Original articles

Intermittent negative pressure ventilation in the treatment of hypoxic hypercapnic coma in chronic respiratory insufficiency

Antonio Corrado, Eduardo De Paola, Massimo Gorini, Andrea Messori, Giovanni Bruscoli, Sandra Nutini, Donatella Tozzi, Roberta Ginanni

Abstract

Background **– In recent years non-invasive ventilatory techniques have been used successfully in the treatment of acute on chronic respiratory failure (ACRF), but careful selection of patients is essential and a comatose state may represent an exclusion criterion. The aim of this retrospective and uncontrolled study was to evaluate whether a non-invasive ventilatory technique such as the iron lung could also be used successfully in patients with hypoxic hypercapnic coma, thus widening the range for application of non-invasive ventilatory techniques.**

Methods **– A series of 150 consecutive patients with ACRF and hypoxic hypercapnic coma admitted to our respiratory intensive care unit were evaluated retrospectively. The most common underlying condition was chronic obstructive pulmonary disease (79%). On admission a severe hypoxaemia (Pa_{O_2} 5.81 (3.01) kPa) and hypercapnia (Pa_{CO_2} 14.88 (2.78) kPa) associated with a decompensated acidosis (pH 7.13 (0.13)) were present, the Glasgow coma score ranged from 3 to 8, and the mean APACHE II score was 31.6 (5.3). All patients underwent intermittent negative pressure ventilation with the iron lung. The study end point was based on a dichotomous classification of treatment failure (defined as death or need for endotracheal intubation) versus therapeutic success.**

Results **– There were 45 treatment failures (30%) and 36 deaths (24%). Nine patients (6%) required intubation because of lack of airway control. The median total duration of ventilation was 27 hours per patient (range 2–274). The 105 successfully treated cases recovered consciousness after a median of four hours (range 1–90) of continuous ventilatory treatment and were discharged after 12.1 (9.0) days.**

Conclusions **– These results show that, in patients with acute on chronic respiratory failure and hypoxic hypercapnic coma, the iron lung resulted in a high rate of success. As this study has the typical limitations of all retrospective and uncontrolled studies, the results need to be formally confirmed by controlled prospective studies. Confirmation of these results could widen the range of application of non-invasive ventilatory techniques.**

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Keywords: intermittent negative pressure ventilation, hypoxic hypercapnic coma, chronic respiratory insufficiency.

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To avoid endotracheal intubation and its attendant complications, in recent years non-invasive techniques of mechanical ventilation have been successfully employed in several studies for the treatment of episodes of acute respiratory failure in patients with chronic respiratory insufficiency.[1–13] However, for some types of ventilatory support (such as those employing a positive pressure at the airway opening through a face or nose mask) careful selection of patients is essential. In particular, a comatose state has been proposed as an exclusion criterion.[14]

At our institution the treatment of patients with chronic respiratory insufficiency (including those in a comatose state) who present with an episode of acute respiratory failure of any degree of severity has been based for many years on a conservative non-invasive ventilatory method which utilises a body ventilator (iron lung) to provide an intermittent negative pressure (INPV).[8] The aim of this uncontrolled and retrospective study was to evaluate the effects of our non-invasive ventilatory method in the treatment of hypoxic hypercapnic coma in patients with acute on chronic respiratory failure (ACRF).

Methods

STUDY DESIGN AND PATIENT SELECTION

This open non-controlled study consisted of a retrospective assessment of all patients with ACRF and hypoxic hypercapnic coma who were treated at our institution with the iron lung between 1983 and 1993. Hypoxic hypercapnic coma was defined as loss of consciousness (Glasgow scale score ≤ 8)[15] with Pa_{CO_2} >10.64 kPa and pH <7.20.[16] The homogeneity

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of this evaluation over time has been facilitated by the presence of the same medical staff in the ward over the whole period of the study.

In most cases the condition underlying the chronic respiratory insufficiency was chronic obstructive pulmonary disease (COPD). The diagnosis of COPD was established when, in a clinical stable condition, forced expiratory volume in one second (FEV_1) was less than 70% predicted and FEV_1/vital capacity (VC) was lower than 0.70 after bronchodilators (salbutamol 200 μg). In the absence of lung function measurements COPD was defined by a history of productive chronic cough and dyspnoea for more than 10 years in heavy smokers and radiological evidence of hyperinflation.

