

Dental arch morphology in children with sleep-disordered breathing

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SUMMARY The aim of the present study was to examine the effects of nocturnal breathing disorders such as obstructive sleep apnoea (OSA) and snoring on developing dental arches. The study group comprised 41 children (22 males, 19 females, mean age 7.2 years, standard deviation 1.93) with diagnosed OSA. Age- and gender-matched groups of 41 snoring and 41 non-obstructed control children were selected. Orthodontic examination was carried out and dental impressions were taken. Malocclusions were diagnosed clinically and 13 linear variables were measured from the dental casts. The differences between the dental arch measurements of the OSA, snoring, and control groups were studied using analysis of variance followed by Duncan's multiple comparison method.

Children with diagnosed OSA had a significantly increased overjet, a reduced overbite, and narrower upper and shorter lower dental arches when compared with the controls. Snoring children had similar but not as significant differences as OSA children when compared with the controls. There were more children with an anterior open bite (AOB) in the OSA group ($P = 0.016$) and with a Class II or asymmetric molar relationship in the groups of OSA ($P = 0.013$) and snoring ($P = 0.004$) subjects compared with the non-obstructed controls. There were more subjects with mandibular crowding ($P = 0.002$) and with an AOB ($P = 0.019$) with an increasing obstructive apnoea–hypopnoea index (AHI).

These findings are in agreement with previous studies of the effects of increased upper airway resistance on dental arch morphology and can be explained by long-term changes in the position of the head, mandible, and tongue in order to maintain airway adequacy during sleep.

Introduction

Snoring is often considered a harmless problem in children (Ali *et al.*, 1994), but it may be a sign of a more serious upper airway obstruction, such as increased upper airway resistance or obstructive sleep apnoea (OSA), a condition involving repeated obstruction of the upper airway during sleep (Guilleminault and Stoohs, 1990). The prevalence of regular snoring is estimated to be approximately 10 per cent and the prevalence of OSA about 0.7–2.9 per cent in pre-school children (Teculescu *et al.*, 1992; Ali *et al.*, 1993, 1994; Gislason and Benediktsdottir, 1995; Löfstrand-Tideström *et al.*, 1999).

Adenotonsillar hypertrophy compromising the upper airway volume is generally considered the most common cause for snoring and OSA in 'normal' children. Development of the disorder depends more on a decreased volume of the upper airways as shown by a magnetic resonance imaging study (Arens *et al.*, 2001) than on the absolute size of the lymphoid tissue. Orovelopharyngeal factors, including enlargement of palatinal tonsils, are dominant in producing obstructive symptoms in children (Fernbach *et al.*, 1983; Brodsky *et al.*, 1989; Suto *et al.*, 1996; Nieminen *et al.*, 1997; Fregosi *et al.*, 2003).

The relationship between adenotonsillar hypertrophy and dentofacial morphology in children has been reported in many studies usually in connection with mouth breathing (Linder-Aronson, 1970; Behlfelt *et al.*, 1989, 1990; Hultcrantz *et al.*, 1991). In OSA, both oral and nasal airflow are prevented by blockage of the upper airway during sleep. It has been speculated that a child with a collapsed airway has to either change sleeping body or head posture, or the position of the tongue and/or the mandible in order to breathe (Behlfelt, 1990). Upper airway volume has been demonstrated to be related to these factors by many previous researchers (e.g. Hellsing, 1989; Lowe, 1990; Jan *et al.*, 1994; Solow *et al.*, 1996; Ono *et al.*, 2000; Hiyama *et al.*, 2002). A number of studies have shown that the same factors have effects on the occlusion (Pirilä *et al.*, 1995; Solow and Sonnesen, 1998; Solow and Sandham, 2002).

