European Journal of Orthodontics 28 (2006) 319–326
doi:10.1093/ejo/cji119
Advance Access publication 28 April 2006

Changes in dentofacial morphology after adeno-/tonsillectomy in young children with obstructive sleep apnoea—a 5-year follow-up study

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SUMMARY The aim of this study was to compare a number of dentofacial variables and airway space in children suffering from obstructive sleep apnoea (OSA) syndrome with the corresponding variables in control children exhibiting a normal breathing pattern, to study the development of these variables prospectively over a 5-year-period following treatment for OSA, and to compare the recorded changes with the corresponding changes occurring in the controls.

The subjects were 17 children (10 boys and 7 girls, mean age 5.6 years) diagnosed with OSA syndrome. The treatment for the OSA was adeno-/tonsillectomy. The control group comprised 17 age- and gender-matched children (mean age 5.8 years) without breathing problems. Lateral cephalograms were taken of the OSA children at baseline and then at 1, 3, and 5 years post-treatment. The control records comprised registrations at baseline and then after 1 and 5 years.

In comparison with the controls, the OSA children exhibited a more posteriorly inclined mandible ($P < 0.05$), a more anteriorly inclined maxilla ($P < 0.001$), a greater lower anterior face height ($P < 0.01$), a shorter anterior cranial base ($P < 0.01$), retroclined upper and lower incisors ($P < 0.05$ and $P < 0.01$, respectively), reduced airway space ($P < 0.05$ and $P < 0.01$), and a less pronounced nose ($P < 0.05$). At 5 years post-treatment, there were no statistically significant differences between the groups except for the lengths of the anterior cranial base and the nose which were still shorter ($P < 0.05$) in the patient group.

OSA in young children has an unfavourable effect on the development of several dental and facial components. However, if OSA is diagnosed and treated at an early age, an almost complete normalization of dentofacial morphology may be achieved.

Introduction

Over the past few decades obstructive sleep apnoea (OSA) has become recognized as the most extreme variety of mouth breathing and snoring on the wide spectrum of symptoms of upper airway obstruction. The most common cause of OSA in children is adeno-/tonsillar hypertrophy. The degree of severity of the OSA is associated with the size of the hypertrophic adenoids (Jain and Sahni, 2002). However, a similar correlation cannot be demonstrated with regard to the size of the tonsils (Ågren *et al.*, 1998; Jain and Sahni, 2002). On the whole, adeno-/tonsillectomy has a dramatically relieving effect on the obstructed breathing (Ågren *et al.*, 1998).

There are few previous investigations concerning the prevalence of OSA in children. A study from Iceland, however, indicates a prevalence of at least 2.9 per cent (Gislason and Benediktsdottir, 1995) while in a Swedish cohort study of the prevalence of breathing obstructions in 4-year-old children, a frequency of 0.9 per cent was recorded (Löfstrand-Tideström *et al.*, 1999). The peak incidence occurs between 3 and 6 years of age (Guilleminault *et al.*, 1981) and in pre-pubertal children both genders are equally affected (Carroll, 1996).

The criteria which should form the basis for the diagnosis of OSA in children are currently under discussion (Lim and McKean, 2003). Although parental anamnesis can be sufficient to confirm the diagnosis in a typical case, a polysomnographic sleep recording in a sleep laboratory is also recommended (Carroll, 1996; Ågren *et al.*, 1998; Mindell *et al.*, 1999; Marcus, 2001). However, such registrations are expensive and can be difficult to undertake because the situation in the laboratory is so unfamiliar for the patient. For this reason a less extensive sleep registration, including measurement of overnight oximetry and respiratory effort, is sometimes carried out at home. However, non-polysomnographic registrations do not give an adequate assessment of the problems since they do not quantify sleep disruption (Carroll, 1996).

In studies of facial morphology in adult apnoeics, it has been shown that the subjects exhibit extended head posture, lowered hyoid bone and tongue, increased lower anterior face height, retrognathic and posteriorly inclined mandible, and retroclined lower incisors (Lowe *et al.*, 1986; Andersson and Brattström, 1991; Solow *et al.*, 1996). Similar dentofacial effects have been observed in children with upper airway obstruction caused by enlarged adenoids

(Linder-Aronson, 1970), atopy (Hannuksela, 1981), allergy (Bresolin *et al.*, 1983), and hypertrophic tonsils (Behlfelt, 1990; Löfstrand-Tideström *et al.*, 1999).

