Efficacy of noninvasive CPAP in COPD with acute respiratory failure

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Efficacy of noninvasive CPAP in COPD with acute respiratory failure. P. Goldberg, H. Reissmann, F. Maltais, M. Ranieri, S.B. Gottfried. ©ERS Journals Ltd 1995.
ABSTRACT: Dynamic hyperinflation and the development of intrinsic positive end-expiratory pressure (PEEPi) are commonly observed in patients with severe chronic obstructive pulmonary disease (COPD) in acute respiratory failure. Previous studies have shown that externally applied PEEP reduces PEEPi and its adverse effects in mechanically-ventilated COPD patients. The purpose of this study was to determine the effects of graded amounts of continuous positive airway pressure (CPAP) on the degree of inspiratory effort, pattern of breathing, gas exchange, and level of dyspnoea in a group of spontaneously breathing, nonintubated COPD patients in acute hypercapnic respiratory failure.

Ten COPD patients admitted to the intensive care unit in acute hypercapnic respiratory failure were studied. Inspiratory effort was measured by the tidal excursions of oesophageal (Poes) and transdiaphragmatic (Pdi) pressure. Inspiratory effort and both the pressure-time product for the diaphragm ($\int P$di·dt) and for the inspiratory muscles ($\int P$oes·dt) were measured during the application of 5, 7.5, and 10 cmH$_2$O of CPAP. Dyspnoea, gas exchange and pattern of breathing were also assessed.

Inspiratory effort and the pressure-time product both for the diaphragm and the inspiratory muscles fell significantly with CPAP in a dose-dependent fashion. Both the pattern of breathing and level of dyspnoea improved with CPAP. End-expiratory lung volume remained stable at the lower levels of CPAP, with only modest increases at the higher levels. Arterial oxygen tension (Pa,O$_2$) and arterial carbon dioxide tension (Pa,CO$_2$) either improved or remained stable with CPAP.

We conclude that the noninvasive application of CPAP to spontaneously breathing patients with severe COPD in acute respiratory failure decreases inspiratory effort and dyspnoea whilst improving breathing pattern. It is conceivable that the early institution of CPAP in this setting may obviate the need for intubation and conventional mechanical ventilation.
Eur Respir J., 1995, 8, 1894–1900.

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Classically, the increase in the work of breathing accompanying an acute exacerbation of chronic obstructive pulmonary disease (COPD) has been attributed primarily to an increased resistive load. Accordingly, therapeutic approaches have focused on its pharmacological reduction. Recently, however, it has become apparent that individuals with COPD, both when stable and especially during an acute decompensation, exhibit dynamic hyperinflation which has profound consequences on the energetics of breathing. Indeed, dynamic hyperinflation, which is reflected by the intrinsic positive end-expiratory pressure (PEEPi), imposes an added inspiratory threshold load whilst decreasing the effectiveness of the inspiratory muscles as pressure generators. Several investigators [1, 2] have shown that positive end-expiratory pressure applied externally can counterbalance PEEPi without causing further hyperinflation.

We undertook this study, therefore, to determine whether the application of continuous positive airway pressure (CPAP) to spontaneously breathing patients in hypercapnic respiratory failure, due to an acute exacerbation of COPD, could decrease inspiratory effort and dyspnoea and improve breathing pattern.

Methods

Ten severely dyspnoeic patients admitted to the intensive care unit (ICU) of the Montreal Chest Hospital Centre in worsening hypercapnic respiratory failure were studied. Of these patients, six were studied during their first day of admission to the ICU. One patient (No. 8) was studied several days after admission, following extubation and subsequent worsening respiratory status, as documented by his arterial blood gases (table 1). The other three patients were initially managed pharmacologically by their treating physicians. When, in the opinion of those physicians, therapy had proved inadequate, these

