

Effect of obesity and erect/supine posture on lateral cephalometry: relationship to sleep-disordered breathing

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Effect of obesity and erect/supine posture on lateral cephalometry: relationship to sleep-disordered breathing. P.E. Brander, I.L. Mortimore, N.J. Douglas. ©ERS Journals Ltd 1999.

ABSTRACT: Craniofacial and upper airway anatomy, obesity and posture may all play a role in compromising upper airway patency in patients with the sleep apnoea/hypopnoea syndrome. The aim of this study was to investigate the relationship between obesity, facial structure and severity of sleep-disordered breathing using lateral cephalometric measurements and to assess the effect of body posture on cephalometric measurements of upper airway calibre variables in obese and non-obese subjects.

Lateral cephalometry was carried out in erect and supine postures in 73 awake male subjects randomly selected from patients referred for polysomnography who had a wide range of apnoea/hypopnoea frequencies (1–131 events·h sleep⁻¹). Subjects were divided into non-obese (body mass index (BMI) <30 kg·m⁻²; n=42) and obese (BMI ≥30 kg·m⁻²; n=31) groups.

Significant but weak correlations were found between apnoea/hypopnoea index (AHI) and measurements reflecting upper airway dimensions: uvular protrusion–posterior pharyngeal wall (r=–0.26, p<0.05) and hyoid–posterior pharyngeal wall (r=0.26, p<0.05). Multiple regression using both upper airway dimensions improved the correlation to AHI (r=0.34, p=0.01). Obese subjects had greater hyoid–posterior pharyngeal wall distances than non-obese subjects, both erect (42±5 versus 39±4 mm, respectively (mean±SD) p<0.01) and supine (43±5 versus 40±4 mm, p<0.05). Skeletal craniofacial structure was similar in obese and non-obese subjects.

In conclusion, measurements reflecting upper airway size were correlated with the severity of sleep-disordered breathing. Differences in upper airway size measurements between obese and non-obese subjects were independent of bony craniofacial structure.

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The sleep apnoea/hypopnoea syndrome (SAHS) is characterized by repeated upper airway narrowing or collapse during sleep, associated with brief arousals which, if sufficiently frequent, cause daytime somnolence [1]. The pathogenesis of upper airway collapse may be multifactorial, with upper airway anatomy [2–4], compliance [4] and muscle function [5, 6] all playing a role. Lateral cephalometry allows visualization of upper airway size, soft tissues and craniofacial structure. SAHS patients have narrower upper airways than control subjects [7–9], enlarged soft palates and tongues [7–10], inferiorly set hyoid bones [7, 9, 11] and retropositioned mandibles/maxillae [2, 7, 10–12]. It has been suggested that bony craniofacial abnormality is more important as a risk factor in non-obese than in obese SAHS patients [13–15]. Relatives of thin SAHS patients have similar craniofacial abnormalities associated with sleep-disordered breathing, suggesting that the risk may be familial [16]. Obesity is also a risk factor for SAHS [13, 17] and may affect upper airway morphology in the absence of bony craniofacial abnormality.

It was hypothesized that if the overall result of craniofacial anatomy, obesity and upper airway physiology is to compromise upper airway patency, there should be lateral

cephalometric measurements that reflect these influences. Previous studies have demonstrated different soft tissue variables to correlate with the severity of SAHS (apnoea/hypopnoea index (AHI)) in obese and non-obese patients [13, 15, 17, 18] but not measurements of upper airway size in an unselected group.

The present study was, therefore, designed to evaluate the relationship between AHI severity as a continuous variable with lateral cephalometric measurements and to assess the effect of body position on cephalometric measurements in non-obese and obese subjects.

Subjects and methods

Subjects

The study population consisted of 73 males referred to the laboratory for investigation of possible sleep-disordered breathing (table 1). Subjects were selected consecutively and prospectively. Each had clinical polysomnography, conducted and scored according to the department's usual

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methods [19], and cephalometry was performed the morning after the sleep study. Subjects without any apnoeas or hypopnoeas were excluded from the analysis, as were subjects with fewer than six teeth per jaw.

