Flow-volume curves and sleep-disordered breathing: therapeutic implications

EDWARD F HAPONIK, PHILIP L SMITH, JOSEPH KAPLAN, EUGENE R BLEECKER

From the Pulmonary Division and the Baltimore Regional Sleep Disorders Center, Department of Medicine, Baltimore City Hospitals and Johns Hopkins Medical Institutions, Baltimore, Maryland, USA

ABSTRACT   To assess the relationship of abnormal flow-volume curves during awake periods to the clinical severity of sleep-disordered breathing and the need for surgical treatment of obstructive sleep apnoea, flow-volume curves were measured in 72 adults with obstructive apnoea. Patients in whom surgery was recommended for standard clinical indications had significantly lower inspiratory flow rates ($p < 0.01$) and a higher incidence of flow-volume curves indicating extrathoracic airway obstruction ($p < 0.01$) than did non-surgical patients. These abnormal flow-volume curves correlated with an increased severity of nocturnal oxygen desaturation ($p < 0.01$). Furthermore, increases in inspiratory flow rates measured serially in 22 patients were related to improvement in their polysomnography ($p < 0.05$), suggesting that alterations of airway function during awake periods correlate with changes in the severity of sleep apnoea.

Physiological abnormalities that might predispose to upper airway occlusion during sleep have been observed in patients with obstructive sleep apnoea while they were awake.[1][2] Flow-volume curves indicating decreased inspiratory flow rates and a configuration consistent with variable extrathoracic airway obstruction (fig 1) are found in about 40% of this population, often in the absence of obvious anatomical upper airway abnormalitites.[3] Sanders and coworkers observed a "sawtooth pattern" in the flow-volume curves of patients with sleep apnoea,[4] and Perks and colleagues noted abnormal flow-volume curves in patients with Scheie's syndrome[5] and acromegaly.[6] These physiological abnormalities of upper airway function in patients with sleep apnoea are associated with anatomical narrowing of the pharynx demonstrable by computerised tomography.[7]

Although the flow-volume curve appears to be useful diagnostically in patients with sleep apnoea, the therapeutic implications of this non-invasive test

of upper airway function have not been evaluated. Therefore to determine whether the presence of abnormal flow-volume curves correlates with the clinical severity of sleep-disordered breathing and the need for surgical treatment, flow-volume curves were obtained in 72 adults with obstructive apnoea. Serial recordings were made in 22 patients to assess whether alterations in airway function while they were awake occurred with treatment and whether these were associated with changes in breathing patterns during sleep.

Methods

The patients comprised 60 men and 12 women, who were studied at the Baltimore Regional Sleep Disorders Center over a two-year period. All had a diagnosis of obstructive sleep apnoea established by polysomnography. This was performed by a standardised technique that included simultaneous recordings of an electroencephalogram ($C_3 - A_2$ lead), electrocardiogram, oxygen saturation measured by an ear oximeter (Model No 472–1A: Hewlett-Packard, Waltham, Montana), thoraco-abdominal motion detected by mercury strain gauges (Parks Electronics, Beaverton, Oregon), nasal and oral airflow measured by thermistors (Grass TCT1R: Grass Instruments Co, Quincy, Montana), and videotape recordings of the patient during sleep. All

physiological variables were recorded on either a Beckman DM dynograph (Beckman Instruments, Schiller Park, Illinois) or a Grass polygraph (Grass Instruments Co). The study was interpreted according to conventional criteria,[8] independently of the results of flow-volume curves.

Sleep-disordered breathing events were categorised as apnoea or hypopnoea. Apnoea was defined as cessation of respiratory airflow for 10 seconds or longer. Central apnoea was defined as simultaneous absence of airflow and thoracoabdominal motion and obstructive apnoea as the absence of airflow despite persistent and usually progressively accentuated thoracoabdominal movements. These events were usually terminated by a resuscitative snort with subsequent restoration of airflow and arousal according to the EEG. Hypopnoea was defined by decreased nasal and oral airflow with continued thoracoabdominal movement, accompanied by EEG signs of arousal and a fall in oxygen saturation of at least 4% from the baseline level while the patients were awake and supine. At least 30 episodes of apnoea or hypopnoea during a six-hour nocturnal sleep period, or a rate exceeding five episodes an hour, were required to make the diagnosis of sleep-disordered breathing. The frequency of all episodes was expressed as the disordered-breathing rate. The severity of oxygen desaturation during the sleep study was expressed as the maximum fall in oxygen saturation from the baseline level when patients were awake and supine($\Delta Sao_2\%$).

