

Mask intermittent positive pressure ventilation in chronic hypercapnic respiratory failure due to chronic obstructive pulmonary disease

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Mask intermittent positive pressure ventilation in chronic hypercapnic respiratory failure due to chronic obstructive pulmonary disease. P. Sivasothy, I.E. Smith, J.M. Shneerson. ©ERS Journals Ltd 1998.

ABSTRACT: Noninvasive ventilation in chronic obstructive pulmonary disease (COPD) has been shown to improve arterial blood gases but its long-term role has not been established.

We retrospectively studied 26 consecutive patients with hypercapnic ventilatory failure due to COPD in whom oxygen therapy caused worsening hypercapnia (defined as a rise in the daytime arterial carbon dioxide tension (P_{a,CO_2}) to >8.0 kPa or nocturnal transcutaneous carbon dioxide tension (P_{tc,CO_2}) to >9 kPa). All were treated with mask ventilation (15 with nasal and 11 face masks) at night and during daytime naps. Additional oxygen therapy was required in 15 patients.

The mean annualized death rate was 10.8% with a 1 and 3 yr survival of 92 and 68%, respectively. After 1 yr the median daytime P_{a,CO_2} had fallen by 1.35 kPa and the arterial oxygen tension (P_{a,O_2}) had risen by 2.4 kPa. In subjects not using additional oxygen the median overnight S_{a,O_2} rose by 12% and the P_{tc,CO_2} fell by 2.8 kPa after 1 yr. The haematocrit was significantly less than pretreatment at 6 months and 1 yr. Quality of life in the domain of role limitation by physical health (measured using the SF-36 questionnaire) improved significantly at 6 months.

Survival in this selected group with clinically stable airflow obstruction unable to tolerate oxygen therapy and treated with noninvasive mask ventilation is better than historical controls and is comparable to those able to tolerate oxygen therapy. Poor survival was associated with a low forced expiratory volume in one second, a low body mass index and a high nocturnal transcutaneous carbon dioxide tension. No difference in survival was found between those treated with mask intermittent positive pressure ventilation alone or with mask intermittent positive pressure and supplementary oxygen therapy.

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The use of noninvasive ventilation in pulmonary disease has been shown to improve the blood gases of patients both while awake [1–3] and asleep [1, 4, 5] but it has proved difficult to identify in which patients survival or quality of life can be improved [5–7]. Mask intermittent positive pressure ventilation (MIPPV) has been introduced in acute respiratory failure in patients with chronic obstructive pulmonary disease (COPD) as a short-term measure to reduce intubation rates and improve survival [8].

Long-term oxygen therapy (LTOT) improves survival in chronically hypoxic COPD patients [9, 10]; however, patients with a high arterial carbon dioxide tension (P_{a,CO_2}) had a poor prognosis both prior to oxygen therapy [11, 12] and (at least in the first year) with oxygen therapy [13]. In some patients, hypercapnia deteriorates with oxygen therapy manifesting with headaches, confusion, drowsiness and difficulty concentrating such that LTOT is not tolerated. Long-term MIPPV could in principle prevent this complication and improve survival.

We report our experience with nocturnal mask ventilation in patients with COPD with hypercapnic respiratory

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failure in whom oxygen therapy resulted in worsening daytime or nocturnal hypercapnia.

Materials and methods

Patient selection

The inclusion criteria were: 1) arterial oxygen tension (P_{a,O_2}) <7.3 kPa and P_{a,CO_2} >6.0 kPa while breathing room air; 2) stable clinical state, arterial blood gases and pulmonary function tests over the 3 weeks prior to the study (with a daytime arterial H^+ concentration between 35–48 $nmol^{-1}$); and 3) supplementary oxygen therapy failed either to raise the daytime P_{a,O_2} to >7.3 kPa without a rise in the awake P_{a,CO_2} to >8 kPa or failed to raise nocturnal oxygen saturations to $>90\%$ without a rise in the transcutaneous carbon dioxide tension (P_{tc,CO_2}) to >9 kPa. Patients with co-existing pulmonary diseases, neuromuscular or chest wall disorders were excluded. Twenty six patients, 13 males and 13 females, fulfilling these criteria were studied retrospectively.

