SCIENTIFIC INVESTIGATIONS

JCSM
Journal of Clinical Sleep Medicine

pii: jc-00237-14
http://dx.doi.org/10.5664/jcsm.4604

Shorter Mandibular Length is Associated with a Greater Fall in AHI with Weight Loss

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Rationale: Obesity is a major risk factor towards the development of obstructive sleep apnea, while significant weight loss (both conservatively managed and surgically assisted) has a variable effect upon its severity. Differences in the effect of weight loss on obstructive sleep apnea may be due to underlying craniofacial characteristics.

Objectives: To determine whether craniofacial characteristics can predict OSA treatment response to significant weight loss.

Methods: We analyzed craniofacial measurements from lateral cephalograms performed at baseline on 57 patients enrolled in a previously reported 2-year randomized clinical weight loss trial (laparoscopic adjustable gastric band surgery versus conservatively [dietician and very low calorie diet] treated). Group mean weight loss was ~ 13% (mean weight loss 131 to 114 kg), with corresponding reduction in mean apnea-hypopnea index (AHI) from 61 to 41 events/h. Computer assisted lateral cephalogram analysis was undertaken by three trained staff blinded to treatment. We analyzed lateral cephalogram and demographic data at baseline (cross-sectional) and change over two years (interventional) in 54 patients.

Measurements and Main Results: Baseline cross-sectional analysis indicated no cephalometric measurement correlated significantly with baseline AHI when corrected for neck circumference. The percentage change in AHI over 2 years correlated with a shorter menton-gonion distance (i.e., mandibular body length). The % change in AHI correlated with the % weight change (R^2 = 0.25, p < 0.001) and mandibular body length (R^2 = 0.19, p = 0.002). The % change in AHI correlated with combined weight change and mandibular body length (combined R^2 = 0.31, p < 0.001).

Conclusions: Weight loss as a therapeutic option for severe OSA with severe obesity may be predicted by shorter mandibular body length as measured by lateral cephalometry.

Keywords: apnea, mandibular length, weight loss

Citation: Naughton MT, Monteith BD, Manton DJ, Dever P, Schachter LM, O'Brien PE, Dixon JB. Shorter mandibular length is associated with a greater fall in AHI with weight loss. *J Clin Sleep Med* 2015;11(4):451–456.

Obstructive sleep apnea (OSA) is a common condition considered a significant cause of impaired quality of life plus cardiovascular, neuropsychological and perioperative morbidity, and possible mortality. Treatment is difficult as indicated by the wide variance (25% to 75%) in adherence to the "gold standard" therapy of continuous positive airway pressure (CPAP).[1] Other treatments such as weight loss, upper airway surgery, oral appliances, medications, nasal valves, and pacemakers may be effective in patient subgroups. Thus patient selection for each of the various treatment options is crucial but supportive data are very limited. Given that obesity and craniofacial abnormalities are two variables that contribute significantly to the apnea-hypopnea index (AHI), a greater understanding of the obesity-craniofacial anatomy interplay may assist in selecting the best therapeutic option for individual patients.

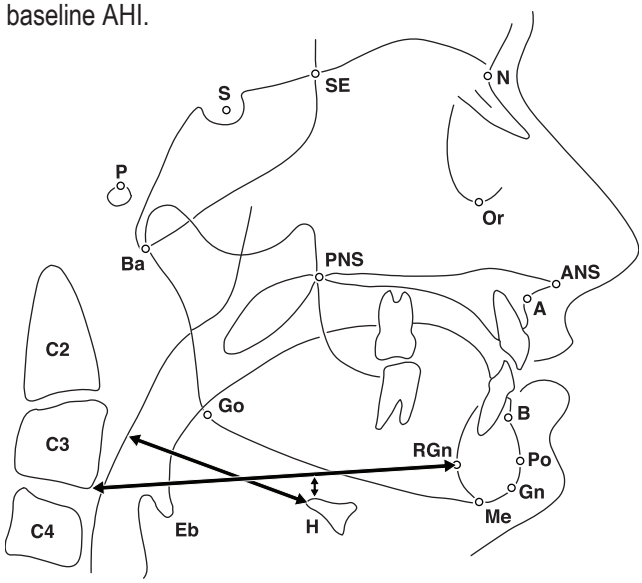
Observational studies of major weight loss following bariatric surgery suggest substantial remission of OSA symptoms in up to 60% to 80%.[2] However, in studies where repeat polysomnography (PSG) was available, remission of OSA was unusual.[3] Three long-term randomized controlled trials of dietary-induced weight loss indicated only small changes in AHI with a low chance of cure (AHI < 5 events/h).[4–6] Recently, we reported a randomized controlled trial of bariatric surgery, using laparoscopic adjustable gastric banding (LAGB) in 60 patients (baseline weight 131 ± 21 kg, BMI 45 ± 5 kg/m^2, and AHI 61 ± 31 events/h).[7] After two years, the bariatric surgery group had significantly more weight loss (−28 vs −5 kg) but there was no difference in change in mean AHI (−26 vs −14 events/h) compared with the conventional program (dietary and exercise advice). Only 10 patients achieved an AHI < 15

