European Journal of Orthodontics 20 (1998) 133–143

Natural head posture, upper airway morphology and obstructive sleep apnoea severity in adults

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SUMMARY Enlarged tonsils, adenoids, and chronic respiratory problems have been associated with the compensatory adaptations of natural head posture (NHP) in children. Recently, it has been shown that adult patients with Obstructive Sleep Apnoea (OSA) also tend to exhibit a craniocervical extension (CCE) with a forward head posture (FHP). This study was designed to search for some characteristics of OSA patients that may be related to these adaptive changes in NHP. Overnight polysomnographic, demographic, and cephalometric records of 252 adult male subjects with various types of skeletal patterns and dental conditions were examined. Apnoea Index (AI) and Apnoea + Hypopnoea Index (AHI) variables were assessed to separate the non-apnoeic snorers (n = 35), and mild (n = 101), moderate (n = 63), and severe (n = 53) OSA groups. Results of the Tukey tests revealed that severe OSA patients had a greater tendency to exhibit a CCE with a FHP ($P \leq 0.05$ to $P \leq 0.001$). Differences in head extension (NSL.VER) between groups could not be identified. Pearson's 'r' correlation coefficients revealed that the CCE and FHP in OSA patients were associated with a higher disease severity, a longer and larger tongue, a lower hyoid bone position in relation to the mandibular plane, a smaller nasopharyngeal and a larger hypopharyngeal cross-sectional area, and a higher body mass index ($P \leq 0.05$ to $P \leq 0.001$). It is concluded that a CCE with a FHP is more likely to be seen in severe and obese OSA patients with certain morphological characteristics of the upper airway and related structures.

Introduction

Natural head posture (NHP) is the upright position of the head of a standing or sitting subject, while it is balanced by the post-cervical and masticatory-suprahyoid-infrahyoid muscle groups, with the eyes directed forward so that the visual axis is parallel to the floor. Generally, there is a consensus in the orthodontic literature that the individual variations of NHP are related to certain characteristics of the craniofacial structure (Solow and Tallgren, 1976; Marcotte, 1981; Cole, 1988; Solow and Siersbæk-Nielsen, 1992; Özbek and Köklü, 1993). However, the mechanisms responsible for the differences in NHP are not fully understood. It has been suggested that it is primarily controlled by the need to maintain a patent pharyngeal airway, and other guiding mechanisms such as sight, hearing and vestibular orientation, and mass and contour of the head (Bosma, 1963; Solow and Kreiborg, 1977; Solow and Greve, 1979; Woodside and Linder-Aronson, 1979; Vig *et al.*, 1980, 1983; Daly *et al.*, 1982; Solow *et al.*, 1984, 1993; Wenzel *et al.*, 1985; Fjellvang and Solow, 1986; Behfelt *et al.*, 1990; Tangugsorn *et al.*, 1995a). Experimental studies demonstrated an immediate head extension and changes in postural EMG activity in the craniofacial muscles following the obstruction of the nasal airways (Vig *et al.*, 1980; Hellsing *et al.*, 1986). Correspondingly, studies in children emphasized the role of enlarged tonsils and adenoids (Solow and Greve, 1979; Woodside and Linder-Aronson, 1979; Behfelt *et al.*, 1990),

and chronic respiratory problems such as asthma and perennial rhinitis (Wenzel *et al.*, 1985), in increased craniocervical extension (CCE = increase in craniocervical angulation by cranial extension and/or forward inclination of the cervical column).

Similar craniofacial structures both in growing subjects and in adults with CCE and forward head posture (FHP = forward positioning of the head mediated by a forward inclination of the cervical column) suggest that some of the trigger factors responsible for the adaptations of NHP in children may persist in adults. On the other hand, as the upper airway problems such as adenoids and/or tonsils, which are frequently observed in children, are less common in adults, further studies of the interaction between NHP and the morphology, and/or physiology of the upper airway in the adult population are needed.