Methods

All patients had initial arterial blood gas measurements (Ciba-Corning 238 Blood gas analyser; Medfield, MA, USA) performed breathing room air. Patients had haemoglobin concentration and haematocrit assessed at presentation. Total lung capacity (TLC) was assessed by body plethysmography (Masterlab, Jaeger, Wurzburg, Germany), forced expiratory volume in one second (FEV₁) and forced vital capacity (FVC) with a rolling seal spirometer (Vitalograph, Buckingham, UK) and the transfer factor of the lung for carbon monoxide (TL_{CO}) with a gas analyser (TT Autolink; P.K. Morgan, Rainham, UK).

Hypercapnia was assessed by continuous P_{tc,CO_2} monitoring (Radiometer TCM 3; Copenhagen, Denmark) and P_{a,CO_2} . Oxygenation was assessed using continuous oxygen saturation (S_{a,O_2}) monitored by a pulse oximeter (Ohmeda Biox 3740; Louisville, CO, USA) and P_{a,O_2} . Data from both the pulse oximeter and transcutaneous capnometer were simultaneously recorded on a chart recorder (Dash 3; Astromed, Warwick, RI, USA). Recordings were made as supplementary oxygen was titrated in increments of no more than 0.25 L·min⁻¹, to raise the oxygen saturation to >90%.

All patients were established on noninvasive MIPPV which was either with the volume preset Monnal D ventilator (TAEMA, Paris, France) or a pressure preset ventilator, either Nippy (Friday Medical Ltd, London, UK) or bi-level positive airway pressure (BiPAP) (Respironics Inc, Murrysville, PA, USA). MIPPV was initiated during an in-patient episode in each case. Oxygen therapy was given on initiation of MIPPV or subsequent review of patients to increase night-time and daytime S_{a,O_2} to >90% if this could not be achieved despite a reduction of the P_{a,CO_2} with MIPPV.

Patients were reviewed at 1 month, 6 months, 12 months, and 2 yrs. At each review, daytime arterial gas, overnight P_{tc,CO_2} and S_{a,O_2} , haemoglobin and haematocrit measurements were made. Patient ventilator settings and oxygen therapy were adjusted according to these results. Data obtained on patients who had an infective exacerbation of their pulmonary disease were excluded from later analysis at that time point. An infective exacerbation was defined as cough productive of mucopurulent or purulent sputum with either new changes on a chest radiograph or a raised white blood cell count.

Quality of life was assessed in 11 patients who were studied prior to MIPPV and after 1, 6 and 12 months of treatment using the SF-36 questionnaire. This measures the patient's perceptions of health in the domains of physical functioning, role limitations because of physical problems, bodily pain, general health perceptions, vitality, social functioning, role limitation because of emotional problems and mental health.

Statistical analysis

Statistical analysis of all data was performed using the Statistical Products and Service Solutions (SPSS) software for Windows (SPSS; Chicago, IL, USA). Comparisons of results between review visits was made using the Wilcoxon matched pairs signed rank test. Arterial blood gas measurement, S_{a,O_2} and P_{tc,CO_2} at differing inspiratory

oxygen fractions (F_{I,O_2}) on the same patient were used for paired analysis of P_{a,CO_2} or P_{tc,CO_2} but not for comparisons of overnight mean S_{a,O_2} . Survival was estimated using the Kaplan-Meier method from the initiation of MIPPV and survival comparisons were made using the log rank test between those using MIPPV alone or MIPPV with supplementary oxygen therapy. Analyses of predictive factors were made using the Mann-Whitney U test looking between: 1) survivors and nonsurvivors; and 2) those using MIPPV alone or MIPPV with supplementary oxygen therapy with regard to the variables of age, smoking history, per cent predicted FEV₁, body mass index (BMI), haematocrit and initial blood gases and blood gases at 1 month. Statistical significance was taken as $p < 0.05$ with the Bonferroni adjustment used to correct for repeat pairwise comparisons.

Results

The median age of patients at entry into the study was 66 (range 41–78) yrs. The median duration of follow-up was 17.5 (range 4–74) months. Self-reported smoking history, available in 25 patients, showed patients had been smoking for 43±25 (mean±SD) pack-years. The mean±SD FEV₁ was 0.68±0.37 L (27±14.6 % pred) and the mean±SD FVC was 1.99±0.49 L (63±17.8 % pred). The gas transfer was reduced with a TL_{CO} of 4.6±2.27 mmol·min⁻¹·kPa⁻¹ (58±25.9 % pred) and carbon monoxide transfer coefficient (K_{CO}) 1.10±0.46 mmol·min⁻¹·kPa⁻¹·L⁻¹. TL_{CO} values were unavailable in seven patients due to insufficient volumes for single breath analysis. Arterial blood gas measurements at presentation were performed on air in all patients. Sixteen patients had a daytime P_{a,CO_2} >8 kPa on daytime arterial measurement with supplementary oxygen therapy. The remaining 10 patients had a P_{tc,CO_2} >9 kPa during a night-time study, with added oxygen.

