

# Nasal intermittent positive pressure ventilation in acute exacerbations of chronic obstructive pulmonary disease – a preliminary study

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Ten patients (two male) suffering from acute exacerbations of long-standing chronic obstructive pulmonary disease and admitted in hypoxic, hypercapnic respiratory failure were treated with Nasal Intermittent Positive Pressure Ventilation (NIPPV) plus supplemental oxygen, on a general medical ward. The median (range) pH on admission was 7.30 (7.20–7.35), the median age was 67 years (47–77) with an FEV<sub>1</sub> (percent of predicted) of 30 (17–39). On admission the median arterial oxygen tension ( $PaO_2$ ) was 4.71 kPa (3.45–6.26) on air, and the carbon dioxide tension ( $PaCO_2$ ) was 7.68 kPa (6.85–9.83). With controlled oxygen therapy there was no significant improvement in  $PaO_2$ , but the median  $PaCO_2$  increased significantly to 9.75 kPa (7.04–11.70) ( $P < 0.05$ ). By using NIPPV with supplemental oxygen it was possible to significantly improve the median  $PaO_2$  to 11.25 kPa (6.70–26.90) ( $P < 0.01$ ) without worsening  $PaCO_2$  levels (8.96 kPa; 6.85–13.10). NIPPV was applied by a senior, respiratory physiotherapist and used intermittently depending on patient tolerance and clinical response. The median total time on NIPPV was 27 h, delivered over 1–5 days. One patient found the mask difficult to tolerate beyond a short period of time. NIPPV was well accepted on a general ward by nursing staff. Three patients later died with progressive hypercapnia, despite an initial response; with one of these patients also receiving intubation and mechanical ventilation. A further patient also received intubation and mechanical ventilation and was eventually discharged.

NIPPV plus supplemental oxygen offers a method to correct hypoxaemia on a general medical ward without worsening hypercapnia for acute on chronic, hypoxic, hypercapnic respiratory failure, and warrants further investigation.

## Introduction

A major clinical problem in acute on chronic, hypoxic, hypercapnic, respiratory failure is the inability to adequately oxygenate without worsening the hypercapnia and therefore incurring the need to support ventilation. When such patients are unresponsive to initial medical therapy one treatment option, which has recently been re-evaluated in Edinburgh (1), is the use of the respiratory stimulant doxapram with all its attendant difficulties and side effects (2,3). Should the patient continue to deteriorate, a further option is intubation and mechanical ventilation on an intensive care unit, which in chronic obstructive pulmonary disease (COPD) can be associated with difficulties in weaning (4). We were therefore interested in a non-invasive method of reversing hypoxia while supporting ventilation.

Nasal intermittent positive pressure ventilation (NIPPV) is established as an effective method of managing nocturnal hypoventilation in the treatment of patients with ventilatory pump failure e.g. neuromuscular diseases and severe kyphoscoliosis (5–8). In one descriptive study from a national centre, the use of NIPPV has been described in a variety of respiratory conditions and was suggested to prevent formal intubation and ventilation in patients with severe respiratory failure (9). More recently, an intermittent positive pressure device, delivered via a face mask, was shown to be capable of partially correcting hypoxaemia over 45 min in acutely ill patients with COPD (10). The use of a facial mask in this study carries with it the disadvantage of being less acceptable to the claustrophobic patient; the device was also not commercially available and therefore the results difficult to reproduce; and the study was based on an intensive care unit in all cases, rather than a general medical ward.

We report the application of a commercially available form of NIPPV in the management of acute

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respiratory failure on a general medical ward in a district general hospital. The group of patients studied were admitted, as a medical emergency in respiratory failure secondary to an exacerbation of pre-existing airway disease, to a general medical ward as is normal practice in the U.K. Nasal ventilation was used in conjunction with supplemental, controlled oxygen. Our objectives, in this open, descriptive trial, were firstly to know whether NIPPV plus supplemental oxygen could reverse hypoxaemia without worsening hypercapnia; secondly we wished to assess the feasibility of using NIPPV on a general medical ward.

