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Habitual snoring with and without obstructive sleep apnoea: the importance of cephalometric variables

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Abstract

Background **The obstructive sleep apnoea syndrome is characterised by an increased apnoea-hypopnoea index and a reduction in the minimal arterial oxygen saturation (SaO_2) values during sleep. The extent to which these variables can be predicted by cephalometric and otorhinolaryngological variables was tested.**

Methods **One hundred consecutive habitual snorers (84% male), with a mean (SD) age of 50·1 (10·1) years, were studied. The 45 patients with less severe sleep apnoea, with an apnoea-hypopnoea index of 10 or less (group A), were compared with the 55 with an index above 10 (group B).**

Results **Body mass index, some cephalometric variables, and some otorhinolaryngological variables differed significantly between group A and group B, in particular the soft tissue measures PNS-P (posterior nasal spine to palate), MP-H (mandibular plane to hyoid bone), degree of oropharynx stenosis, and tongue size. In a multiple regression correlation analysis MP-H, SNB (angle from sella to nasion to subspinale point), SNA (angle from sella to nasion to supramentale point), PAS (posterior airway space), tongue size, and body mass index contributed significantly to the equation explaining the severity of sleep apnoea. Nevertheless, these variables together explained only 33% of the variance of the apnoea-hypopnoea index in the total sample; they were more important for patients with moderate to severe stages of the disease.**

Conclusion **The lack of association between cephalometric variables and mild sleep apnoea suggests that the differences in these variables (soft tissue measures) may be the consequence rather than the cause of habitual snoring and the obstructive sleep apnoea syndrome.**

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Several studies have analysed the characteristics of patients with the obstructive sleep apnoea syndrome, relating clinical features to cephalometric and otorhinolaryngological variables.[1–6] Most of these studies have considered patients with overt disease—that is, patients with a high apnoea-hypopnoea index, frequent oxygen desaturation during sleep, and obesity (a body mass index over 30). These studies have reported either a large series of patients or a small number of patients with age matched controls; some have used multiple stepwise regression models to find which clinical polysomnographic and cephalometric variables are important in explaining the severity of the syndrome.

Quera-Salva *et al*[2] found body mass index and amount of stage 1 non-rapid eye movement (non-REM) sleep to be significant variables in explaining the severity of ventilatory disturbance and falls in oxygen saturation (Sa_{O_2}) during sleep. A cephalometric measurement, posterior airway space, was another important factor for the apnoea-hypopnoea index and forced vital capacity (FVC) was important for the fall in Sa_{O_2}. Among the cephalometric measurements reported by Bacon *et al*[3] there was a smaller sagittal dimension of the cranial base and a greater lower facial height in patients with obstructive sleep apnoea than in normal subjects. In addition, Partinen *et al*[4] showed that the body mass index and upper airway abnormalities, including the distance from the mandibular plane to the hyoid bone (MP-H) and the width of the posterior airway space, were important determinants of the apnoea-hypopnoea index and to a lesser degree oxygen desaturation in patients with severe sleep apnoea.

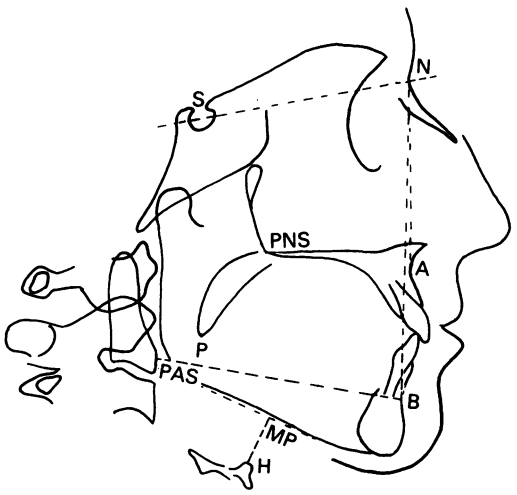
Recently Lyberg *et al*,[6] in a more detailed controlled cephalometric analysis of patients with severe sleep apnoea, found that soft palate length and tongue position may be important in explaining the severity of the disease and constitute an indication for surgery.