Initially 13 patients were treated with MIPPV alone at night since it raised night-time S_{a,O_2} to >90%. In the other 13 patients MIPPV was given with supplementary oxygen therapy initially at night but also during the day when their P_{a,CO_2} had been reduced. Subsequently supplementary oxygen therapy was introduced in two patients from the MIPPV alone group to maintain night-time S_{a,O_2} >90%. In patients prescribed oxygen, a combined total of at least 15 h·day⁻¹ use of nocturnal ventilation and daytime oxygen was recommended. In 13 out of 15 patients started on oxygen therapy it was prescribed at a rate of 8 l·min⁻¹ and six were prescribed 0.5 L·min⁻¹. During subsequent in-patient assessments the F_{I,O_2} was increased to maintain S_{a,O_2} >90% in five patients and decreased in two patients for hypercapnia. The mean duration of the initial stay was 10±2.3 days.

All patients tolerated MIPPV either *via* nasal mask (15 patients) or full face mask (11 patients). Compliance judged by hours of measured ventilation per day, using the internal ventilator clock, was 7.81±1.61 (mean±SD). Six patients died after starting MIPPV at 4, 5, 13, 14, 17 and 22 months. In all six patients death occurred during a chest infection with acute respiratory failure. One patient had developed a bronchial carcinoma but had continued with MIPPV.

The mean±SEM survival time was 55.1±6.92 months. Baseline comparisons between survivors and nonsurvivors with regard to age, FEV₁, P_{a,CO_2} , P_{a,O_2} , BMI, and

Table 1. — A comparison of results between survivors and nonsurvivors at pretreatment and 1 month

	Survivors (n=20)	Nonsurvivors (n=6)
Age yrs	67.0 (41–78.0)	60.0 (49.0–71.0)
FEV ₁ * % pred	26.0 (14.8–64.3)	15.2 (7.7–18.5)
P _a .CO ₂ kPa	8.2 (6.5–9.6)	9.6 (7.2–11.8)
P _a .O ₂ kPa	5.8 (4.0–6.9)	5.0 (4.6–6.9)
P _a .CO ₂ at 1 month kPa	7.5 (6.1–9.1)	7.5 (6.5–14.2)
P _a .O ₂ at 1 month kPa	7.5 (6.1–10.3)	7.0 (5.5–8.5)
S _a .O ₂ daytime %	79.0 (56.0–88.0)	73.0 (65.0–86.0)
P _{tc} .CO ₂ * kPa	8.9 (7.4–11.0)	11.4 (7.8–12.5)
BMI*	29.2 (20.4–43.8)	19.7 (15.5–30.1)
Duration of stay* days	9.5 (6.0–13.0)	12.0 (11.0–14.0)

Values are presented as median with range in parenthesis. FEV₁: forced expiratory volume in one second; P_a.CO₂: arterial carbon dioxide tension; P_a.O₂: arterial oxygen tension; S_a.O₂: arterial oxygen saturation; P_{tc}.CO₂: transcutaneous carbon dioxide tension; BMI: body mass index. *: p<0.05.

Table 2. — A comparison of results between mask intermittent positive pressure ventilation (MIPPV) alone or with oxygen therapy at pretreatment and 1 month

	MIPPV + oxygen (n=13)	MIPPV alone (n=13)
Age yrs	65.0 (41.0–77.0)	71.0 (54.0–78.0)
FEV ₁ % pred	20.0 (7.7–44.2)	23.5 (15.2–64.3)
P _a .CO ₂ kPa	7.9 (6.5–11.8)	8.8 (7.1–9.7)
P _a .O ₂ kPa	5.9 (4.0–6.9)	5.7 (4.7–6.9)
P _a .CO ₂ at 1 month kPa	7.5 (6.1–14.2)	7.45 (6.5–8.4)
P _a .O ₂ at 1 month kPa	7.4 (6.2–11.7)	7.6 (6.5–10.2)
S _a .O ₂ daytime %	79.0 (56.0–88.0)	78.0 (66.0–87.0)
P _{tc} .CO ₂ kPa	10.0 (7.4–12.5)	8.7 (7.6–11.0)
BMI	23.9 (16.1–40.5)	31.0 (15.5–43.8)
Duration of stay days	11.0 (9.0–14.0)	11.0 (6–13)

