Using nasal intermittent positive pressure ventilation on a general respiratory ward

ABSTRACT – Objectives: To assess the use of nasal intermittent positive pressure ventilation (NIPPV) in treating acute-on-chronic respiratory failure in a general medical ward.

- Design: Retrospective analysis of clinical outcome.
- Setting: A general medical ward of a tertiary respiratory medicine referral centre.
- Subjects: Altogether 75 patients admitted with acute exacerbations of chronic respiratory failure and treated with NIPPV.
- Main outcome measures: Blood gas tensions determined at admission to hospital and during NIPPV, tolerance of NIPPV and mortality.

Results: During treatment with NIPPV, the mean (SD) Pao2 increased rapidly by 2.31 (3.58) kPa (p<0.0001), while the mean Paco2 fell by 1.07 (1.74) kPa (p<0.0001) and the mean pH increased by 0.03 (0.07) (p=0.001). Altogether 57 (76%) of patients tolerated NIPPV, and 7 (9.3%) died in hospital. Improvement in Pao2 was more noticeable in patients with chronic obstructive pulmonary disease (+3.13 (3.49) kPa, p<0.0001) than in those with restrictive chest wall disease (+1.20 (3.07) kPa, p=0.25) or obstructive sleep apnoea (+0.18 (3.64), p=0.88). The reduction in Paco2 was similar in all three groups.

Conclusions: In routine treatment of unselected patients with acute-on-chronic respiratory failure who are being cared for on a general ward, NIPPV rapidly improves hypoxaemia and hypercapnia, is well tolerated and is associated with low mortality.

There is considerable interest in using non-invasive positive pressure ventilation (NIPPV) to treat patients with acute respiratory failure[1,2]. Prospective randomised clinical trials of treatment with NIPPV in patients with acute-on-chronic respiratory failure due to chronic obstructive pulmonary disease have shown improvements in blood gases and patients’ symptoms and reduced rates of endotracheal intubation and mortality[3,4]. In clinical practice, the benefits of NIPPV, the avoidance of endotracheal intubation could save intensive care resources. However, in most previously published studies, patients were treated in intensive care wards, thereby lessening any possible economic advantages of the treatment. The results of two published controlled trials in which patients were treated on general wards are contradictory[5,6]. Whether these patients can be treated successfully with NIPPV outside the intensive care unit still remains unclear. Furthermore, although NIPPV is well established as the treatment of choice in chronic respiratory insufficiency due to previous thoracoplasty or kyphoscoliosis[7], there are few data on its use in acute exacerbation of restrictive chest wall disease or obstructive sleep apnoea[8].

At our specialist respiratory referral centre we use NIPPV routinely outside intensive care, as early treatment for acute hypercapnoeic exacerbation of chronic respiratory failure. We have assessed retrospectively the outcome of NIPPV treatment for acute-on-chronic respiratory failure in a broad group of patients treated on a general ward. We also examined the results of NIPPV in three individual diagnostic categories – chronic obstructive pulmonary disease, restrictive chest wall disease and sleep apnoea/hypoventilation.

Methods

Patients

All patients with acute exacerbations of chronic obstructive pulmonary disease, sleep apnoea/hypoventilation or restrictive chest wall disease treated at the London Chest Hospital with NIPPV over a period of 34 months from 1 January 1993 were included in the study. We excluded patients with cystic fibrosis or acute respiratory failure without underlying thoracic abnormalities, or those transferred from another hospital for weaning off intubation. Criteria for using NIPPV for acute-on-chronic respiratory failure were: a Paco2 value greater than 7.0 kPa with an acute increase over baseline level (especially if combined with a low PaO2 despite controlled oxygen therapy) and respiratory distress without cardiovascular instability or upper airways obstruction. There was no upper limit of Paco2 above which patients were automatically intubated. Conventional treatments with bronchodilators, corticosteroids, antibiotics and, if necessary, intravenous respiratory stimulants and methylxanthines were continued.

