Evaluation of Carbon Dioxide Rebreathing During Pressure Support Ventilation With Airway Management System (BiPAP) Devices*

Frédéric Lofaso, MD; Laurent Brochard, MD; Dominique Touchard; Thierry Hang; Alain Harf, MD; and Daniel Isabey, PhD

The purpose of this study was to evaluate whether carbon dioxide (CO₂) rebreathing occurs in acute respiratory failure patients ventilated using the standard airway management system (BiPAP pressure support ventilator; Respironics; Murrysville, Pa) with positive inspiratory airway pressure and a minimal level of positive end-expiratory pressure (PEEP) and whether any CO₂ rebreathing may be efficiently prevented by the addition of a nonrebreathing valve to the BiPAP system circuit. In the first part of the study, the standard device was tested on a lung model with a nonrebreathing valve (BiPAP-NRV) and with the usual Whisper Swivel connector (BiPAP-uc). With the BiPAP-uc device, the resident volume of expired air in the inspiratory circuit at the end of expiration (RVEA) was 55% of the tidal volume (VT) when the inspiratory pressure was 10 cm H₂O and the frequency was at 15 cycles per minute. The BiPAP-NRV device efficiently prevented CO2 rebreathing but resulted in a slight decrease in VT, which was due to a significant increase in external PEEP (2.4 vs 1.3 cm H₂O) caused by the additional expiratory valve resistance. For similar reasons, both the pressure swing necessary to trigger pressure support and the imposed expiratory work were increased in the lung model when the nonrebreathing valve was used. In the second part of the study, seven patients weaned from mechanical ventilation were investigated using a randomized crossover

design to compare three situations: pressure support ventilation with a conventional intensive care ventilator (CIPS), BiPAP system use, and BiPAP-NRV. When we compared the BiPAP system use with the other two systems, we observed no significant effect on blood gases but found significant increases in VT, minute ventilation, and work of breathing. These findings are experimental and are clinical evidence that significant CO₂ rebreathing occurs with the standard BiPAP system. This drawback can be overcome by using a nonrebreathing valve, but only at the expense of greater (CHEST 1995; 108:772-78) expiratory resistance.

BiPAP-uc=BiPAP system using the usual Whisper Swivel connector; BiPAP-NRV=BiPAP system using a nonrebreathing valve; CIPS=conventional intensive care ventilator; CPS=conventional pressure support ventilation; IPS=inspiratory pressure support; N-CPAP=nasal-continuous positive airway pressure; PEEP=positive end-expiratory pressure; Pes=esophageal pressure; RVEA=resident volume of expired air in the inspiratory circuit at the end of expiration; Vr=tidal volume; WOB=work of breathing; WO $\hat{B}_{\text{E-L}}\text{=}\text{expiratory}$ work imposed on the lung model

Key words: bilevel positive airway pressure; CO₂-rebreathing; pressure support; work of breathing

Noninvasive ventilation with a pressure support ventilator is an attractive method for the treatment of acute ventilatory failure and has been used widely, with or without positive end-expiratory pressure (PEEP). Clinicians can choose from a wide array of pressure support devices with fairly marked differ-

*From the Service de Physiologie-Explorations Fonctionelles and Institut National de la Santé et de la Recherche Medical (IN-SERM) U 296 (Drs. Lofaso, Harf, and Isabey and Ms. Touchard); Reanimation Medicale (Dr. Brochard); Hopital Henri Mondor, Creteil, France; and the Association d'Entraide des Polios et Handicapes (Mr. Hang), Puteaux, France.

The experimental part of this study was supported by a grant from the Caisse Nationale d'Assurance Maladie (CNAMTS); the clinical part by the Assistance Publique de Hopitaux de Paris.

Manuscript received October 12, 1994; revision accepted February 2, 1995.