Obstructive Sleep Apnoea (OSA) results from the repeated obstruction of the upper airway during sleep. Recently, Solow *et al.* (1993) and Tangugsorn *et al.* (1995a) demonstrated that OSA patients exhibited an extended and forward NHP when compared with control samples. This may be attributed to the compromised morphology, and/or physiology of the upper airway and related structures observed in OSA patients which persist to some extent even when they are awake (Haponik *et al.*, 1983; Hoffstein *et al.*, 1984; Suratt *et al.*, 1984, 1985; Brown *et al.*, 1987; Shephard *et al.*, 1990; Mezzanotte *et al.*, 1992; Tsuchiya *et al.*, 1992; Wasicko *et al.*, 1993; Lowe *et al.*, 1995; Schwab *et al.*, 1995; Tangugsorn *et al.*, 1995a,b). An evaluation of patients who may have different levels of disease severity, different NHPs, and different morphological and/or physiological characteristics of the upper airway and related structures, may increase our understanding of the factors associated with the individual NHP variations in adults. This may also enhance the sleep physician's understanding of the possible mechanisms that may be responsible for OSA in different patients. Hence, it was the purpose of this study to determine if the severity of this disease, obesity, and/or the cephalometric measurements of the upper airway, tongue, soft palate, and hyoid bone position are related to the individual differences in NHP of OSA patients.

Subjects and methods

The overnight polysomnographic records and NHP cephalometric films of 252 adult male subjects with various skeletal and dental conditions were used. All subjects had been referred to a sleep disorders centre to determine if they had OSA. Details of the polysomnographic procedure have been explained elsewhere (Pae *et al.*, 1994; Lowe *et al.*, 1995). The cephalometric films were taken using the same cephalostat (Counterbalanced Cephalometer Model W-105, Wehmer Co.), in the NHP, by the subject standing and looking straight into a mirror (Solow and Tallgren, 1971). The distance from the X-ray source to the median plane of the head was 165 cm and the median plane to film distance was 14 cm. No corrections were made for the radiographic enlargement. To enhance the outlines of the upper airway tissues, all subjects swallowed a spoonful of a radiopaque barium sulphate oesophageal cream (65 per cent W/W) to coat the dorsum of the tongue and the upper airway.

Apnoea Index (AI = average number of apnoeas/hour during sleep) and Apnoea + Hypopnoea Index (AHI = average number of apnoeas + hypopnoeas/hour during sleep), obtained from overnight polysomnography studies, were evaluated to create the non-apnoeic snorer, and mild, moderate, and severe OSA groups. Apnoea was defined as the cessation of breathing during sleep for 10 seconds or more. Hypopnoea was defined as a greater than 50 per cent decrease in airflow for 10 seconds or more. Of the 252 subjects who underwent overnight polysomnography, 35 subjects aged 23–71 years who were not diagnosed as having OSA comprised the non-apnoeic snorers group (AHI < 10 and AI < 5). Of the remaining 217 subjects who had been determined to have OSA, 101 had mild (aged 27–68 years, AHI = 10–30 or AI = 5–15), 63 had moderate (aged 18–69 years, AHI = 31–50 or AI = 16–25) and 53 had severe OSA (aged 20–72 years, AHI > 50 or AI > 25). In cases where AI and AHI variables suggested overlapping levels of disease severity, the patient was placed in the higher severity group. Therefore, a patient who had an AHI between 31 and 50 was accepted to have a moderate OSA, even if his AI was less

Table 1 Means and standard deviations of AI (Apnoea Index) and AHI (Apnoea + Hypopnoea Index) in four different groups.

	A = controls $n = 35$		B = mild OSA $n = 101$		C = moderate OSA $n = 63$		D = severe OSA $n = 53$	
	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD
AI	1.0	1.3	5.3	4.0	12.0	6.4	34.2	19.2
AHI	4.3	3.5	18.9	5.9	37.1	7.4	63.1	16.3

than 15. The mean values for AI and AHI in different groups are presented in Table 1.

Tracings of the cephalometric films were completed, and traditional contours and points were digitized (Lowe *et al.*, 1986; Tsuchiya *et al.*, 1992; Pae *et al.*, 1994).

Variables

See figure legends for definitions:

1. Natural head posture (NHP) variables (Figure 1). Traditional head and neck posture variables were used (Solow and Tallgren, 1971). The lower border of the films was accepted as the 'true horizontal' (HOR) reference line (at a right angle to the direction of gravity).
2. Upper airway, soft palate, and tongue variables (Figure 2).
3. Hyoid bone position variables (Figure 3).