Despite the research in the field of paediatric OSA during the last few decades, little attention has been paid to the possible effects of sleep-related breathing disorders on the developing dentition. Löfstrand-Tideström *et al.* (1999) studied a group of 4-year-old children with breathing obstruction and sleep apnoea and found that the obstructed children had a narrower maxilla, deeper palatal height, and

a shorter lower dental arch when compared with healthy children with ideal occlusion. They also noted the prevalence of lateral crossbites to be greater in the group of obstructed children. These characteristics are much the same as found previously in older children with lymphoid tissue hypertrophy (Linder-Aronson, 1970; Behlfelt *et al.*, 1989, 1990; Hultcrantz *et al.*, 1991).

The aim of this investigation was to examine dental arch morphology in snoring children and those with OSA with or without enlargement of palatal tonsils. The hypothesis was that in the children with night-time obstructed breathing, the complex combination of the environmental factors affecting occlusal development and dental arch formation is different from normal children, which would lead to variations in dental arch morphology. It was assumed that the findings would be more prominent in children with more severe obstruction.

Subjects and methods

The study protocol was approved by the Ethics Committee of the Oulu University Hospital, Finland. Informed consent was obtained from the parents before the children entered the study.

Subjects

Dental examinations were performed on 138 children referred from primary health care units to the Department of Otorhinolaryngology of Oulu University Hospital because of snoring and symptoms of obstructive sleep disorders for more than 6 months during the years 1994–1996 and 2000–2002. Children with known upper airway anomalies, abnormal development, chronic infections, asthma, or perennial allergy were not included. Previous orthodontic treatment or missing teeth resulted in the exclusion of 23 children. One hundred and fifteen children were eligible for inclusion, but 18 children or parents refused to take part, so the study group comprised 97 children.

All children were evaluated by overnight polysomnography. Four patients were excluded at this stage because of unsuccessful registration. On the basis of these findings, the study group of 41 children with diagnosed OSA was formed. The mean age of these children (22 males and 19 females) was 7.2 years [standard deviation (SD) 1.93; range 4.3–11.4]. From the same group of examined children, an age- and gender-matched group of 41 snoring children without diagnosed OSA was selected. The mean age in the group of snoring children was 7.2 years (SD 1.79; range 3.8–10.8). For 11 snoring children, there was no matched OSA child, and these children were thus excluded.

The control group comprised 41 randomly selected non-obstructed age- and gender-matched children with no history of snoring, upper airway problems, or health-related

complaints. These voluntary children were recruited from schools and the younger children were offspring of hospital or university personnel. The control children presented no apnoeic episodes, habitual snoring, or restless sleep during the night according to their parents' observations. A history of adenoidectomy because of recurrent otitis media was accepted in two control children. Dental examinations were carried out and dental impressions were taken of all control children. Malocclusions were accepted but no orthodontic treatment had been carried out in the control group. During the oral examination, it was confirmed that none of the children in the control group had enlarged tonsils. Asymptomatic control children did not undergo polysomnographic assessment since they had no history of obstructive symptoms as observed by their parents. Obstructive apnoeas are found to be rare in non-snoring children (Marcus *et al.*, 1992; Nieminen *et al.*, 2000). The mean age in the control group was 7.2 years (SD 1.90; range 4.1–11.9).

A history of adenoidectomy was found in 46.3 per cent of the OSA subjects, in 24.4 per cent of the snoring subjects, and in 4.9 per cent of the non-obstructed subjects.

Methods

Polysomnography. All children with snoring and suspected OSA underwent overnight polysomnographic monitoring in the Department of Otorhinolaryngology, and the recordings were manually analysed by the same clinical neurophysiologist (UT) at the Department of Clinical Neurophysiology, Oulu University Hospital. The nocturnal events were monitored with a six-channel computerized polysomnograph with leads for an oro-nasal thermistor, a thoracoabdominal strain gauge, a pulse oximetry, a body position sensor, a leg electromyogram, and a static charge sensitive bed.