BRIEF SUMMARY

Current Knowledge/Study Rationale: Substantial weight loss does not routinely result in resolution of obstructive sleep apnea. Craniofacial characteristics may be useful in predicting which patients have a better response.

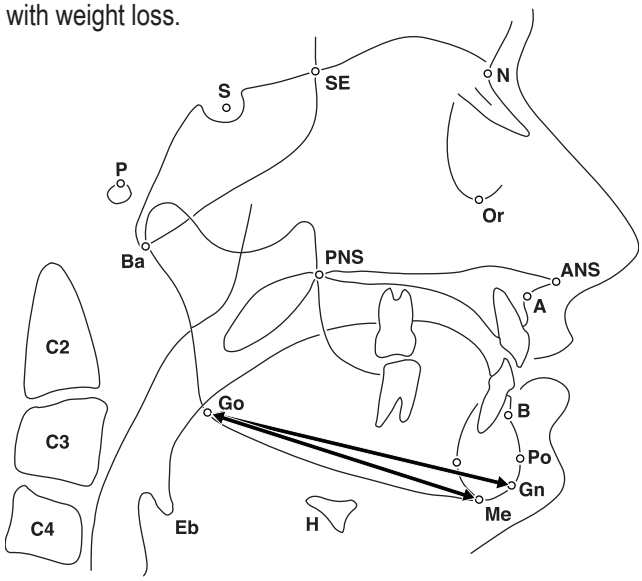
Study Impact: A greater fall in AHI was associated with a shorter jaw length.

Figure 1—Illustration of two measures associated with baseline AHI.



The two measures associated with baseline AHI are shown, hyoid perpendicular to C3-retrognathion (RGn) distance ($r = 0.37$, $p = 0.004$) and hyoid to posterior pharyngeal wall distance ($r = 0.47$, $p < 0.001$). Lateral cephalogram illustrating landmarks (A, supradentale; ANS, anterior nasal spine; Ba, basion; C2-4, Cervical vertebral bodies; Eb, Epiglottis base; H, hyoid; Gn, gnathion; Go, gonion; B, infradentale; Me, menton; N, nasion; Or, orbitale; PNS, posterior nasal spine; P, porion; Po, pognonion; S, sella; SE, spheno ethmoidal point).

Figure 2— Illustration of two measures of mandibular length that correlate with the percentage change in AHI with weight loss.



Two measures of mandibular length correlate with the percentage change in AHI with weight loss in this study, Me – Go ($r = 0.45$, $p = 0.001$) and Gn – Go ($r = 0.28$, $p = 0.034$). Shorter mandibular length associated with a greater percentage fall in AHI. Lateral cephalogram illustrating landmarks (A, supradentale; ANS, anterior nasal spine; Ba, basion; C2-4, Cervical vertebral bodies; Eb, Epiglottis base; H, hyoid; Gn, gnathion; Go, gonion; B, infradentale; Me, menton; N, nasion; Or, orbitale; PNS, posterior nasal spine; P, porion; Po, pognonion; S, sella; SE, spheno ethmoidal point).

and one patient an AHI < 5 events/h. Thus, "cure" of OSA, as defined by an AHI < 5 in this trial was rare. Moreover, the response to weight loss upon AHI was variable in both surgical and conservatively treated groups. The wide variety of responses raised question as to whether craniofacial structure influences response to weight loss.