Administration of NIPPV

NIPPV was initiated by the physiotherapist or doctor and maintained by the nursing staff, with 24-hour support from the physiotherapists and respiratory physicians. All patients were treated on a general ward. The nasal ventilators used were the pressure cycled BiPAP (Respirronics Inc, Murraysville, PA, USA), and the Bromptonpac (PneuPAC UK, Luton, UK) and Monnal D (TAEMA, Paris, France), both of which are volume cycled. The particular ventilator chosen depended on patient acceptance, success with ventilation and the experience of the medical staff. After an initial trial of two hours, blood gases were checked to assess the
adequacy of ventilation and oxygenation. The normal pattern of use of NIPPV was continuous with short rest periods for the first three days, weaning to intermittent use three times a day with continual nocturnal use, then purely nocturnal use before stopping. Patients established on long-term domiciliary nocturnal NIPPV continued nocturnal treatment.

Data collection

Information on demographic details, chest radiograph reports, baseline values for forced expiratory volume in 1 second (FEV₁), length of hospital admission, outcome and blood gas results were obtained from case notes. Tolerance of NIPPV was assessed from the medical and physiotherapy notes. Respiratory function tests and blood gas measurements performed during a stable period shortly before or after the index hospital admission in the study period were used for baseline data. Blood gas measurements taken immediately before NIPPV treatment and once the patient was established on a suitable NIPPV regimen (usually within two hours) were recorded, whenever possible with the same inspired oxygen flow. To avoid skewing the results, analysis of data from patients with recurrent admissions was limited to the first admission for each patient during the study period. Results were assessed for all patients and separately for the following three groups: chronic obstructive pulmonary disease, restrictive chest wall disease and sleep apnoea/hypoventilation.

Statistical analysis

Statistical analysis was carried out using the epidemiology program Epi Info. Continuous variables were assessed by 2-tailed t tests and categorical data by χ² tests for contingency tables and Fisher's exact test.

Table 1. Characteristics of all patients and in relation to disease groups.

|                       | All patients n=75 | Chronic obstructive pulmonary disease n=49 | Restrictive chest wall diseases n=16 | Sleep apnoea/hypoventilation n=10 |
|-----------------------|-------------------|-------------------------------------------|-----------------------------------|----------------------------------|
| Mean (SD) age (years) | 63 (11)           | 66 (9)                                    | 65 (13)                           | 51 (11)                          |
| Sex (male)            | 42 (56)           | 34 (69)                                   | 4 (25)                            | 4 (40)                           |
| Mean (SD) FEV₁ (ml)   | 868 (469)         | 773 (271)                                 | 720 (221)                         | 1662 (839)                       |
| Using long term oxygen therapy | 47 (63) | 38 (77)                                   | 6 (38)                            | 3 (30)                           |
| Using domiciliary NIPPV | 12 (17)         | 9 (18)                                    | 3 (19)                            | 0                                |
| Mean (SD) length of hospital stay (days) | 19 (range 1–63) | 19 (12)                                   | 17 (8)                            | 24 (8)                           |
| Mean (SD) duration of NIPPV (days) | 11 (range 1–42) | 10 (8)                                    | 14 (8)                            | 11 (8)                           |

Values: number (%) unless stated otherwise

Table 2. Characteristics and blood gas values at baseline and at hospital admission for patients with complete and incomplete data who were undergoing NIPPV treatment.