It has been demonstrated that treatment of nasal obstruction in growing individuals results in a more normal pattern of dentofacial development (Linder-Aronson, 1972, 1975; Linder-Aronson *et al.*, 1986, 1993; Behlfelt, 1990; Woodside *et al.*, 1991). For this reason it would appear that there is a cause and effect relationship between nasal airway obstruction and dentofacial development, and that early treatment of children with OSA in order to normalize the mode of breathing is indicated.

In a previous study of young children with OSA syndrome, the effect of adeno-/tonsillectomy on obstructed breathing was described. One year post-operatively it was established that treatment of the breathing problem had been successful (Ågren *et al.*, 1998). Furthermore, a number of dentofacial variables which differed significantly between patients and controls before treatment showed a clear tendency to normalize post-operatively (Zettergren-Wijk *et al.*, 2002). However, the follow-up period was short and only a limited control group was available in that study.

The aims of the present study were:

1. to compare young children suffering from OSA syndrome with non-obstructed children, with respect to craniofacial morphology, soft tissue profile, and airway space;
2. to longitudinally evaluate the development of these structures after successful treatment of the OSA (adeno-/tonsillectomy), and to undertake a comparison with the normal development in non-obstructed children.

Material

This study was approved by the Ethical Committee of Huddinge University Hospital.

The material comprised cephalometric records of 17 Swedish children (10 boys and 7 girls) with OSA syndrome. The age distribution of the subjects is shown in Table 1. The diagnosis of OSA had been established by polysomnographic registration during a whole night's sleep in a sleep laboratory (Ågren *et al.*, 1998). Furthermore, all patients had enlarged tonsils and/or adenoids, and were to undergo tonsillectomy and/or adenoidectomy as the treatment procedure.

The control group comprised the lateral cephalograms of 17 age- and gender-matched children. The dental ages were also matched with one exception. Eleven of these children were Swedish and had been examined by an otolaryngologist who reported no signs of obstructed upper airways. The remaining six children in the control group were selected from a longitudinal cephalometric growth study (Bhatia and Leighton, 1993). This material did not contain any subjects with facial deformity or severe malocclusion.

Among the OSA children, one child had been a finger sucker. In this patient, however, the habit had ceased before the start of the study. Twelve children in this group had been dummy suckers, but the sucking habit had ceased at least 1 year before they entered the investigation. Among the controls, five children were finger suckers when entering the study, and 1 year later three of them maintained this habit.

The cephalographic records in the patient group comprised registrations made at baseline before surgery, and then at 1, 3, and 5 years post-operatively. With the exception of the 3-year registration, corresponding registrations were available for the control group.

At baseline, the mean ages in the patient and control groups were 5.6 and 5.8 years, respectively. This means that primary teeth were exfoliated and the permanent teeth erupted in most of the children during the period of the investigation. As a consequence, a number of observations of variables which included the incisors, the marginal bone, and points A and B could not be made in some of the matched pairs during this transitional stage. In these subjects, therefore, the results of the analyses were based on fewer than 17 measurements. A similar problem applied to the registration of soft tissue point A, which could not be reliably identified on two pairs of radiographs.

Methods

The OSA diagnosis was based on a typical history of sleep apnoea which was subsequently verified by polysomnographic registration (Ågren, 1997; Ågren *et al.*, 1998).

Table 1 Age distribution in the patients and controls at the different time points.

Group	Baseline		1-year follow-up		3-year follow-up		5-year follow-up	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Patient	5.6	1.34	6.8	1.45	9.0	1.40	10.9	1.37
Control	5.8	1.40	6.8	1.41			10.7	1.43

Mean age and standard deviation (SD) in years.

