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# Effect of chronic continuous positive airway pressure (CPAP) therapy on upper airway size in patients with sleep apnoea/hypopnoea syndrome

I L Mortimore, P Kochhar, N J Douglas


## Abstract

***Background*** **– There is evidence to suggest that chronic continuous positive airway pressure (CPAP) therapy may produce reversible changes in upper airway morphology and function in patients with sleep apnoea/hypopnoea. This study was designed to examine the effect of chronic CPAP therapy on upper airway calibre.**

***Methods*** **– Twenty four men with the sleep apnoea/hypopnoea syndrome (mean (SE) apnoea/hypopnoea index 37 (5)) underwent lateral cephalometry with measurement of posterior airway space performed before and at least three months after initiation of CPAP therapy.**

***Results*** **– There was no weight change between the two assessments and mean CPAP use was 4·8 (0·4) hours per night. Posterior airway space (PAS) was measured in erect and supine postures. PAS supine increased with CPAP therapy from a mean (SE) of 11·8 (0·8) mm to 13·4 (0·8) mm, but PAS erect did not. Correlation of the change in PAS (dPAS) before and after CPAP therapy showed an increase with increasing CPAP compliance measured as machine run time both for dPAS supine ($r = 0·68$) and dPAS erect ($r = 0·47$).**

***Conclusions*** **– Patients with the sleep apnoea/hypopnoea syndrome regularly using CPAP for more than four hours per night all showed an increase in dPAS supine. The use of chronic CPAP increases PAS supine probably by a reduction in upper airway oedema, and the change in size is dependent on CPAP use.**


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Keywords: continuous positive airway pressure, upper airway size, sleep apnoea/hypopnoea.

**Respiratory Medicine Unit, University of Edinburgh, Edinburgh EH3 9YW, UK**
I L Mortimore
P Kochhar
N J Douglas

Correspondence to:
Dr I L Mortimore, Scottish National Sleep Laboratory, Royal Infirmary, Lauriston Place, Edinburgh EH3 9YW, UK.

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Patients with the sleep apnoea/hypopnoea syndrome (SAHS) often have erythematous and oedematous pharyngeal soft tissues. Their upper airways are smaller than matched normal controls[1 2] and it has been suggested that snoring causes trauma to the upper airway which may result in oedema and narrowing, thus contributing to the severity of sleep apnoea.[3] However, there is dispute as to the effect of chronic continuous positive airway pressure (CPAP) on upper airway size in patients with SAHS. Ryan *et al*[3] reported a significant increase in pharyngeal volume using magnetic resonance imaging (MRI) but the study was based on only five patients. Collop *et al*,[4] also using MRI, found no consistent change in pharyngeal volume with the use of chronic CPAP in 12 patients with SAHS. However, neither study objectively examined change in upper airway size in relation to CPAP compliance, which is often poor in many patients.[5]

We have therefore examined the effect of chronic CPAP therapy on upper airway calibre, comparing differences observed with CPAP compliance.

## Methods

### SUBJECTS

Twenty four men of mean (SE) age 49 (2·0) years, mean body mass index 30 (0·9) kg/m$^2$, mean apnoea/hypopnoea index 37 (5)/hour were recruited for the study. All were weighed and had lateral cephalometry performed the morning after diagnostic polysomnography. After overnight CPAP titration they were issued with a CPAP machine and continued on therapy for at least three months before repeat weighing and lateral cephalometry. At the time of repeat cephalometry CPAP run time (mean total accumulated CPAP use 2150 (475) hours) was noted, together with the patient's own assessment of CPAP use.

### RADIOLOGY

Cephalometry was performed both seated and supine with patients facing at 90° to the *x* ray beam. The patients were directed to gaze forward for the erect films and upwards for the supine films, holding their heads in a natural position. They were asked to close their mouths with their normal resting occlusion and their lips together to allow the tongue to relax onto the floor of the mouth and not to swallow during the exposures. Radiographs were taken with the patient exhaling slowly from a deep breath. The *x* ray cone was positioned exactly five feet from the radiographic film and was placed against the left side of the face.

Posterior airway space (PAS) shown in fig 1 was measured from both the erect and supine films[2 6] as an index of upper airway size by one observer who was unaware of the order of the cephalometry *x* rays.