## Methods

### PATIENTS

Clinical details of the patients are shown in Table 1. Ten patients (two males) were treated with median age of 67 years (range 47–77). These patients were the first ten, consecutive admissions to fulfil the entry criteria of an emergency admission with acute on chronic respiratory failure (defined as  $PaO_2 < 7.5$  kPa and  $PaCO_2 > 6.0$  kPa) which was secondary to long-standing obstructive pulmonary disease ( $> 10$  yr). Patients were excluded from the trial if there were any other major system disorders or a past history of malignant lesion(s) of the bronchi. Verbal consent was obtained from the patients and if that were not possible, from the next of kin, with the assurance that the patient could be withdrawn from the trial at any time if they so wished. This study was approved by the Southampton Joint Ethics Committee.

### STUDY DESIGN

During this open, descriptive trial, all patients received 'conventional' medical therapy for their respiratory failure (physiotherapy, bronchodilators, antibiotics, steroids and in some cases doxapram). Arterial blood gases were measured on air, after a minimum of 30 min of controlled oxygen therapy (delivered either via a 24–28% venturi mask or nasal prongs at  $1\text{--}2$  l  $\text{min}^{-1}$ ) and following a minimum of 30 min of NIPPV plus supplemental oxygen. Further arterial blood gases were taken according to clinical necessity. Oxygen saturation was continuously monitored by an Ohmeda Biox 3700 pulse oximeter (Ohmeda Ltd, Hatfield, Herts, U.K.).

Constant liaison was maintained between the staff on the general medical ward concerned and the intensive care unit. Failure of NIPPV was decided on clinical grounds. The patient would then be referred to the intensive care unit for intubation and mechanical ventilation if this was deemed appropriate by all concerned. The early discussion of all such patients with

the intensive care unit staff facilitated the referral of those who required mechanical ventilation.

An impression of the ease of application of NIPPV by the operator (physiotherapist) and a member of the nursing staff was assessed daily. Individual patients rated the acceptability of NIPPV on the third day of admission or as soon as was practicable. Clinical outcome was recorded.

### NIPPV

All members of staff involved with the trial were given the appropriate introduction and training on the use of NIPPV.

NIPPV was delivered via a Brompton Pneuapac ventilator (Pneuapac Ltd., Luton, Bedfordshire, U.K.) using a tight-fitting, contoured, nasal mask (Medic-Aid Ltd., Pagham, W Sussex, U.K.). This ventilator delivers a set volume which is time cycled thus ensuring a minimum respiratory rate while allowing the patient to trigger ventilation. The following adjustments on the Brompton Pneuapac are possible: the rate of flow ( $l\ s^{-1}$ ) of compressed air delivered during inspiration, inspired time (s) (Ti) and expired time (s) (TE). The mask was held in place by a head strap. An additional chin strap was used when necessary to prevent mouth breathing. Nasal discharge was kept to a minimum by ephedrine nose drops and pressure sores over the bridge of the nose were prevented with Granuflex dressing (Squib Surgicare Ltd., Hounslow, Middlesex, U.K.).

### APPLICATION OF NIPPV

This was the responsibility of a senior respiratory physiotherapist. The initial settings of the Pneuapac ventilator were a flow rate of  $0.7$  l  $s^{-1}$ , a Ti of 1 s and a TE of 2 s. These initial settings were then adjusted to achieve optimum comfort, whilst at the same time optimizing arterial blood gases and oximetry. Supplemental oxygen was in all cases supplied through a port in the nasal mask from a wall supply. The appropriate flow of oxygen and thus inspired concentration was titrated against oxygen saturation (oximetry) and arterial blood gases.

The first 'session' lasted approximately 1 h, though there was a range of 30 min to 2 h, depending on the clinical state of the patient and on patient acceptance. Further intermittent NIPPV was delivered with maximum time on the ventilator sought but with necessary breaks for physiotherapy, nebulizers, meals etc.

### DATA ANALYSIS

Results were analysed using the Wilcoxon signed rank test. Median values with minimum to maximum ranges are used to illustrate the spread of data.

