

Cervical headgear therapy as a factor in obstructive sleep apnea syndrome

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Abstract

Purpose: Obstructive sleep apnea syndrome (OSAS) has been a subject of increasing interest from the orthodontic point of view, but less attention has been paid to the possible influence of orthodontic treatment on its occurrence. The aim here was to study possible associations between the use of cervical headgear and nocturnal cessations of airflow and the severity of the latter.

Methods: The subjects were 30 children (12 boys, 18 girls, mean age 8.2, sd 1.61 years), divided into three groups: a group of 10 children undergoing headgear therapy, selected for this examination because of symptoms of OSAS while using headgear, an age-matched control group of 10 healthy children and a group of 10 with OSAS. Standard cephalograms of the headgear group prior to the orthodontic therapy and the corresponding cephalograms of healthy controls were analysed. A polygraphic (PG) sleep evaluation was used to assess the tendency for OSAS. Apnea and hypopnea periods were summated as apnea index (AI) and number of desaturations as desaturation index (ODI). All the subjects spent one night sleeping under laboratory conditions, those with orthodontic treatment spending the first half of the night with the headgear and the latter half without.

Results: The position of the mandible was found to be slightly more posterior in the headgear group than in the control group. The children in the headgear group were found to have significantly more apneal/hypopnea periods during the hours when the appliance was used, and the ODI-index showed increased values in this group.

Conclusions: We suggest that headgear therapy may contribute to the occurrence of sleep apnea, when a strong predisposition, such as mandibular retrognathia to the development of upper airway occlusion already exists. (*Pediatr Dent* 21:39–45, 1999)

Obstructive sleep apnea syndrome (OSAS), a condition involving repeated obstruction of upper airway accompanied by persistent respiratory efforts during sleep, has been studied extensively in recent years. OSAS may give rise to del-

eterious, even life-threatening complications.¹ Disabling psychological and social changes, like learning difficulties, behavioural disturbances and lethargy, may occur in children with serious OSAS.²

The diagnosis is made on the basis of laboratory findings of obstructive apneic episodes during sleep as recorded by an overnight polygraphic (PG) evaluation and clinical evidence of nocturnal symptoms of OSAS including heavy snoring, difficulty breathing and possible respiratory pauses.³ Diurnal symptoms, such as excessive daytime somnolence, are difficult to diagnose in young children. Apnea index is used to describe the number of apneic events per hour of sleep. Currently, oxygen desaturation indexes have increasingly been applied to express the severity of the disease.^{4,5}

The mechanism of obstruction is not completely understood, and there is some disagreement concerning the major site of airway closure.^{6,7} Etiologic and predisposing factors, apart from age, gender and body mass index (BMI) are still debated. Special attention has been paid to a possible causal etiologic relationship between the structure and sleep apnea.^{8–12} There are also some OSAS patients having no readily recognizable airway abnormality, and the etiology may remain obscure. It has been suggested, that patients with so-called idiopathic sleep apnea may have an anatomic predisposition to the development of the disorder that may not be detectable on clinical examination.¹³

Sleep apnea treatments are highly based on morphologic criteria, in other words it is assumed that the long-term resolution of OSAS will be gained by altering the structure. The role of dental personnel in the diagnosis and treatment of the patient with OSAS has recently been recognized. The upper airway can be reconstructed by the means of orthognathic surgery, a successful choice of treatment with many advantages.^{14,15} Different kinds of modified intraoral and functional