Reprint requests: Dr. Lofaso, INSERM 296; 8 rue du General Sar-rall, 94010 Creteil Cedex, France

ences in design.¹⁻⁷ Relatively simple devices, initially designed for home mechanical support, may be both easy to use and less expensive than sophisticated ventilators. On the other hand, patients admitted for acute ventilatory failure may have different ventilatory requirements from those of patients treated at home, and home ventilators may have technical limitations. In this study, we examined one of these limitations, which may have a major negative impact on efficacy.^{8,9}

The airway management system (BiPAP; Respironics; Murrysville, Pa) is a nasal-continuous positive airway pressure (N-CPAP) equipped with a solenoid valve system to permit separate adjustment of expiratory positive airway pressure and inspiratory positive airway pressure. This device provides both inspiratory pressure support (IPS) and PEEP. It was first used to

Clinical Investigations in Critical Care

Table 1—Characteristics of the Seven Patients Studied

Patients	Sex	Age, yr	Duration of Ventilation, d	Diagnosis at Admission	Outcome
1	M	61	29	Cardiac surgery	Survived
2	M	67	25	Cardiac surgery	Survived, tracheotomy
3	M	52	5	Pneumonia	Survived
4	M	57	17	Hepatic transplantation	Survived
5	\mathbf{F}	41	12	Kyphoscoliosis	Survived
6	M	69	64	Pneumonia	Survived, tracheotomy
7	M	44	18	Polymyositis	Survived, tracheotomy

improve home treatment of sleep apnea syndrome¹⁰ and severe stable COPD. 11-17 Because the patient inspires and expires through a single circuit, there is a risk of CO₂ rebreathing with the standard BiPAP system. The circuit of the standard BiPAP system usually includes a Whisper Swivel connector (BiPAP-uc, Respironics), which permits venting of expiratory gas toward the atmosphere. With this system, the risk of CO₂ rebreathing presumably is low when sufficient levels of PEEP are used and/or when the expiratory time is long enough to ensure that evacuation of the circuit is complete. However, the high levels of ventilatory requirements and elevated breathing frequency characteristic of patients with acute ventilatory failure may promote the occurrence of CO₂ rebreathing. Furthermore, the use of PEEP during noninvasive pressure support ventilation for acute ventilatory failure is limited by the risk of gastric insufflation, or leaks, or both at the mask.7

The purpose of this study was to evaluate whether CO₂ rebreathing occurs in acute respiratory failure patients ventilated using the standard BiPAP system with positive inspiratory airway pressure and a minimal level of PEEP, and whether any CO₂ rebreathing may be efficiently prevented by the addition of a nonrebreathing valve to the BiPAP system circuit (BiPAP-NRV).

In the first part of our study, we used a physical lung model that simulates spontaneous breathing to evaluate the BiPAP system with and without a nonrebreathing valve. In the second part, we investigated critical care patients undergoing weaning from mechanical ventilation. The patients were ventilated with a BiPAP system with or without a nonrebreathing valve, or with a conventional intensive care ventilator (CIPS). The effects of these three systems were evaluated on the basis of arterial blood gas values, work of breathing (WOB) and respiratory pattern. Large differences were found, which may have major consequences on patients' health statuses.

METHODS

This study comprised two parts, an experimental and a clinical study.

Experimental In Vitro Study

Equipment: The comparison of the BiPAP system with and without a nonrebreathing valve was based on the three following parameters: (1) accumulation of expiratory gas in the circuit at the end of expiration, (2) inspiratory work performed by the ventilator, and (3) external PEEP and expiratory work imposed by the device in each situation studied.

We used the BiPAP-uc device model, which does not prevent CO_2 rebreathing, or the BiPAP system equipped with a Sanders NRV-2 Non-Rebreathing Valve (BiPAP-NRV; Respironics; Murrysville, Pa), which is a one-way machine-to-patient valve connected to an expiratory port that opens when the one-way valve is closed by expiration, permitting venting of expiratory gas toward the atmosphere.

All measurements were made at inspiratory pressure levels of $10 \text{ cm H}_2\text{O}$, at the lowest PEEP levels, and at a respiratory frequency of 15 cycles per minute.