The upper airway, soft palate, tongue, and hyoid bone variables have been defined previously (Pae *et al.*, 1994).

Obesity variables

1. BMI (Body Mass Index): this was calculated by dividing the weight (kg) by the stature squared (m^2; BMI = kg/m^2).
2. PPNC (percentage of the predicted neck size): neck circumference was measured at the level of the cricothyroid membrane. PPNC measurement (Davies and Stradling, 1990) provides a compensation for the increase in neck circumference by height [PPNC = (100 × neck circumference in mm)/{(0.55 × height in cm) + 310}].

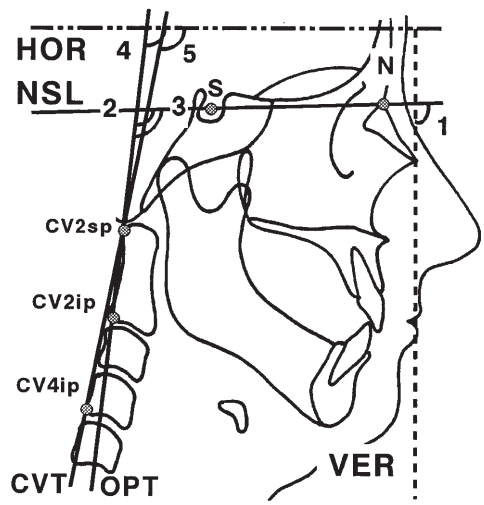

Figure 1 Natural head posture variables. Cranial extension/flexion: (1) NSL.VER; angle between nasion–sella line (NSL) and true vertical (VER = vertical to the lower border of the film and parallel to the gravity forces) reference line. Craniocervical posture: (2) NSL.OPT; angle between NSL and odontoid process tangent which passes through cv2sp (superior posterior point of second cervical vertebra) and cv2ip (inferior posterior point of second cervical vertebra). (3) NSL.CVT; angle between NSL and cervical vertebrae tangent which passes through cv2sp and cv4ip (inferior posterior point of fourth cervical vertebra). Increases in these angles result in a CCE. Cervical posture: (4) OPT.HOR; angle between odontoid process tangent and true horizontal reference line. (5) CVT.HOR; angle between cervical vertebrae tangent and true horizontal reference line. Increases in these angles result in a FHP.

OSA severity variables

AI, AHI, and $MinSaO_2$ percentage (the percentage of minimum oxygen desaturation during sleep).

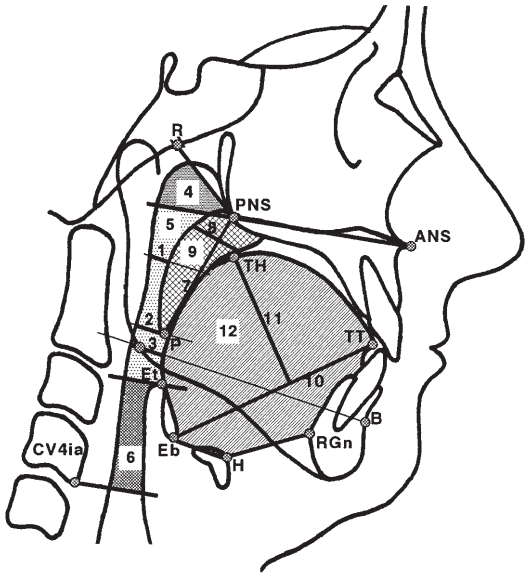

Figure 2 Upper airway, soft palate and tongue variables. (1) SPAS; superior posterior airway space. (2) MAS; middle airway space. (3) IAS; inferior airway space. (4) Naso area; nasopharyngeal airway cross-sectional area. (5) Oro area; oropharyngeal airway cross-sectional area. (6) Hypo area; hypopharyngeal airway cross-sectional area. (7) PNS–P; soft palate length. (8) MPT; maximum palatal thickness. (9) Soft P Area; soft palate cross-sectional area. (10) TGL; tongue length. (11) TGH; tongue height. (12) Tongue area; Tongue cross-sectional area.