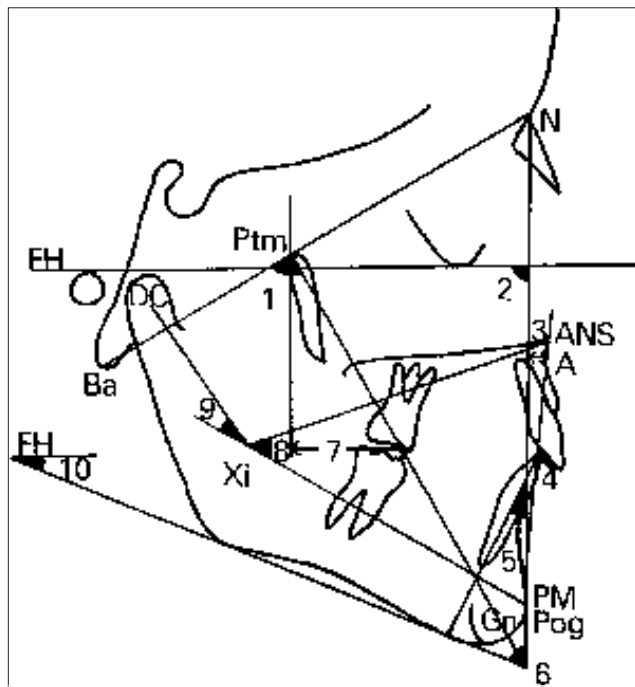


Fig 1. Cephalometric landmarks and measurements (Ricketts standard cephalometric analysis).

1. Facial axis (angle BaN-PtmGn lines).
2. Facial angle (angle FH-NPog anatomic porion).
3. Convexity of point A (mm distance NPog-APog at A).
4. Lower incisor to APog (mm distance APog-incisal edge).
5. Mandibular incisor inclination (NPog-incisor axis).
6. Facial taper (angle NPog-GoGn).
7. Upper molar to pterygoid vertical (mm distance mesiobuccal cusp 6 to vertical line from Ptm perpendicular to Frankfort plane).
8. Lower facial height (angle ANSXi-XiPM).
9. Mandibular arc (angle DCXi-XiPM).
10. Mandibular plane (angle FH-GoGn).

appliances have been used with varying success as treatments of OSAS. The success achieved with these appliances is usually attributed to increased airway space, stable anterior position of the mandible and the tongue and a forward movement of the soft palate.^{16, 17} Attention, however, has been focused on the possibilities of orthodontic therapy in the management of OSAS. There are no previous studies concerning the possible influence of orthodontic appliances on the occurrence of sleep apnea.

Cervical headgear is a removable appliance, which is widely used in orthodontic treatment especially in children. The major indications are crowding, Angle II molar relationship, anchoring and retention purposes. Headgear affects the antero-posterior position of the maxilla and it does not have a direct influence on the mandible. One reason for this study was that some parents of orthodontic patients expressed their concern about their children having evident apneic symptoms after the initiation of headgear therapy.

The aim was to study possible associations between the use of cervical headgear and nocturnal cessations

of airflow and the amount of the latter in children with OSAS symptoms.

Methods

The sample consisted of 30 children (12 boys, 18 girls, mean age 8.2, SD 1.61 yrs) with normal weight divided into three groups; a group of ten children (mean age 8.8, SD 1.33 yrs) undergoing cervical headgear therapy, selected for this examination because of symptoms of OSAS while using headgear; a control group of ten healthy asymptomatic children (mean age 8.8, SD 1.26 yrs) and ten OSAS children (mean age 7.1, SD 1.71 yrs). All groups were matched for sex. The children in both control groups had normal occlusions and no active orthodontic treatment. The children wearing the cervical headgear were sent by their own orthodontists to the Oulu University Hospital for further investigations due to the apneic symptoms while using the headgear.

Standard cephalograms were taken of the headgear group children before the initiation of the orthodontic therapy and analysed by using the standard Ricketts cephalometric analysis.^{18, 19} The control group was an age and sex matched group of healthy children (mean age 8.5, SD 1.39 yrs) with normal occlusion. For ethical reasons the control group for the cephalometric study was different from the control group used in the polygraphic study. All cephalometric landmarks (Fig 1) were located and digitized (Dentofacial Planner 7.0, Dentofacial Software Inc., Canada) by one investigator (KPP).