Values are presented as median with range in parenthesis. No significant differences were demonstrated. For definitions see legend to table 1.

duration of first stay (median and range) are shown in table 1. The patients who died had a significantly lower FEV₁ and BMI, a higher median overnight P_{tc}.CO₂ and a longer duration of first hospital admission. Although P_a.CO₂ was correlated with overnight P_{tc}.CO₂ (p<0.05) there was no association between it and survival. No significant differences between patients using MIPPV alone or those with supplementary oxygen therapy were shown for age, smoking duration, FEV₁ or pretreatment arterial blood gas measurement (table 2). The Kaplan-Meier survival analysis with the log rank test showed no significant differences between the two groups (fig. 1).

There was a statistically significant fall (p<0.05) of daytime P_a.CO₂ and rise in P_a.O₂ at 1 and 6 months and 1 and 2 yrs compared to pretreatment (figs. 2 and 3). There were no significant changes when daytime P_a.CO₂ and P_a.O₂ at 1 month were compared to those at 6 months, 1 and 2 yrs. Similarly overnight median P_{tc}.CO₂ and S_a.O₂ showed statistically significant decreases and increases respectively at 1 and 6 months, and 1 and 2 yrs compared to pretreatment with no significant changes between 1 and 6 months, and 1 and 2 yrs (figs. 4 and 5). The successive median haematocrit estimates up to 2 yrs showed a continuing decrease compared to admission (p<0.05) (fig. 6). The

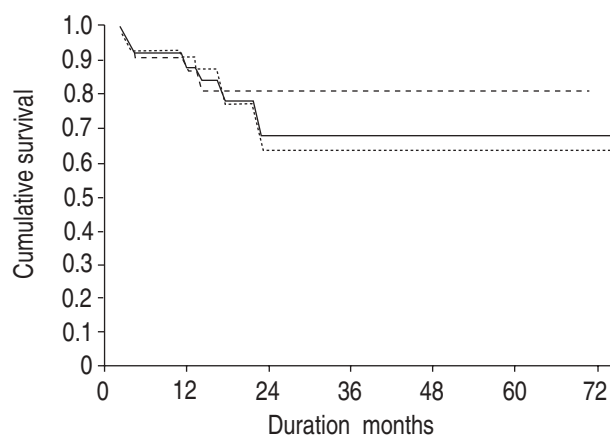


Fig. 1. — Kaplan-Meier survival graph for patients treated with mask intermittent positive pressure ventilation (MIPPV). —: whole group;: with oxygen; ----: without oxygen. Mean (\pm SEM) survival was: for the whole group 55 \pm 6.9 months; for the MIPPV group with supplementary oxygen therapy 53 \pm 8.9 months; and for the MIPPV group alone 55 \pm 7.3 months.

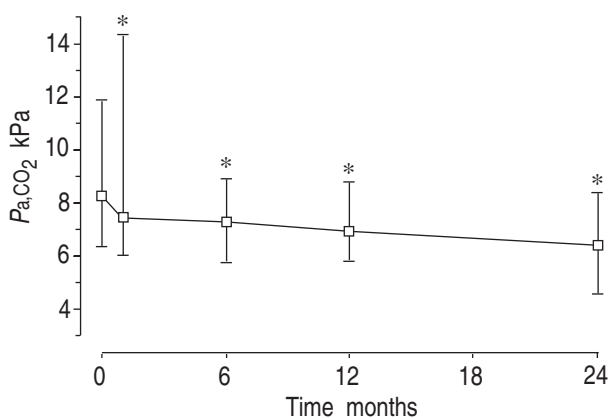


Fig. 2. — Median values and range in arterial carbon dioxide tensions (P_a.CO₂) in patients treated with mask intermittent positive pressure ventilation at: pretreatment (n=26); 1 month (n=20); 6 months (n=19); 1 yr (n=18); and 2 yrs (n=7). *: p<0.05 compared to pretreatment.