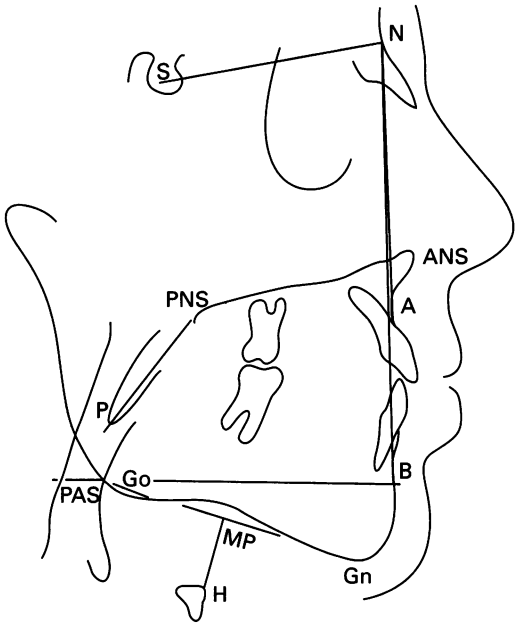


*Figure 1 Diagram of a lateral cephalogram demonstrating posterior airway space (PAS) which is the distance between the tongue base and posterior pharyngeal wall at the level of a line drawn through the supramentale (B) and gonion (Go).*

DATA ANALYSIS

Data were analysed using paired *t* tests and correlation analyses using SPSS-PC.

## Results

Both PAS erect ($r = 0{\cdot}43$; $p<0{\cdot}05$) and PAS supine ($r=0{\cdot}44$; $p<0{\cdot}05$) measured before initiation of CPAP were significantly correlated with the apnoea/hypopnoea index. Overall there was no significant change in mean weight between the performance of the first (92·9 (2·5) kg) and second (93·1 (2·4) kg) lateral cephalometry *x* rays. Mean CPAP run time was 4·8 (0·4) hours per night. Mean PAS erect was not significantly different on CPAP (10·9 (0·8) mm before CPAP and 11·1 (0·7) mm on CPAP), but mean PAS supine was significantly greater ($p<0{\cdot}05$) when patients were on chronic CPAP therapy (13·4 (0·8) mm) than before therapy (11·8 (0·8) mm).

The change in PAS with CPAP (dPAS) supine was significantly correlated with compliance as measured by average CPAP machine run time per night ($r=0{\cdot}68$; $p<0{\cdot}001$) shown in fig 2. The change in PAS (dPAS) erect was also correlated with CPAP compliance ($r=0{\cdot}47$; $p=0{\cdot}02$). dPAS erect ($r=-0{\cdot}57$; $p<0{\cdot}005$) and supine ($r=-0{\cdot}56$; $p<0{\cdot}005$) were both correlated with PAS measured before CPAP therapy. There was no correlation between total accumulated CPAP use (in hours) and dPAS erect or supine. Overall, there was no significant correlation between dPAS supine or erect and change in weight, although three patients (who were poor CPAP compliers) gained 3–6 kg during the study with corresponding decreases in PAS. These patients contributed to the negative values of dPAS supine shown in fig 2.

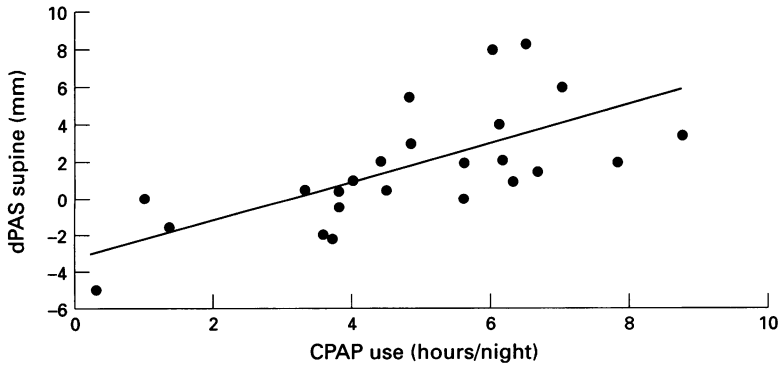


*Figure 2 Relationship between change in posterior airway space (dPAS) supine and compliance with continuous positive airway pressure (CPAP) therapy ($r=0{\cdot}68$; $p<0{\cdot}001$).*