Parents responded to a questionnaire about their perception of the child's nocturnal sleeping and snoring habits, possible difficulties in breathing during sleep and daytime sleepiness. Patient history was also taken and medical and dental examinations were carried out.

Polygraphy (PG)

Overnight polygraphy was used to determine the incidence of breathing abnormalities and oxygen saturation. Children with orthodontic treatment spent



Fig 2a Polygraphic equipment for the recording of apnea periods during one overnight observation period.

TABLE 1. CEPHALOMETRIC VARIABLES IN HEADGEAR CHILDREN PRIOR TO ORTHODONTIC THERAPY AND IN HEALTH CONTROLS

	<i>Headgear with Healthy controls apnea symptoms</i>				
	<i>X</i>	<i>SD</i>	<i>X</i>	<i>SD</i>	<i>P</i>
1. Facial Axis	88.8	2.53	90.8	3.76	0.161
2. Facial Angle	82.3	3.76	86.7	3.91	0.050*
3. A Pt Convexity (mm)	5.3	2.79	4.5	3.08	0.546
4. Lower 1 to APog (mm)	1.2	2.33	0.8	2.71	0.666
5. Lower 1 inclination	21.0	5.00	20.3	8.12	0.666
6. Facial Taper	71.2	2.85	67.9	2.19	0.014*
7. Upper 6 to PTV (mm)	18.9	3.37	18.8	2.17	0.796
8. LFH Angle	45.7	3.17	45.3	2.92	1.000
9. Mandibular Arc	29.7	3.63	28.1	4.50	0.436
10. Mandibular Plane	25.6	3.19	25.5	4.4	0.136

* $P < 0.05$, Mann-Whitney U test.

the first half of the night with the cervical headgear and the latter half without it (Figs 2a and 2b). The recording time varied from 8 to 10 h depending on total sleeping time. PG recordings included arterial oxyhemoglobin saturation, oronasal airflow, thoracic respiratory effort, tibial muscle activity and the amount of time spent sleeping supine.

The following parameters of PG were calculated:

1. Apnea index (AI): the sum of apneas and hypopneas (at least 2/3 decrease in oronasal air flow signal) lasting ten seconds or more per hour
2. Number and duration of obstructive apneas: obstructive apnea indicating cessation of oronasal air flow with continued respiratory effort for ten seconds or more
3. Number and duration of obstructive hypopneas: obstructive hypopnea was determined as at least



Fig 2b Patients in the headgear group spent the first half of the night (4h) with the appliance and the latter half (4h) without.

2/3 decrease in oronasal air flow signal with continued chest wall motion lasting ten seconds or more

4. Number and duration of central apneas: central apnea was defined as a cessation of oronasal air flow with absence of chest wall motion for ten seconds or more
5. Number and duration of mixed apneas: mixed apnea was stated as a cessation in oronasal airflow signal lasting 10 seconds or more in the beginning of the apnea with absence of chest wall motion but in the latter part of apnea with respiratory effort
6. Total apnea time: the percentage of the registration time spent in apneas and hypopneas

7. Time per apnea/hypopnea episode: the mean time (in seconds) of the apneas and hypopneas
8. Oxygen desaturation indexes: ODI_4 describing the number of episodes of 4% or more decrease from the control level in silent rest and correspondingly ODI_{10} the number of desaturation episodes of 10% or greater decrease from the control level in silent rest
9. SAO_2 90–10%: oxygen saturation showing the difference between 90 and 10 percentile saturation values for the whole night
10. Mean value for oxygen saturation: the mean oxygen saturation of the night.

PG analysis was made by a neurophysiologist (UT) in order to assess the tendency for OSAS and their consequences. The neurophysiologist was not aware who belonged to the patient group and who was a healthy control. Although the analysis program included a possibility for automatic analysis of apneas and hypopneas, every apnea and hypopnea was manually checked. Apnea index and oxygen desaturation indexes were used to express the severity for sleep apnea. The reliability for the values of ODI_4 and ODI_{10} , which are also dependent on environmental factors was assured by the fact that all the measurements were done in the same laboratory under similar conditions.