|                       | Complete data n=37 (76%) | Incomplete data n=18 (24%) | p values |
|-----------------------|--------------------------|-----------------------------|----------|
| Mean (SD) age (years) | 63 (12)                  | 64 (10)                     |          |
| Sex (male)            | 30 (53)                  | 11 (61)                     |          |
| Mean FEV₁ (ml)        | 878 (495)                | 847 (400)                   |          |
| Using long term oxygen therapy | 35 (61) | 12 (67)                      |          |
| Using domiciliary NIPPV | 6 (11)                  | 6 (33)                      | 0.023    |
| Compliance            | 14 (25)                  | 3 (17)                      | NS       |
| Poor outcome          | 8 (14)                   | 2 (11)                      | NS       |
| Disease:              |                          |                             |          |
| Chronic obstructive pulmonary disease | 37 (65) | 12 (63)                      |          |
| Restrictive chest wall disease | 10 (18)            | 6 (32)                      |          |
| Sleep apnoea/hypoventilation | 10 (18)        | 1 (5.3)                     |          |
| Mean (SD) blood gases at baseline: | | | |
| ⁰PaO₂ (kPa)            | 7.29 (1.39)              | 7.02 (1.25)                 | NS       |
| ⁰PaCO₂ (kPa)           | 7.14 (0.93)              | 7.34 (0.95)                 | NS       |
| Mean (SD) blood gases on admission: | | | |
| ⁰PaO₂ (kPa)            | 7.24 (2.35)              | 7.62 (2.14)                 | NS       |
| FiO₂ (%)               | 1.7 (1.6)                | 1.1 (1.1)                   |          |
| ⁰PaCO₂ (kPa)           | 9.26 (2.11)              | 8.49 (1.51)                 | 0.19     |
| pH                    | 7.34 (0.08)              | 7.37 (0.06)                 | 0.12     |

Values: number (%) unless stated otherwise
Results

Demographic and baseline data

Over the 34 months of the study, NIPPV was used or attempted in 75 patients with an acute exacerbation of underlying chronic respiratory failure. Twenty-two (29%) patients had been transferred from other hospitals for treatment at our centre and 2 (3%) had been intubated previously at the referring hospital, but were breathing spontaneously at the time of transfer.

Forty-nine (65%) of the study population had chronic obstructive pulmonary disease, 16 (21%) had a restrictive chest wall disorder (9 thoracoplasty, 5 kyphoscoliosis, 2 other conditions leading to restrictive chest wall defect) and 10 (13%) had sleep apnoea/hypoventilation. Baseline details for each diagnostic group are shown in Table 1. Four (5.3%) patients had marked diffuse bronchiectasis as well as airways obstruction. The median age was 65 years (range 23–85) and 42 (56%) patients were men. Stable baseline mean FEV₁ value, available in 69 patients, was 868 ml (range 330–3,400 ml). At the time of admission, 48 (63%) patients were established on domiciliary long-term oxygen therapy. Twelve (16%) patients were using domiciliary nocturnal NIPPV, and a further 12 (16%) had used NIPPV before the study, giving a total of 24 (32%) patients with previous experience of NIPPV. The mean length of their hospital stay was 19 days (range 1–63), and the mean duration of NIPPV treatment was 11 days (range 1–42). The chest radiograph showed new consolidation in 5 (6.6%) patients (all from the chronic obstructive pulmonary disease group).

Blood gases

Complete blood gas data were available for 55 patients. There were no significant differences for age, sex, FEV₁, and blood gas values at baseline and admission in patients with complete and incomplete blood gas data (Table 2). In patients with complete data, the mean changes in \( P_{\text{O}_2} \), \( P_{\text{CO}_2} \) and pH between baseline and admission and between NIPPV and admission are shown in Table 3. The \( P_{\text{CO}_2} \) on admission was higher than at baseline and the pH was lower, confirming that the patients were experiencing a severe exacerbation of their underlying disease. The mean flow of supplemental oxygen (\( F_{\text{io}}\text{O}_2 \)) increased from 0.2 l/min at baseline to 1.7 l/min on admission (\( p<0.0001 \)), but the \( P_{\text{O}_2} \) did not change. During NIPPV treatment \( P_{\text{O}_2} \), \( P_{\text{CO}_2} \) and pH values improved and there was no change in the mean supplemental oxygen flow rate (1.7 l/min versus 1.5 l/min, \( p=0.59 \)). When analysed separately, improvements in \( P_{\text{CO}_2} \) were seen in all three groups, but improvements in