Table 1 Clinical details of patients

Subject No.	Age	Sex	FEV <sub>1</sub> % pred.	pH	On admission				On NIPPV* (Median of all samples)				Outcome	
					With air		With O <sub>2</sub>		Initial sample		PaO <sub>2</sub> PaCO <sub>2</sub>			
					PaO <sub>2</sub>	PaCO <sub>2</sub>	PaO <sub>2</sub>	PaCO <sub>2</sub>	PaO <sub>2</sub>	PaCO <sub>2</sub>	PaO <sub>2</sub>	PaCO <sub>2</sub>		
1	77	F	35	7.25	4.71	8.14	4.76†	8.97	24.46	8.86	26.90	8.90	8.90	Home and well
2	71	F	31	7.32	3.66	9.83	3.91‡	9.75	10.38	8.94	9.59	8.66	8.66	Home and well
3	67	M	17	7.25	4.46	9.07	NA	NA	12.91	9.89	11.28	12.91	12.91	Died
4	67	F	30	7.35	6.26	6.86	8.28‡	7.04	15.40	7.22	14.41	6.89	6.89	Home and well
5	67	F	39	7.30	3.45	7.60	4.60†	9.87	7.88	10.18	8.37	11.02	11.02	Died
6	68	M	25	7.22	4.12	6.85	4.52†	7.40	7.86	7.08	7.86	6.85	6.85	Home and well
7	60	F	NA	7.30	6.10	7.61	4.71†	10.81	6.41	11.32	6.70	13.12	13.12	ITU (Intubated & ventilated)
8	63	F	38	7.20	NA	NA	4.02†	11.73	14.30	10.20	12.60	9.03	9.03	Died
9	47	F	26	7.30	4.75	7.68	3.82†	9.92	6.11	6.60	11.70	9.94	9.94	ITU (Intubated & ventilated)
10	49	F	23	7.35	5.91	8.40	6.70†	9.44	7.80	7.30	11.22	7.41	7.41	Home and well

\*Plus median 2 l min<sup>-1</sup> supplemental oxygen.

†Twenty-four percent fractional inspired oxygen through face mask.

‡Oxygen through nasal cannula (2 l min<sup>-1</sup>).

NA, not available.

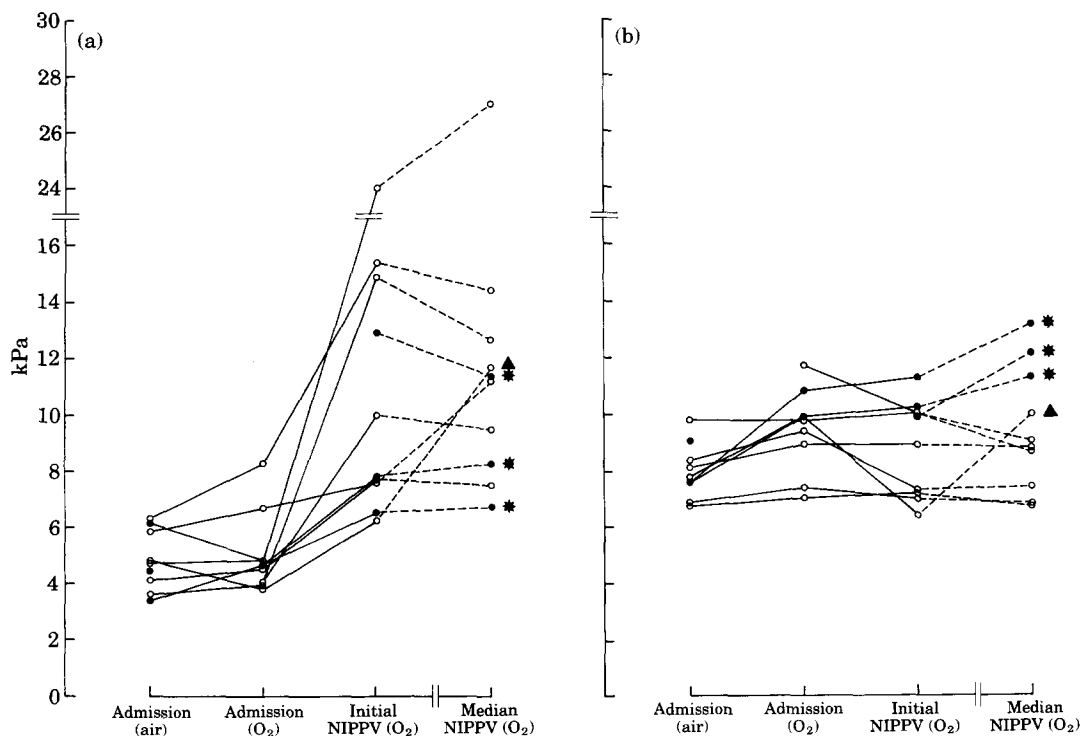


Fig. 1 Arterial blood gases; (a)  $PaO_2$  and (b)  $PaCO_2$  for each subject at four time points: on admission on air; on admission with 24% fractional inspired oxygen through a face mask or  $2\text{ l min}^{-1}$  oxygen through nasal cannulae; following the first session of NIPPV with  $2\text{ l min}^{-1}$  supplemental oxygen; and the median value for all sessions on NIPPV with supplemental oxygen ( $2\text{ l min}^{-1}$ ). The three patients that died (subjects 3, 5 and 7) are denoted by the closed symbol ● and the closed star \*. The one patient that necessitated transfer to intensive care (subject 9) and was later discharged home is denoted by the closed triangle ▲.