On the basis of the detailed anamnestic and diagnostic records in the patient group, it was decided that adeno-/tonsillectomy was the only plausible treatment. It has previously been reported that the status in 70–80 per cent of children with OSA improves after adeno-/tonsillectomy (Shintani *et al.*, 1998; Ward and Thornton, 2002). In the present study 13 patients underwent adeno-/tonsillectomy, three patients tonsillectomy, and one patient adenoidectomy (Ågren *et al.*, 1998). The surgery was successful in all patients and resolved the OSA, a fact which was verified with a reduced overnight sleep registration 1 year after surgery (Ågren *et al.*, 1998).

The cephalometric analyses carried out were based on linear and angular measurements which had been obtained from standardized cephalograms. The cephalometric reference points and lines used are shown in Figures 1 and 2.

The radiographs were taken at three different clinics (the enlargement factors were 6.4, 6.5, and 7.8 per cent, respectively), hence all linear measurements were adjusted for enlargement.

Analysis of the cephalograms

Four cephalograms were available for each individual in the patient group, and three cephalograms for each individual in the control group. A tracing film was placed on the first cephalogram and the points sella, nasion, porion, and orbitale were marked. The Frankfort Horizontal (FH), the perpendicular line to the FH through sella (FHP), the anterior portion of the sella turcica, and easily identified structures of the anterior cranial base were then drawn on the film. A new tracing film was then placed on top of the first and the above-mentioned lines and structures copied. The second tracing film was thereafter placed on the 1-year follow-up cephalogram, and after having been accurately orientated according to the anterior portion of the sella turcica and the cranial base it was secured into position. This procedure was repeated for the 3- and 5-year cephalograms. The reference points were marked on each tracing film and recorded with a digitizer (AccuGrid, Numonics Corp., Montgomeryville, Pennsylvania, USA), which was on-line with a computer and had an accuracy of ±0.1 degrees and ±0.1 mm.

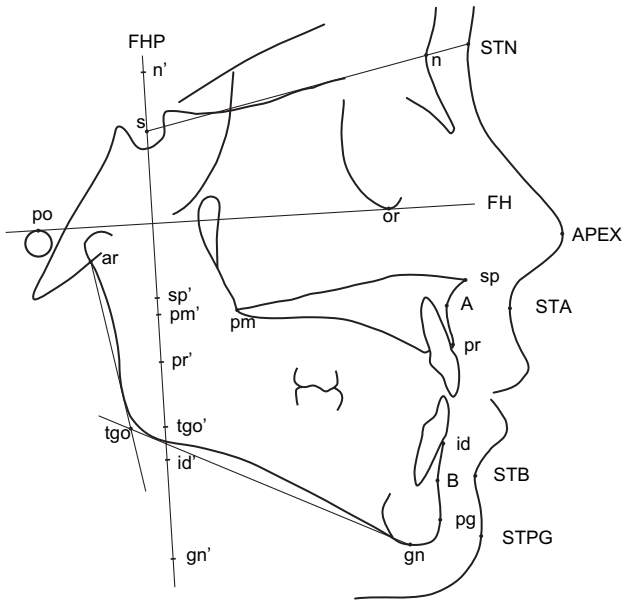

Figure 1 Reference points and lines on lateral cephalograms. The definitions of reference points and the Frankfort Horizontal (FH) are those given by Björk (1960). FHP, the perpendicular line to FH passing through the sella point; STN, soft tissue nasion, the intersection of the nasion–sella line and the contour of the soft tissue profile; APEX, the tip of the nose; STA, soft tissue point A; STB, soft tissue point B; STPG, soft tissue pogonion.