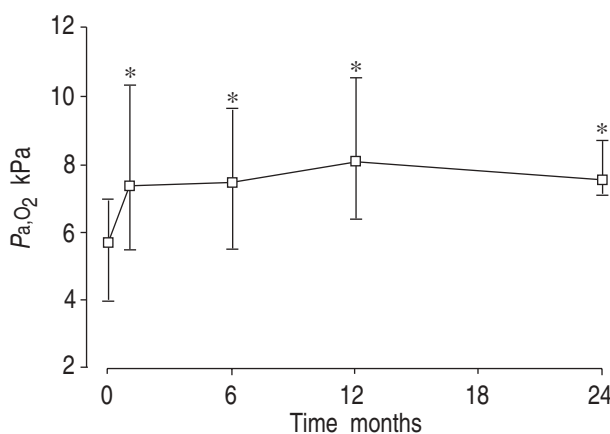


Fig. 3. — Median values and range in arterial oxygen tensions (P_a.O₂) in patients treated with mask intermittent positive pressure ventilation at: pretreatment (n=26); 1 month (n=20); 6 months (n=19); 1 yr (n=18); and 2 yrs (n=7). *: p<0.05 compared to pretreatment.

overall diuretic use for the population was similar at each time point.

Quality of life assessed by the SF-36 questionnaire revealed no significant changes in the patients' perceptions

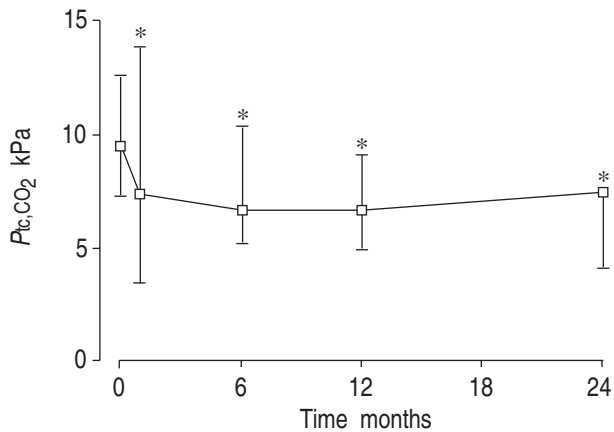


Fig. 4. – Median values and range in overnight transcutaneous carbon dioxide tensions ($P_{t,c}CO_2$) in patients treated with mask intermittent positive pressure ventilation at: pretreatment (n=26); 1 month (n=20); 6 months (n=19); 1 yr (n=18); and 2 yrs (n=7). *: p<0.05 compared to pretreatment.

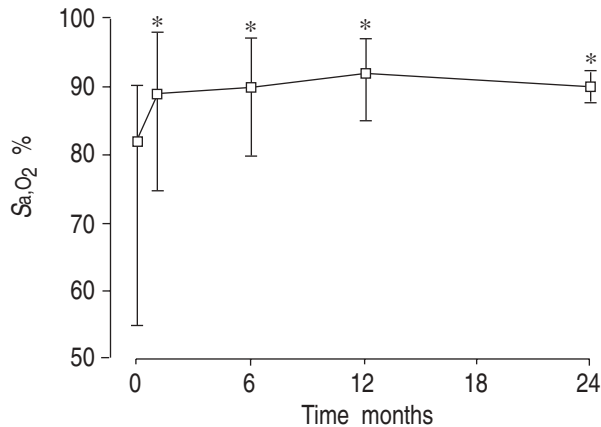


Fig. 5. – Median values and range in overnight arterial oxygen saturations (S_{a,O_2}) in patients treated with mask intermittent positive pressure ventilation at: pretreatment (n=26); 1 month (n=20); 6 months (n=19); 1 yr (n=18); and 2 yrs (n=7). *: p<0.05 compared to pretreatment.

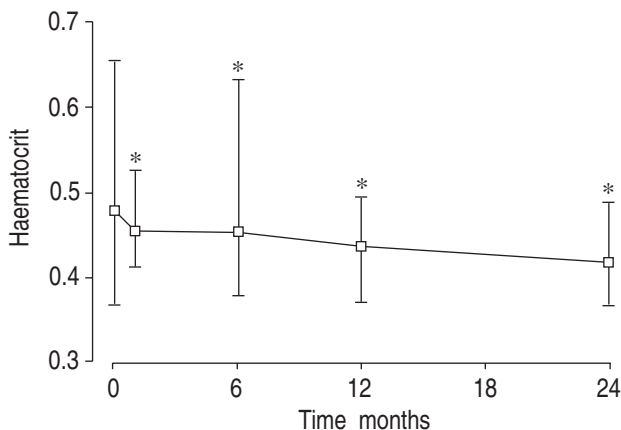


Fig. 6. – Median values and range of the haematocrit in patients treated with mask intermittent positive pressure ventilation at: pretreatment (n=26); 1 month (n=20); 6 months (n=19); 1 yr (n=18); and 2 yrs (n=7). *: p<0.05 compared to pretreatment.