PATIENT EVALUATION, MODALITIES OF TREATMENT, AND OUTCOME ASSESSMENT

On admission each patient underwent arterial blood gas analysis, pH measurement (BMS3 and ABL2 apparatus, Radiometer, Copenhagen), and electrocardiographic (ECG) recordings. The level of coma and the severity of illness at entry were evaluated by the Glasgow coma scale[15] and APACHE II system,[17] respectively. The APACHE II system was applied retrospectively on the basis of all parameters available at entry. Urinary concentrations of benzodiazepines were measured in all patients admitted in a comatose state.

All patients underwent ventilatory treatment with an intermittent negative pressure ventilator (Iron Lung, Models CZ800 and C900, Coppa Co., Biella, Italy) and received oxygen therapy as well as standard drugs (intravenous theophylline, subcutaneous β_2 agonists, intravenous cardiokinetic agents, diuretics, steroids, and antibiotics). All the models of iron lung which we used could not be triggered but were particularly versatile; high levels of positive and negative pressure acting externally to the body could be reached (range -80 to $+80$ cm H_2O), and inspiratory and expiratory time (range 0.8–9.9 seconds), end inspiratory and end expiratory pauses (0.3–1.9 seconds) could be set independently.

The respiratory rate was obtained indirectly by adjustment of inspiration, expiration, and pause times. All these parameters were displayed on the control panel. The ability to adjust these parameters enabled the ventilatory treatment to be individualised. Furthermore, these models had windows that permitted observation of patients and the intervention of nurses when necessary. Portholes were available through which catheters and monitor leads could be passed.

The ventilatory treatment was instituted immediately and carried out continuously until consciousness was recovered with a good level of spontaneous breathing. The ventilatory treatment was then provided intermittently (from a maximum of two hours four times daily to a minimum of one hour four times daily) until stable levels of pH (7.37–7.40) were reached. The ventilator was set to deliver pressures ranging from -40 to -60 cm H_2O (negative pressure) and from $+10$ to $+20$ cm H_2O (positive pressure)[13 18 19] at a rate ranging from 11 to 27 breaths/min. The negative pressures were chosen to elicit a tidal volume (VT) equal to 10 ml/kg (recorded at the mouth by means of Wright's ventilograph).

In all patients a nasogastric tube was inserted to minimise the risk of pulmonary aspiration; oxygen was provided by nasal cannula or Venturi's mask to increase Pa_{O_2} to 8–9.31 kPa. To prevent obstruction of upper airways due to collapse of the tongue an oropharyngeal airway was positioned until patients recovered consciousness. During the ventilatory treatment electrocardiographic activity, systemic blood pressure, and breathing rate were regularly monitored in all patients. Arterial blood samples were also taken at regular intervals during the ventilatory session – a 30 minute sampling frequency was employed during the first cycle of ventilation and thereafter this was reduced to at least one blood sample per ventilatory session.

Ventilatory treatment with the iron lung was judged to be inadequate when there was evidence of insufficient control of the upper airways or when it was impossible to obtain satisfactory ventilation (VT<5 ml/kg) and a substantial improvement in gas exchange (decrease in Pa_{CO_2} of <1.33 kPa and Pa_{O_2} of >8 kPa within one hour of the start of mechanical ventilation) despite optimal setting of the ventilator or there was worsening coma (within 12–24 hours of the start of mechanical ventilation). Under these circumstances endotracheal intubation was performed and mechanical ventilation continued with the iron lung. When this failed, intermittent positive pressure ventilation was given.

After discharge all subjects were entered into a programme of monthly visits at our outpatient clinic. Patients who failed to attend were contacted by telephone in order to ascertain their status.

DEFINITION OF STUDY END POINTS

To assess the efficacy of the iron lung in each individual patient we defined treatment failures as cases who required intubation or who died while in hospital. All cases with other outcomes were considered a "therapeutic success".