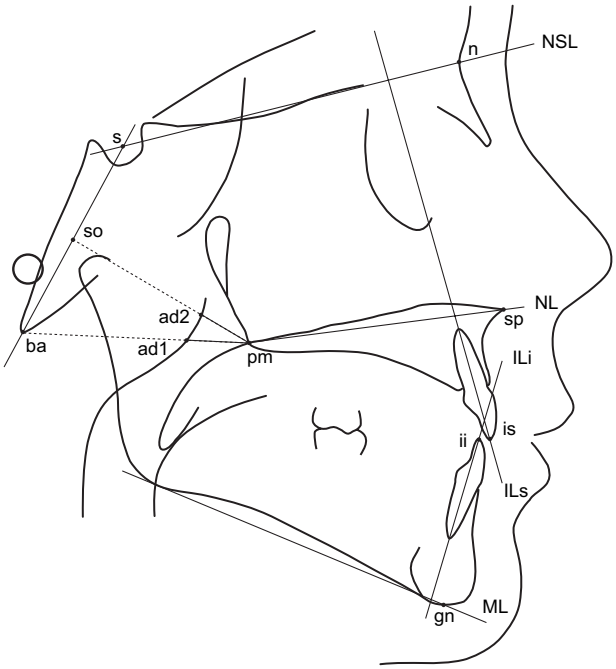

Figure 2 Reference points and lines on the cephalograms. Definition of dental and skeletal reference points and lines are those given by Björk (1960). The points used for measurement of the pharyngeal airway space (ad1, ad2) have been defined by Linder-Aronson and Henriksson (1973).

Measurements on cephalograms

The variables included in the study are presented in Tables 2 and 3.

All skeletal, dental, and soft tissue horizontal dimensions (in millimetres) were measured from the reference points to their respective perpendicular projections on the FHP.

Vertical dimensions (in millimetres) were measured as the distance between the reference points projected on the FHP (Figure 1).

The reference lines which were used for angular measurements (in degrees) are shown in Figure 2. This

Table 2 Cephalometric angular (degrees), linear (mm), and ratio (per cent) variables at baseline values in patients and controls, mean ages 5.6 and 5.8 years, respectively.

Variable	*n*	Patients		Controls		Patients versus controls		
		Mean	SD	Mean	SD	Mean diff	SD	*P* value
Angular								
ML/NSL	17	38.0	4.41	33.5	4.42	4.5	6.50	0.012*
NL/NSL	17	4.2	2.12	6.7	2.19	−2.5	2.48	0.001***
ILs/NL	16	94.5	7.64	99.3	8.38	−4.8	8.66	0.044*
ILi/ML	11	83.6	7.70	89.6	4.74	−6.0	6.17	0.009**
Linear (skeletal)								
A–FHP	16	59.4	3.21	59.8	2.41	−0.4	4.01	0.718
B–FHP	16	53.5	3.59	54.5	3.75	−1.0	4.86	0.429
pg–FHP	17	51.4	4.73	53.7	4.89	−2.3	5.67	0.121
n–FHP	17	58.1	2.49	59.6	2.26	−1.5	2.17	0.009**
n′–sp′	17	38.6	2.95	39.7	3.25	−1.1	2.90	0.114
sp′–gn′	17	53.4	3.57	50.9	3.12	2.5	4.68	0.047*
n′–gn′	17	92.0	5.49	90.7	4.98	1.3	5.56	0.350
sp′–pr′	15	13.9	2.22	13.8	2.55	0.1	2.25	0.770
id′–gn′	9	21.8	1.72	21.6	2.56	0.2	2.74	0.769
s–pm′	17	32.6	2.06	32.0	2.04	0.6	2.47	0.317
pm′–tgo′	17	24.3	2.93	26.6	2.86	−2.3	4.15	0.041*
s–tgo′	17	56.9	4.01	58.5	3.34	−1.6	5.06	0.192
Linear (soft tissue)								
ad1–pm	17	11.6	4.88	14.2	4.65	−2.6	4.37	0.026*
ad2–pm	17	8.4	2.16	11.0	2.84	−2.6	2.44	<0.001***
STN–FHP	17	66.1	2.75	66.7	2.19	−0.6	2.75	0.368
APEX–FHP	17	78.9	3.72	81.0	2.60	−2.1	3.41	0.025*
STA–FHP	15	69.2	3.46	70.3	2.66	−1.1	3.77	0.283
STB–FHP	17	62.2	4.50	62.3	4.24	−0.1	5.08	0.910
STPG–FHP	17	61.7	5.11	63.7	4.95	−2.0	5.78	0.176
Ratio								
(sp′–gn′/n′–gn′) × 100	17	58	1.95	56.2	2.25	1.8	2.87	0.018*

*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

figure also illustrates the variables ad1–pm and ad2–pm, which were used for assessment of the nasopharyngeal airway space (in millimetres).