Visual analogue scales were used as a description of acceptance only.

## Results

Ten patients (two male) with a median age of 67 years (47–77) were admitted to a medical ward at Southampton General Hospital suffering from acute infective exacerbations of long-standing airways disease. No patient was assessed as having pulmonary oedema, and bronchoconstriction was not a major complication of their infective exacerbation. All were treated with NIPPV plus supplemental oxygen. The results of this study will be presented in two ways, firstly for the individual response to NIPPV and secondly an analysis of the group response.

### INDIVIDUAL RESPONSE

Table 1 describes the clinical details, arterial blood gas tensions and outcome for individual patients. Figure 1 depicts the arterial blood gas tensions for the individual patients described in Table 1. The blood gas

tensions presented are those taken on admission on air, on oxygen, following the first session on NIPPV and also the median values of all samples taken whilst on NIPPV.

On admission, all of the patients were in acute, hypoxic, hypercapnic, respiratory failure and presented with a history of long-standing COPD. The lack of improvement in levels of hypoxaemia following conventional oxygen therapy and the increase in hypercapnia due to this therapy is shown in Table 1 and Fig. 1. All arterial blood gases were taken following  $2\text{ l min}^{-1}$  oxygen via nasal prongs or 24% fractional inspired oxygen via a face mask.

The main effect of NIPPV with supplemental oxygen in this group of patients was an immediate and sustained increase in levels of  $PaO_2$ . In most this was not accompanied by a worsening of  $PaCO_2$ . The mean supplemental oxygen concentration used with NIPPV was  $2\text{ l min}^{-1}$ .

Three patients died during the trial (2, 5 and 15 days after entry). These three patients are represented by the symbol \* in Fig. 1.

Patient 3 received NIPPV for 3 days and died on day 5. This patient was well known to the respiratory physicians. A sharp decline in lung function and exercise tolerance had been documented in the preceding year. Following the initial session with NIPPV plus oxygen his hypoxaemia improved from 4.46 kPa (on air) to 12.91 kPa, without a major increase in his hypercapnia ( $PaCO_2$  9.07 kPa to 9.89 kPa with NIPPV). However, his condition deteriorated on day 3, reflected in the median values for arterial blood gases whilst on NIPPV ( $PaO_2$  remaining high at 11.28 kPa but the hypercapnia increasing to 12.91 kPa). After consultation with the family, active treatment was withdrawn on day 4. The patient died on day 5.

Patient 5 had failed to respond to conventional therapy, including i.v. doxapram, before NIPPV was commenced ( $PaO_2$  4.60 kPa,  $PaCO_2$  9.87 kPa) on controlled oxygen). Despite an initial response to NIPPV ( $PaO_2$  7.88 kPa,  $PaCO_2$  10.18 kPa) this patient continued to deteriorate and on day 2 active treatment was withdrawn after taking into consideration the pre-admission quality of life and since it was judged that any further treatment would be ineffective. She died the same day.

Patient 7 was admitted and rapidly deteriorated. This patient was unknown to the respiratory physicians. She failed to respond to conventional therapy which included doxapram ( $PaO_2$  4.71 kPa,  $PaCO_2$  10.81 kPa on controlled oxygen). NIPPV was applied with an initial improvement of hypoxaemia. However sputum retention became the main management problem and for that reason and in view of the severity of this patient's condition she was transferred to intensive care for intubation and mechanical ventilation on day 2. She died in intensive care on day 15.

Patient 9 was the youngest of the group. This patient is represented by the symbol  $\blacktriangle$  in Fig. 1. She too had failed to respond to conventional therapy including doxapram ( $PaO_2$  3.82 kPa,  $PaCO_2$  9.92 kPa on controlled oxygen). She had responded well to NIPPV initially with an improvement in hypoxaemia and a decrease in levels of hypercapnia ( $PaO_2$  6.11,  $PaCO_2$  6.60). However despite this improvement she appeared to be tiring, reflected in the increase in  $PaCO_2$  to 9.94 kPa for the median value of blood gases on NIPPV. In view of her age (47 years) and the severity of her condition she was referred to intensive care for intubation and mechanical ventilation on day 2. This lady was eventually discharged home and has since had a successful, single lung transplant.