ANALYSIS OF DATA

Standard statistical techniques were used (*t* test, Mann-Whitney test, χ^2 test, repeated measurement analysis of variance). A multivariate logistic regression (Css Statistica MS-DOS program, release 3.1, Statsoft Inc, Tulsa, Oklahoma, USA) was used to evaluate the effect of dichotomous variables on the achievement of therapeutic success. Unless otherwise indicated, all data are presented as mean (SD).

Results

PATIENTS

The study population consisted of 150 consecutive patients with ACRF and hypoxic hypercapnic coma admitted to our respiratory

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Table 1 Mean (SD) anthropometric data, APACHE II score, Glasgow scale score, and arterial blood gas analysis in 150 patients in hypoxic hypercapnic coma on admission into the respiratory intensive care unit

Age (years)	68 (11)
Body weight	61.7 (11.8)*
Ratio body weight/ideal weight (%)	103 (25)*
APACHE score	31.6 (5.3)
M/F	106/44
Pa_{O_2} (kPa)	5.81 (3.01)§
Pa_{CO_2} (kPa)	14.88 (2.78)§
pH	7.13 (0.13)§
FEV_1 (% predicted)	31.4 (14.6)**
FEV_1/VC	40.0 (12.8)**
Glasgow coma scale:	
score 3	n = 7
score 4	n = 15
score 5	n = 10
score 6	n = 35
score 7	n = 18
score 8	n = 65

Pa_{O_2}, Pa_{CO_2} = arterial oxygen and carbon dioxide tensions, FEV_1 = forced expiratory volume in one second.
* This information was available in a subgroup of 78 patients.
§ The following blood gas data were measured in the subgroup of 82 patients who were breathing air on admission: Pa_{O_2} = 4.27 (1.42) kPa, Pa_{CO_2} = 13.43 (2.13) kPa, pH = 7.16 (0.09).
** This information was available in a subgroup of 56 patients in whom the diagnosis of COPD was made on the basis of spirometric measurements. In the remaining 62 patients with COPD the diagnosis was made in the absence of spirometric measurements.

intensive care unit between 1983 and 1993. During this period a total of 1280 other patients were admitted to the intensive care unit with acute respiratory failure with hypoxaemia, hypercapnia, and acidosis without impairment of consciousness. The 150 patients reported herein included no cases of readmission. Their characteristics are shown in table 1. The disease underlying chronic respiratory insufficiency was COPD alone in 81 cases, COPD associated with other chronic diseases such as hypertension, ischaemic cardiomyopathy, renal insufficiency, cerebral vascular disease, or chronic lymphatic leukaemia in 37, chest wall deformity in nine, neuromuscular disorders in two, sequelae of pulmonary tuberculosis in 16, obesity in four, and cystic fibrosis in one. Sixty eight patients were receiving oxygen at the time of admission to the intensive care unit. The causes leading to coma were exacerbation of chronic disease in 74, pneumonia in 41, use of sedative agents in 23, congestive heart failure in seven, and pulmonary emboli in five. No cases were seen who were receiving sedatives and in whom another cause of coma was identified. All of the 23 patients whose coma was caused by sedatives had measurable urinary levels of benzodiazepines; in the remaining 127 cases the urinary test for benzodiazepines was negative and their history excluded the use of benzodiazepines or other sedatives.

Table 2 Stratification of treatment failures and fatalities by underlying disease and causes leading to coma

Disease	*No. of patients*	*No. of treatment failures*	*No. of deaths*
Underlying disease*			
COPD (alone)	81	24 (30%)	19 (23%)
COPD associated with other systemic diseases	37	11 (30%)	9 (24%)
Chest wall diseases	9	3 (33%)	2 (22%)
Sequelae of pulmonary tuberculosis	16	6 (37%)	5 (31%)
Other	7	1 (14%)	1 (14%)
Causes leading to coma**			
Exacerbation of chronic disease	74	19 (26%)	14 (19%)
Pneumonia	41	16 (39%)	14 (14%)
Sedative agents	23	3 (13%)	2 (9%)
Pulmonary emboli	5	3 (60%)	3 (60%)
Congestive heart failure	7	4 (57%)	3 (43%)

* Treatment failures ($\chi^2 = 2.01$, df = 4, $p > 0.50$) and mortality ($\chi^2 = 0.85$, df = 4, $p > 0.50$) not influenced by the underlying disease.
** Treatment failures ($\chi^2 = 10.0$, df = 4, $p < 0.05$) and mortality ($\chi^2 = 11.2$, df = 4, $p < 0.025$) significantly influenced by the cause leading to coma.

