Oral airway resistance during wakefulness in patients with obstructive sleep apnoea

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Abstract

Background—Patients with obstructive sleep apnoea (OSA) have a number of upper airway structural abnormalities which may influence the resistance of the oral airway to airflow. There have been no systematic studies of the flow dynamics of the oral cavity in such patients.

Methods—Inspiratory oral airway resistance to airflow ($R_o$) was measured in 13 awake patients with OSA in both the upright and supine positions (neck position constant). Each subject breathed via a mouthpiece while the nasal airway was occluded with a nasal mask.

Results—In the upright position the mean (SE) $R_o$ was 1.26 (0.19) cm H$_2$O/l/s (at 0.4 l/s) which increased to 2.01 (0.43) cm H$_2$O/l/s when supine ($p<0.05$, paired t test). The magnitude of this change correlated negatively with the respiratory disturbance index ($r = -0.60, p = 0.03$).

Conclusion—In awake patients with OSA $R_o$ is normal when upright but abnormally raised when in the supine position.

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Keywords: obstructive sleep apnoea; oral airway resistance; body position

The role of upstream resistance in the pathophysiology of inspiratory narrowing/collapse of the pharyngeal airway in patients with obstructive sleep apnoea (OSA) has been explored by a number of investigators, almost exclusively in terms of the influence of nasal airflow resistance on sleep disordered breathing events.1–3 During nasal breathing the nasal passages constitute the relevant upstream inspiratory resistor whereas during mouth breathing the oral cavity is the potential site of upstream resistance.

The resistance to airflow through the oral cavity ($R_o$) is a major component of total upper airway resistance during oral and oronasal breathing.1–3 Mouth breathing occurs during sleep, even in normal subjects, and may be associated with an increased incidence of sleep disordered breathing events.8 Patients with OSA frequently have mandibular/orthodontic abnormalities8 and enlargement of the tongue,9 all of which could contribute to an increased $R_o$. During periods of oral breathing while asleep a high $R_o$ may be associated with more negative inspiratory intraluminal pressures in the oropharynx and hypopharynx, thus increasing susceptibility to airway narrowing and collapse. Furthermore, if this increased $R_o$ was primarily associated with structural changes in the upper airway—for example, enlargement of the tongue—then $R_o$ might also be increased during wakefulness in patients with OSA.

While total upper airway resistance,9 nasal resistance,1 and pharyngeal resistance10 have all been studied extensively in OSA, there have been no systematic studies of the flow dynamics of the oral cavity in such patients. In the present study we have measured $R_o$ in a group of patients with OSA during wakefulness, examined the influence of posture on $R_o$, and studied the fluid mechanics of oral airflow in OSA.

Methods

SUBJECTS

Inspiratory $R_o$ was measured in both the upright and supine positions in 13 awake patients (10 men) of mean (SE) age 51.0 (3.2) years, body mass index (BMI) 37.3 (1.6) kg/m$^2$, with symptoms of moderate to severe OSA. The diagnosis was confirmed by overnight polysomnography11 and $R_o$ measurements were performed within a four month period of the polysomnography. No patient was undergoing treatment with nasal continuous positive airway pressure (CPAP) at the time the measurements were made and none wore dental plates. Informed consent was obtained from each subject and the protocol was approved by the Western Sydney Area Health Service human ethics committee.

MEASUREMENT OF INSPIRATORY $R_o$

Each study was performed with the subject breathing quietly via a standard mouthpiece (Sensor Medics, internal cross sectional area 300 mm$^2$, Middle Park, Victoria, Australia). The mouthpiece was connected to a heated pneumotachograph (Fleisch #2, Gould, Bilthoven, the Netherlands) which was coupled to a differential pressure transducer ($\pm 10$ cm H$_2$O, Celesco Transducer Products, IDM Instruments, Dandenong, Victoria, Australia) for the measurement of oral airflow. An occluded nasal CPAP mask (Sullivan, Respmed, Sydney, NSW, Australia) was placed over the nose and checked to ensure the absence of leaks. With the occluded nasal mask in place, only oral breathing was possible. Since there was no nasal route airflow, pressure measured inside the mask reflected oropharyngeal pressure. Transoral pressure was then measured using a differential pressure transducer (MP 45, $\pm 100$ cm H$_2$O, Validyne, Northridge, California, USA), one side of which was connected to the mouthpiece while the other side was...
connected to the nasal mask. Both flow and pressure signals were digitised using a sampling frequency of 50–100 Hz and recorded directly on a computer. The data were stored on disk for subsequent analysis.