## Discussion

This study shows that the upper airway calibre of patients with SAHS is increased by chronic CPAP therapy and that the magnitude of the increase is correlated with CPAP compliance. PAS is a measurement of retroglossal airway size and is relevant to SAHS because collapse of the upper airway occurs either at the level of the tongue or retropalatally.[7–9] PAS is reported to be useful as a negative predictor of SAHS severity,[6] which we have confirmed in the present study with significant correlations between PAS (erect and supine) and the apnoea/hypopnoea index. PAS supine increased significantly on CPAP ($p<0{\cdot}05$) and PAS erect showed a non-significant trend. Both dPAS erect and dPAS supine were significantly correlated with average nightly CPAP machine run time and also PAS measured before commencing chronic CPAP therapy. There was no correlation with dPAS and total accumulated CPAP use. This suggests that the change in upper airway diameter is at least partly dependent on average nightly CPAP use and may change more in patients with the smallest upper airways (and therefore the most severe sleep apnoea). Several patients increased their weight during the study with corresponding reductions in PAS. These patients were also poor CPAP compliers which would artificially improve the correlation between dPAS and CPAP compliance shown in fig 1. However, elimination of the patients who gained weight still gave a significant correlation between the two variables ($r = 0{\cdot}47$; $p<0{\cdot}05$; $n = 21$), and overall there was no significant correlation between weight change and dPAS.

Lateral cephalometry of normal subjects and patients with SAHS shows that PAS is greater supine than erect,[2] an effect attributed to increased genioglossus muscle activity.[10] However, retropalatal airway size is decreased in the supine compared with the erect posture in patients with SAHS but not in normal subjects.[2] The results presented here show that PAS is dependent on posture and are comparable with the results of Yildirim *et al.*[2] In the present study PAS supine is better correlated with the apnoea/hypopnoea index and CPAP compliance than PAS erect. This is probably because the supine posture is usually adopted during sleep and may also relate to the ob-

servation that the upper airway is functionally compromised when supine and during sleep as indicated by increased airway resistance,[11] apnoea/hypopnoea index,[12 13] and genioglossus muscle activity.[10] This may enhance the discriminatory effect of PAS supine as an index of upper airway size/function compared with PAS erect.

There are several possible explanations for the change in upper airway size when patients are on chronic CPAP therapy. Weight loss or gain could obviously contribute,[14] but in the present study the mean group weight did not change significantly and dPAS did not correlate with weight change. Reduction of upper airway oedema with chronic CPAP use has been reported in a small study[3] and is an attractive hypothesis which certainly corresponds to our own clinical observations that pharyngeal oedema and erythema are reduced once patients are established on CPAP. Differences in the positional effects of oedema on the upper airway may at least partly explain the changes observed in dPAS with posture. A third explanation is that upper airway dilator muscle function is in some way improved by CPAP therapy as suggested by Sullivan *et al.*[15] A preliminary report[16] shows that reflex palatal muscle activity in response to negative pressure increases with CPAP use in patients with SAHS. This could be due to removal of oedema by CPAP, allowing improved detection of pressure change in the upper airway with a corresponding increase in muscle activity. An alternative explanation is that the upper airway muscles of patients with SAHS are fatigued by constantly trying to maintain a compromised airway[17–19] and CPAP therefore rests these muscles[20] with a corresponding improvement in function. Chronic CPAP therapy in SAHS has also been shown to reduce upper airway collapsibility[21] which may be related to a reduction in sleep fragmentation with a corresponding increase in upper airway muscle activity.[22]

In summary, upper airway calibre increases in patients with SAHS treated with chronic CPAP and the effect is related to average nightly compliance. This may result in improved upper airway function and could be a factor contributing to the reduction in the apnoea/hypopnoea index on the first night after regular CPAP use is stopped[23] and may help to explain why some patients find intermittent use of their CPAP machines beneficial. The results also suggest that the possibility of reducing CPAP pressure after patients are established on CPAP should be examined.