Table 1. – Patient characteristics

Pt No.	Age yrs	Sex	FEV1/FVC L	Pa,O_2 kPa	Pa,O_2 mmHg	Pa,CO_2 kPa	Pa,CO_2 mmHg	pH	FI,O_2
1	58	M	0.45/1.65	6.3	47	9.9	74	7.15	0.21
2	61	F	0.52/1.49	7.2	54	10.1	76	7.26	0.40
3	62	M	0.44/1.67	11.6	87	10.8	81	7.32	0.28
4	28	M	1.16/3.10	11.6	87	6.4	48	7.38	0.35
5	77	M	0.58/1.86	8.8	66	10.4	78	7.39	0.30
6	70	F	0.70/1.36	10.1	76	7.9	59	7.29	0.50
7	61	F	1.16/2.37	8.3	62	9.1	68	7.25	0.28
8	65	M	1.08/2.38	6.8	51	10.9	82	7.35	0.35
9	64	M	0.50/1.95	11.3	85	7.3	55	7.40	0.28
10	68	M	0.38/1.20	8.4	63	7.2	54	7.44	0.30

FEV1/FVC were obtained when stable prior to the present hospitalization. Pt: patient; M: male; F: female; FEV1: forced expiratory volume in one second; FVC: forced vital capacity; Pa,O_2: arterial oxygen tension; Pa,CO_2: arterial carbon dioxide tension; FI,O_2: inspiratory oxygen fraction.

patients were entered into the study. Their arterial blood gas values listed in table 1 represent the period just prior to enrolment in the study. The diagnosis of chronic air-flow obstruction was confirmed by history and physical examination, as well as by previous pulmonary function tests. In all but one patient who suffered from cystic fibrosis (No. 4) this condition was due to tobacco abuse. The precipitating cause of the acute decompensation was acute bronchitis in all patients. Pertinent clinical information is provided in table 1. None of the patients had evidence of myocardial ischaemia or failure, haemodynamically compromising dysrhythmias, or altered mental status. Medical management was continued as prescribed by the primary physicians. The investigative protocol was approved by the Institutional Ethics Committee, and written informed consent was obtained from the patient or family in all cases.

All patients, none of whom was intubated at the time of this study, were studied whilst breathing spontaneously in the semirecumbent position. They breathed either through a mouthpiece with a noseclip in place (eight patients) or a face-mask with inflatable rim (two patients), the choice being based on goodness of fit, absence of leaks, and patient comfort. The mouthpiece or mask was connected to a continuous high flow system (approximately 100 L·min⁻¹) (Downs Flow Generator No. 9250, Vital Signs Inc., Totowa, NJ, USA). The inspired oxygen fraction (FI,O_2) was initially chosen to achieve an arterial oxygen tension (Pa,O_2) of 8.0 kPa (>60 mmHg) and remained constant throughout the remainder of the study.

Flow (V') was measured with a heated Fleisch No. 2 pneumotachograph (three patients) or with a variable orifice pneumotachograph (Cat. No. 279331, Hamilton Medical, Inc., Reno, NV, USA) (seven patients) connected to a differential pressure transducer (MP-45, ±2 cmH₂O, Validyne Corp., Northridge, CA, USA). Pressure at the airway opening (Pao) was recorded proximal to the pneumotachograph using a differential pressure transducer (Validyne MP-45, ±100 cmH₂O). Oesophageal pressure ($Poes$) was measured using balloon-tipped catheter systems. Three patients were studied with a 10 cm long balloon integrated into a gastric tube [3] (Cat. No. 85843, National Catheter Co., Argyle, NY, USA), the

others with a latex balloon of the same length mounted on a polyethylene catheter (length 100 cm; internal diameter 1.7 mm) [4]. In five patients [5], gastric pressure (Pga) was also measured using a second balloon-catheter system. The balloons were filled with 1 mL of air and connected to separate differential pressure transducers (Validyne MP-45, ±100 cmH₂O). The oesophageal balloon was properly positioned using the "occlusion test" as described previously [4]. Where applicable, transdiaphragmatic pressure (Pdi) was determined by subtracting $Poes$ from Pga. A DC-coupled respiratory inductance plethysmograph (RIP) (Respitrace, Ambulatory Monitoring, Ardsley, NY, USA) was used to monitor end-expiratory lung volume. The bands were placed around the rib cage at the level of the nipples and around the abdomen at the level of the umbilicus. Care was taken to avoid the overlap of the two bands.

