

Effects of posture on flow-volume curves during normocapnia and hypercapnia in patients with obstructive sleep apnoea

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Abstract

Background A high ratio of forced expiratory to forced inspiratory maximal flow at 50% of vital capacity (FEF_{50}/FIF_{50}) may identify upper airway dysfunction. Since hypercapnia increases the motor activity of airway dilating muscles its effects on the maximum expiratory and inspiratory flow-volume curves (MEIFV) in patients with obstructive sleep apnoea and in normal subjects in different postures was studied. **Methods** The effects of posture on the maximum expiratory and inspiratory flow-volume curves during the breathing of air and 7% carbon dioxide in 11 patients with obstructive sleep apnoea were compared with those in nine normal subjects. Measurements were made in the sitting, supine, and right lateral recumbent positions. Forced expiratory flow at 50% vital capacity (FEF_{50}), forced inspiratory flow at 50% vital capacity (FIF_{50}) and FEF_{50}/FIF_{50} were determined. **Results** In the normal subjects FEF_{50} , FIF_{50} , and FEF_{50}/FIF_{50} were not affected by change in posture or by breathing carbon dioxide. In the patients there was a fall in FIF_{50} and an increase in FEF_{50}/FIF_{50} when breathing air in the supine position compared with values in the seated and lateral position. While they were breathing carbon dioxide there was a slight increase in FEF_{50} when patients were seated or in the lateral position compared with values during air breathing. Hypercapnia abolished the effects of posture on FEF_{50}/FIF_{50} . Values for FEF_{50}/FIF_{50} in the supine position while they were breathing air correlated with the apnoeic index but not with other polysomnographic data.

Conclusion In patients with obstructive sleep apnoea the upper airway is prone to collapse during inspiration when the patient is supine, even when awake; this tendency can be reversed by breathing carbon dioxide.

Patients with obstructive sleep apnoea have episodic obstruction of the upper airway during sleep.¹⁻⁴ The maximum expiratory and inspiratory flow-volume curve (MEIFV) shows two abnormalities related to upper air-

way dysfunction. A "saw tooth" configuration on the expiratory or inspiratory flow, or both, is thought to be due to pharyngeal fluttering⁵ whereas a high ratio of forced expiratory maximal flow at 50% of vital capacity to forced inspiratory maximal flow at 50% of vital capacity (FEF_{50}/FIF_{50}) may identify upper airway obstruction.⁶

In one report on patients with obstructive sleep apnoea FEF_{50}/FIF_{50} was greater in the supine position than the seated position.⁷ Upper airway resistance is greater in the supine than the sitting posture.⁸ Thus in patients with obstructive sleep apnoea the upper airway is less able to remain patent during inspiration when the patient is supine, even when awake. Since hypercapnia increases the motor neurone activity of the upper airway dilating muscles⁹ and decreases upper airway resistance¹⁰ it may increase maximal flow, particularly inspiratory flow, in the supine position. The first purpose of this study was to determine whether hypercapnia influences the effects of posture on MEIFV curves in patients with obstructive sleep apnoea and normal subjects.

Episodes of obstructive apnoea depend on the posture assumed during sleep, being more frequent in the supine than the lateral position.^{11,12} The degree of upper airway obstruction estimated from the MEIFV curve in the supine position may therefore correlate better with obstructive apnoeic episodes than do estimates from measurements made in other postures. Our second purpose therefore was to examine the relation between the data obtained from MEIFV curves in the supine position and polysomnographic data in patients with obstructive sleep apnoea.

Methods

SUBJECTS

Eleven patients (10 men, one woman) with obstructive sleep apnoea and nine normal men were studied. The table shows the anthropometric data. There were no significant differences in age, height, or weight between the two groups. All patients had a history of snoring and excessive daytime sleepiness, and five complained of morning headache. Four patients had hypertrophy of the palatopharyngeal fold with narrowing of the pharyngeal lumen. No other anatomical abnormalities

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Anthropometric and spirometric data and ventilatory response to hypercapnia in normal subjects and patients with sleep apnoea syndrome. Values are means (SE)