Statistics

The statistical analyses were performed with a computerized statistical program (SPSS version 11.5 for Windows NT, SPSS Inc, Chicago, Illinois, USA). As the OSA children were gender and age matched with the controls, the material was regarded as a paired sample. The differences between the matched pairs at baseline and the 5-year follow-up registrations were tested for statistical significance with the paired *t*-test. The differences in development over the 5-year-period were tested for statistical significance (global *F*-test) using a repeated measures analysis of variance (ANOVA). When sphericity according to Mauchly's test was not obtained, a Greenhouse–Geisser correction (Geisser and Greenhouse, 1958) was carried out. When the developmental change of a variable was found to differ significantly between patients and controls, a *post hoc* multiple comparison test, including Bonferroni corrections, was performed. The significance level $P < 0.05$ was chosen.

Error of the method

For 24 cephalograms, all measurements were repeated at an interval of at least 3 weeks and the error of method was calculated (Dahlberg, 1940) using the formula:

$$s_i = \sqrt{\frac{\sum d^2}{2n}},$$

where s_i is the error of method, d is the difference between the first and second measurements, and n is the number of double determinations.

The greatest error of the method was found to be 0.86 mm (ad1–pm) for linear and 0.80 degrees (ILi/ML) for angular variables. With the exception of the measurement ad1–pm, the error variances of all variables were less than 3 per cent of the biological variances.

In order to determine the possible presence of systematic errors, significance tests of the mean differences ($\bar{d}$) were undertaken according to the formula:

$$t = \frac{\bar{d}}{\sqrt{\frac{\Sigma d^2}{n-1} \bullet \frac{1}{n}}}.$$

Table 3 Cephalometric angular (degrees), linear (mm), and ratio (per cent) variables at the five-year follow-up in patients and controls, mean ages 10.9 and 10.7 years, respectively.

Variable	*n*	Patients		Controls		Patients versus controls		
		Mean	SD	Mean	SD	Mean diff	SD	*P* value
Angular								
ML/NSL	17	34.4	4.91	31.9	4.97	2.5	7.62	0.184
NL/NSL	17	5.8	2.72	7.0	2.95	−1.2	3.49	0.182
ILs/NL	16	109.2	4.35	108.1	6.19	1.1	6.72	0.492
ILi/ML	11	91.9	6.54	91.9	7.79	0	8.35	0.999
Linear (skeletal)								
A–FHP	16	62.9	3.54	64.1	3.82	−1.2	5.19	0.348
B–FHP	16	59.0	4.47	59.7	5.32	−0.7	6.44	0.698
pg–FHP	17	58.9	5.05	60.2	6.32	−1.3	7.05	0.444
n–FHP	17	61.7	2.45	64.0	2.78	−2.3	3.17	0.008**
n′–sp′	17	45.2	3.20	46.1	2.87	−0.9	3.41	0.307
sp′–gn′	17	58.7	4.36	56.3	3.47	2.4	6.44	0.139
n′–gn′	17	103.9	5.37	102.4	4.77	1.5	6.53	0.343
sp′–pr′	15	13.2	2.17	12.8	1.85	0.4	3.14	0.612
id′–gn′	9	24.2	2.39	23.6	1.89	0.6	2.66	0.525
s–pm′	17	37.8	2.15	37.6	2.15	0.2	2.80	0.805
pm′–tgo′	17	29.2	3.36	29.4	3.37	−0.2	5.27	0.906
s–tgo′	17	67.0	4.06	67.6	4.36	−0.6	5.03	0.639
Linear (soft tissue)								
ad1–pm	17	17.5	4.43	19.7	5.46	−2.2	7.25	0.215
ad2–pm	17	14.3	3.35	15.4	3.99	−1.1	5.31	0.403
STN–FHP	17	70.2	2.80	71.3	2.90	−1.1	3.17	0.145
APEX–FHP	17	87.2	4.04	89.7	3.97	−2.5	4.63	0.045*
STA–FHP	15	76.0	3.59	76.5	4.31	−0.5	5.97	0.786
STB–FHP	17	68.7	5.24	68.5	5.56	0.2	6.20	0.896
STPG–FHP	17	69.7	6.51	70.3	6.73	−0.6	7.62	0.776
Ratio								
(sp′–gn′/n′–gn′) × 100	17	56.5	2.54	55	2.04	1.5	3.69	0.113

*$P < 0.05$; **$P < 0.01$.