The first cycle of ventilatory treatment lasted a median of four hours (range 1–120). The median total duration of ventilation (total number of hours over all cycles excluding interruptions) was 27 hours per patient (range 2–274). For the initial cycle of ventilatory treatment the following median settings of the iron lung were used: (a) negative pressure −48 (range −40 to −60) cm H_2O; (b) positive pressure +15 (range +10 to +20 cm H_2O; (c) breath rate 15 (range 11–27) breaths/min; (d) inspiratory time 1.2 (range 0.8–1.7) seconds; (e) expiratory time 1.9 (range 0.8–3.0) seconds; (f) pauses 0.4 (range 0.3–0.4) seconds.

On admission bradypnoea was observed (≥ 10 breaths/min) in 56.7% of the patients and the iron lung was set to a respiratory rate of 15 breaths/min with TI/TTOT of 30% in these patients. In 14% of the patients the respiratory rate was 11–27 breaths/min and in these patients the iron lung was individually set to facilitate adaptation to the ventilator. In this group the values of TI ranged from 0.8 to 1.7 seconds, TE from 0.8 to 3.0 seconds, pauses from 0.3 to 0.4 seconds, and respiratory rate from 11 to 27 breaths/min. In 29% of the patients the respiratory rate was more than 27 breaths/min, a value that corresponds to the highest frequency which could be provided by the iron lung used in the study. These patients were initially ventilated with a frequency of 27 cpm, TI of 0.8 seconds, and TI/TTOT of 0.36. The adaptation of patients to the ventilator was assessed by direct observation and by analysis of tidal volume and blood gas measurements.

OUTCOME OF NON-INVASIVE VENTILATORY TREATMENT

The non-invasive ventilatory treatment with the iron lung was successful in 105 patients (70%), with 45 (30%) treatment failures. All patients classified as "therapeutic successes" were alive after a median follow up period of 23 weeks (range 5–45). Nearly all the cases of therapeutic success were patients in whom the episode of acute respiratory failure was controlled by the INPV and who were then successfully weaned from the iron lung with a subsequent uneventful course of the disease. Of the 45 treatment failures 36 (24%) died and nine needed intubation. The 36 who died included three patients who died after intubation. Table 2 shows that the distribution of treatment failures and fatalities is homogeneous across the various disease groups. Among the causes leading to coma, pulmonary emboli and congestive heart failure were associated with the highest rate of therapeutic failure, followed by pneumonia and exacerbation of chronic disease in patients with COPD.

The rates of treatment failure and mortality stratified according to the level of coma are

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Table 3 Rates of treatment failure and mortality according to level of coma

Level of coma	No. of patients	Treatment failures n (%)	Deaths n (%)	Median (range) survival (hours)*
3	7	7 (100)	6 (86)	72 (24–168)
4	15	12 (80)	9 (60)	96 (8–480)
5	10	4 (40)	3 (30)	72 (48–72)
6	35	9 (26)	6 (17)	96 (14–192)
7	18	3 (17)	3 (17)	120 (13–144)
8	65	10 (15)	9 (14)	216 (48–336)

* These data refer exclusively to the subgroup of 36 patients who died.

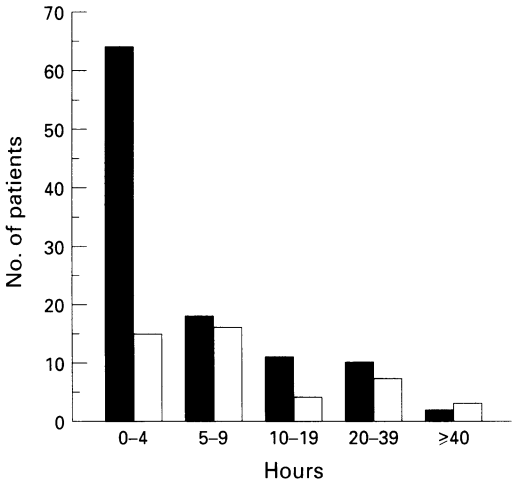

Figure 1 Duration of the initial cycle of ventilation in patients successfully treated with ventilation (■) and those in whom treatment failed (□).