Procedure: The lung model was a two-chamber Michigan test lung (Fig 1). One chamber of the test lung was attached to and powered by a CPU ventilator (Ohmeda) while the other chamber (experimental chamber) was attached to the ventilator being tested. The two chambers were physically connected by a small metal component that allowed the driving chamber to lift the experimental chamber, mimicking spontaneous inspiration. The generation of positive pressure in the driving chamber lowered the pressure in the experimental chamber to subatmospheric levels, triggering a pressure-supported breath. Because the metal component was not secured to the experimental chamber, once triggered, it could rise independently from the driving chamber. The compliance of both chambers was set at 82 mL/cm H₂O. The PEEP was applied to the driving chamber at a level sufficient to prevent chamber separation at end-expiration. A resistance of 10 cm H₂O at 1 L/s was used to connect the tested ventilator to the experimental chamber. The CPU ventilator was set to ensure an inspiratory time of 1.15 s and a constant inspiratory flow rate of 30 L/min. A Fleisch No. 2 pneumotachograph was inserted between the experimental chamber and the circuit of the tested ventilator, and airway pressure was measured at the distal end of the circuit, using a differential pressure transducer (Validyne MP, 45±70 cm H₂O; Northridge, Calif). To determine whether accumulation of expiratory gases occurred in the BiPAP system circuit, we inserted a second Fleisch No. 2 pneumotachograph between the BiPAP system and the usual Whisper Swivel connector or the Sanders NRV-2 nonrebreathing valve (Fig 1). The flow signals were integrated to yield the tidal volume (VT) and the resident volume of expired air (RVEA) in the BiPAP circuit at the end of expiration. Signals were digitized at 128 Hz and sampled using an analogic/numeric system (MP100; Biopac Systems, Goleta, Calif). Pressure-volume loops were analyzed as previously described 18 to determine the inspiratory work performed by the ventilator and the expiratory work imposed on the lung model. These two values were calculated from volume-pressure curves. Inspiratory work performed by the ventilator was calculated

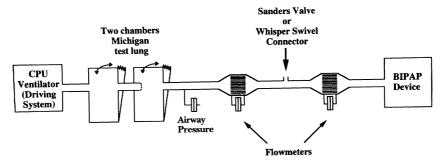


FIGURE 1. Experimental setup. The circuits of tested ventilator devices were connected to a two-chamber Michigan test lung. One chamber (driving chamber) of the test lung was attached to and powered by a ventilator; the other chamber (test chamber) was connected to the ventilator being studied. The driving chamber was able to lift the experimental chamber. Pressure (P) and flow (V) were measured at the extremity of the ventilator circuit. Furthermore, to evaluate possible accumulation of expiratory gas in the BiPAP system circuit, another pneumotachograph (V) was inserted between the BiPAP system and the Whisper Swivel connector or Sanders NRV-2 nonrebreathing valve.

based on the area between the recorded volume-pressure curve during inspiration and the vertical PEEP level line intercepting the volume-pressure curve at the PEEP value; expiratory work imposed by the expiratory circuit was calculated based on the area between the recorded volume-pressure curve during expiration and the same vertical PEEP level line. Inspiratory trigger was evaluated as the difference between the external PEEP level and the peak of airway depression at onset of inspiration. Data from a total of six breaths were obtained. Means and standard deviations were determined.

Clinical Study

Patients: This study was approved by the Research Ethics Committee of our institution. All patients gave informed consent to participate in the study. Seven patients aged 41 to 67 years were included. Their main clinical and respiratory features are listed in Table 1. At the time of the study, all seven patients were intubated or tracheotomized, and none could breathe spontaneously for more than 15 min. Despite differences in the causes of respiratory failure, each patient satisfied the following criteria: (1) respiratory frequency between 15 and 35 breaths per minute without PEEP and with IPS less than 22 cm $\rm H_2O$; (2) $\rm PaO_2$ above 70 mm Hg with an inspired oxygen fraction smaller than 50%, and (3) hemodynamic stability (without vasopressive drugs) and apyrexia.

