Eur Respir J, 1993, 6, 364–370
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European Respiratory Journal
ISSN 0903 - 1936

Comparison of nasal pressure support ventilation with nasal intermittent positive pressure ventilation in patients with nocturnal hypoventilation

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Comparison of nasal pressure support ventilation with nasal intermittent positive pressure ventilation in patients with nocturnal hypoventilation. L.J. Restrick, N.C. Fox, G. Braid, E.M. Ward, E.A. Paul, J.A. Wedzicha. ©ERS Journals Ltd 1993.

ABSTRACT: Nasal intermittent positive pressure ventilation (NIPPV) provides effective ventilatory support in patients with nocturnal hypoventilation. Nasal pressure support ventilation (NPSV), which only provides ventilation in response to patient triggering, may also be effective, simpler, and cheaper, but has not been evaluated.

NIPPV and NPSV were compared in 12 patients with nocturnal hypoventilation, requiring domiciliary ventilatory support. The patients were studied on three consecutive nights, in random order: a control night without ventilation and a night on each mode of ventilatory support using the bilevel positive airway pressure (BiPAP) ventilator.

Both NIPPV and NPSV significantly increased mean arterial oxygen saturation (Sao_2) compared to the control night (NIPPV mean increase 4.1%; 95% confidence interval (CI) 2.2 to 6.1, NPSV 4.4%; CI 2.1 to 6.6) with no significant difference between the two modes. The percentage of the study night spent below 90% Sao_2 was significantly reduced by both ventilator modes compared to the control night (median reduction on NIPPV 37%; CI -54 to -10, reduction on NPSV 31%; CI -53 to -9, with no significant difference between NPSV and NIPPV.

NPSV was as effective as NIPPV in patients with nocturnal hypoventilation, which suggests that these patients are able to trigger the ventilator adequately. The lower cost of NPSV will make it accessible to more patients with chronic lung disease.
Eur Respir J., 1993, 6, 364–370.

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Keywords: Hypoventilation
pressure support
ventilation

Received: January 30 1992
Accepted after revision November 23 1992

The technique of intermittent positive pressure ventilation *via* a nasal mask (NIPPV) is used as a non-invasive method of ventilatory support in patients with nocturnal hypoventilation. It has been used for some years for domiciliary ventilation in patients with nocturnal hypoventilation due to chest wall and neuromuscular disorders [1, 2]. More recently, it has been used in selected patients with chronic airflow obstruction and hypoxaemia [3, 4], and as an aid to weaning [5]. However, other methods of ventilatory support are available, including pressure support ventilation, where each breath is augmented until a preselected pressure is reached. Pressure support ventilation can also be administered *via* a nasal mask, as nasal pressure support ventilation (NPSV).

NPSV has a number of advantages over NIPPV, in that the ventilation is triggered solely by the patient and respiratory patterns can be maintained, increasing the efficacy of spontaneous ventilation [6]. Patients may find a ventilator that they trigger more comfortable and, thus, with NPSV it will be easier to establish patients on ventilatory support. NPSV is much cheaper than conventional NIPPV, which would make it accessible to more patients with chronic lung disease. However, there may be some patients who are unable to trigger the ventilator effectively and, therefore, may require the additional inspiratory cycles provided by the ventilator in the assist/control mode, as in conventional NIPPV.

The aim of this study was to compare two ventilator modes, NPSV and NIPPV, in patients with chronic respiratory disease and documented nocturnal hypoventilation, using a new pressure-cycled pre-set ventilator (Respironics BiLevel Positive Airway Pressure BiPAP), which can function in both ventilator modes.

Patients

Twelve patients (8 males, 4 females; mean age 57 yrs (range 22–71 yrs)) with nocturnal hypoventilation, who used domiciliary ventilatory support, were studied. Patients already established on nasal intermittent positive pressure ventilation (NIPPV) were selected, since the aim of the study was to compare two ventilatory modes, and

these patients were already accustomed to sleeping with home NIPPV. All patients had full assessments, including sleep studies, prior to the introduction of NIPPV, and none had clinical or sleep study evidence of obstructive sleep apnoea. Five patients had chronic airflow obstruction (CAO) with chronic hypoxaemia, while seven patients had chest wall or neuromuscular disease. The patients with CAO were treated with NIPPV, because they were unable to tolerate oxygen therapy alone, due to hypercapnia. The patients with chest wall disease presented with worsening respiratory failure, accompanied by hypercapnia, which was controlled by NIPPV.