Cephalometry

Lateral cephalometric radiographs were carried out with the subjects both erect and supine as described previously [20]. The patients were directed to gaze forward for the erect films and upwards for the supine films, holding their heads in a natural position. A total of 21 standard cephalometric variables, expressed as angular (degrees) or linear (mm) measurements in both erect and supine postures, was analysed for each subject (fig. 1). The investigator who analysed the cephalograms was blinded to the results of the other assessments. Craniofacial measurements included the following five angles: 1) the cranial base angle (NSBa), which is an angle formed by the intersection of lines drawn from nasion (N) to the midpoint of sella (S) and sella to basion (Ba); 2) the SNA and 3) SNB angles, which are the angles between the line from S to N, and the line from N to A (subspinale, the deepest point on the pre-maxillary outer contour) and from N to B (supramentale, the deepest point on the outer mandibular contour); 4) the ANB angle, which is the angle between the lines from A and B to N; and 5) the SN-GoGn angle, which is an angle formed by the intersection of lines drawn from S to N and from gonion (Go) to gnathion (Gn). The SNA and SNB angles give information on the anterior-posterior position of the maxilla (SNA) and mandible (SNB) in relation to the cranial base. The ANB angle is a measure of maxillary/mandibular discrepancy. ANB increases and SNB decreases with increasing retrognathia. The SN-GoGn angle reflects facial divergence.

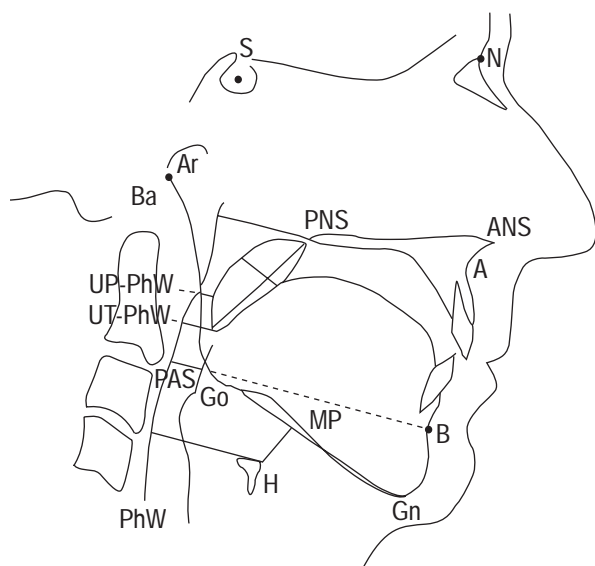


Fig. 1. – Diagrammatic representation of anatomical points and lines used to identify craniofacial and soft tissue parameters on cephalometric radiographs. S: sella; N: nasion; Ar: articulare; Ba: basion; PNS: posterior nasal spine; ANS: anterior nasal spine; A: subspinale; B: supramentale; Gn: gnathion; MP: mandibular plane; H: hyoid; Go: gonion; PAS: posterior airway space; PhW: pharyngeal wall; UP-PhW: uvular protrusion to PhW; UT-PhW: uvular tip to pharyngeal wall.

The distances from the anterior nasal spine (ANS) to Go, Gn and the posterior nasal spine (PNS) were measured (mm) together with the distances from Go to Gn, PNS, S and articulare (Ar; the intersection of the posterior margin of the ascending ramus and the outer margin of the cranial base).

Soft palate size was evaluated by measuring uvular length (UL), calculated as the distance from PNS to uvular tip (UT) and maximum uvular width (UW). The most posterior part of the soft palate was taken as a measure of uvular protrusion (UP). Five measurements of upper airway size were taken: the distances between the posterior pharyngeal wall (PhW) and PNS, UP, UT, tongue base (posterior airway space (PAS)) and the anterosuperior point of the hyoid (H). PAS was measured on a line joining B to Go and the other upper airway size measurements parallel to that line. H bone position was also assessed by measuring the perpendicular distance from H to the mandibular plane (MP), which is a line drawn from Gn to Go, and the distance from H to Gn.

Statistical analysis

Pearson correlation analysis and multiple regression analysis were used to evaluate the relationships between AHI, body mass index (BMI), lowest arterial oxygen saturation (SaO₂) during sleep and cephalometric measurements in both erect and supine postures. In the UK obesity is defined as a BMI >30 kg·m⁻² [21]. The subjects were therefore divided into obese and non-obese groups according to BMI, *i.e.* ≥30 kg·m⁻², and <30 kg·m⁻², respectively. Differences between the two groups and the effects of posture were analysed using paired and unpaired t-tests as appropriate (SPSS for Windows; Chicago, IL, USA).