Three reproducible flow-volume curves were obtained in the sitting position in all patients. These were derived by differentiation of the volume signal from a Stead Wells spirometer equipped with a linear potentiometer (Warren E Collins, Braintree, Montana), which was displayed on a storage oscilloscope (Tektronix, Beaverton, Oregon) and photographed with a polaroid camera. Two reviewers interpreted the studies without knowledge of the outcome of polysomnography or therapeutic recommendations. The flow-volume curves were categorised as consistent with variable extrathoracic airway obstruction if the ratio of forced expiratory flow at 50% of the vital capacity to forced inspiratory flow at 50% of the vital capacity (FEF_{50}/FIF_{50}) exceeded 1.0[9-11] (fig 1). A sawtooth pattern of the flow-volume curve was recognised from the distinct, regular oscillations of flow.[4]

Recommendations for treatment were made without consideration of the flow-volume curve data. Indications for surgery included the presence of an obvious anatomical upper airway obstruction (identified during routine physical examination, indirect laryngoscopy, or fibreoptic nasopharyngoscopy),

potentially life-threatening cardiovascular abnormalities associated with the sleep-related breathing disorders, and excessive daytime somnolence that was both incapacitating and unresponsive to medical treatment. Cardiovascular complications included cor pulmonale and cardiac arrhythmias (atrioventricular block, sinus arrest, premature atrial or ventricular contractions). Shifts from sinus bradycardia to sinus tachycardia were not regarded as an indication for surgery, since these rhythm changes are frequently associated with apnoea episodes.

Serial polysomnography and pulmonary function studies were carried out before and after treatment in 22 patients. Follow-up polysomnograms were compared with the original ones, and were considered to show improvement if there was at least a 20% decrease in disordered-breathing rate and also a 10% improvement in maximum $\Delta Sao_2\%$ during both non-rapid-eye-movement and rapid-eye-movement sleep.

Statistical analysis was performed by χ^2 test and Student's t test for unpaired and paired variables respectively.[12]

Results

Non-surgical treatment was recommended in 47 patients, and included nocturnal oxygen,[13] weight reduction, or drug treatment with protriptyline[14-17] or medroxyprogesterone.[18] Surgical treatment was ad-
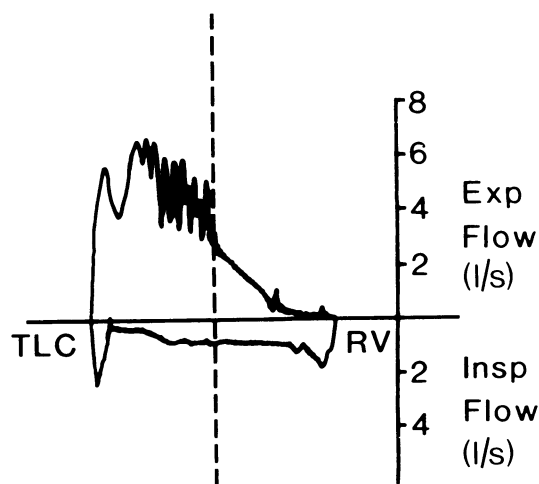

Fig 1 *Flow-volume curve in a patient with obstructive sleep apnoea. The plateau of inspiratory flow rates and an $FEF_{50}/FIF_{50} > 1.0$ are consistent with variable extrathoracic airway obstruction. Sawtoothing is present on the expiratory curve. The vertical broken line denotes 50% of the vital capacity. TLC—total lung capacity; RV—residual volume.*

Table 1 *Obstructive sleep apnoea: indications for surgery*

Indication	No of patients
Anatomical obstruction	6*
Tumour	1
Laryngeal web	1
Micrognathia	2
Tonsillar hypertrophy	2
Cardiovascular complications	7*
Arrhythmias	4
Cor pulmonale	3
Incapacitating hypersomnolence	16*

*More than one major criterion was present in four patients.

vised in the remaining 25 patients and included tracheostomy[19] [20] in 16 patients, tonsillectomy in two patients, and uvulopalatopharyngoplasty[21] in seven patients.