It is well known, however, that the severity of obstructive sleep apnoea (or heavy snorers' disease) varies, starting with a subtle and gradual change from heavy habitual snoring without apnoea or hypopnoea to overt and eventually severe sleep apnoea with pathological changes in respiration during all stages of sleep.[7,8] Factors that are important in the pathophysiology and prognosis of obstructive sleep apnoea should be apparent in the early stages of the illness and not only in the severe stages. So far there is little information on the cephalometric and otorhinolaryngological features of habitual snorers with few or no respiratory events (stage 0 of heavy snorers' disease[7]) or a mild degree of sleep apnoea.

We have investigated a large group of habitual snorers to compare the relation between body mass index, cephalometric variables (in particular soft tissue measures), and otorhynolaryngological characteristics.

Methods

We studied 100 consecutive patients who came



S—sella: the centre of the pituitary fossa; N—nasion; PNS—posterior nasal spine; A—subspinale: the most posterior point on a curve of the maxilla between the anterior nasal spine and the central incisor; B supramentale: the deepest point on the outer mandibular contour between mandibular incisor and pogonion; P—tip of soft palate; PAS—posterior airway space; MP—mandibular plane: a plane constructed from the gnathion through the gonion; H—hyoid: the most anterior-superior point on the body of the hyoid bone.

to the Milan sleep disorders centre for assessment of snoring. Subjects had to have been habitual, heavy snorers for at least three years. The occurrence of snoring was ascertained by interviewing spouses or household members; a *habitual snorer* was someone who snored every night.

INVESTIGATIONS

Each patient underwent:

Clinical and morphometric examinations with particular reference to weight history and specific symptoms, including wakening during the night with choking, breath holding, or pauses in respiration (noted by others), to verify the presence of sleep apnoea and the duration of snoring. The Quetelet or body mass index (= weight (kg)/height(m)^2; accepted normal values 19–25)[9] was calculated for each patient.

An otorhinolaryngological examination to assess the nose (nasal patency and septal deviation), oropharynx (clinical and direct pharyngoscopic examination), and base of the tongue (direct visual pharyngoscopy inspection of the lingual tonsil).

Lateral cephalometric radiography to measure the distance between bone landmarks, craniofacial angles, and dimensions of soft tissues or space widths. We used the technique of Riley *et al*[10]: the mandible was taken in the intercuspal position and the head in a natural posture with the aid of a cephalostat; a digital imaging technique was used to obtain more precise soft tissue measurements by a powerful contrast resolution of anatomical low photon absorption structures.

Nocturnal polysomnography for two consecutive nights with monitoring of the electroencephalogram (EEG), electrooculogram (EOG), electromyogram (EMG) of a submental and an intercostal muscle, and electrocardiogram (ECG) and of oronasal, thoracic, and abdominal respiration and ear lobe Sao_2, according to standard criteria[11]; recordings were continued for seven hours. All subjects slept for at least four hours.

MEASUREMENTS AND DEFINITIONS

Cephalometry The following variables were measured from standard cephalometric radiographs (figure): SNA (angle measurement from sella to nasion to subspinale point); SNB (angle measurement from sella to nasion to supramentale point); PAS (width of posterior airway space defined as the space behind the base of the tongue and limited by soft tissue); MP-H (distance from mandibular plane to hyoid bone); and PNS-P (distance from posterior nasal spine to the tip of the soft palate).

Otorhinolaryngology We used an arbitrary scale to measure the degree of obstruction at each of the following sites: NOSE (from 0—absence of obstruction—to 4—severe stenosis), OROPHARYNX, including uvula and pharyngeal mucosa (from 0—maximum patency—to 3—severe obstruction); and TONGUE (from 0—normal tongue and normal lingual tonsil—to 3—macroglossia and severe hypertrophy of lingual tonsil).

Polysomnography Standard definitions of EEG sleep variables, apnoea and hypopnoea, and oxygen desaturation were used; we also noted the frequency of sleep stage shifts per hour. Sleep was scored according to international criteria[12] and respiratory measurements were made according to Guilleminault *et al*.[11] The second night's recording was analysed.

The cephalometric measurements and the otorhinolaryngological examination were carried out without knowledge of the results of the polysomnographic recordings or the severity of obstructive sleep apnoea.