Results

The characteristics of the study population and sub-groups are shown in table 1. AHI ranged 1–131 events·h sleep⁻¹ (n=73) and was significantly higher in the obese subjects. Fifty-two of the 73 subjects had an AHI ≥15. Overall, 42 of the 73 subjects were non-obese and 31 obese.

Cephalometric measurements

The means and standard deviations for the cephalometric variables reflecting soft palate size, upper airway size,

Table 1. – Patient characteristics

	Whole group	Non-obese	Obese	AHI ≥15
Subjects n	73	42	31	52
Age yrs	49±12	47±11	51±13	43±13
BMI kg·m ⁻²	30±5	27±2	35±5	32±6
AHI	33±26	22±19	48±28***	43±25
LoSa _{o2} %	80±15	83±13	75±17*	77±16

Data are shown as means±SD. Non-obese: body mass index (BMI) <30 kg·m⁻²; obese: BMI ≥30 kg·m⁻²; AHI: apnoea/hypopnoea index; LoSa_{o2}: lowest arterial oxygen saturation during sleep. *: p<0.05; ***: p<0.001, obese *versus* non-obese.

H bone position and bony craniofacial angles and dimensions are shown in table 2 for obese and non-obese subjects in both erect and supine postures.

Obese subjects had significantly longer uvulas than non-obese subjects in the erect posture (table 2). Only one of the five measurements reflecting upper airway size demonstrated significant differences between non-obese and obese subjects. H-PhW was significantly greater in the obese than in the non-obese subjects, both in the erect and supine postures. H-PhW was greater supine than erect in non-obese subjects, but not in obese subjects. There were no differences in PAS or H-MP distance, either with posture or between the non-obese and obese subjects (table 2). There were no differences between non-obese and obese subjects with respect to the intraindividual (within-subject) changes in cephalometric variables with posture.

Subanalysis of subjects with an AHI ≥ 15 showed similarly significant differences between non-obese ($n=22$) and obese ($n=30$) subjects in H-PhW, both erect (non-obese 38 ± 4 mm, obese 42 ± 5 mm, $p=0.006$) and supine (non-

Table 2. – Erect and supine cephalometric measurements of soft palate size, upper airway size, hyoid bone position and bony craniofacial angles and dimensions in non-obese and obese patients

Variables	Non-obese		Obese	
	Erect	Supine	Erect	Supine
UL mm	47 \pm 4	49 \pm 5 ^{††}	50 \pm 3	50 \pm 4
UW mm	13 \pm 2	13 \pm 2	13 \pm 2	13 \pm 2
PNS-PhW mm	30 \pm 4	30 \pm 4	30 \pm 4	30 \pm 5
UP-PhW mm	8 \pm 3	8 \pm 8	7 \pm 3	6 \pm 3 [†]
UT-PhW mm	11 \pm 3	9 \pm 3 [†]	10 \pm 3	9 \pm 4
PAS mm	12 \pm 3	14 \pm 4	12 \pm 3	15 \pm 3
H-PhW mm	39 \pm 4	40 \pm 4 [†]	54 \pm 5**	43 \pm 5*
H-MP mm	27 \pm 7	28 \pm 9	29 \pm 7	29 \pm 6
H-Gn mm	49 \pm 8	48 \pm 10	48 \pm 5	45 \pm 7
NSBa °	126 \pm 6	126 \pm 6	128 \pm 5	127 \pm 5
SNA °	81 \pm 5	81 \pm 4	80 \pm 5	80 \pm 5
SNB °	80 \pm 5	80 \pm 5	78 \pm 4	78 \pm 4
ANB °	1 \pm 3	1 \pm 3	2 \pm 3	2 \pm 3
SNGoGn °	36 \pm 8	36 \pm 7	36 \pm 6	35 \pm 6
Go-Gn mm	81 \pm 5	81 \pm 5	80 \pm 5	80 \pm 5
Go-S mm	92 \pm 7	92 \pm 7	91 \pm 8	92 \pm 8
Go-ANS mm	91 \pm 6	91 \pm 5	92 \pm 6	92 \pm 7
GO-PNS mm	52 \pm 5	52 \pm 5	52 \pm 6	52 \pm 6
Go-Ar mm	57 \pm 7	56 \pm 6	56 \pm 7	56 \pm 6
Gn-ANS mm	82 \pm 7	82 \pm 7	81 \pm 7	81 \pm 7
ANS-PNS mm	51 \pm 4	50 \pm 4	51 \pm 4	51 \pm 4

