mouth-breathing was the highest prevalence with 58.9% of the subjects reported mouth-breathing. However, subjects with no clinical OSA diagnosis had the highest mouth-

breathing and nail-biting percentage with 67% and 48%, respectively. Interestingly, bruxism showed a linear correlation between the different severity of OSA. Appendix 7 shows the questionnaire results for orthodontic recommendation and habits of the entire sample and stratified by OSA severity.

Discussion

The aims of this study were to characterize dentofacial characteristics (overjet, overbite, posterior crossbite, facial convexity) in children referred to a tertiary pediatric sleep center for OSA. Amongst those subjects, comparisons were made between groups based on surgical status (naïve vs adenotonsillectomy), severity of OSA (none, mild, moderate, severe), and with general population control data available from the third National Health and Nutrition Examination Survey (NHANES III).

There was no statistical difference in the measured dentofacial characteristics between the surgically naïve subjects diagnosed with OSA and those subjects with prior exposure to T&A surgery.

As expected, there was a statistically significant difference in the tonsil size between the surgically naïve group and the post-T&A group. Interestingly, we found several subjects with tonsillar size relapses after the surgery (5/30 patients, or 3.3%, who had a tonsil grading of 2+ after undergoing T&A). Zhang et al. in 2014 reported that tonsillar regrowth occurred in 6.1% of the patients who underwent tonsillectomy.[52]

Of the 95 subjects enrolled in the study, 77.9% of the subjects were diagnosed with some level of OSA. The sample size's ratio of males to females was 3 : 1. This ratio was

fairly consistent among all severity groups of OSA. In adult OSA, there is an increased risk factor for males, and the ratio of 3 : 1 matches our sample's ratio. In pediatric OSA, there seems to be conflicting evidence when it comes to sex predilection. There is evidence that the prevalence of pediatric sleep disorder breathing is increased in males as reported by Lumeng et al. in 2008.[53] However, in 2017, Brockmann et al. reported that there are no clear "gender-based differences in OSA prevalence or severity" and evidence remains unclear as there are various confounding factors that contributes to pediatric OSA.[54] One of these factors are subject's level of obesity. Z-score of the BMI was used in lieu of BMI, as it predicts the level of obesity in the pediatric population using external reference, such as national data, rather than internal reference. Must et al. illustrated that when internal reference is used, "exactly 5% of every sample would be above the 95th percentile, and the specific BMI cut point designating the 95th percentile would not be the same across samples. In most research applications, either BMI z-scores or BMI-for-age percentiles can be used to determine cut points and classify weight status of children and adolescents."[55] In our study population, the moderate to severe OSA groups exhibited BMI Z-scores of 2.53 (IQR: 2.1.- 2.8), which was clearly higher compared to the other two groups. This finding supports the literature of obesity being one of the major risk factors for OSA.[16] However, in mild OSA cases, obesity may not play a big role as the Z-score (0.4) of the Mild OSA group was only slightly higher compared to the Z-score (0.6) of the No OSA group.

The facial profile of the subjects with moderate to severe OSA exhibited a tendency for facial concavity. All twelve patients with facial concavity were diagnosed

with OSA, where six were diagnosed with mild OSA and the other six were diagnosed with moderate to severe OSA. However, it cannot be concluded with certainty that facial concavity is associated with pediatric OSA because of the small sample size of the moderate to severe OSA group. Currently, there is limited available literature to support facial morphology associated with pediatric OSA. Roberts et al. reported that pediatric patients with consistent CPAP use can cause changes in midface development.[40] All twelve patients in our study with facial concavity were evaluated for the prescription of previous CPAP wear. Out of the twelve patients, eight patients had undergone a previous sleep study at the Seattle Children's Hospital. Only one subject was recommended use of CPAP for treatment. Two patients were recommended T&A, and the remaining five patients lack patient follow-up data to give a correct treatment rendered. These findings do not indicate that the use of CPAP may have caused the observed facial concavity in our patient pool.