Ten patients were previously established on volume pre-set ventilators; eight on the Bromptonpac (pneuPAC Ltd, Luton, Beds, UK) and two on the Monnal-D ventilator (Deva Medical Ltd, Runcorn, Cheshire, UK). Two patients were already using the pressure-cycled BiPAP ventilator used in this study (details of which are described in "Equipment") to deliver NIPPV. All patients had stable respiratory failure, without any recent change in their clinical condition or respiratory function tests. Table 1 shows the characteristics of the patients studied.

Spirometry was performed in a sitting position using a Morgan dry spirometer, and arterial blood gases taken whilst the patients were breathing air, were analysed on a Radiometer ABL 3 blood gas machine.

The study was approved by the Ethics Committee of the Royal Brompton National Heart and Lung Hospitals.

Equipment

The ventilatory support system used was the Respironics BiPAP S/T nasal positive pressure ventilator (Medicaid Ltd, Pagham, W. Sussex, UK). This is a pressure-cycled pre-set system, cycling from a pre-set inspiratory positive airway pressure (IPAP) to an expiratory positive airway pressure (EPAP). The ventilator is triggered by an inspiratory effort that results in an increased flow of only 40 $cm^3 \cdot min^{-1}$ for a 30 ms period (Respironics, personal communication). The Respironics silicone nasal mask was used, secured with a cap or straps, to ensure that it was tightly fitting.

Unlike other nasal ventilators, the BiPAP S/T can be set to function purely as pressure support (Spontaneous or S mode), where ventilatory support is only provided in response to the patient producing an inspiratory effort ("triggering"). A BiPAP ventilator is in fact available commercially in S mode only. The BiPAP S/T, however, can also be set to function as NIPPV (Spontaneous and Timed or S/T mode) when, in addition, a minimum respiratory rate may be set. Hence, all patients were studied using the BiPAP S/T to deliver NIPPV and NPSV, allowing controlled comparison between the two modes using the same ventilator circuit.

Methods

The patients were admitted to hospital for three consecutive nights and were allocated in random order: 1) a control night with no ventilatory support, although three of the patients required supplementary oxygen, which was continued for all three nights at the same flow rates; 2) a night on NPSV (S mode); and 3) a night on NIPPV (S/T mode) with a minimum respiratory rate set at 10 $breaths \cdot min^{-1}$.

Randomization was performed by sealing each of the six possible permutations into identical unmarked envelopes, selecting one envelope from a hat and allocating the permutation, which was then sealed in a further

Table 1. – Patient characteristics

Pt	Age	Sex	Diagnosis	FEV_1		FVC_1		Pao_2*	$Paco_2$*
no	yrs			*l*	(% pred)	*l*	(%pred)	kPa	kPa
Chronic airflow obstruction (CAO)									
1	51	M	CAO	0.6	21	1.9	51	8.0	7.1
2	53	M	CAO	0.5	18	1.4	39	8.4	6.1
3	56	M	CAO	0.9	30	2.1	53	6.9	8.0
4	60	M	CAO	0.6	23	1.7	49	5.8	9.1
5	71	M	CAO	0.5	18	2.0	50	5.9	9.3
Mean (SD)				0.6 (0.1)		1.8 (0.3)		7.0 (1.2)	7.9 (1.3)
Chest wall/neuromuscular disease									
6	22	M	Kyphoscoliosis/ spina bifida	0.7	16	1.5	29	10.1	7.2
7	52	M	Myotonic dystrophy	0.9	31	1.4	37	7.9	6.9
8	60	F	Kyphoscoliosis/ polio	0.6	38	1.1	61	8.7	6.8
9	61	M	Bil diaphragm paralysis	1.1	37	1.9	45	9.3	6.9
10	66	F	Thoracoplasty	0.6	27	1.2	36	8.1	6.6
11	67	F	Thoracoplasty	0.6	33	1.0	40	5.7	5.9
12	68	F	Thoracoplasty	0.3	17	0.9	36	7.3	7.8
Mean (SD)				0.7 (0.2)		1.3 (0.3)		8.2 (1.4)	6.9 (0.6)

*: arterial blood gases on air. FEV_1: forced expiratory volume in one second; FVC: forced vital capacity; Pao_2: arterial oxygen tension; $Paco_2$: arterial carbon dioxide tension; Bil: bilateral.

identical envelope and replaced, so that each subject had an equal chance of receiving each permutation.