If $-2.07 < t < 2.07$, no systemic errors were considered to be present in the method ($P < 0.05$, Forsberg, 1976). All *t* values were found to be within this range.

Results

The skeletal and soft tissue baseline data are shown in Table 2.

The inclination of the mandible and maxilla differed significantly between the patients and controls. In the patients, the mandible was more posteriorly inclined ($P < 0.05$) whereas the maxilla was more anteriorly inclined ($P < 0.001$) compared with the controls. The relatively greater inclination of the mandibular plane in the patients was also reflected in the variables representing lower anterior (sp′–gn′) and posterior (pm′–tgo′) face heights. As compared with the corresponding dimensions in the control group, anterior face height was greater and posterior face height was smaller in the patients ($P < 0.05$). The anterior facial ratio (sp′–gn′/n′–gn′ × 100) was significantly greater ($P < 0.05$) in the patient group. There was also a significant difference in the length of the anterior cranial base (n-FHP) which was on average 1.5 mm shorter in the patients than in the controls ($P < 0.01$).

The upper and lower incisors were more retroclined in the patients than in the controls ($P < 0.05$ and $P < 0.01$, respectively).

As regards the facial soft tissues (Table 2), the only difference between the groups was the position of the tip of the nose (APEX–FHP) which was slightly more advanced in the control subjects ($P < 0.05$).

The width of nasopharyngeal airways was evaluated with the variables ad1–pm and ad2–pm. Both measurements were significantly reduced in the patients ($P < 0.05$ and $P < 0.001$, respectively).

The mean values and standard deviations 5 years post-treatment are shown in Table 3. At this stage, only two significant differences were recorded between the patients and controls, namely the distances n–FHP and APEX–FHP which were on average 2.3 mm ($P < 0.01$) and 2.5 mm ($P < 0.05$) shorter in the patients.

The ANOVA test of the growth pattern over the 5-year period showed that development of the variables ML/NSL, ILs/NL, and ILi/ML differed significantly ($P < 0.05$)

between the patients and controls (Figure 3). The mandibular plane angle was reduced in both groups, but to a greater extent in the patients (−3.6 degrees compared with −1.6 degrees in the controls). This means that the difference between the groups was reduced to 2.5 degrees (N.S.) after 5 years. The inclination of the upper and lower incisors was approximately 5 degrees greater in the controls at baseline. At the 5-year follow-up, however, these variables were very similar in the patients and controls.

The pattern of change for all other variables was similar in the two groups.

Discussion

The typical feature of OSA is heavy respiratory labour caused by completely blocked or reduced air passage. During these so-called apnoeas or hypopnoeas, the respiratory muscle activity continues. According to Guilleminault *et al.* (1981) the severity of the respiratory obstruction in children may be reflected more often in respiratory labour than in the number of apnoeas/hypopnoeas or the degree of oxygen desaturation in the blood.