An obstructive apnoeic episode was determined as total cessation of oro-nasal airflow with continued respiratory effort for 10 seconds or more. An obstructive hypopnoea period was defined as at least a 50 per cent decrease in oro-nasal airflow signal with continued chest wall motion lasting 10 seconds or more. The severity of OSA was expressed using the obstructive apnoea–hypopnoea index (AHI), which was the sum of apnoeas and hypopnoeas per hour of sleep during polysomnographic registration. An AHI of 1 or higher was considered abnormal based on earlier findings in younger children (Carroll and Loughlin, 1992; Marcus *et al.*, 1992; Rosen *et al.*, 1992; Nieminen *et al.*, 2000). The mean AHI in the OSA group was 3.5 (SD 3.60; range 1–17) and in the snoring group 0.1 (SD 0.20; range 0–0.5).

Clinical assessment of tonsillar size. Both the OSA and the snoring children were examined clinically in order to determine the size of the tonsils on a scale from 1 to 4 (Nieminen *et al.*, 2000). Grade 1 tonsils remain within the

tonsillar fossa, grade 2 tonsils do not reach the midline between the anterior faucial pillar and the uvula, grade 3 tonsils extend medially from the midline, and grade 4 tonsils have a maximum distance of 4 mm between them. Even though the size of both left and right tonsil was registered separately, a single score was used to express tonsillar size in order to simplify statistical analysis. For asymmetric expression of the tonsils (in 23 per cent of the cases), the score of the larger tonsil was used.

Dental cast measurements. Dental examinations, including dental impressions, were all carried out at the Institute of Dentistry, University of Oulu, Finland, by the same investigator (KP-P). The length and breadth of the maxillary and mandibular dental arches were measured on plaster casts using the method of Moorrees (1959) as described in Figure 1. Palatal height was measured to the midline of the palatal vault from a line between the second primary molars. In addition, overjet and overbite were measured. The measurements were undertaken manually using a calibrated digital sliding calliper with an accuracy of 0.01 mm by one author (UP), who was blinded to the study material and protocol.

Intermaxillary occlusal relationships were evaluated both clinically and from the dental casts. Angle classification was performed separately for each side of

the jaw. As none of the examined children had a Class III malocclusion, occlusion of the molars was classified as either Class I (the mesiobuccal cusp of the permanent upper first molar occluded into the buccal fossa of the lower first permanent molar or was less than half cusp mesial or distal) or Class II (the mesiobuccal cusp of the permanent upper first molar occluded from half to a full cusp or more than a full cusp distally relative to the buccal fossa of the lower first permanent molar). Because of the small number of children with an asymmetric Class I/Class II molar relationship, they were combined as Class II malocclusions. If the first permanent molars were not fully erupted, the antero-posterior relationship was determined according to the second primary molar occlusion.

Occlusal discrepancies, such as deep bites, open bites, crossbites, and scissor bites as well as crowding, were recorded and classified modifying the method of Björk *et al.* (1964). Incisor occlusion was judged from the most prominent central incisor. Overbite was categorized as normal, deep (>5 mm), or open (<0 mm, no contact between the upper and lower incisors) bite. Posterior crossbites were recorded if the buccal cusp of the upper tooth occluded lingually to the buccal cusp of the corresponding lower tooth. Posterior crossbites included crossbites of the primary or permanent molars and canines as well as permanent premolars. Scissor bites were registered in the primary or permanent molars or in the permanent premolars if the lingual cusp of the upper tooth occluded buccally to the buccal cusp of the corresponding lower tooth. Crowding was considered moderate or severe if there was >4 mm lack of space in the dental arch, and mild if >2 mm, but <4 mm lack of space existed.

In order to calculate the error of method, the measurement of 20 randomly selected casts was repeated on separate occasions with a 2-week interval. The reliability of Angle classification, deep bite, open bite, crossbite, and scissor bite estimations was also tested.

Statistical analysis

Intra-rater reliability was measured by intraclass correlation coefficients (ICC) for linear dental arch measurements and Cohen's kappa coefficients for malocclusion classifications. The differences between the means of the study and control groups were determined using analysis of variance followed by Duncan's multiple comparison method. The effect tonsillar size on AHI was tested using Fisher's exact test.