The patients were established on each ventilator mode by the physiotherapist during the day. The EPAP was set as low as possible (mean <2 cmH_2O in both groups of patients), and the IPAP was set to patient comfort to the maximum tolerated (mean 15 cmH_2O in patients with CAO, and mean 16 cmH_2O in patients with chest wall disease). All of the patients were able to trigger the ventilator effectively. Blood gas measurements were taken prior to commencing ventilatory support and repeated after 30 min on the appropriate ventilator setting. Arterialized earlobe capillary blood gases [7] were used, since there is a good correlation between these and arterial blood gases in our laboratory (unpublished observations). However, there may not be such close reproducibility with this method in other laboratories.

Each patient received the same preset IPAP and EPAP, on each of the ventilator nights, as determined during the daytime assessment. Overnight measurements of oxygen saturation (Sao_2) using a finger oximeter (Ohmeda Biox III), and of transcutaneous carbon dioxide tension ($Ptcco_2$) were performed (Radiometer TCM20), and were continuously recorded on all three nights. The monitors were validated by comparison with earlobe blood gases for each patient. The mean overnight Sao_2 and mean overnight $Ptcco_2$ levels for each patient, on each night, were derived by analysing each sleep study for the period 2300–0500 h. The means were calculated by averaging the readings of every 5 min period for Sao_2 and $Ptcco_2$ in the 6 h of study. The severity of overnight oxygen desaturation was analysed, by calculating the proportion of time spent under 90% Sao_2. Time spent under 85% Sao_2 during the same period was also recorded. In addition $Ptcco_2$ at 0500 h was recorded, since accumulation of CO_2 is often maximal at this time. At present there is no validated questionnaire to evaluate sleep in this situation and it was not possible to perform overnight electroencephalograms, electromyograms and electrooculograms. However, after each night patients completed standard 10 cm visual analogue scales, to score their degree of tiredness and breathlessness, their quality of sleep, and the comfort of the machine [8].

Statistical analysis

The visual analogue scores and the percentage of time spent beneath 90% and 85% Sao_2 levels were analysed using Wilcoxon's matched pairs signed ranks test. Daytime arterial oxygen tension (Pao_2) and arterial carbon dioxide tension ($Paco_2$), mean overnight Sao_2 and $Ptcco_2$ and 0500 $Ptcco_2$ ($Ptcco_2$ at 0500 h) were analysed using paired t-tests, except for comparisons between patients with chest wall/ neuromuscular disease and patients with COA, where unpaired t-tests were used. Ninety five percent confidence intervals (CI) are used, and refer to differences between paired nights/ventilator settings.

Results

Figure 1 shows the changes in daytime blood gases produced after 30 min on each type of ventilatory support, for each patient. The mean daytime Pao_2 increased significantly on the S mode from 7.9 to 8.9 kPa, (difference 1.0; CI 0.1 to 1.9; p=0.039). The mean daytime Pao_2 also increased significantly on the S/T mode from 7.9 to 9.1 kPa (difference 1.2; CI 0.8 to 1.7; p=0.0001).

The mean daytime $Paco_2$ fell on both modes with a significant fall on the S mode from 7.26 to 7.00 kPa (difference -0.26; CI -0.49 to -0.02; p=0.034). The fall in daytime $Paco_2$ on S/T mode from 7.30 to 7.16 kPa did not reach statistical significance (difference -0.14; CI -0.51 to 0.20; p=0.36). There were no significant differences in the daytime blood gases between the two ventilator modes.

Table 2 shows the results of overnight oxygen saturation (Sao_2) measurements. The mean Sao_2 increased significantly from 87.4% on the control night to 91.8% on the night on the S mode (difference 4.4; CI 2.1 to 6.6; p=0.0012). The increase on S/T mode, to 91.5%, was also significant (difference 4.1; CI 2.2 to 6.1; p=0.0007), with no significant difference between the increases produced by the two modes of ventilation (difference 0.3; CI -0.5 to 1.0; p=0.5).