The three patients who died initially responded favourably to the initial session of NIPPV but could later be distinguished by a relentless and progressive rise in  $PaCO_2$  which was unresponsive to treatment despite

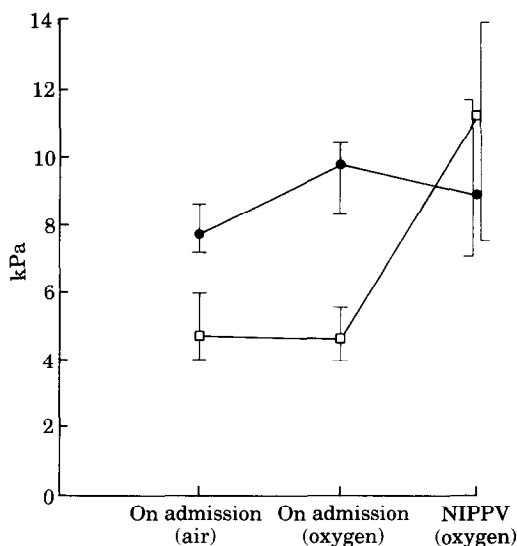


Fig. 2 Median (inter-quartile range) arterial blood gases. Values shown are: on admission on air, on admission with 24% fractional inspired oxygen through a face mask or 2 l  $min^{-1}$  through nasal cannulae, and the median value for all sessions on NIPPV with supplemental oxygen (2 l  $min^{-1}$ ).  $PaO_2$  (—□—),  $PaCO_2$  (—●—).

maximization of conventional therapy and NIPPV. Patient 9 also showed this progressive rise in  $PaCO_2$  despite a good initial response to NIPPV. These four patients (patients 3, 5, 7 and 9) recorded the four highest median levels of  $PaCO_2$  whilst on NIPPV (Table 1, Fig. 1).

Other than the patients described above (patients 3, 5, 7 and 9) two other patients had received doxapram unsuccessfully within 24 h prior to starting NIPPV (patients 1 and 4).

All the remaining patients (1, 2, 4, 6, 8 and 10) showed an immediate and marked improvement in the levels in  $PaO_2$  following the initial session of NIPPV plus supplemental oxygen without an increase in hypercapnia compared with the response to conventional oxygen therapy (Table 1, Fig. 1). This improvement in hypoxaemia without an increase in the levels of hypercapnia was sustained in these patients as described in Table 1 by the median of all samples of arterial blood gases whilst on NIPPV. These patients were all discharged home following a progressive recovery.

#### GROUP ANALYSIS

Figure 2 represents the median (interquartile range) values of arterial blood gas tensions on admission on air, on oxygen and median values of all sessions on NIPPV, for all ten patients.

The median arterial pH of 7.30 (range 7.20–7.35) on admission reflects the acuteness of the admissions, and the severity of the respiratory failure is seen in the degree of hypoxaemia and hypercapnia (Table 1). Analysis of blood gas tensions on admission reveals that for the group the median (range) arterial oxygen tensions on admission were 4.71 kPa (3.45–6.26) on air and 4.60 kPa (3.82–8.28) with controlled oxygen therapy; there was no significant difference between these two sets of values. The corresponding medians (range) for arterial carbon dioxide tensions were 7.68 kPa (6.85–9.83) with air increasing significantly to 9.75 kPa (7.04–11.70) with controlled oxygen ( $P < 0.05$ ).

All of the ten patients demonstrated an improvement in hypoxaemia after a minimum of 30 min of NIPPV plus oxygen compared to controlled oxygen alone (in the case of subject 3 the comparison was with the arterial oxygen tension whilst breathing air). The same comparison was made for carbon dioxide tensions and for the group as a whole there were minor changes (less than 1 kPa) in the level of hypercapnia following the initial session of NIPPV except for cases 8, 9 and 10 where there was a moderate reduction in hypercapnia.