ANALYSIS

As many authors[13–15] consider more than 10 episodes of apnoea or hypopnoea per hour of sleep to be clinically significant, we divided our patients in two groups according to severity: a less severely affected group (A) with an apnoea-hypopnoea index of 10 or less and a more severely affected group (B) with an apnoea-hypopnoea index of more than 10. Group B contained a subgroup of patients with an apnoea-hypopnoea index above 30 and data on these were also analysed for comparison with other studies.[2–6]

Statistical differences were tested for significance by means of analysis of variance with appropriate checks for multiple comparisons and non-parametric distributions.

Multivariate approach With the apnoea-hypopnoea index and minimum Sao_2 as dependent variables, a multiple regression correlation analysis was used to test the relative importance of polysomnographic, cephalometric, and otorhinolaryngological variables, after age and body mass had been taken into account. A two stage procedure was used, including forced entry and backward/stepwise selection of explanatory variables. The variables and the entry order in the model were selected from published data as the basis of a possible explanatory model: age, body mass index, duration of habitual snoring, size of upper airway with soft tissue cephalometric measures, angle values, and otorhinolaryngological measures.

Two analyses were performed for each

dependent variable, with and without allowance for sleep variables (stage 1 and stage 3 and 4 non-REM sleep). At the end of the forced entry stage we ran the stepwise selection of the variables to assess the statistical significance of the model; default SPSS entry criteria were then used ($p > 0.10$ for removal and $p < 0.05$ for re-entry).[16]

We used the same four equation models for group A, group B, and the group B subgroup consisting of the most severely affected patients.

Results

General characteristics of the patients are shown in table 1. The mean age was 50 years, with a preponderance of men, who were overweight but not substantially obese. Forty five patients were in group A and 55 in group B; table 2 shows sleep, respiratory, and cephalometric results for the two groups. The mean (SD) values of the apnoea-hypopnoea index and of min Sao_2 (%) were 4·7 (2·4) and 89·5 (3·5) in group A and 31·3 (20·1) and 77·6 (12·5) in group B. Patients in group B (body mass index 29·2 (5·7)) were significantly more overweight than those of group A (body mass index 26·9 (4·0)) and had a longer history of snoring (17·2 (12·8) versus 12·3 (9·6) years: table 1). Polysomnographic data show that all patients slept relatively well, though patients in group B had more disrupted sleep with an increased percentage of stage 1 non-REM sleep and a lower percentage of stage 3–4 non-REM and of REM sleep; the number of stage shifts per hour was greater in group B.

Table 1 General characteristics of the two groups (mean (SD) values)

Group: Apnoea-hypopnoea index:	*All (n = 100)*	*A (n = 45) ≤10*	*B (n = 55) >10*	*p*
Age (years)	50·1 (10·1)	50·2 (9·2)	50·0 (10·9)	NS*
Body mass index	28·1 (5·1)	26·9 (4·0)	29·2 (5·7)	<0·05*
Snoring (years)	15·0 (11·7)	12·3 (9·6)	17·2 (12·9)	<0·05*
% Male	84·0	82·2	85·5	NS†

*Analysis of variance.
†χ^2 test.

Table 2 Polysomnographic and cephalometric measures (mean (SD) values)

Group:	*All (n = 100)*	*A (n = 45)*	*B (n = 55)*	*p*
POLYSOMNOGRAPHY				
(A + H)I	19·3 (20·0)	4·7 (2·4)	31·3 (20·1)	<0·001
Min Sao_2 (%)	83·0 (11·2)	89·5 (3·6)	77·6 (12·5)	<0·001
SE (%)	80·1 (13·1)	79·7 (13·1)	80·4 (13·2)	NS
Sleep stage				
1 non-REM (% TST)	12·8 (8·9)	9·9 (6·6)	15·1 (9·8)	<0·01
2 non-REM (% TST)	60·0 (8·5)	58·7 (8·3)	61·0 (8·5)	NS
3–4 non-REM (% TST)	10·7 (7·0)	13·4 (6·9)	8·5 (6·3)	<0·001
REM (% TST)	16·5 (6·3)	17·8 (6·1)	15·4 (6·3)	<0·05
St shifts (n)	64·8 (26·6)	56·0 (16·4)	72·0 (30·9)	<0·01
St shifts/h (n)	10·9 (5·1)	9·5 (3·0)	12·1 (6·0)	<0·01
CEPHALOMETRY				
PAS (mm)	9·9 (2·7)	10·1 (2·7)	9·7 (2·7)	NS
PNS-P (mm)	39·3 (4·5)	38·2 (3·7)	40·2 (5·0)	<0·05
MP-H (mm)	20·7 (6·7)	17·8 (5·9)	23·2 (6·5)	<0·001
SNA (°)	82·6 (4·0)	82·1 (3·5)	83·0 (4·3)	NS
SNB (°)	81·2 (4·1)	80·7 (4·1)	81·7 (4·0)	NS