The indications for surgery are outlined in table 1. Six patients had obvious anatomical upper airway obstruction. One patient had a benign tumour (lymphangioma), one had a laryngeal web, two patients had micrognathia with a bird-like face as described by Coccagnia *et al,*[22] and two patients had massive tonsillar hypertrophy.[23] Cardiovascular complications were present in four of these patients, and in three who did not have anatomical abnormalities. Four patients had cardiac arrhythmias associated with episodes of oxygen desaturation; these included frequent sinus arrests of up to 12 seconds in one patient, paroxysmal atrial tachycardia in another, and frequent multifocal premature ventricular contractions in two. All the cardiac arrhythmias occurred in patients with a history of heart disease. Three patients had cor pulmonale, presumably because of obstructive sleep apnoea and the associated nocturnal oxygen desaturation. Thus surgery was advocated as initial treatment in the nine patients with anatomical obstruction or cardiac complications or both. Four of these patients, all with anatomical upper airway obstruction, refused this treatment. Either uvulopalatopharyngoplasty or tracheostomy was recommended in 16 other patients because of persistent and incapacitating daytime somnolence that was unrelieved by medical treatment, and 13 of these had surgery.

The clinical characteristics of the patients, including age, disordered breathing rate, and the maximum fall of oxygen saturation in non-REM sleep from the baseline level when they were awake and supine are shown in fig 2. The surgical and non-surgical groups were of similar age and had a similar number of disordered-breathing episodes per hour of sleep. All patients experienced clinically important falls in oxygen saturation and these were significantly more severe in the patients who had surgery (p < 0·01).
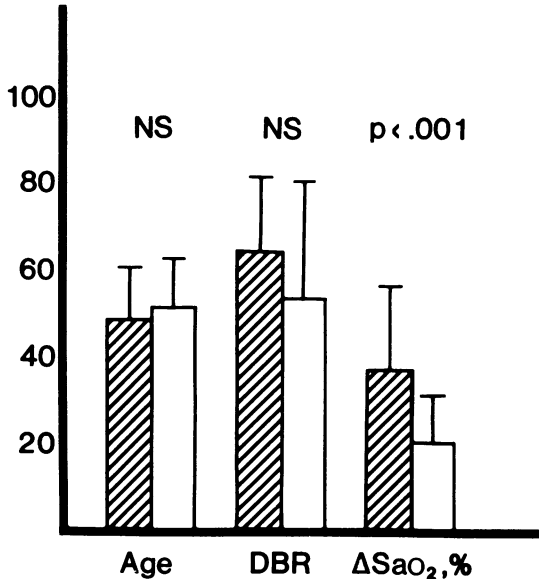

Fig 2 *Clinical characteristics of patients with obstructive sleep apnoea. Surgical and non-surgical patients were similar with regard to age and disordered-breathing rate (DBR), but surgical patients had more severe falls in oxygen saturation (ΔSao_2, %). The values recorded are those obtained during non-rapid-eye-movement sleep.*
▨ *Surgical;* □ *non-surgical. The vertical axis indicates numbers of patients.*

The results of flow-volume curves are shown in table 2. Twenty-five of the 72 patients (35%) had flow-volume curves that were consistent with variable extrathoracic airway obstruction. This finding was not associated with a significantly higher disordered-breathing rate, but was associated with a more severe fall in oxygen saturation. Patients with extrathoracic obstruction had a maximum fall in Sao_2% of 33·0 (20·8) (mean and SD) while those without extrathoracic obstruction had a fall in Sao_2% of only 20·9 (12·3) (p < 0·01). Abnormal curves were obtained in only eight of 47 (17%) referred for medical treatment, but in 17 of 25 (68%) in whom surgery was recommended. This relationship was significant, but might have been accounted for by the patients with obvious anatomical upper airway abnormalities, all of whom had decreased inspiratory flow rates. The association of abnormal flow-volume curves with the requirement for surgery remains significant, however, if analysis is limited to patients without apparent anatomical obstruction (p < 0·01), or if those who refused surgery are excluded (p < 0·01). The relationship between treatment recommendations and the results of flow-volume curve measurements are shown in fig 3. Peak expiratory flow rates (PEF) in surgical patients were