As the groups were gender- and age matched, the material was regarded as paired samples (OSA and healthy groups, OSA and snoring groups, snoring and healthy groups) when analysing malocclusion prevalences. A binomial test was used to examine the malocclusion prevalences of each paired sample. Associations between AHI, age, gender,

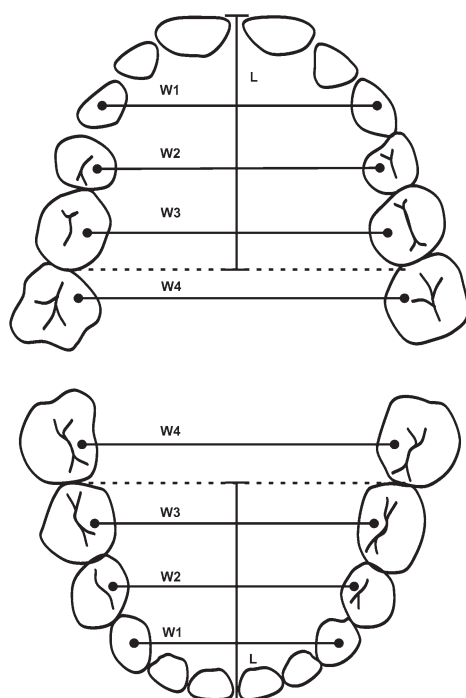


Figure 1 The methods used in dental arch measurements. Maxillary and mandibular arch length (L): arch length measured from the extreme labial surfaces of the incisors perpendicular to a line indicating the extreme distal point of the first primary molars or the second premolars. Maxillary and mandibular arch breadth: a line connecting the crown tips of the canines (W1), the mesiolingual cusps of the primary first (W2) and second (W3) molars and the permanent first molars (W4), and the lingual cusps of the first permanent premolars (W2) or the second permanent premolars (W3).

dental arch measurements, and occlusal and tonsillar variables were tested using multiple regression analysis, with AHI as the dependent variable.

Results

ICC varied from 0.990 to 0.997 for dental arch measurements. Cohen's kappa coefficients varied from 0.889 (Angle classification) to 1.000 (open bite estimation).

In the OSA group 92 per cent, and in the snoring group 76 per cent, of the children had large tonsils (grade 3 or 4). There was no statistically significant relationship between AHI and tonsillar size in the groups of OSA and snoring children ($P = 0.154$, Fisher's exact test).

Linear measurements of the dental arches are shown for the examined groups of OSA, snoring, and control children in Table 1. The differences between the dental arch measurements were studied using analysis of variance followed by Duncan's multiple comparison method. Overjet was found to be larger in the OSA ($P < 0.05$) and snoring ($P < 0.05$) children when compared with the controls (Table 1, Figure 2a). Overbite was significantly reduced in children with OSA when compared with the control children ($P < 0.05$; Table 1, Figure 2b). Maxillary arch width was significantly smaller in the groups of OSA and snoring children than in the control group. Arch width, measured at the level of maxillary canines (W1), was decreased in the OSA ($P < 0.01$) and snoring ($P < 0.05$) groups when compared with the controls (Table 1, Figure 2c). Maxillary arch width was reduced at the level of first primary molars or first permanent premolars (W2) in the OSA group ($P < 0.05$) when compared with the control children (Table 1). Maxillary arch width, measured at the level of second primary molars or second permanent premolars (W3), was

smaller in both the OSA ($P < 0.05$) and snoring ($P < 0.05$) groups when compared with the controls (Table 1). Arch width, measured at the level of the maxillary first permanent molars (W4), was also decreased in the OSA children ($P < 0.05$) when compared with the control children (Table 1). Lower arch length was also found to be significantly smaller in the OSA ($P < 0.001$) and snoring ($P < 0.05$) children than in the control subjects (Table 1, Figure 2d). There was no statistically significant difference in upper arch length, palatal height, or mandibular width between the groups.