Procedure: An arterial line was inserted in each patient before the measurements were taken, to allow repeated analysis of arterial blood gas levels. Blood gas values were measured using an ABL 30 analyzer (Radiometer, Copenhagen, Denmark).

Flow was measured using a Fleisch No. 2 pneumotachograph connected to a differential pressure transducer (Validyne, MP). The flow signal was integrated to yield the volume. The respiratory rate and minute ventilation were calculated from the flow signal. Airway pressure was measured at the proximal end of the endotracheal tube, using a differential pressure transducer. Esophageal pressure (Pes) was measured using a balloon catheter (Marquat; Boissy Saint Léger, France), which was attached to a differential pressure transducer (SDX 001; Sensym; Santa Clara, Calif, [range±70 cm

 $\rm H_2O$]). The esophageal balloon was inflated with 1 mL of air, and appropriate placement of the esophageal balloon was checked by means of an occlusion test.

All signals were digitized at 128 Hz and sampled for subsequent analysis using an analogic/numeric system.

Inspiratory work of breathing (WOB) was computed from Pesvolume loops as previously described. 19 The WOB was calculated from a Campbell diagram by computing the area between the recorded Pes-volume curve during inspiration and the static Pes-volume curve of the chest wall. The values for Pes at zero flow instants were taken as the beginning and the end of inspiration. The theoretical value of chest wall compliance, which theoretically represents about 4% of the predicted value of the vital capacity per cm H₂O, was used to trace the static Pes-VT curve of the chest wall. 19 This curve passed through the value for elastic recoil pressure of the chest wall at end expiration, which was assessed by measuring intrinsic PEEP on the esophageal pressure tracing. The beginning of inspiration was thus separated from the elastic recoil pressure by an amount equal to the intrinsic PEEP on the Campbell diagram. After elimination of abnormal cycles caused by coughing or very small Vrs, the mean value for at least ten breaths was determined for each parameter.

Protocol: Measurements with the three systems were performed in the morning of the same day and lasted 3 to 4 hours. All the patients were studied in a semirecumbent position. Endotracheal suctioning was routinely performed before the study periods. Because the BiPAP system does not allow the inspired fraction of oxygen to be set, all patients were ventilated with room air, and an additional flow of oxygen was added to the ventilatory circuit when necessary and kept constant throughout the study.

Each patient received the same level of pressure support with the three systems, which were tested in random order. The three systems were as follows: (1) pressure support with the conventional intensive care ventilator (CIPS) used during the previous few days (8400 ST; Bird; Palm Springs, Calif), 900C Servo (Siemens; Sweden), Evita ventilator (Dräger; Lubeck, Germany), Advent

Table 2—Experimental Performances of BiPAP-uc or With Prevention of Rebreathing by BiPAP-NRV*

	RVEA, mL	Vt, mL	PIP, cm H ₂ O	PEEP, cm H ₂ O	Te,	Trigger, cm H ₂ O	Work I-V, J/L	WOB _{E-L} , J/L
BiPAP-NRV	0	675±5	10.0±0.0	2.4±0.0	2.3±0.1	2.6±0.2	0.56±0.01	0.18±0.00
BiPAP-uc	400±6	731±4	9.4±0.0	1.3±0.0	2.2±0.1	1.4±0.1	0.57±0.03	0.12±0.00

^{*}Values are means ±SDS. PIP=peak inspiratory pressure; Te=expiratory time; Work I-V=work performed by the ventilator during inspiration; WOB_{E-L} = expiratory work imposed on the lung model.

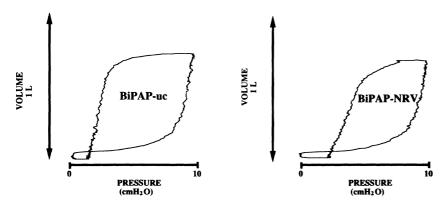


FIGURE 2. Pressure-volume curves in the Bi-PAP-uc and the BiPAP-NRV during a simulated breath produced by the lung model (Fig 1). Note that the PEEP level was higher with the BiPAP-NRV. Consequently, pressure support and VT were slightly lower with the BiPAP-NRV than with the BiPAP-uc.