All of the above variables were recorded on an eight channel strip-chart recorder (7718A, Hewlett-Packard Fullerton, CA, USA) together with an additional volume trace obtained by electrical integration of the flow signal (Hewlett-Packard 8815A). The signals were also simultaneously filtered, digitized (DT 2801A, Data Translation, Marlboro, MA, USA) and stored on a portable computer (SuperSport 286, Zenith Data Systems, St. Joseph, MI, USA) for later analysis.

Procedure and analysis

After a period of accommodation to the experimental apparatus, the patients breathed spontaneously for approximately 10 min at atmospheric pressure (Control). CPAP of 5 cmH₂O was then applied to the circuit by attaching a spring-loaded threshold valve (Vital Signs, East Rutherford, NJ, USA) to the respiratory circuit for approximately 10 min. This was followed by another 10 min control period of breathing at atmospheric pressure. Similar control and CPAP trials were repeated with CPAP of 7.5 cmH₂O (eight patients) and 10 cmH₂O (three patients), the lesser number of patients at the higher levels due to patient discomfort and/or the presence of a leak around the mask or mouthpiece. At the end of each control and CPAP period, arterial blood
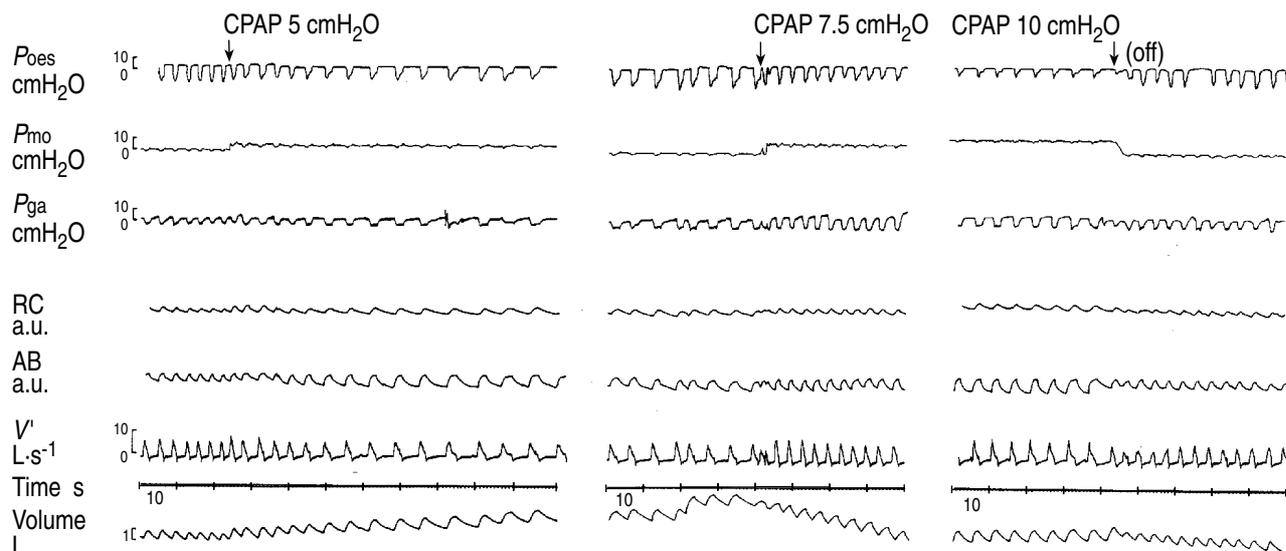
Fig. 1. – Effects of continuous positive airway pressure (CPAP) in a representative patient. From top to bottom, tracings represent oesophageal pressure (P_{oes}), mouth pressure (P_{mo}), and gastric pressure (P_{ga}), volume displacement of the rib cage (RC) and abdomen (AB), and flow (V') and tidal volume. a.u: arbitrary unit. See text for interpretation.