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1 Stauffer J L, Zwillich C W, Cadieux R J, Bixler E O, Kales A, Varano L A, *et al.* Pharyngeal size and resistance in obstructive sleep apnea. *Am Rev Respir Dis* 1987;**136**: 623–7.
2 Yildirim N, Fitzpatrick M F, Whyte K F, Jalleh R, Wightman A J A, Douglas N J. The effect of posture on upper airway dimensions in normal subjects and in patients with the sleep apnea/hypopnea syndrome. *Am Rev Respir Dis* 1991; **144**:845–7.
3 Ryan C F, Lowe A A, Li D, Fleetham J A. Magnetic resonance imaging of the upper airway in obstructive sleep apnea before and after chronic nasal continuous positive airway pressure therapy. *Am Rev Respir Dis* 1991;**144**: 939–44.
4 Collop N A, Block A J, Hellard D. The effect of nightly nasal CPAP treatment on underlying obstructive sleep apnea and pharyngeal size. *Chest* 1991;**99**:855–60.
5 Engleman H M, Martin S E, Douglas N J. Compliance with CPAP therapy in patients with the sleep apnoea/ hypopnoea syndrome. *Thorax* 1994;**49**:263–6.
6 Partinen M, Guilleminault C, Quera-Salva M-A, Jamieson A. Obstructive sleep apnea and cephalometric roentgenograms. *Chest* 1988;**93**:1199–205.
7 Hudgel D W. Variable site of airway narrowing among obstructive sleep apnea patients. *J Appl Physiol* 1986;**61**: 1403–9.
8 Stein M G, Gamsu G, De Greer G, Golden J A, Crumley R L, Webb W R. Cine CT in obstructive sleep apnea. *Am J Radiology* 1987;**148**:1069–74.
9 Shephard J W, Thawley S E. Localization of upper airway collapse during sleep in patients with obstructive sleep apnea. *Am Rev Respir Dis* 1990;**141**:1350–5.
10 Douglas N J, Jan M A, Yildirim N, Warren P M, Drummond G B. The effect of posture and breathing route on genioglossal electromyogram activity in normal subjects and in patients with the sleep apnea/hypopnea syndrome. *Am Rev Respir Dis* 1993;**148**:1341–5.
11 Anch A M, Remmers J E, Bunce H. Supraglottic airway resistance in normal subjects and patients with occlusive sleep apnea. *J Appl Physiol* 1982;**53**:1158–63.
12 Cartwright R D. Effects of sleep position on sleep apnea severity. *Sleep* 1984;7:110–4.
13 McEvoy R D, Sharp D J, Thornton T. The effects of posture on obstructive sleep apnea. *Am Rev Respir Dis* 1986;**133**: 662–6.
14 Rubinstein I, Colapinto N, Rotstein L E, Brown I G, Hoffstein V. Improvement in upper airway function after weight loss in patients with obstructive sleep apnea. *Am Rev Respir Dis* 1988;**138**:1192–5.
15 Sullivan C, Issa F, Berthon-Jones M, McCauley V, Costas L. Home treatment of obstructive sleep apnea with continuous positive airway pressure applied through the nosemask. *Bull Eur Physiopathol Respir* 1985;**20**:49–54.
16 Mortimore IL, Douglas N J. Effect of CPAP treatment on reflex palatal muscle activity in sleep apnea patients. *Am J Respir Crit Care Med* 1995;**151**:A537.
17 Suratt P M, McTier R F, Wilhoit C. Upper airway muscle activation is augmented in patients with obstructive sleep apnea compared with that in normal subjects. *Am Rev Respir Dis* 1988;**137**:889–94.
18 Mezzanotte W S, Tangel D J, White D P. Waking genioglossal electromyogram in sleep apnea patients versus normal controls (a neuromuscular compensatory mechanism). *J Clin Invest* 1992;**89**:1571–9.
19 Tangel D J, Mezzanotte W S, White D P. Influence of sleep on tensor palatini EMG and upper airway resistance in normal men. *J Appl Physiol* 1991;**70**:2574–81.
20 Alex C G, Aronson R M, Onal E, Lopata M. Effects of continuous positive airway pressure on upper airway and respiratory muscle activity. *J Appl Physiol* 1987;**62**:2026–30.
21 Series F, Roy N, Marc I. Effects of sleep deprivation and sleep fragmentation on upper airway collapsibility in normal subjects. *Am J Respir Crit Care Med* 1994;**150**:481–5.
22 Leiter J C, Knuth S L, Bartlett D. The effect of sleep deprivation on activity of the genioglossus muscle. *Am Rev Respir Dis* 1985;**132**:1242–5.
23 Kribbs N B, Pack A I, Kline L R, Getsy J E, Schuett J S, Henry J N, *et al.* Effects of one night without nasal CPAP treatment on sleep and sleepiness in patients with obstructive sleep apnea. *Am Rev Respir Dis* 1993;**147**: 1162–8.