(Ohmeda; Maurepas, France), or Cesar ventilator (Taema-CFPO; Le Plessis-Robinson, France), which served as the reference; (2) pressure support generated by the BiPAP-uc device; and (3) pressure support generated by the BiPAP-NRV.

With each of the three systems, pressure support ventilation (*ie*, the maximum value of inspiratory pressure) was adjusted to the level used before inclusion of the patient in the study. Patients were ventilated with each pressure support system for 20 min. Respiratory parameters were recorded during the last 5 min of each test period. Arterial samples for blood gas analysis were collected at the end of each period.

Statistical Analysis

Data are given as means \pm SD. Comparisons were done using analysis of variance for repeated measurements. When appropriate (F test with a probability value below 0.05), pairwise comparisons were performed using Fisher least statistical difference tests. The level of significance was set at 5%.

RESULTS

Experimental in Vitro Study

Table 2 shows the results obtained using the lung model, and Figure 2 depicts the pressure-volume loops recorded with the BiPAP-uc and the BiPAP-NRV. With the BiPAP-uc, the RVEA was 400 mL, ie, up to 55% of the Vt. Use of the Sanders NRV-2 nonrebreathing valve fully eliminated rebreathing.

When the usual connector was replaced by a

nonrebreathing valve, there was an increase in PEEP $(2.4~vs~1.3~cm~H_2O)$ as a result of a rise in the time constant required to reach the equilibrium of the relaxed system. Because of this auto-PEEP effect, both the VT and the inspiratory work performed by the ventilator per breath decreased by 6 to 8%, and the pressure swing that triggered the ventilator was wider with the BiPAP-NRV (Table 2 and Figure 2). In addition, the expiratory work imposed on the lung model increased by 50%, reflecting the higher expiratory resistances imposed by the nonrebreathing valve.

Clinical Study

Addition of oxygen (1 to 4 L/min) was necessary in four patients. Ventilatory patterns, arterial blood gas values and WOB observed with each of the three devices are shown in Table 3. Figure 3 shows typical signals observed in a representative patient. There were no differences in breathing patterns, gas exchange, or WOB between CIPS and BiPAP-NRV ventilation. By contrast, with the BiPAP-uc device, minute ventilation increased significantly in comparison with the other two situations, because of an increase in VT with no significant change in PaCO₂ or respiratory rate. A

Table 3—Mean Values (±SD) of Breathing Pattern, Arterial Blood Gas Values and Respiratory

Muscle Work Indexes With the Three Systems Tested: Pressure Support With the Conventional Intensive Care

Ventilators, the BiPAP-uc, or the BiPAP-NRV

	CIPS	BiPAP-uc	BiPAP-NRV	Statistical Analysis*
Vt, mL	467±112	599±114	495±100	1-3
VE, L·min ⁻¹	12.1 ± 3.6	15.7 ± 5.8	11.4 ± 4.9	1-3
Respiratory rate, min ⁻¹	27±6	26 ± 7	23±9	ns
PIP, cm H ₂ O [†]	16.3 ± 5.3	15.8 ± 3.6	16.1 ± 4.9	ns
External PEEP, cm H ₂ O	1.1 ± 1.2	1.0 ± 0.5	1.4 ± 0.5	ns
Ti/Ttot, %	44±7	46 ± 7	49±8	ns
WOB/L, J/L	0.63 ± 0.51	1.04 ± 0.7	0.68 ± 0.44	1-3
WOB/min, J/min	7.7 ± 7.9	14.3±9.9	8.2 ± 6.7	1-3
Arterial pH, ui	7.45 ± 0.06	7.44 ± 0.06	7.45 ± 0.05	NS
PaO ₂ , mm Hg	78±37	127 ± 103	79±35	NS
PaCO ₂ , mm Hg	47.0 ± 9.9	49.3 ± 8.5	46.0 ± 9.5	NS

^{*}Statistical analysis: 1=p<0.05 CIPS vs BiPAP-uc; 2=<0.05 CIPS vs BiPAP-NRV; 3=<0.05 BiPAP-uc vs BiPAP-NRV. NS=not significant.