The importance of craniofacial anatomy has been highlighted by a subgroup of the Wisconsin Sleep Cohort in which lateral cephalometry was undertaken.[8] Approximately ~25% of the AHI variance could be explained by body mass index ([BMI] with a further 25% by 6 cephalometric measurements (PV-A, PAS_1, H-MP, Gonion-SE-PNS, PAS_2, S-N-B) out of 55 measurements tested (see **Figures 1** and **2**, **Table 1**). Collectively, these cephalometric measurements indicate a shorter distance between maxillary projection from the cranial base, a smaller posterior airway space, less mandibular protrusion, a narrower space between the hard palate and cranial base, and a more caudally placed hyoid bone predispose to a higher AHI. The most important cephalometric measurement was the "PV-A" (i.e., maxillary projection), which accounted for 14% of the variance; this was greater than the other 54 lateral cephalometric measurements analyzed.

We therefore hypothesized that the variance of change in AHI in our previous study[7] could be explained by craniofacial characteristics. Our aim was to determine which craniofacial measurements were associated with a response to weight loss treatment in a cohort of patients from our previously reported randomized controlled trial of two weight loss programs over two years,[7] with OSA severity as the major outcome.

METHODS

We studied subjects aged 18–60 years who were involved in a recently reported 2-year randomized controlled trial of bariatric surgical versus conservative treatment for obesity (BMI 35–55 kg/m^2, AHI > 20 events/h) who had baseline and 2-year follow-up diagnostic polysomnograms.[7] The study was approved by the Human Ethics committees of the Alfred Hospital and Monash University in accordance with the guidelines of the National Health and Medical Research Council of Australia and the Helsinki Declaration, as revised in 2000 and 2008, respectively. All participants provided written informed consent.

Of 60 subjects randomized in the original trial, 3 patients did not obtain baseline lateral cephalograms and 3 did not attend 2-year polysomnograms; thus, data from 57 subjects were available for cross-sectional baseline analysis and 54 for interventional analysis. In this analysis, we combined both weight loss groups and used the method of weight change as a possible modifier. We then sought to determine the effect of weight change upon AHI, and asked whether craniofacial measurements were able to assist in predicting those subjects in which weight loss was effective in reducing the AHI.

Lateral cephalograms were taken while seated and awake during the baseline assessment prior to surgery. Each was taken at the same center (Alfred Hospital Radiology) using identical equipment with special attention to maintain a horizontal

Frankfort line. Lateral cephalograms were analyzed using Dolphin Imaging 11 software (Dolphin Imaging, Chatsworth, CA, USA) by 3 experienced dental specialists (PD, BDM, and DJM), blinded to the results of polysomnography and change in weight. Reproducibility of measurements was undertaken by 2 trained orthodontists. Any disagreement that arose was resolved by discussion with a third expert so that consensus could be reached. Sixteen measurements were chosen based upon previously published literature[5,8–10] as predictors of OSA (**Table 1**). The following 19 craniometric *landmarks* (anterior nasal spine [ANS], basion [Ba], most anterior and inferior points of cervical vertebral bodies 2-4 [C2-4], epiglottis base [Eb], hyoid [H], gnathion [Gn], gonion [Go], infradentale [B], menton [Me], nasion [N], orbitale [Or], pogonion [Po], posterior nasal spine [PNS], porion [P], supradentale [A], sella [S], spheno ethmoidal point [SE]) from which 6 angles (SNA, SNB, ANB and BSGo) were estimated, 5 *planes* (Frankfort (P-O), Nasion Vertical, Porion Vertical, Mandibular (Gn-Go) and Occlusal Plane) were identified, and 10 *distances* were calculated (see **Table 1**).

As the aim of the study was to look for predictors of change in % AHI in relation to % change in weight, both surgical and conventionally treated groups were combined for analysis and method of treatment was included as a covariate. Of the 60 participants recruited into the previously published study, 57 had baseline lateral cephalograms and 54 completed the study with diagnostic PSG at baseline and 2 years.[7] In view of the finding in our original study that indicated modest weight gain and loss strongly influenced the change in AHI and that greater weight loss had limited beneficial effect, we divided the trial completers (n = 54) into 2 groups: those with ≥ 5% weight loss (n = 38); and those with < 5% weight loss or weight gain (n = 16), and examined for the predictors in these subgroups.