When evaluating the BMI Z-score of subjects with a concave facial profile, the moderate to severe OSA group exhibited a mean BMI Z-score of 2.3 (SD = 1.4). The mild OSA group exhibited a mean BMI Z-score of 1.4 (SD = 1.8). The two groups diagnosed with OSA exhibited higher values vs. the no OSA group, where the mean BMI Z-score was 0.4 (SD = 1.2). The no OSA group did not include any subjects with facial concavity. As mentioned previously, increased weight is a contributing factor for pediatric OSA. Therefore, it is difficult to draw conclusions about subject's facial concavity as increased weight may be a confounding factor associated with the greater sleep problems.

There was no statistically significant difference between the presence of a posterior crossbite between the No OSA group and the different OSA groups. The percentage of crossbite in the entire sample population was 20.0%. The No OSA group had 14.3% of patient exhibiting posterior crossbite while the Mild OSA and Moderate to Severe OSA groups had 22.3% and 20.0%, respectively. This is far greater reporting than the percentages reported by NHANES III, which are 7.10% in the 8-11 years old and 8.80% in the 12-17 years old.

Carvalho et al. found that posterior crossbite was associated with sleep disordered breathing in 7-9 year-olds.[56] Caprioglio et al. in 1999 reported similar findings.[57] Our study confirms these reports, as the younger age group showed significant differences compared to population frequencies reported by NHANES.

There was a slight tendency of the overbite to decrease as the severity of OSA increased. There have been numerous reports that mouth breathing is associated with anterior open-bite. In our sample population, 58.9% of the subjects reported mouth-breathing. A quarter of these patients were not diagnosed with clinical OSA, while the majority (57.1%) of the subjects who reported mouth-breathing were diagnosed with Mild OSA. It is not certain if a decreased overbite increases the risk of OSA or the OSA causes the subjects to have a decreased overbite.

There is some literature that supports an association between Class II malocclusion and a diagnosis of pediatric OSA.[22, 58] In our study population, this

association was not supported. The Mild and Moderate to Severe OSA groups did not exhibit statistically significant differences between the No OSA group in Angle's classification ($p = 0.3$, $p=0.07$).

There were several limitations of this study. First, the sample size of the patients diagnosed with moderate to severe OSA was small (20 patients). Furthermore, the OSA groups had very low numbers when separated into age groups for comparison with the NHANES III data. The lack of a control group of matched children without OSA was also a limiting factor. The NHANES III cross-sectional data could not be matched to our subjects for age/weight. The no-OSA group was also a suboptimal control group since they were referred to the sleep clinic for suspected sleeping disordered breathing. Lastly, only a single assessor was used to analyze the facial convexity of the subjects. Despite being blinded to OSA diagnosis, the risk of systematic misclassification is increased with a single assessor.

In essence, this study supports a whitepaper published by the American Association of Orthodontics in 2019 that stated that "the presence of OSA cannot be determined by craniofacial morphology alone".

Conclusions

No significant differences in dentofacial characteristics were found for subjects who had residual OSA after a T&A procedure vs. the surgically naïve. We found no significant differences in dentofacial characteristics between children diagnosed with

different severities of OSA. A posterior crossbite was associated with age specific groups and OSA, but this should be viewed with caution as the size samples were small. There was a slight tendency for facial concavity to be associated with moderate to severe pediatric OSA. However, this most likely was caused by the confounding factor of obesity. Dentofacial characteristics alone cannot be used to determine which children will be refractory to T&A surgery or the severity of OSA in children.

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Appendices

Groups	oAHI
Non-apneic	< 1
Mild OSA	≥ 1 to < 5
Moderate OSA	≥ 5 to < 10
Severe OSA	≥ 10

Appendix 1: Breakdown of oAHI Scoring to assess OSA Severity

		Surgically Naïve (N=48)	Post-T&A OSA (N=26)
Sex (% of total)			
	Male	36 (69.2%)	13 (50%)
	Female	12 (30.8%)	13 (50%)
Median (IQR) oAHI score		3.4 (1.9-6.4)	2.1 (1.3-4.9)
Median (IQR) Age		11 (9-15)	12 (11-14)
Median (IQR) Mallampati Score		2 (1-2)	2 (1-3)
Median (IQR) BMI Z-Score		1.1 (-0.2 – 2.1)	1.9 (0.6 – 2.5)

Appendix 2: Distribution of demographic data, stratified by the state of adenotonsillectomy surgery prior to the clinic visit.