	No of subjects	Age (years)	Height (cm)	Weight (% predicted)	VC (% predicted)	FEV ₁ /VC (%)	$\Delta\dot{V}_E/\Delta P_{ETCO_2}$ (l/min/mm Hg)	$\Delta P_{0.1}/\Delta P_{ETCO_2}$ (cm H ₂ O/mm Hg)
Normal subjects	9	43.6 (2.9)	168 (2)	103 (5)	113.9 (4.0)	82.6 (2.5)	1.80 (0.28)	0.42 (0.02)
Patients	11	51.6 (2.7)	161 (2)	118 (6)	101.7 (6.0)	84.1 (2.0)	1.94 (0.16)	0.46 (0.08)

VC, slow vital capacity; FEV₁/VC, forced expiratory volume in one second divided by VC; \dot{V}_E , minute ventilation; P_{0.1}, mouth pressure 0.1 second after onset of an occluded inspiration; P_{ETCO₂}, end tidal carbon dioxide tension.

were identified. Written informed consent was obtained from each subject before the start of the experiments.

FLOW-VOLUME CURVES

Slow vital capacity and forced vital capacity in one second (FEV₁) were measured with a spirometer (Benedict-Roth 13.5-L spirometer, Tatebe Seishudo, Tokyo, Japan). After 15 minutes MEIFV curves were measured with a direct writing flow-volume recorder (Chest, Tokyo, Japan)¹³ in the sitting, supine, and right lateral recumbent positions in a random fashion while subjects were breathing air (normocapnia) and in hypercapnia. MEIFV curves during hypercapnia were obtained after the breathing of 7% carbon dioxide in oxygen for five minutes with a non-breathing circuit. The breathing circuit of the flow volume recorder was filled with 7% carbon dioxide in oxygen. At least two flow-volume curves were obtained for each posture, and the one with higher expiratory and inspiratory flows was used for analysis. Ten minutes elapsed between the two trials. Subjects were coached to avoid volume leaks at the mouthpiece, and care was taken to keep the head, neck, and trunk configuration as constant as possible in the three postures.¹⁴ Subjects were taught to avoid neck flexion and extension and to use maximal expiratory and inspiratory effort in all postures.

Four indices were derived from accepted flow-volume curves: firstly, presence or absence of a "saw tooth" configuration, which was defined as three or more consecutive peaks and troughs occurring at regular intervals and not exceeding 300 ml during the middle half of the vital capacity in expiration, inspiration, or both⁵; secondly, forced expiratory flow at 50% vital capacity (FEF₅₀); thirdly, forced inspiratory flow at 50% vital capacity (FIF₅₀); and, fourthly, the ratio of FEF₅₀ to FIF₅₀ (FEF₅₀/FIF₅₀).

VENTILATORY RESPONSE TO HYPERCAPNIA

Ventilatory response to hypercapnia was assessed by rebreathing 7% carbon dioxide in oxygen. Each subject wore a noseclip and breathed through a mouthpiece connected to a carbon dioxide rebreathing circuit.¹⁵ Minute ventilation (\dot{V}_E) and mouth pressure 0.1 seconds after the onset of an occluded inspiration (P_{0.1}) were plotted against end tidal carbon dioxide tension (P_{ETCO₂}) and analysed by linear regression. The slopes of the two regressions ($\Delta\dot{V}_E/\Delta P_{ETCO_2}$, $\Delta P_{0.1}/\Delta P_{ETCO_2}$)

were used as indices of the ventilatory response to hypercapnia.

POLYSOMNOGRAPHY

All patients were studied at least twice by standard polysomnographic techniques, including electroencephalography, electrooculography, monitoring of respiratory cage movement, and electrocardiography.¹⁶ Respiratory movements of the rib cage and abdomen with tidal volume summation were measured by inductive plethysmography (Respirace, Ambulatory Monitoring, Ardsley, New York, United States). Arterial oxygen saturation was measured continuously with a pulse oximeter (Biox 3700, Ohmeda, Boulder, Colorado, United States). Airflow at the nose and mouth was recorded with two thermistors. Tracheal sounds were detected with a microphone attached to the skin over the trachea and were band pass filtered (200 to 1000 Hz), rectified, and integrated to display the sound envelope.^{17,18} All variables were recorded continuously on a polygraph (Model 360, NEC San-ei, Tokyo, Japan) and data recorder (A-109; Sony, Japan). Data from the second night were used in the analysis to avoid the first night effect.¹⁹