Videotape recordings were made in order to study positional changes during the night. The recordings were analysed as described in our previous study²⁰ with special attention paid to the head and trunk postures.

TABLE 2. POLYSOMNOGRAPHIC VARIABLES IN TEN HEADGEAR CHILDREN WITH APNEA SYMPTOMS, TEN CHILDREN WITH DIAGNOSED OBSTRUCTIVE SLEEP APNEA SYNDROME, AND TEN HEALTHY CHILDREN

	<i>Controls with diagnosed apnea</i>		<i>Headgear with apnea symptoms</i>		<i>Healthy controls</i>
	<i>X±SD</i>	<i>P</i>	<i>X±SD</i>	<i>P</i>	<i>X±SD</i>
Apnea index	2.4±2.17	0.280	2.3±2.54	0.007 [†]	0.1±0.32
Number of apnea and hypopnea periods	20.6±21.39	0.352	22.4±24.27	0.003 [†]	1.6±1.90
Number of obstructive apnea periods	8.9±14.03	0.093	16.2±21.90	0.001 [†]	0.2±0.63
Number of central apnea periods	9.4±6.82	0.043 [*]	4.5±4.28	0.038 [*]	1.4±1.84
Number of mixed apnea periods	2.3±6.93	0.107	1.7±2.36	0.006 [†]	0.0±0.00
Total apnea time (%)	0.9±0.85	0.462	1.39±1.49	0.007 [†]	0.1±0.07
Time per apnea /hypopnea episode (s)	13.3±0.91	0.245	15.7±4.27	0.468	16.3±6.50
ODI ₄ /h	0.9±1.62	0.176	3.4±4.35	0.115	0.5±0.71
ODI ₄ /night	8.1±15.40	0.138	32.9±41.93	0.048 [*]	4.1±5.76
ODI ₁₀ /night	0.1±0.33	0.030 [*]	2.0±4.00	0.234	0.9±1.66
Total sleeping time (h)	8.9±0.72	0.054	9.4±0.73	0.457	9.4±0.67
Time spent sleeping supine (%)	35.3±26.07	0.386	26.7±13.58	0.066	57.2±48.90
SaO ₂ 90-10%	2.6±1.01	0.073	3.8±2.24	0.065	2.7±0.59
Mean value for oxygen saturation (%)	97.8±1.89	0.032 [*]	96.4±2.38	0.430	97.0±0.84

**P* < 0.05. †*P* < 0.01, Mann-Whitney U test.

Mann-Whitney U test and *t*-test for paired observations were used for statistical analysis of the data. The reason for using a nonparametric test, when comparing the headgear group with the control groups, was that the control groups differed from each other by their distribution. A parametric test was used to analyze intraclass differences between the first and the second half of the night after the normality of the data was first verified.

Results

When the cephalometric values were analysed, the variables indicating the position of the mandible showed that the mandible was more retrognathic in the headgear group. Facial angle was smaller in the headgear group (82.3° SD 3.91°) than in the controls (86.7°, SD 3.76°) (*P* < 0.05) and facial taper was larger in the headgear patients (71.2°, SD 2.85°) than in the control group (67.9°, SD 2.19°) (*P* < 0.05) (Mann-Whitney's U test) (Table 1).

Patients in the headgear group were found to have less central apnea periods than the control children with apnea (*P* < 0.05) whereas oxygen desaturation index, ODI₁₀ was increased (*P* < 0.05) in the children with headgear. There was a clear tendency for increased value in obstructive apnea periods in the headgear group when compared to the control sample of apnea children. The mean value for oxygen saturation was decreased in the headgear group (*P* < 0.05) (Mann-Whitney U test) (Table 2).