*Analysis of variance. (A + H)I—apnoea-hypopnoea index; Min Sao_2—minimum arterial oxygen saturation; SE—sleep efficiency; TST—total sleep time; PAS—posterior airways space; PNS-P—posterior nasal spine to palate; MP-H—mandibular plane to hyoid bone; St shifts—sleep stage changes; St shifts/h—sleep stage changes per hour of sleep; SNA—sella-nasion-subspinal point; SNB—sella-nasion-submental point.

Cephalometry produced significantly greater values for PNS-P (40·2 (5·0) versus 38·2 (3·6) mm) and MP-H (23·2 (6·5) versus 17·8 (5·9) mm) in group B than group A; the other measures were similar in the two groups (table 2). When the values for the total sample were compared with reference values,[1] only MP-H was significantly longer in our patients (20·7 (6·7) versus 15·4 (3·0); $p < 0.01$).

Otorhinolaryngological results showed greater oropharyngeal obstruction and basal tongue hypertrophy in group B patients (table 3). Increased upper airways stenosis in the nose was observed in both groups, with 76 of the 100 patients showing septal deviation and scoring at least 2 on our arbitrary scale.

The first multiple regression analysis of the apnoea-hypopnoea index, which included measures of sleep architecture, showed that only MP-H, stage 3–4 non-REM sleep, and TONGUE had a significant effect and remained in the equation; these variables alone accounted for only 31% of the variance ($R^2 + 0.314$); the zero order correlation between body mass index and TONGUE was $r + 0.43$, and the selection criteria of our model excluded the former rather than the latter from the final stepwise equation.

When we considered min Sao_2 as the dependent variable, only MP-H, body mass index, and stage 3–4 non-REM sleep remained in the equation, accounting for only 19% of the variance ($R^2 = 0.197$).

When sleep EEG measures were excluded in the multiple regression analysis a similar percentage of the variance was explained (33% for apnoea frequency and 19% for lowest oxygen saturation). No difference was found between this model and the results of multiple regression analyses carried out on groups A and B separately.

Finally, in the group B subgroup of 21 patients with an apnoea-hypopnoea index above 30 the mean apnoea-hypopnoea index was 52·9 (15·8) and the mean min Sao_2 69·0% (15·7%), indicating severe obstructive sleep apnoea. The other anthropometric, polysomnographic, cephalometric, and otorhinolaryngological descriptive results did not differ statistically from those of group B as a whole. In the multivariate approach the variance of the apnoea-hypopnoea index and min Sao_2 was completely explained by all the variables considered.

Discussion

Our patients showed a wide range of severity of obstructive sleep apnoea. They had a mean body mass index of 28·1, and were therefore less overweight than subjects in some North American studies (body mass index 31·0 (SD 6·0))[4] and 31·1 (5·9)[2]) but similar to other European samples (BMI 27·9 (3·0)[15] and 26·9 (3·9)[14]).

Several factors are important in the pathogenesis of snoring and obstructive sleep apnoea. Patients in group B, with more abnormal sleep pattern and more disturbed respiration, were more overweight and had a longer history of snoring than those of group A; they

Table 3 Otorhinolaryngological variables (mean (SD))

Group:	All (n = 100)	A (n = 45)	B (n = 55)	p
Nose (0–4)*	2·3 (1·0)	2·5 (0·9)	2·2 (1·0)	NS
Oropharynx (0–3)*	1·6 (1·1)	1·3 (0·9)	1·9 (1·1)	0·01
Tongue (0–3)*	1·2 (1·1)	0·9 (0·9)	1·4 (1·2)	<0·05

*Ranges refer to arbitrary scales (see text; 0 = no observed obstruction).

also had significantly different cephalometric soft tissue measurements (PNS-P and MP-H), more severe oropharyngeal stenosis, and more pronounced macroglossia. But cephalometric soft tissue measures, facial angles, lingual tonsil hypertrophy, and body mass index explained only one third of the severity of obstructive sleep apnoea as judged by the apnoea-hypopnoea index. In the patients with a non-pathological number of episodes of apnoea or hypopnoea our analysis failed to find any explanatory variable for apnoea and hypopnoea or for Sao_2 among the factors we considered except for body mass index, which showed a significant though weak link with min Sao_2.