On the control night, in the absence of ventilatory support, the patients studied spent a considerable proportion of the night at low levels of oxygen saturation, with frequent further dips in saturation. Figure 2 shows the median percentage of the 6 h period spent beneath 90% and 85% Sao_2. On the control night, 42% of the time was spent at less than 90% Sao_2, which fell on theS mode to 11% of the night (Difference -31; CI -53 to -9; p=0.009); and also fell significantly on the S/T mode to 5% (difference -37; CI -54 to -10; p=0.004). There was no significant difference between the two modes of ventilation at this level (difference 6; CI -2 to 7; p=0.263). A fall in time spent beneath 85% Sao_2 was also seen for both modes and is shown for comparison. Median time under 85% Sao_2 was 10% of the control night, falling to 1% of the S mode night, (difference 9; CI -36.5 to -1) and 0% of the S/T mode night (difference 10; CI -34 to -1.5) with no significant difference between the two modes of ventilation.

The results of the mean overnight $Ptcco_2$ levels are also shown in table 2. The mean $Ptcco_2$ fell significantly on S mode from 7.6 kPa on the control night to 7.2 kPa (difference -0.4; CI -0.7 to -0.1; p=0.01). The fall in overnight $Ptcco_2$ on S/T mode from 7.6 to 7.4 kPa did not reach statistical significance (difference -0.2; CI -0.5 to 0.0; p=0.08). $Ptcco_2$ at the end of each study night, at 0500 h, fell from 7.5 (1.1) kPa on air to 7.2 (1.1) kPa on S mode (p=0.081), but only fell to 7.3 (0.9) kPa on S/T mode (p=0.396). The differences in mean overnight $Ptcco_2$ and 0500 h $Ptcco_2$ between the two modes were not significant.

Patients found both ventilator modes acceptable, in terms of comfort and quality of sleep. On the visual analogue scales there were no significant differences between the scores for the different ventilator modes and the control night for any of the questions asked. The results are shown in table 3.