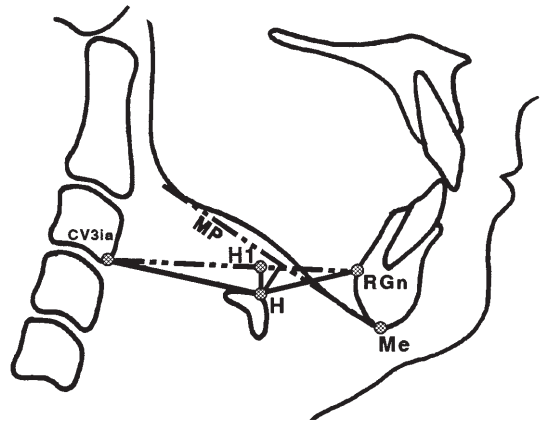

Figure 3 Hyoid bone position variables. H–MP; hyoid bone to mandibular plane distance. H–H1; vertical hyoid position. H–C3; hyoid bone to cervical column distance. H–RGn; hyoid bone to mandibular symphysis.

Statistical methods

Analysis of variance (ANOVA) was used to determine if the differences between NHP variables were statistically significant in the four groups. Tukey tests (Zar, 1984) were performed to calculate the level of OSA severity at which significant changes in postural variables occurred. Pearson's '*r*' correlation coefficients were used to find out if any cephalometric measurements, severity of OSA, and/or obesity may be related to changes in NHP of OSA patients.

Results

NHP and OSA severity (Tables 2–4)

Table 2 presents the Pearson's '*r*' correlation coefficients between NHP and OSA outcome variables in OSA patients. The extension of the head in relation to the true vertical reference plane (NSL.VER) did not show any significant correlations with the OSA variables. However, craniocervical and cervical posture measurements exhibited significant correlations with AHI ($P \leq 0.001$), AI ($P \leq 0.01$–0.001) and $MinSaO_2$ percentage ($P \leq 0.05$–0.01). Correspondingly, results of the variance analysis (Table 3) suggested that the differences of the means of craniocervical (NSL.OPT, NSL.CVT) and cervical (OPT.HOR, CVT.HOR) posture variables were statistically significant between the four groups ($P \leq 0.001$, $P \leq 0.05$), whereas the differences in head extension (NSL.VER) were not. Results of Tukey tests (Table 4) suggested that significant changes could be detected between moderate and severe groups for the variables NSL.OPT, NSL.CVT and OPT.HOR ($P \leq 0.05$, $P \leq 0.01$; Table 4), although the level of significance of the differences between mild and severe OSA groups was naturally higher ($P \leq 0.001$).

Upper airway dimensions (Table 5)

Only the hypopharyngeal airway cross-sectional area (Hypo area) was significantly correlated with all NHP variables ($P \leq 0.05$, $P \leq 0.001$). This reveals that a larger hypopharyngeal airway can

Table 2 Matrix of probabilities and Pearson's 'r' correlation coefficients between NHP and OSA variables.

	OSA patients									
	NSL.VER		NSL.OPT		NSL.CVT		OPT.HOR		CVT.HOR	
	r	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*
AI	0.080	NS	0.230	0.001	0.200	0.003	0.242	0.000	0.216	0.002
AHI	0.009	NS	0.224	0.001	0.224	0.001	0.253	0.000	0.234	0.001
$MinSaO_2$%	–0.117	NS	–0.183	0.009	–0.206	0.003	–0.146	0.039	–0.163	0.020

Table 3 Means and standard deviations of cranial extension (NSL.VER), craniocervical posture (NSL.OPT, NSL.CVT) and cervical posture (OPT.HOR, CVT.HOR) measurements, and variance analysis showing the level of significance of differences in NHP variables between the four groups.

