Control of nocturnal hypoventilation by nasal intermittent positive pressure ventilation

N CARROLL, M A BRANTHWAITE

From Brompton Hospital, London

ABSTRACT Ten patients with respiratory failure and nocturnal hypoventilation were treated for three to nine months by nasal intermittent positive pressure ventilation. Four patients had chronic obstructive lung disease (median FEV₁ 19% predicted) and six restrictive chest wall disorders (median FVC 25% predicted); eight of the patients also had cardiac failure. The median daytime arterial oxygen tension, measured before and after at least three months' treatment, increased from 6.2 (range 5.4-9.6) to 9.1 (7.1-9.8) kPa in those with restrictive disease ($p < 0.05$), and from 6.0 (5.7-6.5) to 7.1 (6.3-7.7) kPa in the four with airflow limitation (NS). Median values for arterial carbon dioxide tension over the same time fell from 8.2 (range 6.7-9.8) to 6.5 (6.0-6.9) kPa in the group with restrictive disease ($p < 0.05$) and from 8.2 (7.0-9.2) to 7.1 (4.9-7.7) kPa in those with airflow limitation ($p < 0.02$). Total sleep time while patients were using nasal positive pressure ventilation varied from 155 to 379 (median 341) minutes, and included 4-26% rapid eye movement sleep (median 14%). The percentage of monitored time during the night in which the arterial oxygen saturation was less than 80% fell from a median (range) of 96 (3-100) to 4 (0-9) in the six patients with restrictive disease and from 100 (98-100) to 40 (2-51) in those with airflow limitation. There were no changes in spirometric values but exercise tolerance improved in all patients. The technique may prove an acceptable alternative to long term domiciliary oxygen therapy in selected patients.

Introduction

Nocturnal hypoventilation is a feature of respiratory muscle weakness,¹ restrictive chest wall disease,² and chronic obstructive lung disease,³ and is believed to contribute to the development of respiratory and cardiorespiratory failure. Control of this sleep related breathing disturbance relieves symptoms, improves daytime ventilatory function, and prolongs survival.⁴ Patients with respiratory muscle weakness have been treated successfully with intermittent positive pressure ventilation delivered through a well fitting nasal mask,^{5,6} and we have used the same technique to control nocturnal hypoventilation in patients with restrictive defects or chronic obstructive pulmonary disease.

Patients and methods

We selected 10 patients (eight of them men) aged 41-65 years. Four have chronic airflow limitation and six

restrictive disorders of the chest wall. Two of the six with restrictive disease (patients 5 and 9) have pulmonary fibrosis secondary to tuberculosis and ankylosing spondylitis respectively, and patient 5 also has airflow limitation (FEV₁/FVC ratio 55%). Clinical details are given in table 1. Hypercapnic respiratory failure was found in all the patients at the outset, and eight presented with signs of cor pulmonale. The six patients with restrictive chest wall disease had had their disease inadequately controlled with protriptyline,⁷ three developed an unacceptable degree of upper airway obstruction when ventilated mechanically in a tank ventilator, and two who had used negative pressure ventilation at home for many months had poor control of their disease and required frequent readmission to hospital. One ambulant outpatient (No 10) had been using a tank ventilator every night in a local hospital for more than a year. Two of the four patients with chronic obstructive lung disease had shown some improvement in diurnal arterial blood gas tensions while using protriptyline but long term treatment was precluded by anticholinergic side effects. Three of these four refused treatment with long term domiciliary oxygen because of the constraint of using it for 15 hours each day, and the fourth developed

Address for reprint requests: Dr M A Branthwaite, Brompton Hospital, London SW3 6HP.

Accepted 15 January 1987

Table 1 Details of patients on recruitment to the study

Patient No	Diagnosis	Age (y)	Sex	Weight (kg)	Lung volumes	Pao ₂ † (kPa)	Paco ₂ ‡ (kPa)
					FEV ₁ (% pred†)		
1	Chronic	65	M	69	16	5.9	9.2
2	obstructive	62	M	62	19	5.7	8.1
3	lung disease	63	M	88	19	6.5	7.0
4		65	M	90	28	6.2	8.2
					FVC (% pred†)		
*5	Pulmonary tuberculosis, lobectomy, thoracoplasty	53	M	67	22	5.4	8.1
*6	Congenital scoliosis	41	F	50	11	5.8	8.2
7	Congenital scoliosis						
	ankylosing spondylitis	41	F	39	27	8.3	8.0
8	Paralytic scoliosis	56	M	37	12	6.6	8.3
9	Ankylosing spondylitis						
	pulmonary fibrosis	60	M	57	45	5.5	9.8
*10	Paralytic scoliosis	54	M	55	39	9.6	6.7

*Used negative pressure ventilation at night before transition to nasal intermittent positive pressure ventilation.