[†]PIP=peak inspiratory pressure; WOB=work of breathing.

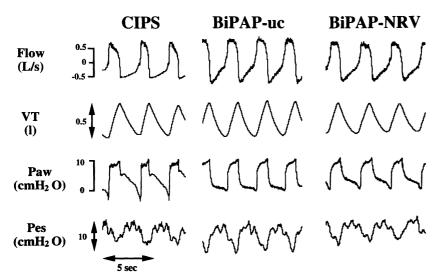


FIGURE 3. Tracing obtained in a representative patient during ventilation with a CIPS, during pressure support with the BiPAP system and the BiPAP-uc, and during pressure support with the BiPAP-NRV. Note that the BiPAP system was associated with wider Pes swings and higher VT values during the inspiratory phase. Paw=airway pressure.

small, nonsignificant rise in PaO_2 was seen. These changes in breathing pattern were accompanied by a significant increase in the WOB expressed per minute or per unit of ventilation.

DISCUSSION

Both our *in vitro* experiment (lung model) and our clinical randomized crossover study demonstrate that use of a standard BiPAP system to deliver inspiratory pressure support to ICU patients can result in a substantial amount of CO2 rebreathing with a marked increase in the WOB. Two simple factors were required to produce these adverse effects: (1) use of a standard tubing system with the widely used Whisper Swivel connector, and (2) a level of expiratory pressure set at the minimal value, ie, less than 2 cm H₂O. Under these conditions, we found that VT and ventilation increased significantly as compared with a conventional intensive care pressure support system, while Pco₂ remained unchanged or increased slightly. Thus, the VT and ventilation increases succeeded in maintaining normal PaCO₂ levels despite the substantial amount of rebreathing. This pattern of breathing resulting in a 1.3fold increase in minute ventilation was associated with a nearly twofold increase in the WOB, to the level (10 I/min), at which the increase in power evaluated using diaphragmatic electromyography suggested excessive loading in a previous study.19

We used an *in vitro* setup to evaluate rebreathing during BiPAP with standard tubing. This rebreathing phenomenon is VT-dependent. Indeed, for an identical leak flow, the volume actually washed out is constant, whereas the expiratory volume remaining in the tubings will increase with the magnitude of VT. In order to obtain physiologic VTs in the *in vitro* condition, we used an inspiratory pressure support level lower than that used in the clinical study to take into account the passive mechanical properties and simulated inspiratory activity. We found a large RVEA at the

end of expiration (55% of expiration (55% of VT). However, this value may overestimate the rebreathing that can be observed in clinical situations, since RVEA does not represent exactly the rebreathing volume (a part of it is washed toward the atmosphere during inspiration) and also because VT remained higher in the *in vitro* than in the *in vivo* study. Indeed, in the clinical study with the standard BiPAP system, considering the 30% increase in ventilation and the slight insignificant increase in PaCO₂, the rebreathing can be evaluated to be no more than 30% of VT.

Pressure support ventilation with BiPAP system has been used successfully in selected patients with respiratory failure.^{2,3} The 5 cm H₂O or greater external PEEP levels used in these studies may have minimized CO₂ rebreathing. We did not evaluate the clinical consequences of CO2 rebreathing or the effect of applying 5 cm H₂O of external PEEP. In a separate study aimed at estimating the time required to expel accumulated CO₂, we found that the leak flow of the Whisper Swivel connector was 200 mL/s when the pressure in the internal circuit was 5 cm H_2O . This suggests that complete venting of expiratory gas requires an expiratory time above 2 s when the external PEEP is 5 cm H₂O and the VT above 400 mL. Only if this requirement is met will there be no CO₂ rebreathing. Although the expiratory time can reach or exceed 2 s in stable chronic respiratory failure, 12 lower values are seen in acute conditions. Expiratory times were under 2 s in our study. Thus, application of 5 cm H_2O of external PEEP may not completely prevent CO₂ accumulation in the circuit when the respiratory frequency is slightly above normal.