The prevalence of recorded malocclusions in the OSA, snoring, and control groups are presented in Table 2. The prevalence of malocclusions in the examined groups were compared using a binomial test (Table 3). The number of subjects with an anterior open bite (AOB) was significantly increased ($P = 0.016$) in the OSA group when compared with the controls (Table 3). The number of subjects with an AOB in the snoring group showed an increasing tendency when compared with the controls ($P = 0.063$; Table 3). The number of subjects with a Class II or asymmetric molar relationship was increased in OSA ($P = 0.013$) and snoring ($P = 0.004$) children compared with the control children (Table 3). A tendency for increasing maxillary ($P = 0.057$) and mandibular ($P = 0.077$) crowding was seen in the OSA children when compared with the control children (Table 3).

In the multifactorial analysis, the effects of age, gender, tonsillar size, and the dental arch variables were calculated on AHI. There were significant associations between mandibular crowding and AOB with AHI. There were more subjects with mandibular crowding ($P = 0.002$) and more with an AOB ($P = 0.019$) with increased AHI. There was also a tendency for the overjet to be larger with increasing AHI ($P = 0.069$).

Table 1 Mean values, standard deviations (SDs), and significances of the difference for dental arch measurements in children with diagnosed obstructive sleep apnoea (OSA), snoring children, and controls. The superscripts (A–C) refer to the table of comparison.

Variable (in mm)	Snoring group ^A ($n = 41$)	OSA group ^B ($n = 41$)	Control group ^C ($n = 41$)	Statistical significance		
	Mean (SD)	Mean (SD)	Mean (SD)	A–B	A–C	B–C
Overjet	3.7 (2.10)	3.5 (1.51)	2.6 (1.01)	—	*	*
Overbite	2.1 (1.47)	1.8 (1.81)	2.6 (1.00)	—	—	*
Upper arch length	30.4 (2.07)	30.2 (1.77)	30.5 (2.17)	—	—	—
W1 (maxilla)	30.4 (2.53)	29.6 (2.43)	31.2 (2.10)	—	*	**
W2 (maxilla)	30.4 (2.31)	30.1 (2.09)	31.3 (1.82)	—	—	*
W3 (maxilla)	34.2 (2.10)	34.3 (2.70)	35.5 (2.03)	—	*	*
W4 (maxilla)	38.7 (2.70)	38.7 (2.74)	40.0 (2.73)	—	—	*
Palatal height	12.2 (2.32)	12.4 (2.34)	12.4 (1.51)	—	—	—
Lower arch length	26.1 (1.42)	25.6 (1.29)	26.8 (1.44)	—	*	***
W1 (mandible)	24.9 (2.23)	24.5 (2.17)	24.4 (2.38)	—	—	—
W2 (mandible)	27.2 (2.08)	26.7 (1.88)	26.9 (1.64)	—	—	—
W3 (mandible)	30.2 (2.24)	29.7 (1.82)	30.2 (1.77)	—	—	—
W4 (mandible)	34.1 (2.23)	33.5 (2.40)	34.3 (2.15)	—	—	—

Statistically significant difference between the groups as determined by analysis of variance with Duncan's multiple comparison method, * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