Statistical Methods

Patients in this trial were analyzed as one group as the 2 modes of weight loss had similar effects on AHI with group of randomization a modifier. Pearson or Spearman correlation coefficients were used, as appropriate, to access univariate associations. Linear regression analysis was used to identify independent factors associated with baseline AHI, and change in AHI between baseline and 2 years. In addition a hierarchical stepwise approach was used for all analyses using demographic, anthropometric, and cephalometric measurements sequentially.[11] The whole group was divided into those with clinically significant weight loss ≥ 5%, and those with < 5% weight loss or weight gain and the analysis for prediction change in AHI was reexamined. Univariate relationships have been expressed as r-values, linear regression as R^2 values, and odds ratio ± 95% CI. All statistical analysis was performed using IBM SPSS Statistics20.

RESULTS

Cross-Sectional Associations with Baseline Diagnostic AHI (n = 57).

Baseline AHI was associated with baseline BMI and male sex independently and together provided an adjusted R^2 of 0.17 (p = 0.002), whereas there was no association with age, or a history of diabetes, hypertension, or depression. When age, sex, BMI and neck circumference were modeled together, only neck circumference was associated with baseline AHI (adjusted R^2 = 0.21, p = 0.001). Of the 16 lateral cephalogram measures, 2 were associated in a univariate manner (p < 0.05) with baseline AHI: the hyoid perpendicular to C3-retrognathion distance (r = 0.37, p = 0.004) and hyoid to posterior pharyngeal wall distance (r = 0.47, p < 0.001; **Figure 1**). However, when modelled with BMI, sex, and neck circumference, neither lateral cephalometric measure above added significant variance at predicting baseline AHI. Baseline neck circumference correlated with the hyoid perpendicular to C3-retrognathion length[8] (r = 0.51, p < 0.001) and hyoid to posterior pharyngeal wall length (r = 0.60, p < 0.001). Neck circumference provided the most relevant single measure for predicting the baseline AHI.

Table 1—Sixteen lateral cephalogram measurements.

Angles:
- Sella Nasion Supradental (SNA) angle (°)
- Sella Nasion Infradentale (SNB) angle (°)
- Supradental Nasion Infradentale angle (°)
- Cranial base angle (Basion to Sella to Nasion) (°)
- Angle of the planes of Sella to Nasion and Gonion to Menton (°)
- Gonion – spheno ethmoidal point – posterior nasal spine (°)

Lengths:
- Mandibular Body Length 1 (Menton-Gonion) (mm)
- Mandibular Body Length 2 (Gnathion-Gonion) (mm)
- Soft Palate Length (posterior nasal spine (PNS) to uvula tip) (mm)
- Anterior Facial Height (Anterior nasal spine to Menton) (mm)
- Hyoid – Mandibular Plane perpendicular distance (mm)
- Hyoid perpendicular to most anterior and inferior point of C3 vertebral body - Retrognathion distance (mm)
- Hyoid to pharyngeal wall distance (mm)
- Palatal Air Space distance (mm)
- Porion vertical to A distance (mm)
- Pogonion to Orbitale distance (mm)

Associations with the Percentage Change in AHI at 2 Years (n = 54)

The characteristics of these participants are shown in **Table 2**. The median percentage change in AHI was −41.1% (interquartile range [IQR] −63.5 to −2.8%), and the mean was −24.7% (SD 53.7). Despite 2 outliers with > 100% increase in AHI, both of whom gained weight throughout the study and skewed the distribution to the right, the distribution of data was still appropriate for linear regression analysis with skewness and kurtosis < 2.0.