†Based on age, sex, and either height or span.

‡Before intermittent positive pressure ventilation.

FVC—forced vital capacity; Pao₂, Paco₂—arterial oxygen and carbon dioxide tension.

symptomatic hypercapnia after one night's sleep using controlled oxygen therapy.

Arterial blood gas analysis and spirometry were performed in all subjects at the outset, when their condition was stable in terms of exercise tolerance, control of weight in those with cor pulmonale, and lack of infected sputum in those with chronic obstructive pulmonary disease. Measurements were made in the late afternoon with the subjects seated and breathing air spontaneously.

Oxygen saturation and transcutaneous carbon dioxide tension were recorded continuously during sleep with a Biox III oximeter and Hewlett-Packard 47210A capnometer coupled to a Linseis pen recorder. A Prime computer was used to digitise the trace and display condensed data graphically, as well as a histogram of the proportion of time spent at successive deciles of oxygen saturation. All the patients breathed air while asleep, including two (Nos 5 and 6) who were studied initially while using a negative pressure jacket because they were unable to sleep unaided. Patient 10, who had been using a tank ventilator at night for the previous year, was studied while breathing spontaneously during sleep.

The four patients with chronic obstructive pulmonary disease were investigated in more detail before treatment began. A two night sleep study was carried out, with recordings of the electroencephalogram (biparietal electrodes), electro-oculogram, electrocardiogram, chest and abdominal movement (magnetometers), oronasal gas flow (thermistors), and oximetric and capnographic measurements. Records from the second night were analysed and sleep was

staged according to conventional criteria.⁸

After the preliminary assessment all patients were acclimatised to intermittent positive pressure ventilation by nasal mask with the apparatus and technique described previously.^{9*} Once they were able to sleep comfortably with the apparatus, oxygen saturation and transcutaneous carbon dioxide tension were recorded during a single night study to conform that optimum settings had been achieved. The patient was then discharged home. One patient (No 5), who developed unacceptable hypercapnia with added oxygen while breathing spontaneously or using negative pressure ventilation during sleep, remained hypoxaemic during nasal intermittent positive pressure ventilation with air and his peripheral oedema failed to resolve. Oxygen with a flow rate of 1 l/min was added through a port on the nasal mask from six months onwards.

All patients were reassessed from three to nine months (median four months) later, when all stated that they had complied with the advice to use the ventilator at home while sleeping at night. Arterial blood gas analysis and spirometry were repeated in the late afternoon while the resting patient breathed air spontaneously. Polysomnography was carried out as described previously on all patients. All subjects were ventilated with air, patient 5 using added oxygen at 1 l/min. Data were analysed with Student's paired *t* test.

*Respironics nasal mask, available from Medicaid Ltd, West Sussex, and Lifecare PLV 100 ventilator (Thomas Respiratory Systems, London) or Monnal D ventilator (Deva Medical Ltd, Runcorn, Cheshire).

Results

The six patients with restrictive disorders had a median forced vital capacity (FVC) 25% of the value predicted from their arm span (range 11.2–45%). Those with chronic airflow limitation had a median FEV₁ of 19.3% of the predicted value (range 16–28%). Analysis of arterial blood confirmed the presence of hypercapnic respiratory failure by day before treatment (median arterial oxygen tension (PaO₂) 6.0, range 5.4–9.6 kPa; median carbon dioxide tension (PaCO₂) 8.2, range 6.7–9.8 kPa). During sleep arterial oxygen saturation fell and arterial carbon dioxide tension increased in all eight spontaneously breathing subjects, and oxygen saturation fell in the two subjects with chest wall restriction (patients 5 and 6) who were studied while using negative pressure ventilation.

When reassessed at least three months after treatment with nasal intermittent positive pressure ventilation at night, all patients reported symptomatic improvement and an increase in exercise tolerance. In the patients with chronic airflow limitation the six minute walking distance was unchanged in one, fell slightly in another, and increased by more than half in

the other two. The median (range) distance walked increased from 254 (135–508) to 359 (123–508) metres. Four patients with a restrictive disorder who had been housebound previously had become mobile, and only one of the 10 has required hospital admission (for a respiratory infection) while using nasal intermittent positive pressure ventilation during a 15 month follow up. Patient 1 voluntarily discontinued treatment after three months; 10 weeks later he was admitted with a severe, almost fatal exacerbation of infection and cor pulmonale.