Table 2 *Relationship between flow-volume curve abnormalities and clinical severity of sleep apnoea*

	No of patients	Disordered breathing rate (mean (SE) episodes/h)	Maximum ΔSao_2 (%, mean (SE))[2]	No with surgical indications
Extrathoric airway obstruction				
Present	25	56·6 (28·8)	33·0 (20·8)	17
Absent	47	54·2 (28·7)	20·9 (12·3)	8
p		NS	p < 0·01	p < 0·0˙
Sawtooth pattern				
Present	36	59·3 (26·9)	25·9 (17·2)	15
Absent	36	51·3 (28·9)	24·4 (16·8)	10
p		NS	NS	NS

ΔSao_2—change in percentage oxygen saturation of arterial blood; NS—not significant.

not significantly different from those of non-surgical patients. Their peak inspiratory flow rates (PIF), however (4·08 (1·8) l/s), were lower (p < 0·01) and the ratio of FEF_{50} to FIF_{50} (1·41 (0·88)) was higher (p < 0·001) than that of non-surgical patients (5·55 (2·3) l/s and 0·82 (0·36) respectively). The surgical patients were slightly more obese, with higher weight:height ratios than non-surgical patients, but obesity itself did not correlate with the flow volume measurements.

A sawtooth pattern of the flow-volume curve[4] was present on the expiratory curves of 36 patients (50%), and occurred more often in surgical (15/25, 60%) than non-surgical patients (21/47, 44·7%). Sawtoothing did not correlate with either the disor-

Fig 3 *Flow-volume curve measurements in patients with obstructive apnoea. Peak expiratory flow rate (PEF) was similar in both groups, but peak inspiratory flow rate (PIF) was lower and the ratio of forced expiratory flow at 50% vital capacity to forced inspiratory flow at 50% vital capacity (FEF_{50}/FIF_{50}) was higher in surgical patients. Surgical; non-surgical. Units of PEF and PIF are l/s.*

dered-breathing rate or the severity of oxygen desaturation (table 2).

Serial pulmonary function studies and polysomnography were performed in 22 patients. All had excessive daytime somnolence, abnormal disordered-breathing rates, and large falls in oxygen saturation during sleep. Treatment consisted of the following: 16 patients received drug treatment (14 protriptyline, two amitriptyline), three had upper airway surgery (one tonsillectomy, two uvulopalatopharyngoplasty), one with obvious tonsillar hypertrophy refused surgery and was treated with a cervical collar, and two refused all treatment. According to the criteria previously described, 11 of these patients (seven having drugs, three who had surgery, and one who had a collar) had improved polysomnograms. Their mean disordered-breathing rates fell significantly from 63·8 (20·8) episodes an hour to 30·8 (19·7) episodes an hour (p < 0·01) and the maximum percentage fall in oxygen saturation during apnoeic periods in non-REM sleep was reduced from 36·9 (19·2) to 12·6 (10·2) (p < 0·01). Eleven patients had no improvement in polysomnography: their disordered-breathing rates did not change (55·3 (20·7) baseline, 52·6 (33·8) follow-up; p > 0·5), and their maximum percentage fall in oxygen saturation was unchanged (25·1% (16·1%) baseline, 26·1 (18·7%) follow-up, p > 0·5).

The relationship of serial measurements of inspiratory flow rates when the patients were awake to the results of polysomnography is summarised in table 3. PIF and FIF_{50} increased significantly (p < 0·05) in patients with improvement in their sleep-disordered breathing. PIF worsened (p < 0·05), and FIF_{50} was unchanged in patients without polysomnographic improvement at the time of their follow-up examination. Mean $FEF_{50}:FIF_{50}$ was unchanged in both groups, but changes in the configuration of flow-volume curves occurred in eight patients. In six the baseline flow-volume curves were consistent with upper airway obstruction and reverted to a normal pattern after treatment, and all of these had improved polysomnograms. One patient whose sleep
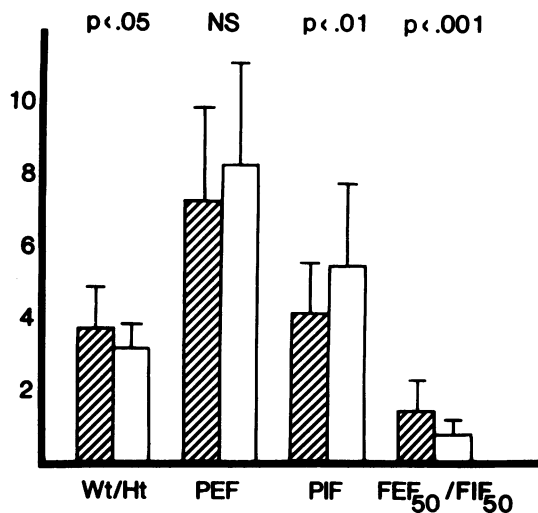
Table 3 *Association of serial flow-volume curve measurements* (means and SD) with polysomnographic findings*