When comparing the headgear group with the healthy controls, significant differences were found. In the headgear group there were increased values in apnea indexes (*P* < 0.01), obstructive (*P* < 0.001), central (*P* < 0.05) and mixed apnea periods (*P* < 0.01), total apnea time (*P* < 0.01), oxygen desaturation index, ODI₄ (*P* < 0.05) when compared to the healthy controls (*P* < 0.05) (Mann-Whitney U test) (Table 2).

The occurrence of apnea and desaturation periods were compared between the first and the second half of the night. The children in the headgear group were

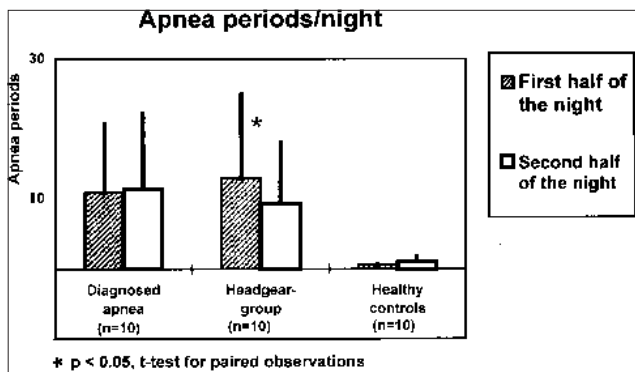


Figure 3. The total count of hourly apnea periods shown as intraclass differences between the first and the second half of the night. The patients in the headgear group were not awakened when the appliance was removed (t-test for paired observations).

found to have more apnea and hypopnea events during the first half of the night, when the appliance was in use (13.0, SD 12.74) than during the second half of the night without the appliance (9.4, SD 9.25) ($P < 0.05$) (t-test for paired observations) (Fig. 3). No significant results were found in the distribution of desaturation periods between the first and the second half of the night.

Discussion

Upper airway obstructions with snoring and sleep apnea symptoms are seen in children of all ages, even though little is still known about their exact prevalence. Estimated frequency of significant sleep and breathing disorders is about 0.7% in 4–5 year olds.²¹ Any closer inquiry about the prevalence of OSA in the age range studied here was not found.

The true normal value for the apnea index in this age range is not well established, but according to the latest reports it approaches zero.²² This has been taken into consideration in our examination so that every index ≥ 1 was regarded as abnormal.

There was an intentional sampling bias in the headgear group. These were the children who did not have any clinical symptoms at all prior the beginning of the orthodontic treatment but after the initiation of the therapy while using headgear the symptoms of OSA occurred as observed by their parents. For this reason the existence of OSA at the initiation of the orthodontic treatment cannot be excluded, since no polygraphy could be performed in these children before the treatment. Therefore the group was chosen to serve as its own control. The comparison of the two parts of the night, one with and one without the appliance, was carried out for practical reasons, as a monitoring time of two nights could not be justified.

It has not been shown that the apnea index would normally be at the higher level during the first period of the night. In the present investigation apnea periods in the headgear group appeared more severe during

the hours when the appliance was used, i.e., during the first half of the night. The reason for that is not clear. It can be supposed that these investigated children have a predisposition to the development of OSAS, which is triggered off by an external factor such as cervical headgear treatment. This hypothesis raises two further questions: do these children have a common clinically detectable anatomical or degenerative susceptibility to the development of obstruction and which factor in headgear therapy may contribute to the occurrence of sleep apnea.

In the light of current knowledge OSAS appears to have a multifactorial etiology. Alterations in craniofacial form,²³ disturbance in the growth of the mandible,⁸ soft tissue abnormalities and discrepancy in muscle activities²⁴ have been reported concomitantly with sleep apnea. Micrognathia, retrognathia, macroglossia, nasal obstruction, vocal cord dysfunction, adenotonsillar hypertrophy, and decreased airway lumen are examples of the pathological changes, which may result in OSAS. The main cause of the obstruction in children is generally considered to be enlarged adenoids and tonsils.²⁵ Repeated upper airway infections in childhood have been considered as a predisposing factor in upper airway obstruction.²⁶

Cephalometric assessment has been widely used as an adjunctive procedure for identifying the patients at risk for OSAS.⁹ The present cephalometric examination indicated that children in the headgear group had more retrognathic mandibles than the control ones, which in itself may act as a predisposing factor for sleep apnea. Clinically seven out of ten children were found to have distal or cusp to cusp molar relationship. Class II occlusal relationship and crowding were the main reasons for beginning the headgear treatment.