Data are means \pm SD. Non-obese: body mass index (BMI) <30 kg \cdot m $^{-2}$; obese: BMI ≥ 30 kg \cdot m $^{-2}$. UL: uvular length; UW: uvular width; PNS-PhW: posterior nasal spine to pharyngeal wall; UP-PhW: uvular protrusion to PhW; UT-PhW: uvular tip to PhW; PAS: posterior airway space; H-PhW: hyoid to PhW; H-MP: H to mandibular plane; H-Gn: H to gnathion; NSBa: angle between lines from nasion (N) to sella (S) and from S to basion (Ba); SNA: angle between lines from S to N and from subspinale (A) to N; SNB: angle between lines from S to N and from suprmentale (B) to N; ANB: angle between lines from A and B to N; SNGoGn: angle formed by intersection of lines from S to N and from gonion to Gn; Go-Gn: Go to Gn; Go-S: Go to S; Go-ANS: Go to anterior nasal spine; Go-PNS: Go to PNS; Go-Ar: Go to articulare; Gn-ANS: Gn to ANS; ANS-PNS: ANS to PNS. *: $p<0.05$; **: $p<0.01$, obese *versus* non-obese; †: $p<0.05$; ††: $p<0.01$, erect *versus* supine.

obese 39 ± 4 mm, obese 43 ± 5 mm; $p=0.006$) and in UL erect (non-obese 47 ± 4 mm, obese 50 ± 3 mm, $p=0.05$). There were no bony craniofacial differences between non-obese and obese subjects in those with an AHI ≥ 15 .

Correlation analysis

AHI was found to be significantly correlated with BMI ($r=0.3$, $p<0.01$), lowest S_aO_2 during sleep ($r=-0.37$, $p<0.001$), H-PhW erect ($r=0.26$, $p<0.05$) and UP-PhW erect ($r=-0.26$, $p<0.05$). There were no other significant correlations between AHI and cephalometric variables in either the erect or the supine posture. Multiple regression analysis using H-PhW erect and UP-PhW erect improved AHI prediction ($r=0.34$, $p=0.01$). If PAS erect, which alone was not significantly correlated to AHI ($r=-0.23$, $p=0.85$), was included in a multiple regression with H-PhW and UP-PhW, the prediction of AHI was marginally improved ($r=0.39$, $p=0.01$). The addition of BMI and lowest S_aO_2 during sleep sequentially to prediction equations including the two upper airway variables showing significant correlations to AHI (H-PhW and UP-PhW) improved AHI prediction ($r=0.43$, $p=0.003$ and $r=0.5$, $p<0.001$, respectively).

BMI was found to be highly correlated with H-PhW both erect ($r=0.41$, $p<0.001$) and supine ($r=0.48$, $p<0.001$), PNS-PhW erect ($r=0.24$, $p<0.05$) and PAS supine ($r=0.32$, $p<0.01$), but not with PAS erect.

Discussion

This study demonstrated significant correlations between measurements of upper airway size and severity of sleep-disordered breathing. There were no differences in bony craniofacial structure between obese and non-obese subjects. The changes in upper airway calibre with posture were broadly similar.

The present study differs from many previous studies [7, 10, 12–14, 17] with respect to the subject group. Subjects with low AHI were included because the severity of SAHS is a continuum and, therefore, it is important to correlate anatomical dimensions for the whole range of AHI and not just for high AHI, which might skew the distribution. In addition, the definition of SAHS with an AHI of ≥ 15 events \cdot h $^{-1}$ is arbitrary and not uniformly accepted. By choosing subjects consecutively, a wide range of AHI and BMI was gained.