| Table 3. Blood gas values, mean (SD), at admission to hospital and during NIPPV treatment for all patients and separately for each disease category. |
|------------------|------------------|------------------|------------------|
| **Blood gases**  | **Disease category** | **Disease category** | **Disease category** |
|                  | Chronic obstructive pulmonary disease | Restrictive chest wall disease | Sleep apnoea/hypoventilation |
|                  | \( n=49 \) | \( n=16 \) | \( n=10 \) | All patients \( n=75 \) |
| \( P_{\text{O}_2} \) (kPa) | | | | |
| Baseline | 7.07 (1.32) | 7.74 (1.56) | 9.0 (2.4) | 7.16 (1.34) |
| Admission | 7.04 (2.0) | 7.80 (3.11) | 7.40 (2.87) | 7.24 (2.35) |
| NIPPV | 10.17 (3.0) | 9.0 (2.24) | 7.59 (1.21) | 9.55 (2.84) |
| NIPPV versus admission | +3.13 (3.49) | +1.20 (3.07) | +0.18 (3.64) | +2.31 (3.58) |
| \( p \) value for mean change on NIPPV | \(<0.0001\) | 0.25 | 0.88 | \(<0.0001\) |
| \( P_{\text{CO}_2} \) (kPa) | | | | |
| Baseline | 7.03 (0.94) | 7.55 (0.74) | 8.25 (1.08) | 7.21 (0.93) |
| Admission | 9.31 (2.02)* | 9.54 (2.29)† | 8.76 (2.42)‡ | 9.26 (2.11)* |
| NIPPV | 8.35 (1.69) | 8.25 (1.08) | 7.48 (1.41) | 8.19 (1.57) |
| NIPPV versus admission | –0.96 (1.87) | –1.29 (1.53) | –1.28 (1.55) | –1.07 (1.74) |
| \( p \) value for mean change on NIPPV | 0.004 | 0.026 | 0.038 | \(<0.0001\) |
| \( pH \) | | | | |
| Baseline | 7.41 (0.04) | 7.39 (0.04) | 7.37 (0.04) | 7.40 (0.04) |
| Admission | 7.35 (0.08)* | 7.34 (0.06)§ | 7.32 (0.1) | 7.34 (0.08)* |
| NIPPV | 7.38 (0.08) | 7.37 (0.04) | 7.39 (0.09) | 7.38 (0.07) |
| NIPPV versus admission | +0.03 (0.07) | +0.03 (0.06) | +0.07 (0.09) | +0.03 (0.07) |
| \( p \) value for mean change on NIPPV | 0.017 | 0.17 | 0.06 | 0.001 |

For changes from baseline to admission: * \( p<0.0001 \), † \( p=0.023 \), ‡ \( p=0.008 \), § \( p=0.03 \)
\(P_{aO_2}\) and pH were limited to the chronic obstructive pulmonary disease group. In patients with chronic obstructive pulmonary disease, the mean supplemental oxygen flow rate between admission and NIPPV treatment was unchanged (1.7 l/min versus 1.8 l/min respectively).

**Tolerance of NIPPV**

NIPPV was poorly tolerated by 13 patients and was not tolerated at all by five patients, giving a total of 18 (24%) patients who were poorly compliant with NIPPV. Factors associated with tolerance are shown in Table 4. Of the 51 patients who had no previous experience of NIPPV, 36 (71%) tolerated the treatment.

**Death and intubation**

During the index admission 5 (6.6%) patients underwent endotracheal intubation and 7 (9.3%) died, 2 of whom had been intubated. Taking both these end-points together, 10 (13%) patients had a poor outcome. Factors affecting the outcome are shown in Table 5.