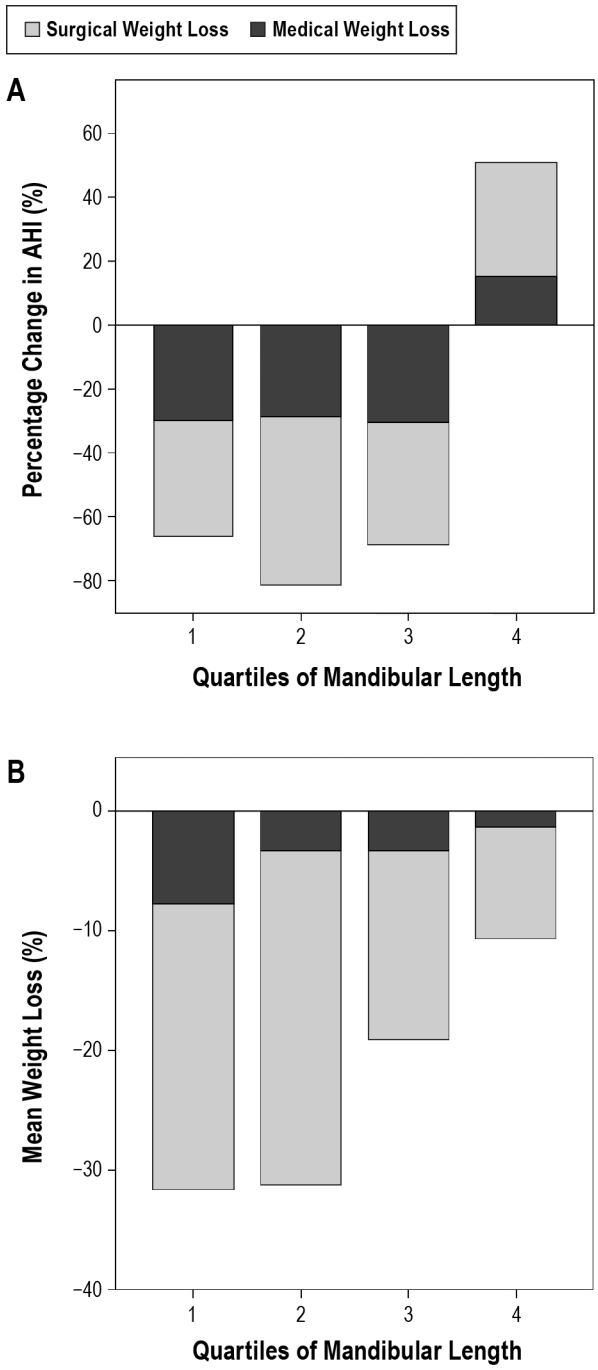
Mean percentage weight change for the combined groups was −12.4% (SD 13.7%), and median −11.6% (IQR −22 to 4.0%). As indicated in the primary manuscript,[7] we found a positive relationship between percentage change in weight and percentage change in AHI (r = 0.49, p < 0.001). We examined for preoperative variables that may influence the effect of percentage weight change on the percent change in AHI; baseline

Table 2—Baseline and 2-year characteristics of 54 study participants with baseline lateral cephalogram examination who completed the 2-year study.

Characteristic	Baseline n = 54	2 Years n = 54
Age, years	46.1 (8.4)	
Men, n (%)	29 (54%)	
Randomized to surgery, n (%)	28 (52%)	
Hypertension, n (%)	29 (54%)	
Diabetes, n (%)	21 (39%)	
Metabolic Syndrome - ATP III, n (%)	40 (74%)	
Depression, n (%)	21 (39%)	
BMI, kg/m^2	44.9 (5.5)	39.4 (6.5)*
Weight, kg	131.1 (22.1)	114.3 (23.8)*
Change in weight (kg)		−16.8 (20.0)
Percentage change in weight (%)		12.2 (14)
Neck circumference, cm	46.8 (4.7)	42.3 (6.0)*
Apnea-hypopnea index	60.1 (27.0)	40.4 (27.0)*
Change in AHI		−19.7 (30.6)
Percentage change in AHI (%)		−24.3 (54.9)
Arousal Index, events/h	46.1 (26.0)	30.8 (15.9)*
Lowest SpO_2 (%)	70.8 (6.9)	82.3 (6.8)*

Continuous variables presented as mean (standard deviation). *All changes at 2 years were significant ($p < 0.001$, paired student t-test). Data for the additional 3 participants in the baseline cross-sectional analysis are not shown. BMI, body mass index; AHI, apnea-hypopnea index.

Figure 3



(A) Relationship between % change in AHI over 2 years and quartiles of mandibular length. **(B)** Relationship between % weight loss and quartiles of mandibular length. Mandibular length: menton – gonion (i.e., mandibular body) length Quartile 1, 63.7–67.4 mm; Quartile 2, 67.5–70.1 mm; Quartile 3, 70.2–73.6mm; and Quartile 4, 73.7–82.4 mm.

age, sex, BMI, and neck circumference did not influence this relationship. In none of the analyses did weight loss method influence the results when analysis was controlled for percentage weight loss.

Univariate correlation analysis indicated the menton – gonion (i.e., mandibular body) length (**Figure 2**) was associated with %AHI change ($r = 0.45$, $p = 0.001$) and in % weight change ($r = 0.37$, $p = 0.006$) (**Figure 3**). After controlling for the percentage weight loss shorter mandibular body length was associated with a reduction in %AHI ($R^2 = 0.11$, $p = 0.02$). These effects were unchanged after additionally controlling for the subjects' height ($r = 0.47$, $p < 0.001$) and gender ($r = 0.29$, $p < 0.001$) respectively. A shorter mandibular body length was associated with greater % weight loss and greater reduction in %AHI. The second measure of mandibular length also correlated with the change in %AHI, Gn – Go, $r = 0.28$, $p = 0.034$.