	A = snorers *n* = 35		B = mild OSA *n* = 101		C = moderate OSA *n* = 63		D = severe OSA *n* = 53		
	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD	
NSL.VER	99.3	7.0	101.3	7.3	101.5	6.9	102.4	6.1	NS
NSL.OPT	101.8	7.0	103.1	8.0	103.3	8.7	108.4	7.8	0.000
NSL.CVT	107.0	6.8	108.2	7.4	109.4	8.1	113.1	7.2	0.000
OPT.HOR	93.0	6.9	91.8	7.5	92.4	9.3	97.1	7.6	0.001
CVT.HOR	98.2	6.4	97.1	7.1	98.4	8.3	101.7	6.6	0.003

Table 4 Tukey tests showing the level of significance of changes in NHP with increases in OSA severity. A = Non-apnoeic snorers; B = mild OSA; C = moderate OSA; D = severe OSA.

	A–B	B–C	C–D	B–D
NSL.OPT	NS	NS	0.003	0.001
NSL.CVT	NS	NS	0.043	0.001
OPT.HOR	NS	NS	0.009	0.000
CVT.HOR	NS	NS	NS	0.000

be expected in OSA patients who have a CCE in the NHP. A smaller airway size behind the soft palate (SPAS) was correlated with increases in NSL.CVT (CCE) and a smaller nasopharyngeal airway cross-sectional area (naso area) was correlated with increases in OPT.HOR and CVT.HOR (FHP; $P \leq 0.05$).

Soft palate size and shape (Table 5)

The only soft palate measurement which was found to be correlated to a NHP variable (CVT.HOR) was the maximum palatal thickness (MPT; $P \leq 0.05$).

Tongue size and shape (Table 6)

All tongue measurements showed statistically significant correlations with NHP variables ($P \leq 0.05$–0.001). A longer (TGL), but thinner tongue (TGH) with a larger cross-sectional area (tongue area) can be expected in OSA patients with a CCE.

Hyoid bone position (Table 6)

A lower hyoid bone in relation to the mandibular plane (H–MP) was found to be statistically

Table 5 Matrix of probabilities and Pearson's *r* correlation coefficients between NHP variables and upper airway/soft palate measurements.

	OSA patients							
	NSL.OPT		NSL.CVT		OPT.HOR		CVT.HOR	
	r	*P*	*r*	*P*	*r*	*P*	*r*	*P*
SPAS	–0.114	NS	–0.163	0.017	–0.045	NS	–0.098	NS
MAS	–0.016	NS	–0.051	NS	0.058	NS	0.045	NS
IAS	0.119	NS	0.066	NS	0.073	NS	0.033	NS
Naso area	–0.062	NS	0.068	NS	–0.140	0.040	–0.174	0.011
Oro area	0.004	NS	–0.078	NS	0.040	NS	–0.024	NS
Hypo area	0.312	0.000	0.359	0.000	0.172	0.012	0.235	0.001
PNS-P	0.130	NS	0.099	NS	0.070	NS	0.023	NS
MPT	0.045	NS	0.078	NS	0.132	NS	0.156	0.023
PNS-P/MPT	0.059	NS	0.023	NS	–0.068	NS	–0.105	NS
Soft P area	0.117	NS	0.125	NS	0.120	NS	0.124	NS

Table 6 Matrix of probabilities and Pearson's *r* correlation coefficients between NHP variables and tongue/hyoid bone position/obesity measurements.

	OSA patients							
	NSL.OPT		NSL.CVT		OPT.HOR		CVT.HOR	
	r	*P*	*r*	*P*	*r*	*P*	*r*	*P*
TGL	0.329	0.000	0.393	0.000	0.200	0.003	0.276	0.000
TGH	–0.278	0.000	–0.319	0.000	–0.119	NS	–0.151	0.027
TGL/TGH	0.395	0.000	0.462	0.000	0.197	0.004	0.263	0.000
Tongue area	0.176	0.010	0.182	0.008	0.211	0.002	0.214	0.002
H–MP	0.272	0.000	0.297	0.000	0.100	NS	0.111	NS
H–H1	–0.022	NS	–0.060	NS	–0.014	NS	–0.075	NS
H–C3	0.270	0.000	0.174	0.011	0.305	0.000	0.230	0.001
H–RGn	0.359	0.000	0.443	0.000	0.155	0.023	0.221	0.001
BMI	0.220	0.001	0.220	0.001	0.251	0.000	0.247	0.000
PPNC	0.136	NS	0.162	0.043	0.112	NS	0.113	NS

related to CCE ($P \leq 0.001$), whereas the measurement H–H1, which represents its vertical position in relation to mandibular symphysis (RGn) and the third cervical vertebra (Cv3ia), was not significantly correlated to NHP measurements. Its sagittal distances to RGn and Cv3ia increased significantly with CCE and FHP ($P < 0.05$–0.001).