of health in the domains of physical functioning, bodily pain, general health perceptions, energy, social functioning, role limitation because of emotional problems and mental health at pretreatment baseline compared to 1 and 6 months and 1 yr. Despite no perceived change in phys-

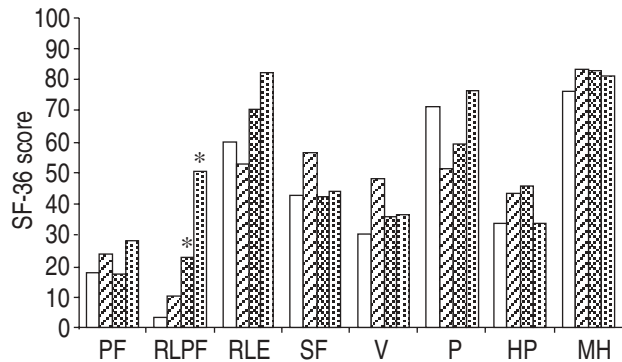


Fig. 7. – Median values of the SF-36 scores for 11 patients assessed at: □ : pretreatment; ▨ : 1 month; ▩ : 6 months; ▪ : 1 yr. PF: physical function; RLPF: role limitation due to physical function; RLE: role limitation due to emotional function; SF: social function; V: vitality; P: bodily pain; HP: health perception; MH: mental health. *: p<0.05 compared to pretreatment.

cal function there was a significant improvement in role limitation due to physical problems before and after 6 months and 1 yr of treatment. The overall changes in the median values for the above parameters are shown in figure 7.

Discussion

MIPPV is a well-established treatment for restrictive disorders [14, 15], but its role in the long-term treatment of COPD is not settled, with few and conflicting data with regard to survival, physiological changes and quality of life [8]. The present retrospective study of 26 patients was not controlled but the group is comparable to patients in several other studies of LTOT, tracheostomy ventilation and mask ventilation [6, 7, 9, 11, 13, 16, 17]. All the patients in the present study were hypoxic ($P_{a,O_2} < 7.3$ kPa), had severe airflow obstruction, were in a stable clinical state prior to therapy and were aged ≤ 35 yrs. The selection criteria for the present study resulted in a higher mean P_{a,CO_2} at entry and a lower FEV₁ % pred in patients than in previous trials of LTOT, home mechanical ventilation *via* tracheostomy or mask ventilation, where data are available (table 3).

In the present study of 26 patients, none of whom could tolerate oxygen therapy alone but were treated with MIPPV \pm supplementary oxygen therapy, the 3 and 5 yr survival rate was greater than the Medical Research Council (MRC) control group, the 15 h-day⁻¹ oxygen treatment group in the MRC trial and the Association Nationale pour le Traitement à Domicile de l'Insuffisance Respiratoire Chronique (ANTADIR) study "COPD" patients [9, 11]. The 3 yr survival was similar to the continuous oxygen therapy group in the Nocturnal Oxygen Therapy Trial (NOTT) study (table 3) [10]. The benefits on survival were probably even greater since eight patients in the present study had been on LTOT prior to starting MIPPV and would have already utilized some of the survival benefit that LTOT offered. These results suggest that in this selected group of patients with hypercapnic respiratory failure, MIPPV \pm supplementary oxygen is at least as effective as continuous oxygen therapy in improving survival rates.