Using a hierarchical regression model to assess the influence of preoperative factors on % change in AHI after controlling for % weight loss, no demographic or anthropometric measure including neck circumference, study group, or any of the lateral cephalographic measures other than menton-gonion was associated with outcomes (**Table 2**). In addition, % weight loss ($R^2 = 0.25$, $p < 0.001$) and menton-gonion length ($R^2 = 0.19$, $p = 0.002$) were independently associated with %AHI change ($R^2 = 0.31$, $p < 0.001$).

The 54 subjects were divided into those who lost ≥ 5% (n = 38) of body weight and those who lost < 5% body weight or gained weight (n = 16). Using the same regression model the only predictor of %AHI change in those that lost more than 5% of body weight was the menton-gonion length (adjusted $R^2 = 0.17$, $p = 0.01$), and for those with weight gain or < 5% weight loss it was % weight change (adjusted $R^2 = 0.27$, $p = 0.034$).

In view of the association between mandibular body length and both improved % AHI and % weight change it is possible that improved sleep with CPAP (i.e., lower AHI) was assisting better weight loss, so we tested the association between CPAP compliance (see **Figures 3A** and **3B**). We found

Table 3—Univariate and multivariate linear regression for variables associated with percentage change in AHI at 2 years.

	Variables Associated with the % Change in AHI at 2 Years	
	Univariate Odds Ratio	Multivariate Adjusted OR
BMI	−0.9 (−3.6 to 1.7)	1.4 (−1.2 to 4.0)
Male sex	−1.6 (−31.3 to 28.1)	−18 (−58 to 22.4)
Age	0.24 (−1.6 to 2.1)	−0.1 (−1.9 to 1.5)
Neck circumference	0.8 (−2.5 to 4.1)	2.1 (−3.0 to 7.2)
Hyoid to PhW (Brander)	−0.4 (−2.6 to 2.5)	−0.6 (−3.8 to 2.7)
Mandibular body length (Me-Go)(mm)	4.5 (2.0 to 7.0)**	3.8 (1.1 to 6.5)**
Mandibular body length (Gn-Go)(mm)	3.8 (1.4 to 6.2)**	Not included
Randomization group (medical = 1)	18.7 (−10.5 to 48)	Not included
% weight change at 2-years	1.9 (1.0 to 2.8)*	1.7 (0.6 to 2.8)**

*p > 0.001. **p < 0.01. Adjusted R^2 for the multivariate model = 0.31. Factors not included as they were each strongly related to factors included: Me-Go and Gn–Go, and randomization group to percentage weight change. AHI, apnea-hypopnea index; BMI, body mass index.

no association between CPAP compliance and the change in either weight or AHI.

Percentage weight loss at 2 years was associated with three independent pre-intervention predictors, namely (a) randomization to the surgical weight loss program, (b) a higher baseline BMI, and (c) a shorter mandibular body length (Me-Go, mm), and together provided an adjusted $R^2 = 0.52$ (**Table 3**).

DISCUSSION

The main findings from this study of patients with severe OSA and severe obesity are twofold. First, using cross-sectional baseline data, 21% of the AHI variance could be explained by neck circumference and no other anthropometric, demographic, or lateral cephalographic measures provided additional explanation of variance. Second, although % weight loss correlated with % change in AHI, the only other variable that correlated with % change in AHI was the mandibular body length. However while highly statistically significant, the menton-gonion length provided an explanation for only 20% of variance. By implication, a shorter jaw was associated with a greater effect of weight loss upon the AHI. A shorter jaw was also associated with better weight loss. However shorter jaw and greater weight loss had independent influence on the reduction in AHI.