Apnoea was defined as the stopping of airflow at the nose and mouth and of breath sound at the trachea for longer than 10 seconds.¹⁶ Obstructive apnoea was identified as an absence of airflow signals despite continuing thoracoabdominal movement (Respirace), and central apnoea as stopping of airflow signals with complete absence of thoracoabdominal movements.¹⁶

DATA ANALYSIS

Data from normal subjects and patients with obstructive sleep apnoea were compared by unpaired Student's *t* test, and matched paired data within each group by paired Student's *t* test. Correlation coefficients were calculated by linear regression analysis and linear regression by the least squares method. Values of *p* below 0.05 were considered significant (two tailed test). Data are expressed as means and standard errors of the mean (SE).

Results

Spirometric data and ventilatory response to hypercapnia are summarised in the table. The slow vital capacity (VC) and FEV₁/VC in the patients with obstructive sleep apnoea were within the normal range and did not differ from

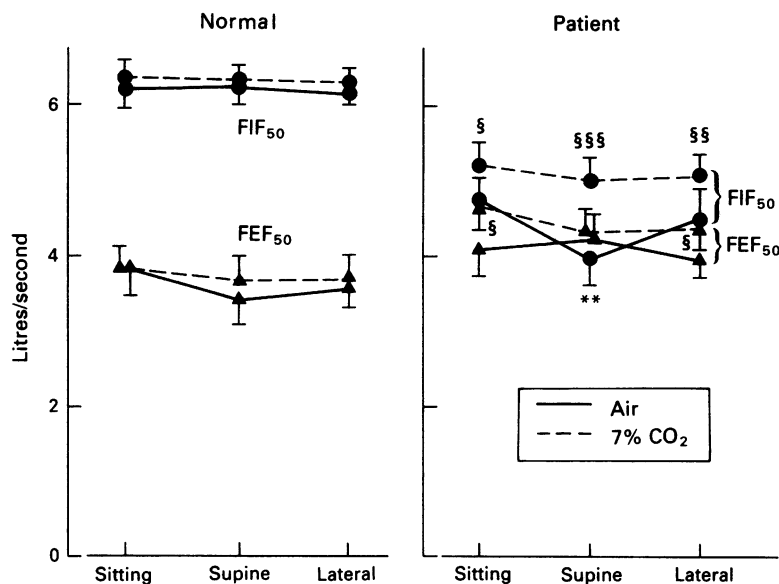


Figure 1 Effects of posture on forced expiratory (▲) and inspiratory (●) flow rate at 50% of vital capacity during breathing of air (continuous line) and 7% carbon dioxide (dashed line) in normal subjects (left) and in patients with obstructive sleep apnoea (right). ** $p < 0.01$ compared with sitting and lateral positions; § $p < 0.05$, §§ $p < 0.01$, §§§ $p < 0.001$ compared with corresponding values during air breathing. Bars show SE of mean.

those in normal subjects. There was no significant difference in ventilatory response to hypercapnia between the two groups.

Figure 1 shows the mean (SE) flow-volume measurements. In the normal subjects FEF₅₀ and FIF₅₀ did not differ for the three postures during normocapnia or hypercapnia, nor were there any differences in either measurement between hypercapnia and normocapnia.

The FEF₅₀ in patients did not significantly differ from that in the normal group for any posture during normocapnia or hypercapnia. FEF₅₀ was significantly higher, however, during hypercapnia when patients were in the sitting and lateral postures compared with the corresponding values during normocapnia. The FIF₅₀ was significantly lower in the supine position than values in the other two positions during normocapnia, and the FIF₅₀ in all three positions was significantly higher during hypercapnia than during normocapnia; this was most noticeable in the supine position. The FIF₅₀ was significantly lower in patients than in the normal subjects for all postures during both normocapnia and hypercapnia.

A saw tooth configuration was observed in only four obese patients (body weight 120–150% of ideal) and was seen in both expiratory and inspiratory flows. The saw tooth shape was not affected by posture or hypercapnia.