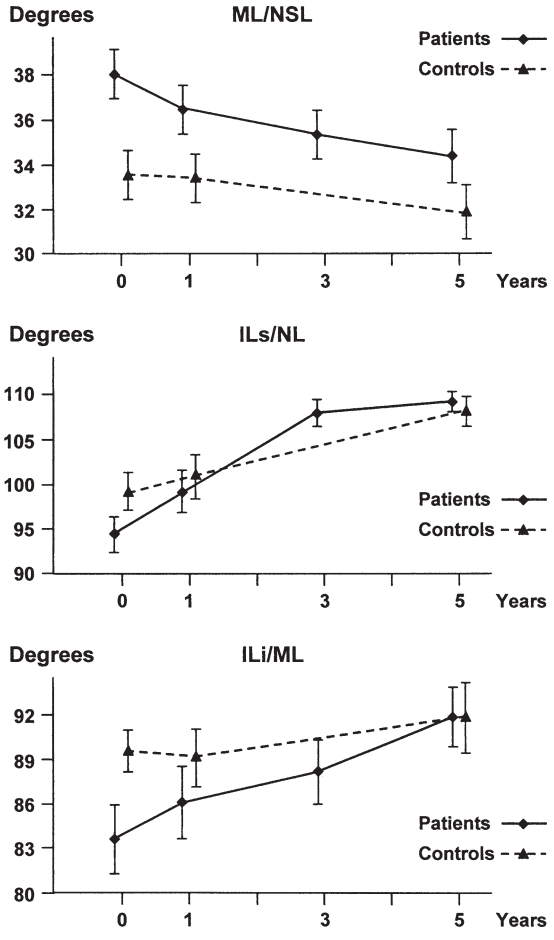

Figure 3 Diagrams illustrating the means, the standard errors, and the changes of the variables ML/NSL, ILs/NL, and ILi/ML during the 5-year follow-up in the obstructive sleep apnoea and control groups. In order to avoid overlapping of the error lines, the breakpoints of the patient curves have been slightly displaced to the left in relation to the corresponding breakpoints of the controls.

Face and neck muscles are normally inactive during rapid eye movement and slow-wave sleep. From video recordings, however, it could be assessed that the OSA patients in this study exhibited both mouth breathing and an extended head posture (Ågren *et al.*, 1998). It was not surprising, therefore, that the most striking results from the polysomnographic recordings were the comparatively high degree of obstructive breathing during a full night's sleep and an increased electromyographic activity in the neck and chin muscles (Ågren *et al.*, 1998). It has previously been stated that the neuromuscular response to the obstructive breathing is one important factor which may cause dentofacial changes (Linder-Aronson and Woodside, 2000).

The most common cause of OSA in children is enlargement of the lymphoid tissues in Waldeyer's ring. The adenoids reach their maximum relative size at 5 years of age (Linder-Aronson and Leighton, 1983). It seems logical, therefore, that the occurrence of OSA in children is most common in preschool ages (Guilleminault *et al.*, 1981; Mindell *et al.*, 1999). The mean age of the OSA children in the present study was 5.6 years at the time of surgery.

The patients and control children were closely matched with respect to gender and chronological and dental age. It proved impracticable to obtain a sufficient number of longitudinal cephalographic records of normal Swedish children which included ages as low as 3 to 5 years. It was therefore necessary to complete the control group with six records from a British longitudinal study. The Swedish children had undergone an examination by an otolaryngologist, and it had been established that they all had a normal breathing pattern and no airway obstructions. Such data were not available for the British children. There was, however, no cephalographic indication that the control children were affected by any breathing problems related to narrow upper airways.

The purpose of this investigation was firstly to study whether dentofacial morphology in children suffering from OSA differed in any respect from that of non-obstructed controls. Secondly, the dentofacial development in patients and controls was compared longitudinally following adeno-/tonsillectomy of the patients.

At baseline, dentofacial morphology in the patients showed statistically significant differences when compared with the controls. The mandible (ML/NSL) was posteriorly inclined, the maxilla (NL/NSL) was anteriorly inclined, lower anterior face height (sp′–gn′ and sp′–gn′/n′–gn′ × 100) was greater, posterior lower face height (pm′–go′) was smaller, and the incisors in the upper (ILs/NL) and lower (ILi/ML) arch were retroclined. With the exception of the anterior inclination of the nasal plane, all these characteristics are seen in older children with nasal obstruction of different aetiology, for

example, enlarged adenoids (Linder-Aronson, 1970), atopy (Hannuksela, 1981), allergies (Bresolin *et al.*, 1983), and enlarged tonsils (Behlfelt, 1990). It should be pointed out that the values for incisor inclination in the present study are based in some cases on primary teeth and on permanent teeth in others. With one exception, however, the individuals forming each pair had comparable dental status.

With regard to anterior inclination of the maxilla, it has been reported that finger- and dummy-sucking habits may result in anterior growth rotation of the nasal plane (Larsson, 1972, 1986, 1987). In the present investigation, none of the OSA patients exhibited any sucking habits at the start of the study. In the control group, on the other hand, at least five children had an ongoing finger- or dummy-sucking habit. Considering these facts, it seems logical to relate the reduced NL/NSL values in the OSA patients mainly to the breathing problem.