Another important consideration when evaluating noninvasive ventilation in acute respiratory failure is the effect of PEEP on the ability of patients to tolerate ventilation. Pennock et al^{2,3} observed 10% of failures due to nonadaptation to inspiratory pressure support with PEEP. However, PEEP may increase the

likelihood of failure by causing excessive leakage around the nasal or facial mask. Fernandez et al. 7 found that two of three patients who had severe acute respiratory failure and who were observed required a high level of IPS and were unable to tolerate PEEP because of excessive leaks. These data are consistent with the hypothesis that use of the lowest possible level of PEEP and prevention of CO_2 rebreathing by means of a nonrebreathing valve may improve the tolerability and efficacy of BiPAP system IPS ventilation in patients with acute respiratory failure.

A twofold increase in the WOB was observed with the standard BiPAP system despite an increase in ventilation of only 30%. Tidal volume generation depends on both inspiratory activity and IPS. ²⁰⁻²² Therefore, the sole increase in ventilation underestimates the increase in inspiratory WOB done by the patient when these values are expressed as percentages.

Interestingly, the observed ventilatory pattern changes and excessive WOB were not associated with the occurrence of respiratory warning signals, such as an increase in respiratory frequency, an important increase in PaCO₂, or a decrease in PaO₂. These results suggest that rebreathing and its clinical consequences may be largely underestimated by intensivists working under everyday conditions.

We demonstrated that adding a nonrebreathing valve to the BiPAP system during pressure support and use of PEEP levels under 2 cm H₂O effectively prevented rebreathing. However, the time constant required to reach the equilibrium of the relaxed system was increased as a result of the added resistance of the nonrebreathing valve. The consequences of this effect were an increase in external PEEP, a smaller level of IPS, and a larger effort of the patient to trigger IPS. In our study patients, these unwanted changes were not so marked as to offset the beneficial effect of ventilation in terms of inspiratory WOB. Thus, the reduction in inspiratory WOB achieved with the BiPAP-NRV did not differ significantly from that seen with the conventional intensive care ventilators. Furthermore, the increase in expiratory WOB seen with the nonrebreathing valve may have facilitated the occurrence of expiratory muscle activity. Although our impression was that our study patients had no clinical expiratory activity, we did not perform an objective assessment of expiratory muscle activity (based for instance on expiratory muscle electromyography or gastric pressure measurements). Further studies are needed to settle this point.

In our clinical study, use of the BiPAP-uc device was associated with a nonsignificant but measurable increase in Po₂. In fact, Po₂ increased only in those patients who received additional oxygen. Although the same oxygen flow was used with the three devices in a given patient, the oxygen fraction in the circuit was

greater with the BiPAP-uc device because oxygen evacuation was less effective when rebreathing was detected. This additional deleterious effect can occur when rebreathing is not prevented and can aggravate acute hypercapnia. ²³

In conclusion, using the BiPAP system with the standard circuit equipped with a Whisper Swivel connector to deliver pressure support without external PEEP, we found experimental and clinical evidence of CO₂ rebreathing. Furthermore, we demonstrated that use of a nonrebreathing valve effectively prevented CO₂ rebreathing and thus reduced the inspiratory WOB to a level similar to that seen with CIPS. We strongly recommend routine use of a three-way valve instead of the usual Whisper Swivel connector. Use of a three-way valve is especially desirable when low levels of PEEP are used. However, further improvements are still required to minimize valve resistance and additional studies are needed to evaluate the impact of the increase in resistance on expiratory activity. Our data confirm that CO₂ rebreathing has adverse consequences in terms of the WOB imposed on the patient.