Figure 2 shows the mean (SE) values for FEF₅₀/FIF₅₀ during normocapnia and hypercapnia in patients and normal subjects. In the normal subjects there were no significant differences in FEF₅₀/FIF₅₀ between the three postures and between normocapnia and hypercapnia in each posture. During normocapnia FEF₅₀/FIF₅₀ in patients was significantly greater in the supine than in the sitting and lateral postures, with values exceeding one

(suggesting extrathoracic obstruction) in four patients when seated and in seven when supine. The FEF₅₀/FIF₅₀ was not significantly different in the lateral and sitting posture during normocapnia. During hypercapnia the FEF₅₀/FIF₅₀ in the supine position decreased to values seen in the sitting and lateral position. FEF₅₀/FIF₅₀ did not differ between normocapnia and hypercapnia in either sitting or lateral postures. FEF₅₀/FIF₅₀ was significantly greater in patients than in normal subjects.

Ventilatory responses to hypercapnia did not differ significantly between patients and normal subjects (table) and were similar to control values.¹⁵

There was a significant correlation between FEF₅₀/FIF₅₀ in the supine position and the apnoea index (apnoeic episodes per hour) but no correlation between FEF₅₀/FIF₅₀ and longest duration of apnoea or lowest arterial oxygen saturation overnight (fig 3). The FEF₅₀/FIF₅₀ values in the sitting and lateral positions did not significantly correlate with the polysomnographic data. FEF₅₀ and FIF₅₀ in any posture did not correlate significantly with polysomnographic data or percentage of ideal body weight.

Discussion

We compared maximum flow values measured from flow-volume curves from normal subjects and patients with obstructive sleep apnoea in different postures and under hypercapnic stimulation. FEF₅₀, FIF₅₀, and FEF₅₀/FIF₅₀ were not affected by posture during normocapnia or hypercapnia in normal subjects. During normocapnia the FEF₅₀/FIF₅₀ in patients was

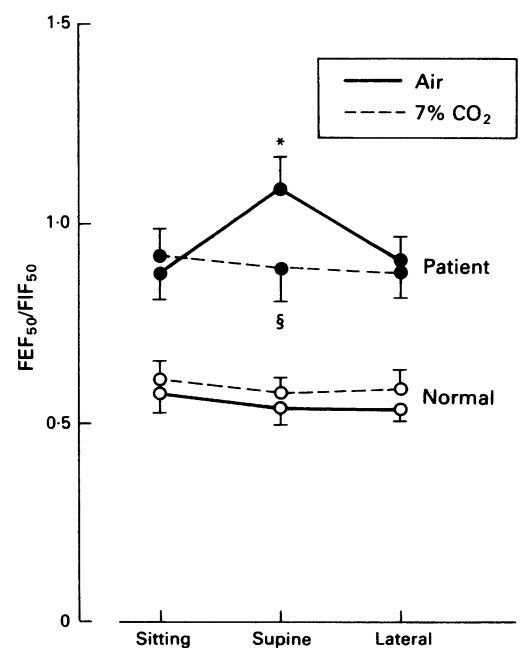


Figure 2 Postural effects on ratio of forced expiratory flow to forced inspiratory flow at 50% of vital capacity (FEF₅₀/FIF₅₀) during breathing of air (continuous line) and 7% carbon dioxide (dashed line) in normal subjects (○) and in patients with obstructive sleep apnoea (●). * $p < 0.05$ compared with sitting and lateral positions; § $p < 0.05$ compared with corresponding values during air breathing. Bars show SE of mean.

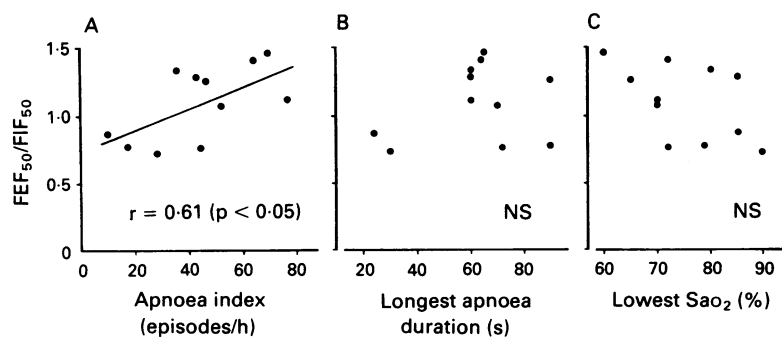


Figure 3 Relations between ratio of expiratory flow to inspiratory flow at 50% of vital capacity (FEF_{50}/FIF_{50}) in supine posture during normocapnia and polysomnographic data in patients with obstructive sleep apnoea.

greater in the supine than in the sitting or lateral postures because of a reduced FIF_{50} . During hypercapnia the supine value for FEF_{50}/FIF_{50} decreased, with an increase in FIF_{50} to the level of that obtained in the other two postures. The saw tooth configuration was not affected by postural change or hypercapnic stimulation. In supine patients FEF_{50}/FIF_{50} during normocapnia correlated with the apnoea index.