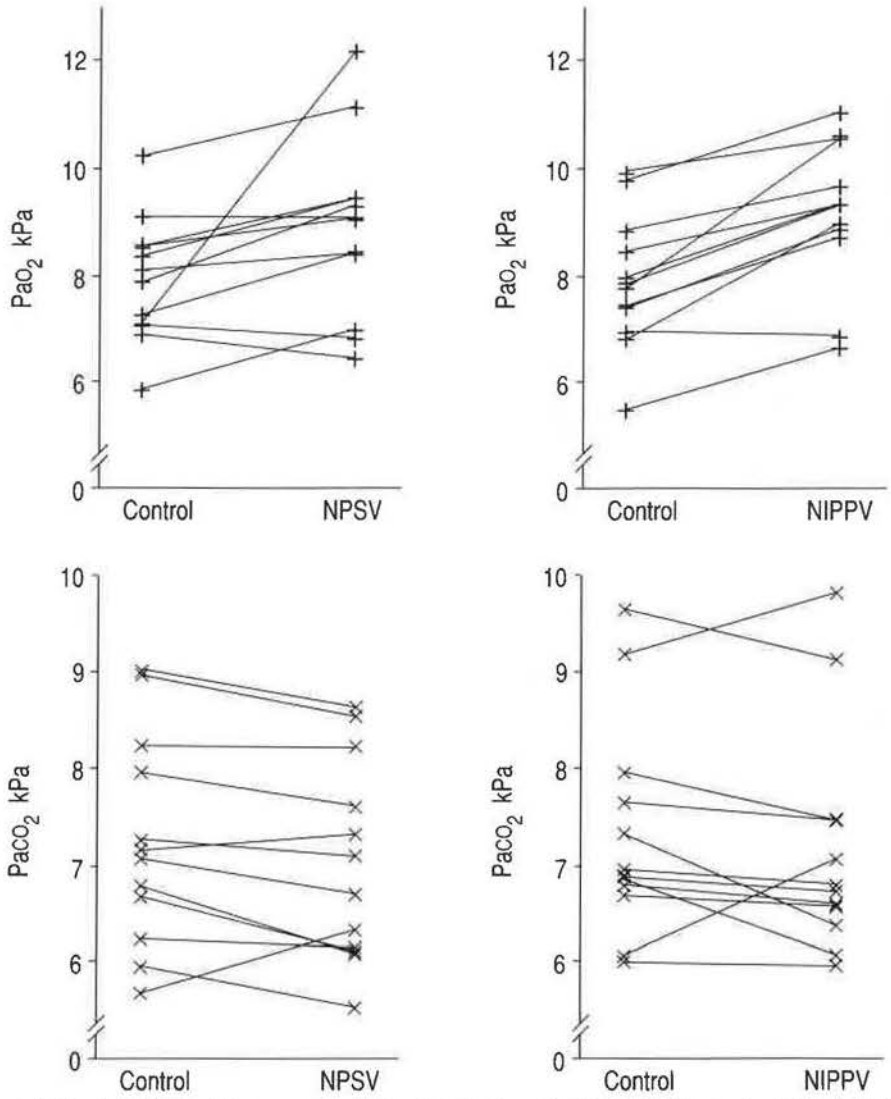

Fig. 1. – Changes in arterialized earlobe capillary oxygen tension (Pao_2) (+) and carbon dioxide tension ($Paco_2$) (x) (kPa) from control, for each of the 12 patients on nasal pressure support ventilation (NPSV), and nasal intermittent positive pressure ventilation (NIPPV).

Table 2. – Results of overnight mean Sao_2 and $ptcco_2$

Pt no	Age yrs	Sex	Mean overnight Sao_2 %			Mean overnight $Ptcco_2$ kPa		
			Air	NPSV	NIPPV	Air	NPSV	NIPPV
1	51	M	88.7	91.4	93.5	6.7	6.1	6.5
2	53	M*	90.9	94.4	94.0	6.5	6.4	6.9
3	56	M	84.9	91.0	91.0	7.9	7.6	7.5
4	60	M	73.0	82.3	79.7	9.5	9.7	8.9
5	71	M*	94.0	90.5	91.1	10.3	9.2	9.3
6	22	M	89.0	95.2	94.9	7.7	6.9	6.9
7	52	M	91.2	94.1	95.3	7.2	7.2	7.3
8	60	F	91.5	94.9	94.5	6.9	6.4	6.7
9	61	M	94.3	95.2	94.5	7.1	7.1	7.5
10	66	F	86.8	93.3	91.2	7.2	6.0	6.9
11	67	F*	82.6	90.0	89.0	6.5	6.3	6.1
12	68	F	81.3	89.2	89.1	8.1	7.9	8.1
	Mean (SD)		87.4 (6.1)	91.8 (3.7)	91.5 (4.3)	7.6 (1.2)	7.2 (1.2)	7.4 (1.0)

*: patients on overnight O_2 on all three nights. Sao_2: arterial oxygen saturation; $Ptcco_2$: transcutaneous carbon dioxide tension; air: control night; NPSV: nasal pressure support ventilation; NIPPV: nasal intermittent positive pressure ventilation. For further abbreviations see legend to Fig. 1.

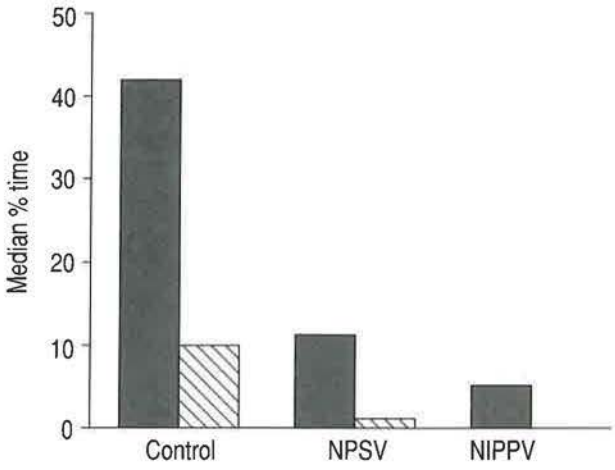

Fig. 2. – Median percentage time of 6 h period overnight spent below 90% Sao_2 (■) and 85% Sao_2 (▨) on the three study nights, for controls, patients on nasal pressure support ventilation (NPSV), and on nasal intermittent positive pressure ventilation (NIPPV). Sao_2: arterial oxygen saturation.

correcting hypoxaemia and reducing hypercapnia, and as well-tolerated, as nasal intermittent positive pressure ventilation (NIPPV).