REFERENCES

- 1 Meduri GU, Abou-Shala N, Fox RC, et al. Noninvasive face mask mechanical ventilation in patients with acute hypercapnic respiratory failure. Chest 1991; 100:445-54
- 2 Pennock BE, Crawshaw L, Kaplan P. Noninvasive nasal mask ventilation for acute respiratory failure. Chest 1994; 105:441-44
- 3 Pennock BE, Kaplan PD, Carlin BW, et al. Pressure support ventilation with a simplified ventilatory support system administered with a nasal mask in patients with respiratory failure. Chest 1991; 100:1371-76
- 4 Vitacca M, Rubini F, Foglio K, et al. Noninvasive modalities of positive pressure ventilation improve the outcome of acute exacerbations in COLD patients. Intens Care Med 1993; 19:450-55
- 5 Wysocki M, Tric L, Wolff MA, et al. Non invasive pressure support ventilation in patients with acute respiratory failure. Chest 1993; 103:907-13
- 6 Brochard L, Isabey D, Piquet J, et al. Reversal of acute exacerbation of chronic obstructive lung disease by inspiratory assistance with a face mask. N Engl J Med 1990; 323:1523-30
- 7 Fernandez R, Blanch L, Valles J, et al. Pressure support ventilation via face mask in acute respiratory failure in hypercapnic COPD patients. Intens Care Med 1993; 19:456-61
- 8 Hill NS. Noninvasive ventilation: does it work, for whom, and how? Am Rev Respir Dis 1993; 147:1050-55
- 9 Brochard L. Non-invasive ventilation: practical issues. Intens Care Med 1993; 19:431-32
- 10 Sanders MH, Kern N. Obstructive sleep apnea treated by independently adjusted inspiratory and expiratory positive airway pressure via nasal mask. Chest 1990; 98:317-24
- 11 Renston JP, DiMarco AF, Supinski GS. Respiratory muscle rest using nasal BiPAP ventilation in patients with stable severe COPD. Chest 1994; 105:1053-60
- 12 Nava S, Ambrosino N, Rubini F, et al. Effect of nasal pressure support ventilation and external PEEP on diaphragmatic activity in patients with severe stable COPD. Chest 1993; 103:145-50
- 13 Lien TC, Wang JH, Chang MT, et al. Comparison of BiPAP nasal ventilation and ventilation via iron lung in severe stable COPD. Chest 1993; 104:460-66

- 14 Strumpf DA, Carlisle CC, Millman RP, et al. An evaluation of the Respironics BiPAP Bi-Level CPAP device for delivery of assisted ventilation. Respiratory Care 1990; 35:415-22
- 15 Strumpf DA, Millman RP, Carlisle CC, et al. Nocturnal positive pressure ventilation via nasal mask in patients with severe chronic obstructive pulmonary disease. Am Rev Respir Dis 1991; 144: 1234-39
- 16 Carrey Z, Gottfried SB, Levy RD. Ventilatory muscle support in respiratory failure with nasal positive pressure ventilation. Chest 1990; 97:150-58
- 17 Ambrosino M, Nava S, Bertone P, et al. Physiologic evaluation of pressure support ventilation by nasal mask in patients with stable COPD. Chest 1992; 101:385-95
- 18 Kacmarek RM. The role of pressure support ventilation in reducing work of breathing. Respiratory Care 1988; 33:99-120

- 19 Brochard L, Harf A, Lorino H, et al. Inspiratory pressure support prevents diaphragmatic fatigue during weaning from mechanical ventilation. Am Rev Respir Dis 1989; 139:513-21
- 20 Fenner A, Jansson EH, Avery ME. Enhancement of the ventilatory response to carbon dioxide by tube breathing. Respiration Physiology 1968; 4:91-100
- 21 MacIntyre NR, Leatherman NE. Ventilatory muscle load and the frequency-tidal volume pattern during inspiratory pressureassisted (pressure-supported) ventilation. Am Rev Respir Dis 1990; 141:327-31
- 22 Scheid P, Lofaso F, Isabey D, et al. Respiratory response to inhaled $\rm CO_2$ during positive inspiratory pressure in humans. J Appl Physiol 1994; 77:876-82
- 23 Prime FJ, Westlake EK. The respiratory response to ${\rm CO_2}$ in emphysema. Clin Sci 1954; 13:321-32