**PROTOCOL**

Subjects were studied first in the upright (seated) position and then supine. Neck position was maintained constant throughout the study by ensuring that the measured distance from the chin (tip of mandible) to the manubriosternal notch remained unchanged (9–11 cm).

**DATA ANALYSIS**

Inspiratory $R_O$ was calculated directly from pressure-flow plots reconstructed from the stored data. An inspiratory transoral pressure-flow plot was constructed from data obtained during 4–5 consecutive stable and representative breaths from each run. A power function of the form $P = aV^b$ (where $P$ is transoral pressure, $V$ is oral flow, and $a$ and $b$ are constants) was then fitted to the inspiratory transoral pressure-flow curve by the method of least squares. Only data exhibiting no phase lag between the pressure and flow signals (that is, no looping of the transoral pressure-flow plot around zero flow) were accepted for analysis. In this manner data which may have been influenced by partial narrowing of the nasopharyngeal airway were excluded. Inspiratory $R_O$ was then calculated from this relationship.

The power function fitted the data with an $r^2$ value of >0.94 for the upright position and >0.91 for the supine position across all the runs. The values for the $a$ constant ranged from 1.43 to 4.49 when upright and from 1.16 to 11.43 when supine. For the whole group the mean $R_O$ was 1.26 (0.19) cm H$_2$O/l/s (CV 55.4%) in the upright position and this increased significantly to 2.01 (0.43) cm H$_2$O/l/s (CV 76.4%; p<0.05) in the supine position.

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There was no relationship between awake upright $R_O$ and RDI ($r = -0.09$, p>0.7). However, when supine there was a borderline significant trend ($r = -0.52$, p = 0.07) for those individuals with a higher $R_O$ (>2.0 cm H$_2$O/l/s) to be the least severely affected by their disease (RDI < 5 events/hour). This negative relationship between a high $R_O$ when supine and disease severity was stronger when the correlation between the absolute change in $R_O$ (in moving from upright to supine) and RDI was examined ($r = -0.60$, p = 0.03, fig 2). Thus, those individuals with no change or only a small increase or decrease in $R_O$ when moving to the supine position tended to have a higher RDI than did those patients in whom the $R_O$ increased substantially.

There was also a significant positive relationship between BMI and RDI ($r = 0.62$, p = 0.03) and a significant negative relationship between BMI and $R_O$ when supine ($r = -0.56$, p = 0.05), as well as the change in $R_O$ in moving from upright to supine ($r = -0.63$, p = 0.02).
Oral airway resistance in OSA

425

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The increase in R0 found with change in body position in patients with OSA is in contrast to results obtained in normal subjects using the same technique. In this latter study there was no significant difference in R0 when upright and supine in a group of 17 normal men. The mean value for upright R0 measured in the present study (1.26 (0.19) cm H2O/l/s) was slightly higher than that obtained in the normal subjects (0.86 (0.23) cm H2O/l/s), perhaps because of slightly more head and neck flexion in the patients (chin to manubrio-
sternal notch distance of 9–11 cm in patients compared with 14 cm in normal subjects). Alternatively, the trend for a higher R0 in patients with OSA might reflect real anatomical differences between the patients and the normal subjects. The previously studied nor-
mal subjects were younger (36 (2) years) and had a smaller BMI (26.4 (0.9) kg/m2) than the patients in the present study. It is therefore possible that the difference between the two studies is a reflection of anthropometric characteristics rather than OSA per se. In any case, the mean supine value (2.01 (0.43) cm H2O/l/s) in the patients was double that measured in the normal subjects (0.90 (0.16) cm H2O/l/s) [25]. Thus, when awake and upright, patients with OSA and a high BMI have a relatively normal R0. However, unlike normal subjects, on assuming the supine position the R0 increases.

It has long been recognised that patients with OSA tend to have a reduced upper airway cross sectional area [26–27] compared with matched control subjects, even while awake and upright. When the anatomy of the upper airway of patients with OSA is compared with that of normal subjects, most attention has been focused on the retropalatal and retroglossal airway segments since these regions are the principal sites of occlusion during obstructive apnoeas. In general, patients with OSA have smaller pharyngeal airways which are more collapsible, are shaped differently, and are more likely to be narrowed in the supine position than those of normal subjects.