Obesity and neck size (Table 6)

A higher BMI was found to be significantly correlated with increases in CCE and FHP ($P \leq 0.001$), whereas the increase in neck size was only correlated with NSL.CVT ($P \leq 0.05$).

Discussion

This study demonstrates that there is a significant relationship between NHP and OSA syndrome (Table 2). It also suggests that severe OSA patients may have a greater tendency to exhibit a craniocervical extension with a forward head posture (Tables 3 and 4). Studies have shown that there is a threshold level of nasopharyngeal airway capacity (determined by nasal resistance

or nasal cross-sectional area) at which subjects start mouth breathing (Watson *et al.*, 1968; Warren *et al.*, 1988, 1991). The higher tendency for a forward and extended head posture in severe OSA patients may be an indication of a similar threshold level at which certain anatomical and/or physiological characteristics of the upper airway and related structures trigger changes in NHP. On the other hand, results concerning the means and standard deviations of the NHP variables also demonstrate that there are subjects with CCE and FHP in all groups, including the non-apnoeic snorer group (Table 3). This implies that different mechanisms may be responsible for the individual variations in NHP.

At this point, it might be questioned why an awake, upright, natural head posture should be related to OSA severity measurements which are obtained when the subjects are asleep with various head positions determined by factors such as the sleeping position and the pillow height. The obvious relationship between NHP and OSA, which has been demonstrated by Solow *et al.* (1993), and further supported by Tangugsorn *et al.* (1995a) and by the results of this study, clearly implies that certain physiological and anatomical factors that cause the nocturnal respiratory problems persist when the patients are awake. A number of cephalometric (Partinen *et al.*, 1988; Tsuchiya *et al.*, 1992; Pracharktam *et al.*, 1994; Lowe *et al.*, 1995; Tangugsorn *et al.*, 1995a,b), computer tomographic (Haponik *et al.*, 1983; Tsuchiya *et al.*, 1992; Lowe *et al.*, 1995), magnetic resonance imaging (Horner *et al.*, 1989; Shelton *et al.*, 1993a,b; Schwab *et al.*, 1995), and acoustic reflectance (Hoffstein *et al.*, 1984; Rivlin *et al.*, 1984) studies have determined significant differences in craniofacial, upper airway and related structures between awake OSA patients and controls. Furthermore, several studies have demonstrated that the neuromuscular properties of pharyngeal (Suratt *et al.*, 1984, 1985; Brown *et al.*, 1987; Stauffer *et al.*, 1987; Shephard *et al.*, 1990; Wasicko *et al.*, 1993) and genioglossus muscles (Mezzanotte *et al.*, 1992) are also compromised in awake OSA patients when compared with controls. These anatomical and physiological characteristics of the upper airway and related structures in OSA patients may trigger the chain of interactions between the muscles of the craniomandibular complex (including the pharyngeal and post-cervical muscles), resulting in a CCE and FHP.

Previous studies of the interactions between airway adequacy and head posture have demonstrated that minor adaptations in NHP to a changed mode of breathing were mainly caused by cranial extension (NSL.VER) (Solow and Greve, 1979; Woodside and Linder-Aronson, 1979; Vig *et al.*, 1980; Hellsing *et al.*, 1986). Correspondingly, in our sample, the initial response of the NHP to a mild OSA occurred primarily by cranial extension, together with an upright cervical column (decrease in cervical posture angles) (Table 3). However, these differences did not reach a statistical level of significance. Changes of the craniocervical posture in severe OSA patients were merely caused by the forward inclination of the cervical column (FHP), probably because a large amount of cranial extension in the NHP cannot be accomplished without compromising the horizontal visual axis. Concomitantly, Solow *et al.* (1993) demonstrated that in the OSA sample, the large increase in CCE was mediated mainly by a forward inclination of the cervical column, whereas the change in cranial extension only contributed about 2.5 degrees. In our study, the differences between the NHP variables in the control and OSA groups were lower than those found in previous studies (Solow *et al.*, 1993; Tangugsorn *et al.*, 1995a). This may be due to the differences in control samples. Our control sample consisted of subjects who had been referred to a sleep disorders centre and, therefore, included subjects with complaints such as snoring, although they were not diagnosed as having OSA (non-apnoeic snorers).