samples were drawn for determination of P_{a,O_2}, arterial carbon dioxide tension (P_{a,CO_2}), and pH. At the same time, patients were asked to indicate any change in their level of dyspnoea between control and CPAP conditions using a bidirectional ordinal scale (0=no change; -1/+1= very slight worsening/improvement; -2/+2=slight worsening/improvement; -3/+3=moderate worsening/improvement; -4/+4 marked worsening/improvement; -5/+5=very marked worsening/improvement) [5]. Heart rate, blood pressure, and oxygen saturation were monitored throughout the study.

Approximately 10–15 consecutive breaths were analysed for each experimental condition once a stable breathing pattern was observed. Tidal volume (V_T), digitally integrated from the flow signal, and respiratory rate were both determined. The duration of inspiration (t_I), expiration (t_E), and total breathing cycle (t_{tot}) were also calculated. As an indication of inspiratory effort, tidal excursions of P_{oes} and P_{di} were determined. The area under the pressure-time relationship, *i.e.* the pressure-time product for both the inspiratory muscles ($\int P_{oes} \cdot dt$) and diaphragm ($\int P_{di} \cdot dt$) were determined and expressed per minute and per litre of minute ventilation [6].

Results are expressed as mean value±standard error. As measurements made in the control periods both preceding and following the application of CPAP were no different their results were averaged

All comparisons between CPAP and the corresponding averaged control period were made using Student's t-test for paired data. Statistical significance was defined as a p-value less than 0.05.

Results

A representative experimental record of one patient at baseline and at CPAP of 5, 7.5 and 10 cmH$_2$O is presented in figure 1. As can be appreciated, each successive level of CPAP is associated with a progressive decrease in P_{oes} swings in an apparent dose-dependent fashion, whilst tidal volume appears to remain constant or increases slightly. Importantly, end-expiratory lung volume appears to remain constant at all levels of applied CPAP, as suggested by the absence of any appreciable shift in the end-expiratory positions of the rib cage, abdominal, and P_{oes} signals.

Table 2. – Ventilatory parameters before and during CPAP

	V'_E L·min^{-1}		V_T mL		R_f breaths·min^{-1}		t_I/t_{tot} %		V_T/t_I mL·s^{-1}		V_T/t_E mL·s^{-1}	
Control	8.35	(0.69)	373	(32)	23.4	(2.3)	30.7	(1.39)	448	(34)	195	(17)
CPAP 5 cmH$_2$O (n=10)	8.85	(0.94)	443	(36)**	21.1	(2.7)**	29.6	(1.16)	500	(53)	211	(23)
Control	9.00	(1.01)	424	(39)	22.3	(3.3)	30.2	(1.43)	494	(49)	217	(27)
CPAP 7.5 cmH$_2$O (n=8)	9.67	(0.75)	463	(30)	21.8	(2.7)	31.2	(1.06)	520	(46)	234	(18)
Control	7.13	(0.97)	396	(81)	20.7	(7.3)	28.5	(1.98)	412	(50)	165	(25)
CPAP 10 cmH$_2$O (n=3)	8.05	(2.24)	474	(99)*	18.9	(8.7)*	29.1	(4.55)	456	(96)	193	(59)

Values are presented as mean, and SEM in parenthesis. CPAP: continuous positive airway pressure; V'_E: minute ventilation; V_T: tidal volume; R_f: respiratory frequency; t_I: inspiratory time; t_E: expiratory time; t_I/t_{tot}: duty cycle; V_T/t_I: mean inspiratory flow rate; V_T/t_E: mean expiratory flow rate. *: p<0.05; **: p<0.01. CPAP *vs* control. Control is the average of before and after. Numbers in brackets refers to the number of subjects.