Our observation that craniofacial characteristics influence impact of weight loss upon obstructive sleep apnea expands upon previous work by Sutherland, Cistulli, and colleagues.[12] They used CT derived computerized 3D reconstructions of the upper airway in 54 males, (47 years of age, weight 108 kg, BMI 34, and AHI 41/h) to determine the effects of weight loss on the upper airway. Following a mean fall in weight of 8 kg with sibutramine and calorie restriction over 24 weeks, associated with a 16/h fall in AHI, they reported an increase in velopharyngeal airway volume (5.4 to 6.4 cm^2) and reduced facial (mid and lower) and parapharyngeal fat volume, while there was no change in upper airway length nor oropharyngeal or hypopharyngeal measurements. Hyoid to PNS and hyoid to C3 distances decreased, suggesting there was posterior and superior movement of the hyoid bone with weight loss. Using multiple linear regression analysis, 30% of the reduction in AHI could be explained by a reduction in airway length and the reduction in visceral abdominal fat. We were unable to find a significant relationship between baseline airway length and change in AHI. Moreover the group mean airway length did not significantly change before and after weight loss (10 vs 9.9 cm). Therefore the clinical applicability of their study in determining which patients with OSA might benefit from weight loss remains unclear. Allowing for the different techniques used (CT vs cephalogram), study populations, positions in which studies are performed (seated vs supine) and degree of weight loss, the two studies do confirm the complexity of predicting the effects of weight loss upon obstructive sleep apnea severity.

Although there are no previous randomized controlled trials of surgical weight loss, three medical weight loss trials have been undertaken. Tuomilehto et al. (n = 72, weight 97 kg, BMI 32 kg/m^2 and AHI 7/h) reported the effects of intensive healthy lifestyle change to have a greater change in weight (−11 ± 7 vs −2 ± 6 kg) and AHI (−4 ± 6 vs 0 ± 8/h), and more patients were cured at one year as defined by AHI < 5/h (63% vs 13%) than control subjects.[4] Johansson et al. (n = 63, weight 113 kg, BMI 35 kg/m^2 and AHI 37/h) reported a greater weight loss (−19 ± 4 vs 1 ± 2 kg) and lower AHI (−25 ± 17 vs −2 ± 1/h), with more cured (18% vs 0%) in the group randomized to 9 weeks of very low calorie diet compared with a control group.[5] Finally, Foster et al. (n = 264 weight 102 kg, BMI 37 kg/m^2, and AHI 23/h) reported greater weight loss (11 ± 1 vs 1 ± 1 kg) and a greater fall in AHI (−5 ± 2 vs 4 ± 1/h) with OSA cure (36% vs 10%) in those randomized to intensive behavioral modification over one year compared with a control group.[6] Each of these trials has a greater cure than that observed in the current trial; however. populations in these medical trials had lower baseline BMI and AHI.

The fact that significant weight loss (~13% reduction) was not associated with greater numbers of OSA patients being cured may be explained by our entry criteria that generated a mean BMI at entry of 45 kg/m^2 and AHI of 60/h, both much higher than the studies detailed above. In addition our study clearly indicates a strong linear association between modest weight loss and gain with the change in AHI, but weight loss beyond 10% provided little additional benefit.[7] A nonlinear association between weight loss and obesity comorbidity change

was well recognized and highlights the value of modest weight loss rather than the attained BMI and suggests non-mechanical (neural, metabolic, or inflammatory) factors may provide the attenuated benefit of greater weight loss.[3]

Several limitations to this study include the following. Patient positioning in the cephalostat (seated rather than supine and while patient was awake) might be considered a limitation of our study; it should be borne in mind, however, that with the lateral cephalogram being taken with the mandible in a set position, whether the patient was standing or supine should have no effect on any of the bony measurements. Lateral cephalograms do not accurately measure soft tissue and are therefore of limited utility. An additional limitation of this study is an absence of a control group: either non-obese OSA or obese non OSA populations.

In conclusion, in a population with severe OSA and obesity, we have observed that the impact of weight loss upon severity of OSA, as measured by the AHI, is greater in those patients with shorter mandibular body length.

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ACKNOWLEDGMENTS

The authors acknowledge Malcolm Monteith for the medical illustrations used in Figures 1 and 2.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication June, 2014
Submitted in final revised form October, 2014
Accepted for publication November, 2014
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DISCLOSURE STATEMENT

Funding source for this study was National Health and Medical Research Council of Australia Project Grant 436728. Trial was registered with the Australian New Zealand Clinical Trials Registry: ACTRN 12605000161628. URL http://www.anzctr.org.au/trial_view.aspx?ID=183. The authors acknowledge equipment support from Allergen Health for this project. Monash University was the grant recipient. Drs. O'Brien and Dixon have received grants from Allergan Health to conduct investigator initiated research. The other authors have indicated no financial conflicts of interest.