AHI was significantly correlated with two measurements of upper airway size, UP-PhW erect and H-PhW erect, the former reflecting upper airway size at the retro-palatal level and the latter at the level of H bone. The positive correlation of H-PhW with AHI probably reflects the fact that there is more soft tissue encroaching the upper airway. The use of both variables in a multiple prediction equation improved the correlation to AHI and inclusion of the retroglossal measurement, PAS, further improved the prediction marginally. However, inclusion of all these variables only explained 17% of the observed variance. Thus, structural characteristics of soft tissues explained only partially the variance in AHI. This indicates that other factors such as functional characteristics of upper airway muscles may play a more critical role in the variance of AHI.

Pharyngeal collapse in sleeping SAHS patients may occur at different levels or multiple levels in the same patient [22]; therefore, by taking upper airway measurements reflecting different anatomical levels the critical upper airway measurement for each individual will be included in the regression equation. This study demonstrates for the first time significant correlations between measurements incorporating upper airway size at different levels and severity of sleep-disordered breathing in a group of subjects referred for sleep studies. Three studies have reported a significant correlation between severity of sleep-disordered breathing and retroglossal airway size [17, 23, 24], but not other measurements of upper airway size. FERGUSON *et al.* [14] were unable to demonstrate any correlation between cephalometric variables and AHI. In some studies soft tissue cephalometric measurements have been shown to correlate with AHI when subjects were split into non-obese and obese groups [13, 15].

Post hoc analysis of subjects with AHI ≥ 15 in the present study showed only H-PhW erect to be significantly correlated with AHI ($r=0.28$, $p=0.047$). The loss of a significant correlation with UP-PhW may, therefore, reflect the narrower range of AHI available for the correlation which could apply to some other published studies.

AHI was not correlated with any of the measurements reflecting bony craniofacial structure. The best correlation with AHI was shown by erect and supine SNB angles (r -values -0.13 erect SNB and -0.16 supine SNB), which gave p -values of 0.26 (erect) and 0.19 (supine). Comparison of angular measurements and bony craniofacial distances did not demonstrate any differences between the obese and non-obese subject groups or between the obese and non-obese SAHS patients ($n=52$), which is a finding at variance with studies from another group [13, 14]. These two studies comprised 84 and 161 SAHS patients. However, the present findings may not be related only to a smaller number of subjects because, out of all the comparisons of bony craniofacial measurements in this study, the lowest p -value was 0.19 . Thus, the significance level of the comparisons would be unlikely to change dramatically with an increased number of subjects. This suggests that non-obese subjects may have factors other than bony craniofacial abnormalities which are important in determining upper airway size and, therefore, predisposition to collapse or narrowing during sleep. This view is supported by the observation that non-obese SAHS patients have increased fat deposits adjacent to the upper airway compared with controls matched for BMI and neck circumference [25].

Obesity, as defined by BMI, was significantly correlated with AHI, H-PhW erect and supine and PAS supine. Several other studies have demonstrated similar correlations between BMI and AHI, with BMI accounting for at most 30% of the variance in AHI [10, 13, 17, 18, 20, 26]. The relationship between H-PhW and BMI probably reflects the effect of adipose tissue deposition in the tissues anterior to the upper airway, which increases the H-PhW distance overall, but decreases the airway size, thus explaining the observed correlation with AHI.

The present study was carried out before publication of the study by FERGUSON *et al.* [14] and differs from that of FERGUSON *et al.* [14], who defined obesity in terms of neck circumference, whereas BMI was used in the current study. In addition, FERGUSON *et al.* [14] studied 161 SAHS

patients compared to the present 73 apnoeic/hypopnoeic subjects, some with AHI <15 events \cdot h $^{-1}$. These differences may account for some of the differences in the results. However, the absence of cephalometric differences between obese and non-obese patients in the present study suggests that any such disparity is relatively small. It may be, however, that neck circumference rather than BMI should be used to determine whether or not cephalometry is likely to be clinically useful. The present study also differs from that of FERGUSON *et al.* [14] by assessing correlations between cephalometric measurements and both BMI and AHI and by studying the effects of posture.

In summary, the present study demonstrates that measurements reflecting upper airway size (measured retro-palatally, and at the level of the hyoid bone) is correlated with apnoea/hypopnoea index severity when taken as a continuum in an apnoeic/hypopnoeic subject group including both obese and non-obese subjects. Differences in upper airway size between obese and non-obese subjects were determined by soft tissue rather than bony craniofacial structure.

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