Neck posture can affect the flow curve, with neck extension increasing maximum expiratory flows because of increased longitudinal tension on the trachea causing an increase in wave speed limited flows.^{20,21} Our subjects were trained to avoid neck extension and flexion and to keep the configuration of the head, neck, and trunk as constant as possible in the three postures; the effect of neck configuration on our results seems to be negligible. Hypoxic dip rate correlates most closely with neck size,²² and this may explain the differences in the configuration of the flow-volume curve between normal subjects and patients. We did not measure neck size.

Castile *et al* reported that changes in posture result in significant changes in the maximum expiratory flow-volume configuration in normal subjects, which are best seen in slope ratio-volume plots.¹⁴ In our study flow-volume configuration was not calculated. FEF_{50} did not differ significantly between the three postures during normocapnia and hypercapnia either in normal subjects or in patients. FEF_{50} may not reflect the small changes in local airway stresses but can be detected from the flow-volume configuration.

We found no significant change in FEF_{50} , FIF_{50} , or FEF_{50}/FIF_{50} in normal subjects in relation to posture or hypercapnic stimulation. These findings partly support the results of Rodarte and Hyatt, who found no change in maximal expiratory flow in subjects breathing carbon dioxide.²³ They suggested that postural changes or adding extra respiratory drive do not alter the maximum respiratory manoeuvre in normal subjects because a maximum effort has been made and there is no capacity to increase the patency of the upper airway further.

The fall in maximum inspiratory flow that

occurred when the patients adopted a supine posture may be caused by movement of mobile soft tissues, such as the tongue and soft palate, towards the posterior pharyngeal wall or by an increase in mucosal and submucosal thickness due to vascular congestion.⁸ Hypercapnia increased both expiratory and inspiratory flow and abolished the effects of posture on FEF_{50}/FIF_{50} . The mechanism governing the effect of carbon dioxide on the flow-volume curve is not clear. One possible explanation is that hypercapnia is helping to maintain upper airway patency during forced inspiration, either indirectly by stimulating respiratory control through central and peripheral chemoreceptors or directly by stimulating upper airway muscles.^{9,10} Alternatively, hypercapnia may affect alertness and eliminate drowsiness by inducing an arousal response and increasing upper airway dilating muscle activity, resulting in upper airway dilation in the supine posture.

All flow-volume curves were measured while the subjects were awake. The observation concerning hypercapnic effects on upper airway muscle activity therefore may not be applicable to patients when asleep. In patients with obstructive sleep apnoea during sleep, not only the arousal response to hypercapnia or hypoxia, or both,^{24,25} but the negative intraoesophageal pressure during each frustrated inspiration of an obstructed episode is a powerful stimulus to breathe.²⁶

The value of the flow-volume curve in the diagnosis of obstructive sleep apnoea is controversial.^{7,27-29} Although several authors have reported that the flow-volume curve was related to upper airway dysfunction in patients with obstructive sleep apnoea,^{7,27,28} recent studies suggest that the flow-volume curve is not useful in the diagnosis of obstructive sleep apnoea.²⁹ We found that the FEF_{50}/FIF_{50} during normocapnia in the supine posture correlated with apnoeic episodes. This measurement in awake patients might be useful in predicting apnoeic episodes in patients with obstructive sleep apnoea, as suggested previously.^{7,27,28} As the saw tooth appearance was not affected by posture or by breathing carbon dioxide it may not be specific for obstructive sleep apnoea.

In summary, we studied maximum expiratory and inspiratory flow-volume curves during both normocapnia and hypercapnia in normal subjects and patients with obstructive sleep apnoea. Our findings show that the upper airway in patients with obstructive sleep apnoea is less able to remain patent during inspiration when the patient is supine, even when awake, and this tendency can be reversed by breathing carbon dioxide.

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