	Surgically Naïve (N=48)	Post-T&A OSA (N=26)	P-value
Crossbite (% of total)			0.62
Yes	10 (20.8%)	6 (23.1%)	
No	38 (79.2%)	20 (76.9%)	
Facial Profile (% of total)			0.08
Balanced	10 (20.8%)	11 (42.3%)	
Concave	7 (14.6%)	5 (19.2%)	

Convex	31 (64.6%)	10 (38.5%)	
Tonsil size (% of total)			>0.001
0	5 (10.4%)	21 (80.8%)	
1+	13 (27.1%)	1 (3.8%)	
2+	17 (35.4%)	4 (15.4%)	
3+	13 (27.1%)	0	
Angle classification (% of total)			0.50
Class 1	21 (43.8%)	15 (56.7%)	
Class 2	21 (43.8%)	9 (36.7%)	
Class 3	6 (12.5%)	2 (6.7%)	
Overjet (mm) (% of total)			0.25
> 2 (Increased OJ)	31 (64.6%)	17 (65.4%)	
1-2 (Normal)	13 (27.7%)	6 (23.1%)	
< 0 (Underbite)	1 (2.1%)	3 (11.5%)	
Overbite (mm) (% of total)			0.17
> 2 (Deep bite)	30 (62.5%)	12 (46.2%)	
1-2 (Normal)	14 (29.2%)	8 (30.8%)	
< 0 (Open bite)	3 (8.3%)	6 (23.1%)	

Appendix 3: Distribution of dentofacial features, stratified by the state of Tonsillectomy and Adenoidectomy surgery prior to the clinic visit for diagnosed OSA subjects.

	Total	**No OSA**	**Mild OSA**	**p-value**	**Moderate to severe OSA**	**p-value**
	(N=95)	**(N=21)**	**(N=54)**		**(N=20)**	
Crossbite (% of total)				0.44		0.68
Yes	19 (20.0%)	3 (14.3%)	12 (22.2%)		4 (20.0%)	
No	76 (80.0%)	18 (85.7%)	42 (77.8%)		16 (80.0%)	
Facial Profile (% of total)				0.26		0.02
Balanced	28 (29.5%)	7 (33.3%)	14 (25.9%)		7 (35.0%)	
Concave	12 (12.6%)	0	6 (11.1%)		6 (30.0%)	
Convex	55 (57.9%)	14 (66.7%)	34 (63.0%)		7 (35.0%)	
Tonsil size (% of total)				0.75		0.16
0	30 (31.6%)	5 (23.8%)	18 (33.3%)		8 (40.0%)	
1+	21 (22.1%)	5 (23.8%)	10 (18.5%)		4 (20.0%)	
2+	28 (29.5%)	8 (38.1%)	19 (35.2%)		3 (15.0%)	

3+	13 (13.7%)	1 (4.8%)	7 (13.0%)	5 (25.0%)	
Missing	3 (3.2%)	2 (9.5%)	0	0	
Angle classification (% of total)			0.30		0.07
Class 1	45 (47.4%)	9 (42.9%)	27 (50.0%)	9 (45.0%)	
Class 2	42 (44.2%)	12 (57.1%)	23 (42.6%)	7 (35.0%)	
Class 3	8 (8.4%)	0	4 (7.4%)	4 (20.0%)	
Overjet (mm) (% of total)			0.97		0.99
> 2	66 (69.5%)	15 (71.4%)	37 (68.5%)	14 (70.0%)	
1-2 (Mild)	24 (25.3%)	5 (23.8%)	14 (25.9%)	5 (25.0%)	
< 0 (Xbite)	5 (5.3%)	1 (4.8%)	3 (5.6%)	1 (5.0%)	
Overbite (mm) (% of total)			0.56		0.43
> 2 (Deep bite)	54 (56.8%)	12 (57.1%)	30 (55.5%)	12 (60.0%)	
1-2 (Mild)	30 (31.6%)	8 (38.1%)	17 (31.5%)	5 (25.0%)	
< 0 (Openbite)	11 (11.6%)	1 (4.8%)	7 (13.0%)	3 (15.0%)	

Appendix 4: Distribution of dentofacial features among those with no OSA, mild OSA, and moderate to severe OSA, stratified by the severity.