Flow-volume curve	Polysomnogram result	
	Improved (n = 11)	*Unimproved (n = 11)*
Peak inspiratory flow rate		
Baseline	5·5 (2·1)	5·4 (1·9)
Follow-up	6·4 (1·8)†	4·4 (1·5)†
FIF_{50}		
Baseline	5·2 (2·1)	4·8 (1·9)
Follow-up	6·2 (1·8)†	4·1 (1·6)
FEF_{50}/FIF_{50}		
Baseline	0·97 (0·3)	1·1 (1·1)
Follow-up	0·83 (0·4)	1·3 (1·0)

*Flow rates expressed in l/s.
†Difference between baseline and follow-up results significant: $p < 0.05$.
FIF_{50}—forced inspiratory flow at 50% vital capacity; FEF_{50}—forced expiratory flow at 50% vital capacity.

apnoea worsened had curves at follow-up suggesting extrathoracic obstruction that was not present at the time of the initial study. Another patient had less apnoea but an increased FEF_{50}/FIF_{50}, an apparent discrepancy related to the patient's severe obstructive pulmonary disease (which considerably lowered the FEF_{50} in his baseline expiratory curve). In the 21 patients without obstructive pulmonary disease conversion of the flow-volume curve to a normal pattern correlated ($p < 0.05$) with polysomnographic improvement.

Discussion

These data further confirm the relationship that exists between upper airway function while patients are awake and the presence of an abnormal breathing pattern with oxygen desaturation during sleep.[3][4][7] Abnormalities of flow-volume curves not only were associated with obstructive sleep apnoea but also correlated with its severity. Patients with airway dysfunction while awake had more severe nocturnal hypoxaemia and more medical complications that required surgical intervention. Furthermore, improvement in upper airway function while they were awake was associated with a decrease in the severity of the sleep-related breathing disorder.

Increased awareness has resulted in the more frequent diagnosis of obstructive sleep apnoea. Treatment for this disorder continues to evolve, however, and there are no absolute guidelines for surgery. Tracheostomy[19][20] has become the established mode of treatment for patients with obvious anatomical upper airway obstruction or life-threatening cardiovascular complications. The latter occurred in only seven (9·7%) of our patients, and was less

common than previously suspected.[24] In patients with less severe clinical manifestations of sleep apnoea tracheostomy may be unacceptable. Furthermore, the availability of less invasive forms of treatment[13-18][21][25-29] may further modify the need for surgery. In our group of patients hypersomnolence was the major reason for surgery and accounted for 64% of those in whom such treatment was recommended. The fact that the degree of oxygen desaturation was more severe in this group than in the non-surgical patients with fewer symptoms supports the hypothesis that hypoxaemia may be responsible for much of the excessive somnolence associated with obstructive apnoea.[30][31]

The relationship between the results of flow-volume curves and polysomnography might have been anticipated in patients with obvious anatomical upper airway abnormalities, but they represented only 8% of our patients, and only 24% of those in whom surgery was advised. This low frequency of upper airway abnormalities apparent in the patients when awake probably reflects the relative insensitivity of conventional techniques for the assessment of the upper airway. When computed tomography is used for evaluation of pharyngeal structure, airway narrowing is detectable during awake periods in most patients with obstructive sleep apnoea.[7] We believe that the flow-volume curves reflect this upper airway narrowing and, in addition, the tendency for the airway to collapse during forced inspiration.