Nocturnal hypoventilation, which may lead to respiratory failure and cor pulmonale, was originally recognized as a problem in patients with CAO [9, 10]. It also occurs in patients with chest wall and neuromuscular disease [11–13], and initial correction of hypoventilation in these patients was by negative pressure ventilation [14]. The introduction of ventilation *via* the nasal route, as in NIPPV, has proved to be an alternative, effective, non-invasive method of home ventilatory support [1, 2]. The applications of NIPPV have been extended, with its recent use in selected patients with CAO and hypoventilation, where it has been shown to improve quality of sleep and arterial blood gases [4]. Nasal ventilation may have an important role in facilitating the use of oxygen therapy to correct hypoxaemia, by preventing the associated rise in arterial carbon dioxide levels occurring in patients with type II respiratory failure. Nasal ventilation may, thus, enable higher inspired oxygen flow rates to be used safely if required, and has advantages over respiratory stimulants. The use of NIPPV, if tolerated, in patients with CAO has significant resource implications, due to the large number of patients with this condition.

Table 3. – Visual analogue scores for nights on air, NPSV and NIPPV; the larger the numerical value, the worse the symptom

Question	Air	NPSV	NIPPV
How well do you feel compared to normal?	4.7 (0.2–9.3)	3.8 (0.2–5.2)	3.1 (0.4–4.8)
How well did you sleep?	4.8 (0.0–9.6)	1.3 (0.0–8.6)	2.1 (0.0–8.3)
Did you have difficulty waking up this morning?	0.3 (0.0–1.1)	0.6 (0.0–1.3)	0.5 (0.0–1.9)
How breathless have you felt this morning?	1.7 (0.0–7.3)	1.4 (0.0–9.6)	1.9 (0.0–7.7)
Did you have a headache this morning?	0.6 (0.2–9.6)	0.7 (0.0–1.8)	1.0 (0.0–6.0)
Did the noise of the machine disturb you last night?	N/A	0.5 (0.0–7.8)	0.5 (0.0–8.1)
Was the machine last night comfortable?	N/A	0.8 (0.1–7.6)	1.6 (0.0–7.2)

Results are expressed as median (range). NPSV: nasal pressure support ventilation; NIPPV: nasal intermittent positive pressure ventilation; N/A: not applicable.

There were no significant differences in the improvements in blood gases, mean overnight Sao_2 or mean overnight $Ptcco_2$ produced by the BiPAP ventilator between the two groups of patients studied, those with chest wall/neuromuscular disease and those with CAO, although comparisons were between small numbers.

Discussion

The aim of this study was to compare two ventilatory techniques, in stable patients with nocturnal hypoventilation, who were already established on domiciliary ventilatory support. In the patients studied, nasal pressure support ventilation (NPSV) was as effective in

Until now, the accepted method of nasal ventilation has been by intermittent positive pressure ventilation. In this mode, the ventilator augments a breath in response to patient triggering, but will also deliver supplementary breaths to maintain a pre-set minimum respiratory rate. Pressure support ventilation, by contrast, is entirely patient initiated, without a pre-set respiratory rate, and ventilatory support is only delivered if the machine is triggered by the patient.

Pressure support has gained widespread acceptance in

the intensive care unit, where it is used, *via* a tracheal tube, to wean patients from conventional assisted ventilation [15]. More recently, it has been used *via* a face mask to avoid intubation in patients with acute on chronic respiratory failure [16]. Pressure support, when delivered nasally, as in NPSV, has the advantage over NIPPV of maintaining the patient's respiratory cycle. Previous studies in intubated patients have shown improvements in tidal volume and arterial blood gases, with some evidence of reduced respiratory muscle activity, using pressure support ventilation, compared to other weaning methods [6]. It may be easier to establish patients on a ventilator that is only patient triggered, and breath stacking is likely to be less of a problem. Nasal pressure support ventilation may be more comfortable, especially if dyspnoea is a problem, or if respiration is irregular, as occurs in patients with CAO, although we were unable to demonstrate a difference between the two modes of nasal ventilation, or between the control night and nasal ventilation, in this study. This may have been due to studying patients already accustomed to sleeping on nasal ventilation. A further advantage of pressure support is that equipment is available that is solely dedicated to the provision of NPSV (the BiPAP S ventilator), which is 50% of the cost of conventional NIPPV, with obvious economic advantages.

The disadvantages of pressure support ventilation are that it does not guarantee a stable tidal volume, and there is no provision of ventilation if the patient fails to trigger the ventilator. This may occur due to leaks around the nasal mask and may be a problem in a few patients with such severe respiratory muscle weakness that they are unable to trigger effectively.