There was an improvement in both arterial oxygen and arterial carbon dioxide tensions in all patients except for one with chest wall restriction, whose PaO₂ fell by 0.3 kPa (fig). Arterial Po₂ measured during the day increased from a median (range) of 6.2 (5.4–9.6) to 9.1 (7.1–9.8) kPa in the group with restrictive disease, and from 6.0 (5.7–6.5) to 7.1 (6.3–7.7) kPa in those with airflow limitation. These changes are significant at the 1% level for the group as a whole and at 5% for those with restriction. For patients with airflow limitation the changes in PaO₂ were not significant but there was a more obvious improvement in daytime PaCO₂, which fell from a median (range) of 8.2 (7.0–9.2) to 7.1

Fig Changes in diurnal arterial oxygen tension (PaO₂) and carbon dioxide tension (PaCO₂).

Table 2 Changes in arterial oxygen saturation (SAO_2) during sleep before and during treatment with intermittent positive pressure ventilation (IPPV)

% time SAO_2 was:	Before nasal IPPV (median (range))	With nasal IPPV (median (range))	
<90%	100 (all patients)	100 (77–100)	Subjects with chronic lung disease
<80%	100 (98–100)	40.5 (2–51)	
<70%	36 (12–100)	2.5 (0–13)	
<90%	100 (64–100)	26 (0–70)	Subjects with restriction
<80%	96 (3–100)	4 (0–9)	
<70%	21 (0–50)	0 (0–2)	

(4.9–7.7) kPa ($p < 0.02$). The median (range) daytime $Paco_2$ for the group with a restrictive disorder fell from 8.2 (6.7–9.8) to 6.5 (6.0–6.9) kPa ($p < 0.05$). There were no significant changes in spirometric values in either group, the median forced vital capacity for the restrictive group being 850 ml before treatment and 950 ml afterwards and the corresponding values for median FEV_1 for the four with airflow limitation being 550 ml and 525 ml.

Oxygenation improved during sleep in all patients. The time spent with oxygen saturation below 80% fell from a median (range) of 96% (3–100%) to 4% (0–9%) of the total monitoring time in those with restrictive disease ($p < 0.05$) and from 100% (98–100%) to 40% (2–51%) in those with airflow limitation ($p < 0.05$). Control of lesser degrees of desaturation was not achieved in the four with chronic airflow limitation (table 2). Hypercapnia during sleep was reduced in all subjects, including the patient who had oxygen added through his nasal mask. The six patients with restrictive disease slept well while using nasal positive pressure ventilation, with a total sleep time of over 330 minutes in all and with rapid eye movement (REM) sleep occupying a median of 16.5% (range 6–26%). The patients with airflow limitation slept less well, both before and during nasal positive pressure ventilation, REM sleep occupying a median (range) of 12.1% (10.3–24%) and 5.3% (4–23%) respectively before and during treatment.

In general the equipment was comfortable and easy to use. Two patients developed minor abrasions over the bridge of the nose, which healed when a light dressing was applied before they used the mask each night. Unacceptable loss of tidal volume occurred in two patients, who slept with their mouths open; but this was controlled by means of a stretch towelling chin support. One patient complained of abdominal distention initially but this symptom resolved with time. One man aged 65 with advanced cor pulmonale secondary to chronic obstructive pulmonary disease had trouble falling asleep while using the apparatus and elected to discontinue treatment despite the benefit he had experienced. The other nine have continued to use it for up to 18 months, and have been

joined by 25 further patients, recruited consecutively to treatment with nasal intermittent positive pressure ventilation without recourse to other forms of domiciliary ventilation; there have been no voluntary withdrawals.

Discussion

Nocturnal ventilatory support relieves hypoxaemia, controls hypercapnia, and may contribute to the relief of respiratory muscle fatigue.^{10,11} Methods using negative pressure applied to the thoracoabdominal wall are clumsy and often inefficient, and predispose to the development or worsening of upper airway obstruction.¹² Positive pressure ventilation delivered through a tracheostomy is efficient but is associated with all the undesirable consequences of the stoma.

Intermittent positive pressure by nasal mask eliminates the hazards and discomforts of tracheostomy while preserving control over the upper airway.⁵ This is of particular value in patients with coexisting chronic obstructive lung disease and obstructive sleep apnoea,¹³ and in those whose upper airway obstruction is manifest when worsening respiratory failure reduces central chemosensitivity.¹⁴ The findings reported here show that it can be used successfully to control nocturnal hypoventilation from widely varying causes. There was a sustained improvement in daytime arterial blood gas tensions and coincident symptomatic benefit, although no spirometric changes were seen. The quality of sleep while patients were using the apparatus was good in those with restrictive disorders but less so in patients with chronic airflow limitation. A diminution in REM sleep time has been noted previously in this group of patients,^{15,16} who tend to be older, so that some diminution in REM sleep might be anticipated. Control of nocturnal oxygenation was less successful during nasal intermittent positive pressure ventilation in the patients with chronic airflow limitation, and the addition of oxygen might improve both oxygen saturation and quality of sleep.¹⁵ The findings in patient 5, who had severe airflow limitation as well as restriction, suggest that oxygen can be used safely in these circumstances, even in patients who

become hypercapnic when breathing spontaneously or when using less efficient forms of ventilatory support. Signs of heart failure in this patient diminished in that the dose of diuretic was halved without return of peripheral oedema or increase in jugular venous pressure, suggesting that pulmonary vascular resistance may have fallen.