**Discussion**

Although several clinical trials of NIPPV for acute-on-chronic respiratory failure due to exacerbation of chronic obstructive pulmonary disease have shown favourable results\(^1\)\(^-\)\(^5\), the technique is not used routinely at most hospitals. Proved benefits of NIPPV include lower rates of intubation\(^2\)\(^-\)\(^3\), rapid correction of abnormal blood gases\(^1\)\(^-\)\(^2\), improvement in patients’ sensation of dyspnoea\(^1\)\(^-\)\(^3\), and lower mortality\(^1\)\(^-\)\(^2\). However, without a practical method of sham ventilation, bias between treatment groups could have occurred. It is therefore important to reassess the physiological and clinical outcome of NIPPV in routine practice. In most published work patients have been treated in intensive care. Thus, any financial or resource advantages of NIPPV over endotracheal intubation have been reduced. In two controlled trials of NIPPV for acute exacerbation of chronic obstructive pulmonary disease, patients were treated on general medical wards\(^6\)\(^-\)\(^8\). Bott et al\(^8\) reported results similar to those in more recent trials based on intensive care wards\(^2\)\(^-\)\(^3\). The more recent study by Barbé et al showed no differences in blood gas data or length of stay with NIPPV. However, since no data were given for blood gas results during NIPPV treatment in this study, the adequacy of ventilation is unknown. In addition, there were only 10 patients in each arm of the trial. Furthermore, NIPPV was given for only six hours a day, considerably less than is routine in our patients. For these reasons, the results of the study of Barbé et al\(^8\) may not have general validity and further information on the use of NIPPV on general medical wards is needed.

As far as we know, this is the largest outcome study of patients from a single centre treated with NIPPV for acute-on-chronic respiratory failure. At our centre all consenting patients who meet the criteria for NIPPV are offered treatment, and NIPPV is given routinely in a general ward. Patients are only treated in intensive care when they are being weaned from endotracheal intubation or are deteriorating appreciably. In line with published evidence on the efficacy of NIPPV for acute-on-chronic respiratory failure, the policy at our centre is to start this treatment immediately on admission\(^2\)\(^-\)\(^9\). Although we included all the patients who met the broad criteria for selection, our results are similar to those in positive clinical trials\(^1\)\(^-\)\(^3\). Improvements in \(P_{aO_2}\), \(P_{aCO_2}\), and pH occurred within a few hours of beginning NIPPV. The hospital mortality of 9% is similar to that in the treatment groups in clinical trials of NIPPV\(^1\)\(^-\)\(^3\).

Although relatively few of our patients were intubated, differences in indications for intubation between countries make comparisons between studies difficult. In common with Ambrosino et al\(^11\), we found that a lower pH on admission to hospital was related to endotracheal intubation and death. Mortality has been associated with a low pH in

**Table 4. Patients’ characteristics in relation to their ability to tolerate NIPPV.**

|                                | Well tolerated n=57 (76%) | Poorly tolerated n=18 (24%) | \(p\) values |
|--------------------------------|---------------------------|-----------------------------|--------------|
| Mean (SD) age (years)          | 63 (12)                   | 65 (10)                     | 0.01         |
| Mean (SD) FEV\(_1\) (ml)       | 786 (317)                 | 1124 (753)                  | 0.15         |
| Previous use of NIPPV         | 21 (37)                   | 3 (16)                      |              |
| Disease category               |                           |                             |              |
| Chronic obstructive pulmonary disease | 38 (67)                   | 11 (61)                     | 0.8          |
| Restrictive chest wall disease | 14 (25)                   | 2 (11)                      | 0.3          |
| Sleep apnoea/hypoventilation   | 5 (9)                     | 5 (28)                      | 0.05         |
| Pneumonia                      | 3 (5)                     | 2 (11)                      | 0.6          |
| Mean (SD) blood gases on admission |                         |                             |              |
| \(P_{aO_2}\) (kPa)            | 6.9 (2.12)                | 8.49 (2.46)                 | 0.012        |
| \(FiO_2\) (l/min)             | 1.4 (1.6)                 | 1.9 (1.2)                   | 0.27         |
| \(P_{aCO_2}\) (kPa)           | 8.95 (1.88)               | 9.59 (2.33)                 | 0.25         |
| pH                             | 7.35 (0.1)                | 7.33 (0.07)                 | 0.38         |