The anatomical abnormalities of the upper airway in patients with OSA are associated with an increased upper airway resistance to airflow [28]. While normal subjects maintain a constant upper airway resistance between the upright and supine positions, pharyngeal resis-
tance tends to increase in patients with OSA when they are supine [29, 30]. The present study demonstrates that oral airway resistance be-
haves in a similar manner provided the head, neck, lip, and jaw position is maintained constant. This is in agreement with a brief report by Kawano et al [30] who also found an increase in R0 (measurement method not described) when patients with OSA moved from the upright to the supine position.

A feature of the difference in upper airway anatomy between patients with OSA and normal subjects is tongue size, patients with OSA having a greater tongue cross sectional area which may be related to airway inflam-
mation and/or oedema or an adaptive increase in muscle mass related to upper airway muscle hyperactivity. In addition, Pae et al [31] have shown that the cross sectional area of the tongue of patients with OSA increased by 4.3% while the oropharyngeal area decreased by 36.5% when changing from the upright to the supine position, but no changes were observed in normal subjects. These findings suggest that changes in tongue size or position may be responsible for the increase in R0 found in the patients in the present study when in the supine position.

A feature of our findings was the negative relationship between the change in R0 when in the supine position and the severity of OSA as measured by RDI. A potential explanation for this finding may lie in the response of the tongue to changes in posture. Tongue position depends on the degree of recruitment of genioglossus muscle activity. Assumption of the supine position has been shown to recruit genioglossus muscle activity in both normal subjects and patients with OSA. This response is thought to help preserve oropharyngeal dimensions. Indeed, oropharyngeal diam-
eter has been shown to increase in normal subjects and patients with OSA [32] in the supine position. However, in other studies the oropharynx has been found to narrow in some OSA patients in the supine position. Thus, there appears to be a heterogeneous response by patients with OSA to the supine position, the oropharynx widening in some individuals.

Figure 2 Change in oral resistance (R0) at 0.4 l/s from upright to supine positions during wakefulness in 13 OSA patients plotted against respiratory disturbance index (RDI). Linear regression line, correlation coefficient (r), and p value are shown.

Discussion
Measurements of R0 were obtained with patients awake and breathing on a standard mouthpiece. A mouthpiece was used in order to standardise the degree of mouth opening. This approach provides a measurement of R0 which is reflective of structures posterior to the teeth. We hypothesised that, if R0 is raised in awake subjects with OSA, it would be because of anatomical abnormalities posterior to the dental arcades—for example, tongue enlarge-
ment. Consequently, our study focuses on R0 measurements obtained with the lips and teeth in a constant and standardised position and in awake subjects. It should therefore be empha-
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and narrowing in others. In their study of genioglossus muscle recruitment Douglas et al. found that, while most patients with OSA had substantially increased genioglossus electromyographic activity in the supine position, some patients did not. We speculate that patients who maintain their pharyngeal dimensions in the supine position do so by recruiting genioglossus muscle activity which moves the base of the tongue forward into the oral cavity and away from the posterior pharyngeal wall. Indeed, voluntary tongue protrusion does lead to an increase in cross sectional area of the oropharynx in awake patients with OSA when supine. When lip and teeth position are fixed, this movement of the tongue may result in a narrowing of the oral cavity (although a widening of the oropharynx) and an increase in $R_n$, (although a decrease in pharyngeal resistance), especially in individuals with a large tongue. Since during sleep airflow is predominantly via the nasal pathway and occlusive apneas are predominantly related to pharyngeal collapse, patients in whom the $R_n$ increases in the supine position may protect their pharyngeal airway from collapse more effectively than subjects who are unable to mount such a response and therefore preserve $R_n$, but with narrowing of the pharyngeal airway. These proposed mechanisms, however, need to be validated with direct experimental testing. In addition, it is not clear if the response is related to anthropometric characteristics (since there was also a significant negative relationship between BMI and supine $R_n$) or to the severity of OSA per se.

In contrast to our previous study in normal subjects, values for the $a$ constant of the fitted power function in the present study also increased significantly when patients moved from the upright to the supine position. This finding confirms that $R_n$ increases in the supine position at all the flow rates encountered. The values for the $b$ constant were in the range indicating a turbulent to orifice flow regime and were unaffected by body position. This contrasts with our previous study of normal subjects in which $b$ values did increase in the supine position. Thus, during mouthpiece breathing a turbulent flow regime exists in the oral cavity in patients with OSA, as it does in normal subjects. 

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