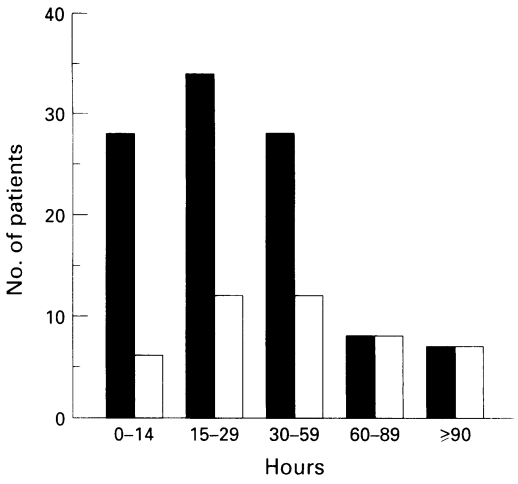

Figure 2 Total duration of ventilation in patients successfully treated with ventilation (■) and those in whom treatment failed. (□).

reported in table 3. While these rates are obviously found to improve as the depth of coma decreases, it is interesting to note that the most striking difference is seen between patients with a coma level of 8 to 6 who have a relatively homogeneous pattern and those with a level of 5 or less in whom mortality increased steeply as the coma worsened.

In the subgroup of 36 patients who died (all while in hospital), 15 had not been weaned off the ventilator and 21 who had been weaned died of extrarespiratory complications including myocardial infarction (three), cardiac arrest (seven), cerebral haemorrhage (three), renal failure (two), and gastrointestinal haemorrhage (six). In these 21 patients the median interval between discontinuation of ventilatory treatment and death was 48 hours (range 7–242). Survival information for the subgroup of 36 patients who died is shown in table 3. Those with a deeper level of coma (levels 3–5) survived 72–96 hours while those with a lower depth of coma (levels 5–8) survived 96–216 hours.

Of the nine patients who required intubation because of the lack of airway control, one was ventilated by intermittent positive pressure ventilation (IPPV), three were intubated and maintained under INPV, and five were first ventilated with the iron lung and then with IPPV. Of these nine patients, three died without being weaned while the remaining six survived. Five patients developed clinical and radiographic findings suggestive of aspiration pneumonia during INPV but all were successfully treated.

Figures 1 and 2 show the duration of the initial cycle of ventilation and total duration of ventilation in the two groups. In the 105 successfully treated cases the first cycle of continuous ventilatory treatment lasted a median of four hours (range 1–90) and led to the recovery of consciousness. In the group of 45 failures the first cycle of continuous ventilatory treatment lasted a median of seven hours (range 2–120). The difference in the duration of the first cycle of continuous ventilatory treatment between the two groups was significant ($p = 0.002$, Mann–Whitney test). The difference in the total duration of ventilation (median 40 hours (range 2–274) in patients with an unfavourable outcome compared with 25 hours (range 2–230) in those treated successfully) was also significant ($p = 0.003$, Mann-Whitney test).

Table 4 shows the arterial blood gas tensions and pH for the therapeutic successes and failures. The values on admission are influenced by the fact that 68 patients (46 in the successfully treated group and 22 in the treatment failure group) were receiving oxygen therapy when blood gas analysis was performed. The data at discharge of the former group reveal a global chronic respiratory insufficiency in metabolic compensation. The statistical analysis showed that the improvement in arterial blood gas tensions and pH was significant in both groups. Table 5 shows the changes in arterial blood gases and pH recorded on an hourly basis during the initial eight hours of the first cycle of INPV in patients with a favourable outcome and in those in whom treatment failed.

The mean (SD) hospital stay was 12.1 (9.0) days in the 105 patients classified as therapeutic successes compared with 6.6 (5.9) days in the 45 patients who did not respond.