With regard to cephalometric and demographic measurements, our results suggest that an extended and forward NHP in OSA patients are mainly related to a larger hypopharyngeal airway cross-sectional area, a smaller nasopharyngeal airway cross-sectional area, a larger and longer tongue, a lower hyoid bone position in relation to the mandibular plane, and obesity (a higher BMI). At this point, it should be noted that any pair of measurements made in the same

individual can be correlated in some way, and that one must be cautious that some of these correlations may show the geometrical relationships, rather than the biological interactions. While evaluating the results, the cross-sectional nature of this study should also be taken into consideration.

The only upper airway size measurement that demonstrated statistically significant correlations with all NHP variables was the hypopharyngeal airway cross-sectional area. Although this seems to be in agreement with Hellsing (1989) who showed that the greatest effect of an experimental head extension occurs at the lower oropharyngeal and hypopharyngeal levels of the upper airway, two significant issues require further consideration. First, the effects of head extension versus a FHP on upper airway dimensions may be different. Secondly, and more important, the mechanisms responsible for the associations between the short-term experimental changes in head posture and upper airway dimensions may be different than the long-term, adaptive interrelations between upper airway and NHP.

Polo *et al.* (1991, 1993) demonstrated that a large hypopharyngeal size might be an aggravating factor for OSA. It was suggested that the proportionately larger hypopharynx might leave the palate to direct inspiratory suction, promoting its collapse. This may be a reason why the hypopharyngeal airway size was significantly correlated with CCE and FHP, which were primarily detected in severe OSA cases.

The low, but statistically significant relationship between a small nasopharyngeal cross-sectional area and a FHP in adult OSA patients is in agreement with the results of studies demonstrating the effects of nasopharyngeal obstruction on changes in NHP in children (Solow and Greve, 1979; Woodside and Linder-Aronson, 1979; Behfelt *et al.*, 1990). On the other hand, the lack of statistically significant correlations between oropharyngeal airway dimensions (except SPAS) and NHP does not necessarily indicate that they are not interrelated. Optimum oropharyngeal airway dimensions may have been maintained by the long-term adaptations in NHP, reducing the wide differences between patients and thereby causing the statistically lower correlations.

The most significant set of correlations in this study was observed between tongue size and shape, and the NHP measurements. A longer ($P \leq 0.01$–0.001) and thinner ($P \leq 0.05$–0.001) tongue with an increased cross-sectional area ($P \leq 0.01$) was related to an increased CCE and FHP. The vital need to maintain an adequate space between the mandible and the cervical column has been stressed previously (Bosma, 1963; Koski and Lähdemäki, 1975). A CCE with a FHP may aid as a compensatory mechanism in pulling the hyoid bone away from the posterior pharyngeal wall, and thereby accommodate the over-sized and longer tongue without interfering with the upper airway. In OSA patients, the cessation of this mechanism during sleep and the effect of gravity may cause the larger, longer, and therefore possibly heavier tongue, to drop back and obstruct the airway. The present findings with regard to the associations between tongue and NHP measurements also suggest that certain facial characteristics, such as an increased lower anterior facial height with a posterior rotation pattern of the mandible and a tendency for a sagittal skeletal Class II relationship, that have been observed in subjects with CCE and FHP (Solow and Tallgren, 1976; Marcotte, 1981; Cole, 1988; Solow and Siersbæk-Nielsen, 1992; Özbek and Köklü, 1993) may be due to a larger and longer tongue, which has also been suggested to be related to similar facial patterns (Lowe *et al.*, 1985).