Since flow-volume curves are useful in the assessment of upper airway function, we might predict that improved flows would correlate with improvements in obstructive apnoea. As can be seen, increased inspiratory flow rates were associated with decreases in the disordered-breathing rate and in the degree of nocturnal oxygen desaturation. Furthermore, the conversion of an abnormal configuration of the flow-volume curve to a normal pattern, suggesting a decrease in upper airway obstruction, correlated with polysomnographic improvement. Even though the changes in inspiratory flow rates were small, they were associated with significant improvement in the severity of obstructive apnoea, emphasising the delicate balance that exists between the forces maintaining upper airway patency and those producing airway occlusion.

It was not surprising that improvement both of inspiratory flow rates when patients were awake and of sleep-disordered breathing occurred after the surgical correction of obvious anatomical upper airway obstruction in three of our patients. Similar postoperative changes in flow-volume curves have been seen in patients without sleep apnoea.[32] The increased PIF and FIF_{50} of the patient treated with a cervical collar are also consistent with improvement

in anatomical upper airway obstruction and with reported effects of head position on upper airway resistance. Inspiratory flow rates also increased in some of the patients treated with protriptyline, a nonsedating tricyclic antidepressant, which has been used successfully for the treatment of patients with moderately severe sleep-disordered breathing.[14-17] Although this drug is thought to block competitively presynaptic α-receptors and has major anticholinergic properties, its mechanism of action in patients with obstructive apnoea remains unclear. The increases in inspiratory flow rates while they were awake in patients whose sleep-disordered breathing improved with protriptyline might be explained by altered tone of the upper airway musculature, improved function or co-ordination of other inspiratory muscles, or changes in neural or cortical control.

Anatomical or functional upper airway obstruction detectable while patients are awake occurs more commonly in obstructive sleep apnoea than is generally appreciated. We do not suggest that the abnormal flow-volume curve represents an independent indication for surgical treatment; but we believe that this simple, reproducible test provides prognostic as well as diagnostic information and might prove useful in guiding decisions about management. Experience with patients with obstructive sleep apnoea indicates that objective analysis of sleep is required to assess the effects of treatment. Flow-volume measurements may also have a role in monitoring the clinical course of patients with obstructive apnoea, although at present there is no substitute for the careful physiological assessment of sleep.

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Book notice

Pulmonary Function Testing Guidelines and Controversies.
JL Clausen (ed). (Pp 368; $39·95.) Academic Press, 1982.

This is essentially a practical manual covering the detailed methods of pulmonary function testing with recommended normal reference data. Particularly welcome are the emphasis on quality control and trouble shooting and honest discussion of areas of uncertainty and controversy. The style is necessarily didactic, but largely based on common sense. Although written primarily for an American audience, most of the information and opinions travel well and the references are comprehensive and up to date. The criterion for inclusion of a test is its popularity in various laboratories rather than a critical appraisal of its clinical role, and a further volume is promised on the more difficult and controversial area of clinical interpretation and usefulness. Unconventional inclusions such as radiographic estimation of lung volume and measurements in the intensive care unit are offset by surprising omissions, including peak flow, Kco, and mixed venous carbon dioxide tension as measured by rebreathing; and the important area of radioisotope studies is not covered. Despite these deficiencies, physicians and senior technicians concerned with clinical lung function testing will find much of interest and value in this book and they will be reassured to discover that their everyday practical problems are shared by others.

Notice

Annual meeting of the European Academy of Allergology and Clinical Immunology

The annual meeting of the European Academy of Allergology and Clinical Immunology 1984 will be held in Brussels on 16–19 May. The themes will be the immunological aspects of asthma, occupational allergy, and immunomodulation, with a postgraduate course on the treatment of allergic diseases. Further information from Professor R Pauwels, Department of Respiratory Diseases, De Pintelaan 185, B 9000 Ghent, Belgium.

Corrections

Flow-volume curves and sleep-disordered breathing
We regret the following errors in the paper by Dr EF Haponik and others (August 1983, pp 609–15): On p 609, col 2, para 2, lines 10 and 14; and p 610, col 1, para 3, line 6, "Montana" should be "Massachusetts." In table 2, 2nd and 3rd column headings, "SE" should be "SD."

Respiratory muscle and pulmonary function in polymyositis and other proximal myopathies
We regret the following errors in the paper by Dr NMT Braun and others (August 1983, pp 616–23): On p 617, col 1, para 2, line 3, "Montana" should be "Massachusetts." In table 4, 2nd line of heading, "observations" should be "subjects."