The results of our multivariate analysis based on logistic regression showed that two variables (coma level ≤ 6 on the Glasgow scale and age ≥ 70 years) had a negative prognostic value at levels of statistical significance. The relative risk of treatment failure was 7.1 (95% CI 2.2 to 22.6) for patients with a coma level of ≥ 6 compared with those with a coma level of 7 or 8 and 3.3 (95% CI 1.1 to 9.7) for patients aged 70 years or more compared with those aged less

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Table 4 Mean (SD) blood gas values and pH in the 150 patients

	Favourable outcome (n=105)				*Treatment failures (n=45)*		
	On admission	*At consciousness recovery§*	*At discharge*	*p value**	*On admission*	*At consciousness recovery§*	*p value***
Pa_{O_2} (kPa)	6.0 (3.10)	7.4 (2.17)	8.3 (1.82)	<0.001	5.4 (2.74)	7.4 (3.94)	0.01
Pa_{CO_2} (kPa)	14.7 (2.78)	9.3 (1.82)	7.0 (1.42)	<0.001	15.4 (2.74)	8.6 (1.74)	<0.001
pH	7.14 (0.10)	7.32 (0.07)	7.38 (0.05)	<0.001	7.11 (0.18)	7.36 (0.09)	<0.001

§ Values measured at the end of ventilatory treatment during spontaneous breathing.
* Assessed by repeated measurement analysis of variance.
** Assessed by paired *t* test.

than 70 years. The other variables examined (Pa_{CO_2} on admission, Pa_{O_2} on admission, pH on admission, pulmonary emboli, congestive heart failure) did not reach statistical significance. It is of interest to note that the level of coma implied a markedly enhanced risk of treatment failure (more than sevenfold increase).

Discussion

Intubation and mechanical ventilation of patients (especially those with COPD) may cause significant complications[16 20 21] which may in turn be associated with a poor outcome.[16] Mortality rates following intubation and mechanical ventilation range from 22%[22] to 80%.[23]

In recent years non-invasive ventilation (usually using positive pressure via a well fitting nasal or face mask) has been successfully employed in the treatment of acute on chronic respiratory failure.[1-13] The rate of treatment failure – that is, the need for endotracheal intubation and/or death – using non-invasive techniques has ranged from 18%[10] to 40%.[7] However, nearly all of the patients reported in these studies were not comatose, and even in the study which reported the highest rate of treatment failure (40%) only three of the 30 patients were comatose.[7] It has been suggested that the presence of coma is a contraindication to non-invasive ventilation.[14 24]

All our patients were comatose, severely acidotic, and with a high APACHE II score at the time of admission. In the other studies of non-invasive ventilation[2-5 7 9-13] the pH on admission ranged from 7.23 to 7.38, Pa_{CO_2} from 6.5 to 11.0 kPa, and in the three studies[9 10 13] in which it was reported the APACHE II scores were 21.0 (6.0), 18.4 (1.2), and 17.9 (1.2), respectively. The mean pH of 7.13, Pa_{CO_2} of 14.9 kPa, and APACHE II score of 31.6 in our patients are all worse than those reported in these studies. Using the iron lung our overall treatment failure rate was 30%, with a mortality of 24% and a rate of endotracheal intubation of 6%, and for patients with COPD the figures were 30%, 24%, and 6%, respectively. These results are comparable with other studies using both invasive and non-invasive techniques. Both Brochard *et al*[12] and Ambrosino *et al*[25] have shown that failure of non-invasive positive pressure ventilation was more likely with more severe acidosis. Our study has shown that even very severely acidotic patients with hypoxic hypercapnic coma can be successfully ventilated non-invasively with the iron lung. Even in the treatment "failures" satisfactory control of arterial blood gas tensions was achieved, suggesting that factors other than ventilatory failure were responsible.

However, the study was retrospective and uncontrolled making comparison with other techniques difficult, and the limitations of this mode of ventilation should be kept in mind. For example, pulmonary aspiration of material from the pharynx may occur, especially in unconscious patients, due to the lack of protection of the upper airway. The placement of a nasogastric tube can minimise this risk, and we observed clinical and radiographic findings suggestive of aspiration pneumonia in only five patients. Upper airway obstruction due to collapse of the tongue on the posterior pharyngeal wall or to the lack of pre-inspiratory upper airway muscle activation may occur during INPV in unconscious patients.[26] Only 6% of our patients needed endotracheal intubation because they failed to obtain a satisfactory tidal volume with INPV. This suggests that upper airway collapse was uncommon in our patients during ventilation with the iron lung and in part this relates to the insertion of an oropharyngeal airway.