Statistical analysis of the blood gas tensions following the initial session of NIPPV plus supplemental oxygen demonstrates this immediate and significant improvement in arterial hypoxaemia compared to the blood gas tensions following controlled oxygen alone; with the median value increasing to a  $PaO_2$  of 9.13 kPa (range 6.11–24.46) on NIPPV from a  $PaO_2$  of 4.60 kPa (range 3.82–8.28) on controlled oxygen ( $P < 0.01$ ). There was no significant difference in the levels of hypercapnia.

The overall effect of NIPPV was judged by calculating the median of all arterial blood gas tensions whilst on NIPPV. Individually it can be seen that the improvement in hypoxaemia is maintained in all cases. The levels of hypercapnia were little changed except for the three subjects who later died (patients 3, 5 and 7) and for subject 9, where an increase in hypercapnia is described. Analysis of the median effect of NIPPV compared to controlled oxygen, showed that following NIPPV with supplemental oxygen the median arterial carbon dioxide tension decreased to 8.96 kPa (6.85–13.12) whilst the oxygen tension increased significantly to 11.25 kPa (6.70–26.90) ( $P < 0.01$ ). The median total time on NIPPV was 27 h (range 10–53) over 1–5 days. The median flow of supplemental oxygen delivered to the nasal mask of the NIPPV was  $2.1 \text{ min}^{-1}$ .

Patients 5, 7 and 9 were unable to rate the acceptability of NIPPV due to the severity of their condition and their transference to the intensive care unit. The remaining seven patients found NIPPV to

be comfortable with a reported improvement in the sensation of breathlessness whilst on the NIPPV to when off it. Five patients were able to sleep whilst using NIPPV whilst for two patients sleep was disturbed. One patient with a history of claustrophobia found the mask unacceptable beyond short periods of time (1 h maximum).

NIPPV was applied by the operator with moderate ease to all patients. The patients were assessed by the operator as accepting NIPPV well; with the rating for acceptance as increasing daily. Comfort of the patient was similarly assessed by a senior nursing staff as good with acceptance increasing daily.

## Discussion

The intention of this open, descriptive study was firstly to quantify the effect of NIPPV with supplemental oxygen on hypoxaemia in acute on chronic, hypoxic, hypercapnic respiratory failure and secondly we wished to assess the feasibility of using NIPPV on a general medical ward. In this group of ten patients admitted as medical emergencies, conventional oxygen therapy had no significant effect on the levels of  $PaO_2$  but was accompanied by a significant ( $P < 0.05$ ) increase in the levels of hypercapnia. Following the first session of NIPPV plus supplemental oxygen (minimum time of 30 min) hypoxaemia was significantly improved ( $P < 0.01$ ) compared with conventional oxygen therapy alone. Most importantly, there was no significant increase in the levels of hypercapnia accompanying the first session of NIPPV. Analysis of the levels of hypoxaemia on all occasions whilst on NIPPV also showed a significant improvement ( $P < 0.01$ ). Levels of hypercapnia were not significantly altered. Therefore the main therapeutic advantage of using NIPPV, combined with added oxygen is the correction of severe hypoxaemia without increasing hypercapnia.

The patients presenting to our study were severely unwell. In the U.K. such patients are routinely admitted to a medical ward rather than an intensive care unit. The purpose of this study was to see if NIPPV could be used with success in a ward setting, within the present clinical framework. Doxapram is used in the U.K. as a respiratory stimulant for extreme cases in an attempt to avoid mechanical ventilation (1), and was therefore included as a possible, conventional therapy.

The severity and the acute nature of the presentation of this group of patients was reflected in the levels of hypercapnia, hypoxaemia and acidosis on admission (Table 1). There was no correlation between the outcome and the degree of airways obstruction. In two