In the present study, there was no difference between patients and controls regarding maxillary or mandibular prognathism, either at baseline or 5 years post-treatment. This is in contrast to the findings of Shintani *et al.* (1998) who reported that 5- to 9-year-old OSA children had a more retrognathic maxilla and mandible (expressed as SNA and SNB) than age-matched controls. Retrognathism has also been demonstrated in 8-year-old children with nasal airway obstruction (Linder-Aronson, 1970). In adult apnoeics, a retrognathic maxilla and mandible is also a common finding (Lowe *et al.*, 1986; Andersson and Brattström, 1991). It could be questioned if the OSA children in this study would also have developed a more retrognathic facial type if they had been left untreated.

At the 3-year follow-up, cephalometric registrations were only available in the OSA group. Comparisons within this group showed that the favourable development observed at the 1-year follow-up had continued. However, the greatest changes took place during the first year after treatment, while the changes during the next 2 years were less extensive.

At 5 years post-treatment, the cephalometric values in the treated and control groups were similar. These results suggest that the normalization of the breathing pattern has a favourable effect on dentofacial development. Treatment of older children with airway obstruction also tends to lead to a normalization (Linder-Aronson, 1975) but not to the same extent as in these young OSA children.

The length of the cranial base (n–FHP) exhibited a statistically lower value in the OSA group. In contrast to other recorded differences, this variable still differed significantly between patients and controls at the 5-year follow-up assessment. A similar but not significant trend was reported by Linder-Aronson (1970) when comparing children with enlarged adenoids and control children without adenoids. In a study on adult apnoeics, Andersson and Brattström (1991) reported a shorter length of the anterior cranial base when compared with the controls.

The OSA children, not unexpectedly, exhibited narrower nasopharyngeal airways than the controls. At the 5-year follow-up, this difference was not significant. None of the patients exhibited any reported apnoea problems at this stage.

Regarding the soft tissue profile, the nose was less pronounced in the OSA patients than in the controls, both at baseline and at the 5-year follow-up. The reduced APEX–FHP distance in the patients could be a reflection of the comparatively short anterior cranial base dimension recorded in this group.

An interesting question is whether or not there is a risk that a relapse of the OSA will occur later in childhood or during adulthood. In general, this risk could be judged as being small. With increasing age, the nasopharyngeal lymphoid tissue decreases (Linder-Aronson and Leighton, 1983) while the size of the bony nasopharynx increases (Linder-Aronson and Henriksson, 1973; Linder-Aronson and Leighton, 1983). The combined effect of these changes should result in a reduction in nasal airway resistance and such a development could be expected to diminish the risk of future OSA and breathing problems. However, relapse of OSA may occur during adolescence due to hormonal factors (Guilleminault *et al.*, 1981), and the aetiology of OSA in adults is multi-factorial. It is not possible, therefore, to predict with any certainty whether or not OSA which has been successfully treated in early childhood will relapse during adolescence or adulthood.

Conclusion

This study has shown that:

1. young children suffering from OSA have a different dentofacial morphology to non-obstructed control children;
2. early treatment of OSA was successful and dentofacial morphology was normalized after adeno-/tonsillectomy;
3. it is important that children with OSA are diagnosed early and evaluated both from a medical and dentofacial point of view. This demands close co-operation between paediatricians, otolaryngologists, orthodontists, and paedodontists.

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Acknowledgements

The authors would like to thank Dr Britt Nordlander, Department of Oto-rhino-laryngology, Head- and Neck Surgery, Karolinska University Hospital, Solna; Professor

Eva Swanborg, Department of Clinical Neurophysiology, University Hospital, Linköping; and Dr Karin Ågren, Oto-surgery Clinic, University Hospital, Uppsala, for valuable advice and assistance. We would also like to express our gratitude to Mr Hans Högberg, Center for Research and Development, Uppsala University/County Council of Gävleborg, Gävle, for statistical guidance.

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