Several questions remain unanswered. Most important is whether nocturnal ventilatory support will prolong life as well as relieve symptoms in those with pulmonary as distinct from chest wall disease. If it does, the relative merits of long term domiciliary oxygen for at least 15 hours each day^{17,18} and nocturnal positive pressure ventilation would need assessment. The role of relieving respiratory muscle fatigue in the long term warrants study and possible patients with intrinsic pulmonary disease, who are oxygenated less readily than those with restrictive lesions, would benefit more if oxygen were added. The consequences in terms of cost and complexity would be considerable but, if added oxygen is unnecessary in most cases, costs are likely to be similar to those of the oxygen concentrator. On the basis of these findings a more extensive trial may be warranted.

References

- 1 Newsom-Davis J, Loh J. Alveolar hypoventilation and respiratory muscle weakness. *Bull Eur Physiopathol Respir* 1979;**15**:45–51.
- 2 Guilleminault C, Kurland G, Winkle R, Miles LE. Severe kyphoscoliosis, breathing and sleep. The "Quasimodo" syndrome during sleep. *Chest* 1981;**79**:626–30.
- 3 Catterall JR, Douglas NJ, Calverley PMA, Shapiro CM, Brezinova V, Brash HM, Flenley DC. Transient hypoxemia during sleep in chronic obstructive pulmonary disease is not a sleep apnea syndrome. *Am Rev Respir Dis* 1983;**128**:24–9.
- 4 Sawicka EH, Loh L, Branthwaite MA. Domiciliary ventilatory support: an analysis of outcome. *Thorax* 1988;**43**:31–5.
- 5 Ellis ER, Bye PTB, Bruderer JW, Sullivan CE. Treatment of respiratory failure during sleep in patients with neuromuscular disease. *Am Rev Respir Dis* 1987;**135**:148–52.
- 6 Kerby GR, Mayer LS, Pingleton SK. Nocturnal positive pressure ventilation via nasal mask. *Am Rev Respir Dis* 1987;**135**:738–40.
- 7 Simonds AK, Parker RA, Branthwaite MA. Effects of protriptyline on sleep related disturbances of breathing in restrictive chest wall disease. *Thorax* 1986;**41**:586–90.
- 8 Rechtschaffen A, Kales A. *A manual of standardized terminology, techniques and scoring systems for sleep stages of human subjects*. Los Angeles: University of California, 1968:4–11.
- 9 Carroll N, Branthwaite MA. Intermittent positive pressure ventilation by nasal mask. Technique and applications. *Intensive Care Med* (in press).
- 10 Hoeppner VH, Cockcroft DW, Dosman JA, Cotton DJ. Nighttime ventilation improves respiratory failure in secondary kyphoscoliosis. *Am Rev Respir Dis* 1984;**129**:240–3.
- 11 Rochester DF, Braun NMT, Laine S. Diaphragmatic energy expenditure in chronic respiratory failure. The effect of assisted ventilation with body respirators. *Am J Med* 1977;**63**:223–32.
- 12 Simonds AK, Branthwaite MA. Efficiency of negative pressure ventilatory equipment [abstract]. *Thorax* 1985;**40**:213.
- 13 Goldstein RS, Ramcharan V, Bowes G, McNicholas WT, Bradley D, Phillipson EA. Effect of supplemental nocturnal oxygen on gas exchange in patients with severe obstructive lung disease. *N Engl J Med* 1984;**310**:425–9.
- 14 Longobardo GS, Gothe B, Goldman MD, Cherniack NS. Sleep apnea considered as a control system instability. *Respir Physiol* 1982;**50**:311–33.
- 15 Calverley PMA, Brezinova V, Douglas NJ, Catterall JR, Flenley DC. The effect of oxygenation on sleep quality in chronic bronchitis and emphysema. *Am Rev Respir Dis* 1982;**126**:206–10.
- 16 Fleethan J, West P, Mezon B, Conway W, Roth T, Kryger M. Sleep, arousals, and oxygen desaturation in chronic obstructive pulmonary disease. The effect of oxygen therapy. *Am Rev Respir Dis* 1982;**126**:429–33.
- 17 Cooper CB, Waterhouse J, Howard P. Twelve year clinical study of patients with hypoxic cor pulmonale given long term domiciliary oxygen therapy. *Thorax* 1987;**42**:105–10.
- 18 Anonymous. Long-term domiciliary oxygen therapy [editorial]. *Lancet* 1985;ii:365–7.