Cephalometric comparative study of craniofacial and upper airway variables in adults has indicated that remarkable differences exist between the OSA patients and the control subjects as related to their skeletal subtype and gender.¹⁰ The most atypical structure has been found in OSA male patients with Class I occlusal relationship, while fewer craniofacial and upper airway abnormalities have been found in OSA patients with distal molar relationship. A quantitative and qualitative analysis of the literature, however, has recently revealed that most studies suffer from several methodologic deficiencies and therefore the etiologic association between craniofacial structure and OSAS remain unsupported by the literature.¹¹ Even though the present control sample was unmatched in terms of their craniofacial structure, this does not explain the fact that apnea periods were more severe in the headgear group during the first part of the night when the appliance was used.

Sleep posture has been, especially in adults, found to have a significant influence on sleep apnea severity; apnea index being twice as high when sleeping supine

than during the time spent sleeping in other positions.²⁷ It has been concluded on the basis of cephalometric and electromyographic studies that upper airway structure and muscle activity are essentially influenced by the body position. This postural effect was found to be different in patients with sleep apnea than in control subjects.^{10, 12}

We tried to account for these results by examining positional changes during the time spent sleeping with appliance. Head postures and body positions during sleeping were studied by using PG and video recordings. We expected, that during the appliance use, children would prefer sleeping predominantly in the supine position. However, it was found that they tended to sleep on their backs even less with the headgear than without it.

Some parents expressed their opinion that during the use of headgear the child tilted the head in a more downward position. Orthopedic intermittent cervical traction is known to be heavy, i.e., about 300–500 g on both sides, which was the force applied in this headgear group. We presume that this force has an effect not only on teeth, but that it also presses the nape of the neck and cheeks. It can be speculated that these children are likely to adopt a different head position to avoid the pressure on the nape of the neck caused by heavy traction. The changes in the head position may be involved in the occurrence of OSAS symptoms as the headgear alters the head posture to a more comfortable position—which may not be ideal for breathing function. Two children actually slept more head-flexed with the headgear than without it, but statistically significant differences were not found and we could not explain our results by differences in sleeping positions.

It is evident that headgear treatment does not directly cause the symptoms of OSAS, but at least in some cases contributes to them. It is reasonable to assume that an existing predisposition, which is not strong enough to cause a marked disorder by itself, combined with additional external factors, can produce clinically significant OSAS. Mandibular retrognathia has been shown to be a consistent predisposing factor in OSAS.¹⁰ Most of the children in the headgear group had a history of recurrent upper airway infections, and large tonsils were detected on clinical examination. Half of the group had a history of adenotomy because of loud snoring and about the same number of the children had established allergic rhinitis. All these manifestations can be regarded as predispositional factors to the development of OSAS.^{26, 27}

The dentist, who is responsible for orthodontic treatment, should be aware of the possible effect of such treatment on sleep apnea occurrence. Due to the multifactorial etiology of the syndrome, it will be difficult, on the basis of clinical examination, to exclude

those children particularly susceptible to the OSAS, prior to the appliance therapy. It normally takes some weeks before the patient becomes completely comfortable with the appliance, and clinical examination is advised after the initial weeks. Polygraphic recording is indicated, if a positive finding is suspected.

Conclusion

Based on the findings of this study, the following conclusions can be made:

1. Headgear therapy may contribute to the occurrence of sleep apnea especially in children susceptible to the disorder.
2. Cervical headgear therapy in children with retrognathic mandibles should be carried out with special care.
3. If a patient already has apnea symptoms, proposed cervical headgear treatment may aggravate the syndrome.

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