	NHANES III		**PostT&A OSA Population (N=26)**			
	8-11 y/o	**12-17 y/o**	**8-11 y/o (N=13)**	**P-value**	**12-17 y/o (N=13)**	**P-value**
Crossbite (% of total)				<0.001		0.76
Yes	7.1%	8.8%	5 (38.5%)		1 (7.7%)	
No	92.9%	91.2%	8 (61.5%)		12 (92.3%)	
Overjet (mm) (% of total)				0.02		0.02
> 10 (Extreme)	0.2%	0.2%	0		0	

	7-10 (Severe)	3.4%	3.5%	0		0	
	5-6 (Moderate)	18.9%	11.9%	2 (15.4%)		3 (23.1%)	
	3-4 (Mild)	45.2%	39.5%	6 (46.2%)		6 (46.2%)	
	1-2 (Ideal)	29.6%	39.3%	3 (23.1%)		3 (23.1%)	
	0 (Mild)	2.2%	4.6%	1 (7.7%)		1 (7.7%)	
	-1 to -2 (Moderate)	0.7%	0.5%	1 (7.7%)		0	
	-3 to -4 (Severe)	0.0%	0.6%	0		0	
	> -4 (Extreme)	0.0%	0.0%	0		0	
Overbite (mm) (% of total)					<0.001		<0.001
	> -4 (Extreme)	0.3%	0.2%	0		0	
	-4 to -3 (Severe)	0.6%	0.5%	0		0	
	-2 to 0 (Moderate)	2.7%	2.8%	3 (23.1%)		3 (23.1%)	
	0 to 2 (Ideal)	40.2%	45.0%	5 (38.5%)		3 (23.1%)	
	3 to 4 (Moderate)	36.2%	34.7%	3 (23.1%)		3 (23.1%)	
	5 to 7 (Severe)	18.8%	15.5%	2 (15.2%)		4 (30.7%)	
	> 7 (Extreme)	1.2%	1.3%	0		0	

Appendix 5: Distribution of selected dental features in the NHANES III population and the Post-T&A OSA group (N=26), stratified by age group.

	NHANES III		**No OSA Population (N=21)**			**Mild OSA (N=54)**			**Mod/Severe OSA (N=20)**		
	8-11 y/o	12-17 y/o	8-11 y/o (N=8)	P-value	12-18 y/o (N=13)	P-value	8-11 y/o (N=25)	P-value	12-18 y/o (N=29)	P-value	8-11 y/o (N=1)	P-value	12-18 y/o (N=19)	P-value
Crossbite				0.20		0.15		<0.001		0.08		<0.001		0.13
Yes	7.10%	8.80%	1 (12.5%)		2 (15.4%)		7 (28%)		5 (17.2%)		1 (100%)		3 (15.8%)	
No	92.90%	91.20%	7 (87.5%)		11 (84.6%)		18 (72%)		24 (82.8%)		0		16 (84.2%)	

Appendix 6: Crossbite data between NHANES III and study population, stratified by diagnosed OSA severity.

		Total	No OSA	Mild OSA	Moderate to severe OSA
		(N=95)	(N=21)	(N=54)	(N=20)
Ortho Recommendation					
	Yes	37 (38.9%)	10 (47.6%)	20 (37.0%)	7 (35.0%)
	No	58 (61.1%)	11 (52.4%)	34 (63.0%)	13 (65.0%)
Habits					
	Nail biting	42 (44.2%)	10 (47.6%)	25 (46.3%)	7 (35.0%)
	Mouth Breathing	56 (58.9%)	14 (66.6%)	32 (59.3%)	10 (50.0%)
	Thumb Sucking	7 (7.4%)	1 (4.8%)	5 (9.3%)	1 (5.0%)
	Bruxism	36 (37.0%)	2 (9.5%)	16 (29.6%)	8 (40.0%)

Appendix 7: Questionnaire Results for Orthodontic Recommendation and Habits by the Study Sample