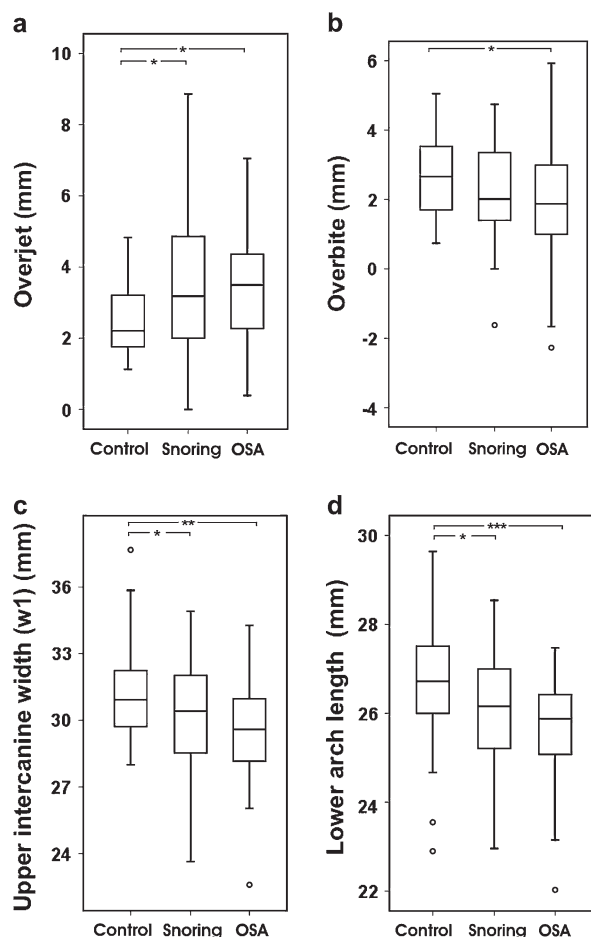


Figure 2 Box plot presentation of measurements of (a) overjet, (b) overbite, (c) maxillary intercanine width, and (d) mandibular arch length in control, snoring, and obstructive sleep apnoea (OSA) children and the significances of the difference (multiple comparisons test according to Duncan's method, $*P < 0.05$, $**P < 0.01$, $***P < 0.001$). Each box plot represents the median and 25th and 75th percentile.

Discussion

Dental arch dimensions are affected by a combination of both heritable and environmental factors. Occlusal variables have been reported to have low genetic contributions, and most of the variation in dental arch measurements may be explained by non-genetic causes (Harris and Smith, 1980; Harris and Johnson, 1991). The spectrum of environmental factors affecting the dentition is more complex and variable than was earlier recognized, and controversies exist concerning the importance of different factors (Mossey, 1999).

The present study indicates that children with sleep-related breathing disorders have differences in dental arch dimensions. Children with diagnosed OSA had a significantly narrower upper dental arch, increased overjet, reduced overbite, and shorter length of the lower dental arch when compared with the non-obstructed control children. This is in accordance with previous findings in 4-year-old children

Table 2 Prevalence of malocclusion types in obstructive sleep apnoea (OSA), snoring, and control children.

Type of malocclusion	OSA group (n = 41)	Snoring group (n = 41)	Control group (n = 41)
	%	%	%
Class II or asymmetric malocclusion	29.3	36.6	4.9
Deep bite	31.7	26.8	31.7
Anterior open bite	17.1	12.2	0
Lateral crossbite	12.2	12.2	2.4
Scissors bite	0	4.9	0
Maxillary crowding			
Mild	19.5	9.8	4.9
Moderate or severe	14.6	7.3	4.9
Mandibular crowding			
Mild	24.4	29.3	24.4
Moderate or severe	17.1	7.3	2.4

with breathing obstruction (Löfstrand-Tideström *et al.*, 1999). One of the main findings of the present study was that less serious nocturnal obstruction of the upper airway seems to have an association with developing dental arches. The results showed that snoring has a similar, albeit weaker, influence than OSA on dental arch morphology when compared with the non-obstructed control children. Dental arch dimensions in the snoring group tended to be between those of the OSA and control groups but importantly there was no significant difference between the snoring and OSA children.

The size of the adenoids has been demonstrated to be correlated with the severity of OSA, but no such association has been found between the size of the tonsils and increased AHI (Jain and Sahni, 2002). The present results also showed no significant relationship between the size of tonsils, as judged by clinical observation, and AHI in the groups of snoring and OSA children. Although the majority of children in the OSA and snoring groups had enlarged tonsils, the severity of the OSA was not associated with the absolute size of the tonsils. The relationship between the size of the adenoids and AHI was not supported by the findings since almost half of the children in the OSA group had a history of adenoidectomy. Fregosi *et al.* (2003) reported enlarged tonsils and increased soft palate behaviour in children with OSA, a finding which supports the hypothesis that the oropharynx might be the primary site of obstruction. It is obvious that enlarged adenoids may aggravate the disorder by interfering with nasal airflow. Hypertrophic tonsils, however, are not always obstructive, and children with normal oropharyngeal anatomy may suffer from OSA (Boudewyns and Van de Heyning, 1995). It is probable that the anatomical and physiological properties of the upper airway together with enlarged pharyngeal lymphoid tissue contribute to the degree of obstruction and the development of OSA (Ågren *et al.*, 1998; Isono *et al.*, 1998).