Our results do not seem to support the usual association between body mass index and obstructive sleep apnoea, except for subjects with the most severe syndrome. A direct measurement of local fat in the neck and the upper airways may have correlated better with obstructive sleep apnoea,[17] but this was not available. Increases in body fat around the neck may be more important than truncal obesity and this could explain the failure of the body mass index in general to contribute in our multiple regression model.[18]

In our patients nasal stenosis (septum deviation, turbinate hypertrophy, and nasal cavity restriction) may be important in determining snoring history: most patients had nasal stenosis, though this was independent of the severity of obstructive sleep apnoea. These data confirm those of De Berry-Borowiecki *et al*[5] in a smaller and ill balanced group of patients with different degrees of severity of obstructive sleep apnoea.

Cephalometric results similar to those of our group B patients were reported recently by Maltais *et al*,[19] who found significantly greater MP-H and PNS-P values in patients with mild to moderate obstructive sleep apnoea than in those who only snore or in normal control subjects. An important additional factor in their study was the positive correlation between MP-H and age. Unfortunately multiple regression analysis in the various groups was not carried out.

The PAS, SNA, SNB, and TONGUE variables, which correlated significantly with severity of obstructive sleep apnoea in our total sample, also had an explanatory role in the analysis of data on group B: this model explained less than 30% of the variance related to the variable apnoea-hypopnoea index, while only body mass index was significant in explaining min Sao_2. Some interpretations may be drawn from our results.

Firstly, the number, the type, and the entry order of the variables in the multiple regression analyses influence the explained variance of the apnoea-hypopnoea index. Including measures of sleep architecture can modify the result of the backward/stepwise procedure substantially. The conclusions of previous work in this field have therefore to be viewed with some caution.[1–6] Secondly, the proportion of variance explained by this kind of model is not great except for patients with the most severe disease. For the whole sample the unexplained variance has to be attributed to factors that we have not considered; these factors include other cephalometric bone measures.

The differences we saw between habitual snorers with an apnoea-hypopnoea index of 10 or less and with an apnoea-hypopnoea index above 30 suggest two possible hypotheses: (1) abnormal MP-H, PAS, PNS-P, and otorhinolaryngological variables and other factors tend to be found in patients with a long history of habitual snoring and obstructive sleep apnoea; these findings may be a consequence of having the severest stages of the disease. (2) Alternatively, patients with the most severe disease may have had more severe cephalometric abnormalities before the onset of obstructive sleep apnoea, and increasing weight, alcohol consumption, aging, or external factors act to precipitate the syndrome.

We think that the second explanation is less likely as the youngest and most severely affected patients also report very long histories of habitual snoring, and have very abnormal cephalometric soft tissue measurements. Obviously only a prolonged follow up of young habitual snorers without apnoea will establish whether the soft tissues of the pharynx have a pathogenic role in obstructive sleep apnoea.

Habitual snoring and obstructive sleep apnoea are likely to be two aspects of the same disease, which is a multifactorial problem having some morphological and functional cofactors that interact during the long course of the disease. We believe that soft tissue changes in the upper airways and the body mass index are important in causing the sleep apnoea syndrome but that other morphometric factors, such as distribution of fibre type in upper airway constrictor muscle (that is, medial pharyngeal constrictor muscle)[20] or structural modifications of pharyngeal tissues unrelated to body weight and documented by magnetic resonance imaging measurements,[21] are also important.

Craniofacial morphology, and skeleton landmarks in particular, may be important in the development of obstructive sleep apnoea, as documented by others,[3 5] and we are investigating this possibility.

In conclusion, our data suggest that some factors, such as body mass index, tongue hypertrophy, and several cephalometric measures (both soft tissue measures such as MP-H and PAS and skeletal measures such as SNA and SNB), may be important determinants of the severity of obstructive sleep apnoea. These variables explain only one third of the variance of the apnoea-hypopnoea index, however. Follow up studies can help to determine which elements are important in the onset of the disease and its progression and which are a consequence of it.

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