Values: number (%) unless stated otherwise
patients with an acute exacerbation of chronic obstructive pulmonary disease patients\(^2\), and similar results when treating these patients with NIPPV may simply reflect the poorer prognosis of severe acidosi.

NIPPV depends critically on patient cooperation, and if the technique is poorly tolerated its widespread adoption is unlikely. We have shown a high rate of tolerance (71%) in patients with no previous experience of NIPPV – comparable to figures quoted for more highly selected groups treated in intensive care.

As our study was retrospective we can not be sure that all patients admitted and treated with, or offered, NIPPV have been included. However, we have used several methods for case ascertainment and the risk of excluding appreciable numbers of cases is minimal.

The London Chest Hospital has no casualty department and most patients admitted are already known to the respiratory team. By contrast, patients treated at a district general hospital may not be known to the admitting team or ward staff, and this may affect the efficacy of NIPPV and patients' tolerance of the treatment. Furthermore, the ward staff at our hospital have considerable experience in caring for these patients. If our results are to be reproduced by other hospitals, patients treated with NIPPV may need to be cared for by comparably experienced staff. Success with NIPPV will probably require patients to be treated on a specified ward under the care of the respiratory team. The ward will need to be staffed adequately to enable close observation of the patients, and at least one nurse per shift must have experience in caring for patients using NIPPV. For a moderately large district general hospital we estimate there would be, on average, two inpatients at any one time requiring NIPPV. Each would need their own ventilator, at a cost of between £2,500 to £5,000 per ventilator, plus a small extra amount for tubing and masks. The additional nursing workload would vary over the year but would be approximately equal to one whole-time equivalent post.

Domiciliary NIPPV is the accepted treatment of choice for chronic respiratory failure due to restrictive chest wall diseases and nasal constant positive airway pressure (CPAP) for sleep apnoea\(^9\). Few data are available on the efficacy of NIPPV for acute exacerbation of these conditions\(^9\). In our patients, improvement in \(P_{A_O_2}\) was limited to the chronic obstructive pulmonary disease group, with smaller non-significant increases in those with restrictive chest wall disease and sleep apnoea/hypoventilation. In contrast, the \(P_{A_C_O_2}\) fell by a similar magnitude in all three disease categories. Early reductions in \(P_{A_C_O_2}\) have been shown in some studies but not in others\(^4\). This variability may result from differences in study populations or ventilator type and settings\(^6,13,14\). Results of nasal constant positive airways pressure treatment for acute deterioration in patients with sleep apnoea/hypoventilation determined after 24 hours\(^15\) are similar to our results with NIPPV at two hours. Compliance with NIPPV in the sleep apnoea/hypoventilation group was relatively poor, possibly because these patients had a greater degree of acidosi\(^13\).

In conclusion, we have shown that in patients with acute-on-chronic hypercapnoeic respiratory failure, early intervention with NIPPV as part of routine treatment on a general respiratory ward can achieve results similar to those in published clinical trials that showed a good outcome. The treatment allows safe oxygen therapy for patients with chronic obstructive pulmonary disease; increasing the \(P_{A_O_2}\) rapidly with a concurrent fall in \(P_{A_C_O_2}\) and improvement in pH. NIPPV also improves blood gas values when used to treat decompensation in patients with restrictive chest wall diseases and sleep apnoea/hypoventilation. Our results
support the routine use of NIPPV on non-intensive care wards for patients with acute-on-chronic respiratory failure.

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