Table 3 Case control comparison of malocclusion types in obstructive sleep apnoea (OSA) ($n = 41$), snoring ($n = 41$), and control children ($n = 41$). Distribution of pairs in three paired samples divided into three categories: <, prevalence of malocclusion smaller in the first group; =, prevalence of malocclusion equal in both groups; and >, prevalence of malocclusion higher in the first group compared with the second group.

Type of malocclusion	OSA versus control group		OSA versus snoring group		Snoring versus control group	
	Paired sample		Paired sample		Paired sample	
	<=	P	<=	P	<=	P
Class II or asymmetric	2/27/12	0.013*	9/27/5	0.424	2/25/14	0.004**
Deep bite	11/19/11	1.000	6/27/8	0.791	10/23/8	0.815
Anterior open bite	0/34/7	0.016*	5/29/7	0.774	0/36/5	0.063
Lateral crossbite	1/35/5	0.219	5/31/5	1.000	0/37/4	0.125
Scissors bite	0/41/0	1.000	2/39/0	0.500	0/39/2	0.500
Maxillary crowding	3/27/11	0.057	6/22/13	0.167	4/30/7	0.549
Mandibular crowding	4/25/12	0.077	11/17/13	0.839	8/20/13	0.383

* $P < 0.05$, ** $P < 0.01$ two tailed; binomial test.

Even though there were no control children with adenotonsillar hypertrophy, the absolute size of the tonsils was not assessed in the control group, since the major aim of the study was to determine the relationship between the severity of nocturnal breathing disorder and dental arch morphology. Behlfelt *et al.* (1989) found significant differences in the dentition of children with enlarged tonsils compared with control children with ideal occlusion. They suggested that enlarged tonsils may give rise to obstruction of the oropharyngeal airway during sleep, which may lead to mouth breathing and postural changes of the mandible, the head, and the tongue. The present results can also be explained by the postural changes in the head, mandible, and tongue as a consequence of airway obstruction. Adenotonsillar hypertrophy has been shown to be related to an extended posture of the head, an antero-inferior posture of the tongue, and a downward inclination of the mandible (Adamidis and Spyropoulos, 1983; Behlfelt, 1990).

Maxillary arch width was constricted in both OSA and snoring children, probably due to mouth breathing and an altered position of the tongue, which changes the muscular balance between the tongue and the cheeks. It is interesting that the prevalence of lateral crossbites was not increased in obstructed children. A narrower upper dental arch may also be explained by the sleeping position. It has been suggested that a supine sleeping posture is related to AHI (Cartwright, 1984) and that a prolonged supine sleeping position correlates with reduced intermaxillary width (Pirilä *et al.*, 1995).

The decrease in the length of the lower arch and mandibular crowding may be explained by increased lip pressure affecting the lower arch as a consequence of a change in posture of the mandible. Extension of the head and an altered mode of breathing have also been reported to increase lip pressure (Hellsing and L'Estrange, 1987). Extended craniocervical posture has also been found to be associated with anterior crowding of the upper and lower dental arches (Solow and Sonnesen, 1998).