The relatively short hospital stay in the cases treated successfully was another important feature. Patients receiving prolonged mechanical ventilation (for more than seven days) consume a high proportion of intensive care resources.[27 28] The use of neuromuscular blocking drugs to facilitate mechanical ventilation in the intensive care unit is very common. However, some patients who receive a neuromuscular blocking drug continuously for more than two days remain profoundly weak long after the drug has been discontinued and weaning from mechanical ventilation may therefore be delayed and the residual weakness may persist for 3–6 months or longer.[29] Difficulty in weaning results in a long hospital stay and is costly. In one series of patients with COPD who required standard mechanical ventilation

Table 5 Changes in Pa_{O_2}, Pa_{CO_2}, and pH recorded hourly for the two groups of patients during the initial eight hours of the first cycle of ventilation†

*Time**	*Favourable outcome group*				*Treatment failure group*			
	n	Pa_{O_2}	Pa_{CO_2}	*pH*	*n*	Pa_{O_2}	Pa_{CO_2}	*pH*
0	105	6.0 (3.10)	14.7 (2.78)	7.14 (0.10)	45	5.4 (2.74)	15.4 (2.74)	7.11 (0.18)
1	105	9.0 (1.29)	11.8 (2.41)	7.23 (0.08)	45	9.1 (1.19)	12.3 (2.26)	7.20 (0.09)
2	99	9.2 (1.37)	10.7 (2.26)	7.27 (0.08)	44	8.2 (1.64)	10.8 (2.28)	7.26 (0.09)
3	72	9.1 (1.43)	10.3 (2.24)	7.29 (0.08)	36	8.2 (1.65)	10.5 (1.91)	7.28 (0.07)
4	55	8.7 (1.15)	10.1 (1.92)	7.30 (0.07)	31	8.1 (1.71)	9.5 (1.55)	7.31 (0.08)
5	32	8.6 (0.99)	9.7 (1.50)	7.32 (0.07)	24	7.7 (1.57)	9.2 (1.84)	7.33 (0.09)
6	18	9.0 (1.04)	9.8 (1.41)	7.33 (0.04)	17	8.0 (0.77)	8.3 (1.86)	7.36 (0.11)
7	13	9.0 (1.44)	9.7 (1.91)	7.35 (0.06)	12	7.7 (1.52)	8.7 (1.42)	7.34 (0.08)
8	5	9.1 (0.96)	10.0 (2.20)	7.36 (0.05)	5	7.0 (1.48)	9.4 (1.97)	7.32 (0.09)

* Hours from start of mechanical ventilation.
† Values measured during mechanical ventilation.

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for acute respiratory failure 24% were not weaned, while the survivors had a mean duration of intubation of 20 days (range 2–900 days).[22] Only 10% of our patients were not weaned and ventilation lasted, on average, 7.7 days in survivors (with a mean hospital stay of 12.1 days). The relatively short duration of ventilation and hospital stay suggest that a non-invasive ventilatory technique could also be economically advantageous; controlled studies, however, are needed to investigate this important issue.

Major risks associated with intubation and positive pressure ventilation include ventilator-associated pneumonia, pulmonary barotrauma, laryngotracheal complications associated with intubation, and difficulty in weaning.[20] Although all comparisons of a particular patient population with previous historical data are known to be influenced by various biases, the better results obtained in our patients compared with previous series[22 23] in which conventional ventilation was used could be explained by a reduction in the frequency of the aforementioned risks.

In conclusion, our data show that, in this patient population with acute on chronic respiratory failure and hypoxic hypercapnic coma, ventilation with an iron lung had a high rate of success. Non-invasive techniques of assisted ventilation require considerable expertise. The fact that the same medical staff were present over the 10 year period and the high level of nursing experience may have contributed to the good outcome in our patients. This study has the limitations of all retrospective and uncontrolled studies, and our results need to be formally confirmed by prospective controlled studies. In particular, negative pressure ventilation should be compared with non-invasive positive pressure ventilation.

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