Adequate oxygenation was achieved in our patients with MIPPV alone or with low oxygen flow rates. In 86%

Table 3. – A comparison between the present study and previous studies using oxygen, tracheostomy or mask ventilation

	Year	Sub- jects n	Acute cases included?	Age yrs	P_{a,CO_2} kPa	P_{a,O_2} kPa	FEV ₁ L	FEV ₁ %	Survival		
									1 yr n	3 yrs n	5 yrs n
Present study	1997	26	No	64.7	8.61	6.00	0.68	27.2	92	68	68
Oxygen therapy											
MRC (control males) [9]	1981	33	No	56.2	7.07	6.85	0.65	-	78	42	18
MRC (treated males) [9]	1981	33	No	58.2	7.30	6.7	0.76	-	88	52	44
NOTT (nocturnal oxygen) [10]	1980	102	No	65.7	5.77	6.85	-	29.5	80	44	-
NOTT (continuous therapy) [10]	1980	101	No	65.2	5.77	6.76	-	29.5	87	66	-
ANTADIR+ [9]	1996	7370	Yes	68.2	6.22*	7.12*	-	35.3	74	47	30
Tracheostomy ventilation											
ROBERT <i>et al.</i> [16]	1983	50	Yes	60	5.85*	7.05*	0.72	33	88	54	18
MUIR <i>et al.</i> [17]	1994	259	Yes	63.3	7.44*	6.70*	0.73	-	88	60	44
Mask ventilation											
LEGER <i>et al.</i> [6]**	1994	50	Yes	63	7.05*	6.26*	-	36	85	75	43
SIMONDS <i>et al.</i> [7]**	1995	33	Yes	57.4	8.3*	6.10*	0.58	-	88	70	43

Values are presented as absolute value or as mean. *In the ANTADIR study 94.9% of COPD patients were treated with oxygen therapy alone although 3.0% and 2.1% were treated with mask or tracheostomy ventilation, respectively [13]. In the ANTADIR study the "COPD" group included patients with bronchiectasis and asthma and these have been excluded from comparison; **: survival figures are those patients continuing ventilation; *: these results included patients with an acute exacerbation in whom these values may be worse than their stable state. MRC: Medical Research Council; NOTT: Nocturnal Oxygen Therapy Trial; ANTADIR: Association Nationale pour le Traitement à Domicile de l'Insuffisance Respiratoire Chronique. For further definitions, see legend to table 1.

of patients the oxygen flow rates prescribed was $\delta 1$ L·min⁻¹. The flow rates were individually titrated to achieve $S_{a,O_2} > 90\%$ once P_{a,CO_2} was reduced. In contrast, patients in the MRC trial were treated with a uniform oxygen flow rate of 2 L·min⁻¹ and in the NOTT study the minimum oxygen flow rate was 1 L·min⁻¹ during the day and 2 L·min⁻¹ at night [9, 10]. The prescription of low oxygen flow rates in our patients minimized the risk of hypercapnia developing.

Survival comparisons between the patients in the present study treated with MIPPV \pm oxygen therapy and those in other studies using mechanical ventilation *via* a mask or tracheostomy, or oxygen therapy alone are complex because patients can start treatment at differing degrees of severity and stages in the natural history of their disease. In previous large series of the use of tracheostomy or MIPPV in COPD, the populations have been heterogeneous with a range of severity of airflow obstruction, P_{a,O_2} and P_{a,CO_2} (table 3) [6, 7, 16, 17]. In some studies patients started on treatment during an acute deterioration [6, 17] or while transferring from naso- or oro-tracheal ventilation to tracheostomy or MIPPV [7, 17] have been included. The inclusion of acute cases is a confounding factor possibly resulting in an apparent improved long-term survival in these series. Although the hospital survival of patients with an acute exacerbation is poor, the survival of those who reach discharge from hospital without home ventilation was 86 and 91% at 1 yr in two large studies [18, 19] which is superior to stable patients treated with LTOT. In our study, survival was better than the tracheostomy series with none of the morbidity associated with that treatment. Compared with other MIPPV series, results from the present study were worse at 3 yrs, probably due to our exclusion of acute cases, but despite this, the 5 yr survival was superior to previously reported studies.

The predictors of poor survival in the present study were a low FEV₁, a low BMI, a long duration of initial

hospital stay and a high overnight P_{t,CO_2} . Our finding of an association between low BMI or body weight with poor survival agrees with other studies [10, 20, 21]. A low BMI has been correlated in a study of 126 patients with stable COPD with a low FEV₁ suggesting that they are not independent predictors of poor survival [22]. A high daytime P_{a,CO_2} has been shown to be a predictor of poor survival either with [9, 23] or without [12, 13] oxygen therapy. In the ANTADIR study "COPD" patients with a high P_{a,CO_2} had a high initial mortality but after 1 yr had a greater survival chance than those with a low or normal P_{a,CO_2} . However the "COPD" patient group in this study included subjects with asthma or bronchiectasis, and 15% of patients had a $P_{a,O_2} > 8$ kPa [11].