The ventilatory parameters at the different levels of applied CPAP in all subjects are outlined in table 2. CPAP improved the pattern of breathing at each level of applied pressure, in that respiratory rate tended to fall whilst tidal volume rose, maintaining minute ventilation constant. At 5 and at 10 cmH$_2$O, the changes in both respiratory rate and tidal volume were highly significant, while at 7.5 cmH$_2$O, the increase in tidal volume just failed to reach significance (p=0.059). CPAP produced little or no change in the other ventilatory parameters measured.

Inspiratory effort, as evidenced by the tidal excursions of both oesophageal and transdiaphragmatic pressure, both on and off CPAP, are represented graphically in figure 2. The inspiratory exclusions of Poes and Pdi fell at all levels of CPAP, in what appears to be a dose-dependent fashion. Although reduced by approximately 40%, the change in Pdi at 7.5 cmH$_2$O failed to reach statistical significance, probably because of the small number of subjects.

The mean pressure developed by the inspiratory muscles during the breathing cycle has been described as reflecting the energy cost of breathing more closely than the actual measurements of the external work performed [7, 8]. Poes·dt and ∫Pdi·dt, calculated per minute and per litre of minute ventilation, are presented graphically in figures 3 and 4, respectively. ∫Poes·dt fell significantly in a dose-dependent manner at all levels of applied CPAP,

whilst ∫Pdi·dt fell significantly at CPAP 5 cmH$_2$O. Despite a decrease of approximately 45% in Pdi·dt at CPAP 7.5 cmH$_2$O, both when calculated per minute and per litre,
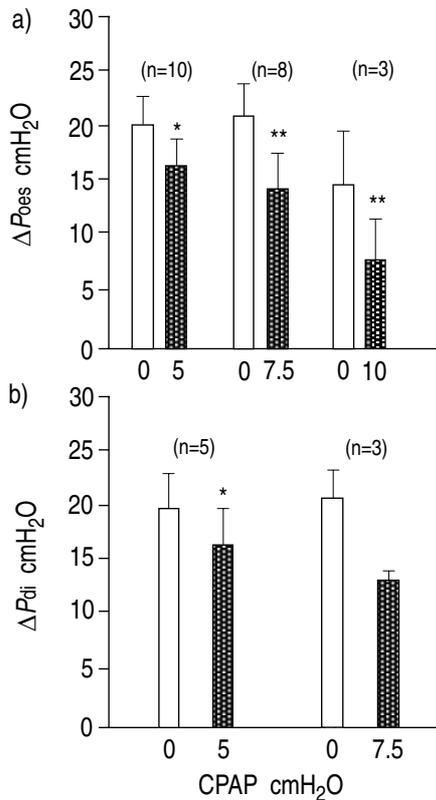
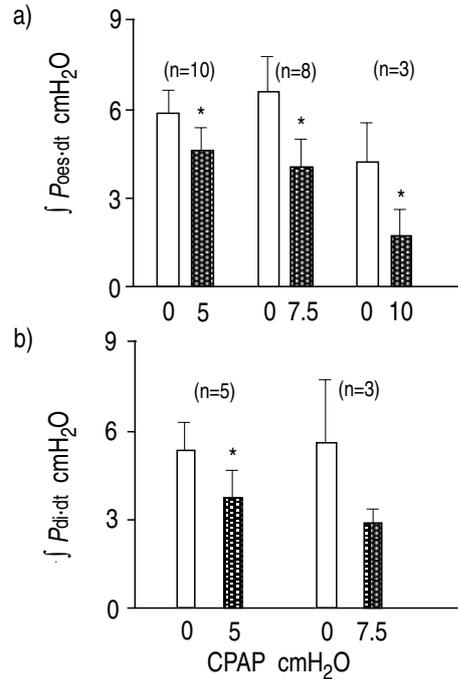

Fig. 3. – Average values (±SEM) for the pressure-time product calculated for a period of 1 min of the a) inspiratory muscles (∫Poes) before (□) and during (▦) the application of continuous positive airway pressure (CPAP) 5, 7.5 and 10 cmH$_2$O and of b) the diaphragm (∫Pdi·dt) before and during the application of CPAP of 5 and 7.5 cmH$_2$O. *: p<0.05 before *vs* during CPAP.