The selection of non-obstructed controls was not based on occlusal criteria, since malocclusions are relatively common in children. The purpose was to compare the occlusal variables of children with sleep-disordered breathing with normal controls, not those with an ideal occlusion. The prevalence of a Class II or asymmetric molar relationship was increased in the snoring and OSA children when compared with the non-obstructed controls. A retrognathic mandible, which is often related to Class II malocclusions, has often been reported in conjunction with obstructed sleep in children (Guilleminault and Stoohs, 1990; Shintani *et al.*, 1997; Kawashima *et al.*, 2002) and has been regarded as a predisposing factor for the development of OSA. This assumption is supported by the fact that craniofacial skeletal variables, such as mandibular retrognathia, are suggested to have moderate to high genetic contributions (Harris and Johnson, 1991). On the other hand, occlusal variables, such as molar relationship, have low familial correlations (Harris and Johnson, 1991). Thus, the increased number of patients with a Class II malocclusion may also be explained by environmental factors. For instance, it has been suggested that prolonged head extension may be associated with the development of a Class II malocclusion (Schwarz, 1928). An increased overjet in the OSA and snoring subjects can be explained by the increased number of subjects with a Class II malocclusion in both groups when compared with the control subjects.

An interesting finding was that mandibular crowding and AOBs were significantly associated with increased severity of OSA, as expressed by AHI. Lower arch crowding and AOB in snoring children are more suggestive of OSA than, for instance, the size of the tonsils. AOBs have earlier been reported in children with enlarged tonsils (Behlfelt *et al.*, 1989; Hultcrantz *et al.*, 1991). Cephalometric studies have revealed that the mandible is posteriorly inclined and lower anterior face height increased in children with OSA (Kawashima *et al.*, 2002; Zettergren-Wijk *et al.*, 2006),

which are typical findings in subjects with a skeletal open bite.

There has been criticism concerning the strength of evidence between nasal obstruction and dentofacial growth (O’Ryan *et al.*, 1982; Vig, 1998), mostly because there has been a lack of objective clinical methods to assess nasal obstruction, with emphasis being on inaccurate subjective methods. Clinical examination may not reveal OSA, and these children may have normal respiratory findings when observed during the day and when in an upright position. The diagnosis of OSA is established by overnight laboratory findings (Boudewyns and Van de Heyning, 1995). The present findings are in agreement with previous studies on the effects on impaired nocturnal breathing on dental arch morphology (Ågren *et al.*, 1998; Löfstrand-Tideström *et al.*, 1999).

The polysomnographic device used in this study meets the minimum requirements of the consensus statement of the American Thoracic Society (1996). Since eye movements, electroencephalographic and electromyographic activity were not recorded, a full-scale polysomnography was not performed. In many studies, the same recording system has been called an overnight cardiorespiratory polygraphy (de Miguel-Diez *et al.*, 2003). It has been validated in relation to the real polysomnography and also used largely to define sleep apnoeas in instances where the effect of these on sleep structure has not been examined (de Miguel-Diez *et al.*, 2003). The disadvantage of the full-scale polysomnography is that it is relatively invasive and may disturb the child’s natural sleep.

It has been shown that obstructive symptoms as reported by the parents are important risk factors for OSA even though polysomnography is still needed to separate primary snoring children from those with OSA and to determine the severity of the disease (Nieminen *et al.*, 1997). The number of obstructive apnoeas in especially younger children has in many studies been shown to be normally below one (Carroll and Loughlin, 1992; Marcus *et al.*, 1992; Rosen *et al.*, 1992; Nieminen *et al.*, 1997). For this reason, an AHI value of 1 or more was considered abnormal in the present study. The mean AHI in the OSA group was 3.5, indicating that the majority of the OSA children had mild OSA.

Conclusions

The results indicate that disturbed nocturnal breathing has significant effects on developing dental arches, which supports previous findings. It is important that snoring children were found to have similar and significant differences in dental arch measurements when compared with the non-obstructed controls, although the differences were not as remarkable as in OSA children. The findings in dental arch dimensions can be explained by long-term postural changes of the head, mandible, and tongue as a consequence of insufficient airway capacity. Snoring and OSA lie on a continuum of obstructive breathing disorders,

which explain the tendency to similar changes in dental arch development, correlating with the severity of obstruction.

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