This study compared nasal pressure support and nasal intermittent positive pressure ventilation by using the BiPAP ventilator, which is able to function in both modes, so permitting direct comparison. For both NPSV and NIPPV, the trigger sensitivity, resistance of the ventilator circuit, and inspiratory and expiratory airway pressures were the same for each patient. The BiPAP ventilator does have a sensitive trigger, with a short response time, which is an essential feature of a nasal ventilator, particularly when providing NPSV. It does not, however, have any safety alarms.

In the absence of ventilatory support, the patients studied spent a considerable proportion of the night at low levels of oxygen saturation, with frequent further dips in saturation. Both ventilator modes were effective at raising the mean overnight saturation and, more importantly, equally effective at reducing the hypoxaemic dips. Full polysomnography was not performed, so it is possible that differences in sleep may have occurred. However, the visual analogue scores of quality of sleep were not significantly different on the three nights, and all the patients were accustomed to nocturnal nasal ventilation, so it is likely that they were asleep for similar periods on the three nights. In this study, NPSV was as effective as NIPPV, suggesting that these patients were able to trigger the ventilator, and did so at an adequate rate throughout the night. This is consistent with the evidence that the problem in these patients is predominantly one of hypoventilation rather than apnoea [17–20].

The changes in daytime $Paco_2$, mean overnight and 0500 h $Ptcco_2$ levels were relatively small, which may be due to the fact that these were patients with stable respiratory failure, who were already using domiciliary nasal ventilation. Such patients were chosen since they were accustomed to the technique of nasal ventilation and, hence, would be easier to establish on the BiPAP for one night. If nasal ventilation had been discontinued for a period prior to the study, larger changes in carbon dioxide levels, mean overnight oxygen saturation, and visual analogue scores might have been obtained. This was not felt to be ethical in patients already established on nasal ventilation, because of the associated likely symptomatic deterioration. Nonetheless, both daytime and mean overnight carbon dioxide levels were significantly reduced on NPSV compared to control and, although they also fell on NIPPV, this did not reach statistical significance.

The inspiratory positive airway pressure (IPAP) used in this study was modest (mean 16 cmH_2O) since 10 of the patients were only introduced to the BiPAP ventilator during the study. It may be possible to increase the IPAP once patients are established on a new ventilator, which may then further reduce hypercapnia. The relatively greater improvement in oxygenation on nasal ventilation, compared to reduction in hypercapnia, suggests that nasal ventilation may also reduce ventilation perfusion inequality [21]. Decreased respiratory muscle activity during nasal ventilation may reduce the high oxygen consumption observed in patients with CAO and, thus, also increase arterial oxygenation [22]. In this study, the expiratory positive airway pressure (EPAP) was minimized in order to assess the effect of pressure support alone. Application of a higher level of EPAP may be beneficial in overcoming the "intrinsic" positive end expiratory pressure caused by the positive elastic recoil pressure in patients with CAO [22], which may reduce the work of breathing and further improve oxygenation [23].

In this study, NPSV was as effective as NIPPV in patients with nocturnal hypoventilation, due to CAO and chest wall and neuromuscular disease, who required domiciliary ventilatory support. The specific features of NPSV may be particularly useful in CAO, and this needs further evaluation. Long-term oxygen therapy has been shown to improve survival in patients with CAO [24, 25], but hypercapnia may be associated with a poorer prognosis [26]. The addition of NPSV to oxygen therapy in this group may have advantages over oxygen therapy alone, and studies to investigate this are in progress.

Acknowledgements: The authors would like to thank L. John and the staff of the Respiratory Function Laboratory for their assistance in this study.

References

1. Ellis ER, Bye PTP, Bruderer JW, Sullivan CE. – Treatment of respiratory failure during sleep in patients with neuromuscular disease. *Am Rev Respir Dis* 1987; 135: 148–152.