A relationship between NHP, craniofacial structure and hyoid bone position, which also reflects the vertical position of the tongue (Behfelt *et al.*, 1990), has been demonstrated by Tallgren and Solow (1987). Correspondingly, in this study, higher correlations were found between a lower hyoid bone position in relation to the mandibular plane (H–MP) and increases in craniocervical extension ($P \leq 0.001$). As suggested by Thurow (1977), a low hyoid with a low tongue posture puts the geniohyoid at a mechanical disadvantage by creating a need for tongue elevation, which results in more downward and backward postural forces on the mandible. These, together with a larger tongue, may cause an increase in the mandibular load and thereby an interruption of the postural balances of the craniomandibular

region (Thurow, 1977). The increased load on the postural muscles of the mandible (mandibular closing muscles) and the head (post-cervicals) may cause a CCE.

The vertical position of the hyoid bone in relation to the third cervical vertebrae and the symphysis (H–H1) was not found to be related to NHP. This may again indicate either a lack of association between these characteristics or an adapted vertical hyoid position by the compensatory changes in NHP. Unfortunately, it is not possible to precisely determine these interrelations in static, cross-sectional studies such as this one. Dynamic, biomechanical simulation studies may provide a better understanding of these interactions.

The mechanisms which may be responsible for the associations between obesity and NHP are not clear. Ferguson *et al.* (1995) found that, in OSA patients, tongue length and cross-sectional area, and the distance between the hyoid bone and the mandibular plane increased as the neck size increased. Lowe *et al.* (1995) also demonstrated significant relationships between CT tongue and soft palate volumes, and obesity (BMI). The relationship between obesity and a compromised upper airway physiology has also been demonstrated (Schwartz *et al.*, 1991). Therefore, it can be proposed that the relatively larger and longer tongue, the lower hyoid bone position in relation to the mandibular plane, and the compromised upper airway physiology may be some of the factors triggering the adaptive changes in NHP in obese patients.

The results of this study indicate that, although severe OSA patients have a greater chance of having an extended and forward head posture, patients with different NHPs can be observed in all severity groups. In other words, CCE and FHP do not necessarily result from a severe OSA. In spite of the limitations of our method (static, two-dimensional, cross-sectional), it can be proposed that the contribution of certain anatomical characteristics of upper airway structures, such as a smaller nasopharyngeal airway cross-sectional area, a larger and longer tongue, a lower hyoid bone position, and obesity may trigger the adaptations in the NHP of OSA patients (Figure 4). Although the size of the soft palate does not seem to be a direct cause of CCE, its relatively closer position to the posterior pharyngeal wall due to the larger and longer tongue may be a reason for the reduction of velopharyngeal airway size (superior posterior airway size = SPAS) which was also found to be significantly correlated with craniocervical posture variables. Future research, including three-dimensional dynamic biomechanical simulation studies, may provide more information regarding the anatomical factors and interactive mechanisms that may be related to the individual differences of NHP in different population groups.

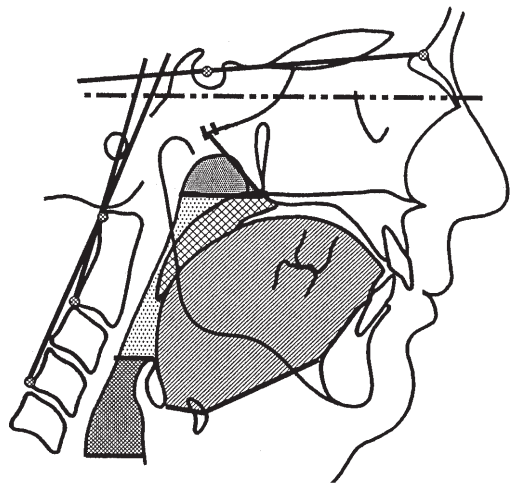

Figure 4 A typical severe OSA male patient to diagrammatically portray some cephalometric characteristics observed in CCE and FHP.

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Acknowledgements

The authors are indebted to Mrs Mary Wong for her statistical guidance and software assistance.

We are also grateful to Mrs Ingrid Ellis for her editorial assistance in the final preparation of the manuscript. This project was supported by the Canadian Federal Government through the Inspiraplex Respiratory Health Network of the Centers of Excellence.

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