In the present study, P_{a,CO_2} was not found to be of prognostic value partly as result of entry criteria which excluded patients with a low or normal P_{a,CO_2} . More importantly, the initiation of MIPPV reduced P_{a,CO_2} by 1.8 kPa in patients thus eliminating or ameliorating this risk factor for mortality.

Other studies of MIPPV in COPD have shown that it can reduce P_{a,CO_2} and increase P_{a,O_2} [2, 6, 7] though in one series it had no effect [5]. In the current study, a rise is also shown in the median daytime P_{a,O_2} of 2.4 kPa and nocturnal S_{a,O_2} (excluding patients started on oxygen). The differences in daytime arterial blood gas measurement were significant after only 1 month and were maintained thereafter.

The significant decrease in haematocrit noted after treatment with MIPPV \pm oxygen therapy probably reflects improved oxygenation and subsequent reduction in secondary polycythaemia. This could not be explained by an alteration in the plasma volume caused by diuretic therapy as there was no overall change in diuretic therapy for the study population. The continued reduction up to 1 yr may be attributable to the gradual increase in the median P_{a,O_2} up to 1 yr and the delayed haematological

response caused by the life span of the erythrocyte which is 100–120 days.

All patients continued to comply with MIPPV, with 81% using it for at least 7 h·day⁻¹. Other studies of MIPPV in COPD have emphasized difficulties tolerating the mask, abdominal distension, poor sleep and anxiety, which usually arise within 3 months of starting ventilation [1, 4, 5]. In two large long-term studies of MIPPV in COPD 6 and 15% of patients discontinued therapy [6, 7]. The superior results in the present study might be due to a longer in-patient initiation of therapy: patients were only discharged when they had fully adjusted to using MIPPV. This took between 6 and 14 days compared to 2 and 5 days allocated in the study by Leger *et al.* [6]. Domiciliary visits were available to patients who lived within a 30 mile radius of the hospital. All patients had regular in-patient review with the option of using a 24 h telephone helpline for any problems encountered with mask ventilation.

The only previous study of the SF-36 in patients with COPD on ventilation was a single estimation when patients were already on therapy [7]. Subjects in the present study were more incapacitated with a greater degree of physical impairment but had similar mental health, social function, bodily pain and energy scores (using pretreatment data). One previous study of the neuropsychological function and physical function of MIPPV in COPD using different tests revealed improved attention, verbal and visual memory, constructional apraxia and self regulation but no change in physical function [5]. The quality of life on MIPPV measured by the SF-36 in patients in the present study was unchanged except for the domain of role limitation by physical health which significantly improved at 6 months and 1 yr. This was derived from questions on the physical health of patients over the preceding 4 weeks concerning: 1) Had they cut down the amount of time spent on activities? 2) Had they accomplished less than they would have liked? 3) Were they limited in the activities they could perform? and 4) Had they experienced difficulty performing their work or other activities. The results suggest that patients treated with MIPPV show an acceptance of their severely impaired quality of life or perhaps an adaptation to it allowing them to circumvent this disability despite no change in their self-reported physical function.

It is recommended that patients with hypercapnic respiratory failure commenced on oxygen therapy should be assessed with arterial blood gas measurements, pulse oximetry and P_{tc,CO_2} during sleep both before and after therapy. If supplementary oxygen therapy fails to raise the daytime P_{a,O_2} to >7.3 kPa without a rise in the awake P_{a,CO_2} to >8 kPa or raise nocturnal oxygen saturations to >90% without a rise in the transcutaneous P_{tc,CO_2} to >9 kPa, MIPPV should be instituted. Oxygen therapy may be required to supplement MIPPV if this alone fails to raise P_{a,O_2} despite a fall in P_{a,CO_2} .

The findings indicate that mask intermittent positive pressure ventilation in hypercapnic patients with chronic obstructive pulmonary disease unable to tolerate oxygen therapy is associated with a prognosis which is at least as good as eucapnic patients treated with long-term oxygen therapy and better than hypercapnic patients treated with long-term oxygen therapy and less well-defined groups treated with tracheostomy ventilation. The results were achieved with 86% of patients receiving $\delta 1$ L·min⁻¹ oxy-

gen and all patients tolerating mask ventilation. The high compliance and the emphasis on attempting to normalize the arterial blood gases probably contributed to the prolonged survival in this series.

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