2. Heckmatt JZ, Loh L, Dubowitz V. – Night-time nasal ventilation in neuromuscular disease. *Lancet* 1990; 335: 579–582.
3. Carroll N, Branthwaite MA. – Control of nocturnal hypoventilation by nasal intermittent positive pressure ventilation. *Thorax* 1988; 43: 349–353.
4. Elliott MW, Simonds AK, Carroll MP, Wedzicha JA, Branthwaite MA. – Domiciliary nocturnal nasal intermittent positive pressure ventilation in hypercapnic respiratory failure due to chronic obstructive lung disease. *Thorax* 1992; 47: 342–348.
5. Restrick LJ, Scott AD, Ward EM, *et al.* – Nasal intermittent positive pressure ventilation in weaning intubated patients with chronic respiratory disease from assisted intermittent positive pressure ventilation. *Respir Med* 1993; (in press).
6. Brochard L, Pluskwa F, Lemaire F. – Improved efficacy of spontaneous breathing with inspiratory pressure support. *Am Rev Respir Dis* 1987; 136: 411–415.
7. Spiro SG, Dowdeswell IRG. – Arterialized ear lobe blood gas samples for blood gas tensions. *Br J Dis Chest* 1976; 70: 263–268.
8. Bond A, Lader M. – The use of analogue scales in rating subjective feelings. *Br J Med Psychol* 1974; 47: 211–218.
9. Douglas NJ, Calverley PMA, Leggett RJE, *et al.* – Transient hypoxaemia during sleep in chronic bronchitis and emphysema. *Lancet* 1979; 1: 1–4.
10. De Marco FJ, Wynne JW, Block AJ, Boysen PG, Taasan VC. – Oxygen desaturation during sleep as a determinant of the "blue and bloated" syndrome. *Chest* 1981; 79: 621–265.
11. Mezon BL, West P, Israels J, Kryger M. – Sleep breathing abnormalities in kyphoscoliosis. *Am Rev Respir Dis* 1980; 122: 617–621.
12. Guilleminault C, Kurland G, Winkle R, Miles LE. – Severe kyphoscoliosis, breathing, and sleep. *Chest* 1981; 79: 626–630.
13. Skatrud J, Iber C, McHugh W, Rasmussen H, Nichols D. – Determinants of hypoventilation during wakefulness and sleep in diaphragmatic paralysis. *Am Rev Respir Dis* 1980; 121: 587–593.
14. Sawicka EH, Loh L, Branthwaite MA. – Domiciliary ventilatory support: an analysis of outcome. *Thorax* 1988; 43: 31–35.
15. Goldstone J, Moxham J. – Weaning from mechanical ventilation. *Thorax* 1991; 46: 56–62.
16. Brochard L, Isabey D, Piquet J, *et al.* – Reversal of acute exacerbations of chronic obstructive lung disease by inspiratory assistance with a face mask. *N Engl J Med* 1990; 323: 1523–1530.
17. Catterall JR, Douglas NJ, Calverley PMA, *et al.* – Transient hypoxaemia during sleep in chronic obstructive pulmonary disease is not a sleep apnea syndrome. *Am Rev Respir Dis* 1983; 128: 24–29.
18. Hudgel DW, Martin RJ, Capehart M, Johnson B, Hill P. – Contribution of hypoventilation to sleep oxygen desaturation in chronic obstructive pulmonary disease. *J Appl Physiol: Respirat Environ Exercise Physiol* 1983; 55: 669–677.
19. Douglas NJ, Flenley DC. – Breathing during sleep in patients with obstructive lung disease. *Am Rev Respir Dis* 1990; 141: 1055–1070.
20. Sawicka EH, Branthwaite MA. – Respiration during sleep in kyphoscoliosis. *Thorax* 1987; 42: 801–808.
21. West JB. – Pulmonary Pathophysiology: The Essentials. 3rd edn. Baltimore/London. pp. 19–41, Williams & Wilkins, 1987.
22. Haluszka J, Chartrand DA, Grassino AE, Milic-Emili J. – Intrinsic PEEP and arterial P_{CO_2} in stable patients with chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1990: 140: 1194–1197.
23. Lanigan C, Moxham J, Ponte J. – Effect of chronic airflow limitation on resting oxygen consumption. *Thorax* 1990; 45: 388–390.
24. Medical Research Council Working Party. – Long-term domiciliary oxygen therapy in chronic hypoxic cor pulmonale complicating chronic bronchitis and emphysema. *Lancet* 1981; i: 681–686.
25. Nocturnal Oxygen Therapy Trial Group. – Continuous or nocturnal oxygen therapy in hypoxemic chronic obstructive lung disease. *Ann Intern Med* 1980; 93: 391–398.
26. 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: 105–110.