patients (patients 3 and 5) the severity of the underlying disease resulted in the withdrawal of active management with the subsequent deaths of these two patients. In two others (patients 7 and 9) formal ventilation on an intensive care unit was considered appropriate: of these one died (patient 7) and one was eventually discharged home and has subsequently been the successful recipient of a single lung transplant (patient 9). The approximate incidence of patients admitted to our unit and who go on to receive lung transplantation is one per year. Therefore this sample of patients could be considered atypically severe, and does highlight the need for a larger controlled trial. In all four cases (patients 3, 5, 7 and 9) although there was an initial response to ventilatory support with NIPPV plus oxygen, the underlying pathology was such that this form of ventilation was inadequate; this being reflected in all four by a relentless rise in the levels of hypercapnia which was unresponsive to conventional medical therapy of NIPPV. At that point, the decision had to be made to refer the patient to the intensive care unit or to stop active management. In the two patients where active management was withdrawn (on days 4 and 2 for patient 3 and 5 respectively) NIPPV had allowed enough time for the assessment of their respiratory condition and quality of life to be considered. NIPPV is not a substitute for mechanical ventilation indeed in patients 7 and 9 intubation and mechanical ventilation became necessary. It can therefore be suggested that nasal ventilation is a useful treatment option between conventional oxygen therapy and before recourse to intubation and full mechanical ventilation on an intensive care unit. Our study further suggests that a relentless rise in  $PaCO_2$  is a sign of failure of NIPPV as in other forms of therapy for respiratory failure.

It was possible to use nasal ventilation on a busy general medical ward which was 'on take' for acute medical emergencies. No additional nursing staff were needed. The nasal mask was applied by a senior, respiratory physiotherapist. Settings on the ventilator were aimed at patient acceptability and comfort rather than mandatory ventilation levels. Nasal ventilation was achieved in this group of acutely ill patients despite the presence of distress, and in some, confusion and dyspnoea. One factor in the successful treatment of such acutely ill patients with NIPPV was the unique handling and positioning skills of the respiratory physiotherapists. When familiarized with NIPPV, most patients were able to tolerate the ventilator during sleep. This method of ventilation has the advantage that between periods of treatment the patient was not immobilized. It is possible for patients to eat, receive physiotherapy, use a nebulizer and, were

possible, to care for themselves. One of the group (patient 2) found the mask difficult to tolerate beyond a short period of time (1 h maximum), although despite this, improvement of her respiratory failure occurred.

An advantage of NIPPV, unlike endo-tracheal ventilation, is that paralysing and sedating agents are not required to achieve ventilatory assistance; and since NIPPV is patient triggered, the intrinsic need and drive to breath is not removed. We postulate that this is why no patient had problems with weaning. Removal of NIPPV did not precipitate an immediate respiratory crisis in any individual. Furthermore in no patient was there a precipitous rise in  $PaCO_2$  on NIPPV plus oxygen; where there was a rise it occurred slowly and accompanied a relentless worsening of the clinical state.

The mechanisms behind the improvements in blood gases with NIPPV are probably multiple. NIPPV allows the support of ventilation by timed and/or triggered positive pressure, with the option of delivering supplemental oxygen. The marked increase in  $PaO_2$  may therefore be due to a more effective delivery of oxygen using this system. A further possible mechanism for the raised levels of  $PaO_2$  found in this study following NIPPV is an increase in ventilation perfusion matching caused by opening of atelectatic areas with positive pressure. The potential advantages of correcting hypoxaemia are numerous and include improvement of mental state, cardiac and renal function, tissue perfusion and the ability to overcome infection. In contrast, a possible disadvantage of the hyperoxic state we achieved is the occurrence of ventilation perfusion mismatching as a result of reflex, hyperoxic vasodilation in areas of poor or no ventilation.

Another possible advantage of NIPPV is the achievement of respiratory muscle rest. In acute respiratory failure it is thought that respiratory muscles are severely stressed. It is therefore feasible that respiratory muscle rest occurs whilst on NIPPV. Furthermore, patients were observed to regularize their patterns of breathing whilst on the nasal ventilator. The timed, intermittent nature of the ventilation very probably acts as a regular stimulus to breath.

This study has described a possible, new technique in the management of acute on chronic respiratory failure. However, in this group of patients we cannot exclude a delayed benefit of 'conventional therapy' to explain our findings. It is also necessary to evaluate the effect of an improvement in respiratory failure on clinical course. To test the hypothesis that NIPPV could be advantageous in the management of acute, hypoxic, hypercapnic respiratory failure, a controlled,

randomized, three-centre trial comparing NIPPV plus conventional treatment, with conventional treatment alone, is in progress. The outcome parameters will include, length of stay in hospital, admission to ITU and eventual outcome.

We conclude that the use of NIPPV with supplemental oxygen has a potential place in the management of hypoxic, hypercapnic, respiratory failure due to acute exacerbations of chronic obstructive pulmonary disease. Secondly it is possible to use NIPPV on a general medical ward if there is a critical group of trained staff including senior respiratory physiotherapists, nurses and physicians; with ready access to an intensive care unit.

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