Fig. 2. – Average values (±SEM) for the tidal swing in a) oesophageal pressure (ΔPoes) before (□) and during (▦) the application of continuous positive airway pressure (CPAP) 5, 7.5 and 10 cmH$_2$O and in b) transdiaphragmatic pressure (ΔPdi) before and during the application of CPAP 5 and 7.5 cmH$_2$O. *: p<0.05; **: p<0.01 before *vs* during CPAP.
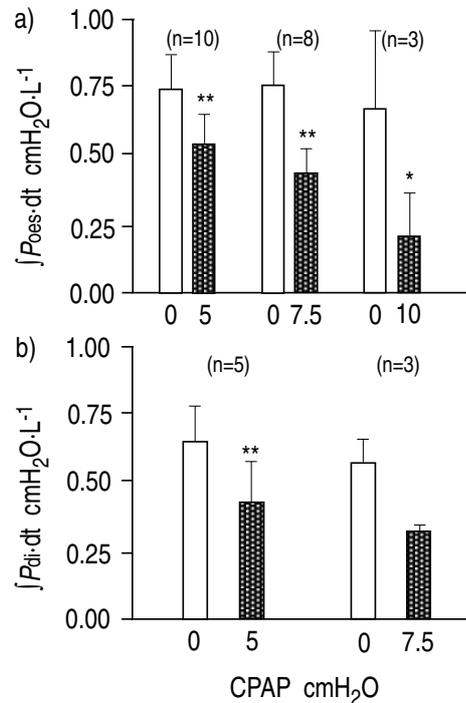

Fig. 4. – Average values (±SEM) for the pressure-time product calculated per litre of minute ventilation of the a) inspiratory muscles (∫Poes·dt) before (□) and during (▦) continuous positive airway pressure (CPAP) 5, 7.5 and 10 cmH$_2$O and b) of the diaphragm (∫Pdi·dt) before and during the application of CPAP 5 and 7.5 cmH$_2$O. *: p<0.05; **: p<0.01 before *vs* during CPAP.

Table 3. – Arterial blood gases before and during CPAP

	P_{a,O_2}		P_{a,CO_2}		pH	
	kPa	mmHg	kPa	mmHg		
Control	11.1 (1.0)	83 (7.8)	8.7 (0.7)	65 (5.2)	7.38 (0.03)	
CPAP 5 cmH$_2$O (n=9)	12.4 (0.8)	93 (6.3)	8.3 (0.6)	62 (4.2)	7.40 (0.02)	
Control	11.5 (1.4)	86 (10.7)	8.1 (0.8)	61 (5.9)	7.39 (0.03)	
CPAP 7.5 cmH$_2$O (n=7)	13.2 (1.5)*	99 (11.2)*	7.9 (0.7)	59 (4.9)	7.41 (0.03)*	
Control	8.3 (1.2)	62 (8.7)	9.3 (1.4)	70 (10.2)	7.33 (0.06)	
CPAP 10 cmH$_2$O (n=3)	9.6 (0.8)	72 (5.7)	8.5 (1.3)	64 (9.8)	7.35 (0.07)	

Values are presented as mean and SEM in parenthesis. *: p<0.05, CPAP vs control; control is average of before and after CPAP (paired t-test). Numbers in brackets refer to the number of subjects. For abbreviations see legends to tables 1 and 2.

statistical significance was not achieved, probably due to the small number of patients studied under these conditions.

The effect of CPAP on arterial blood gases is shown in table 3. P_{a,O_2} tended to improve at all levels of CPAP, whilst P_{a,CO_2} tended to remain constant or fall slightly. Similarly, arterial pH remained stable at all levels of applied CPAP.

When asked to evaluate their sense of breathlessness, all patients reported either an improvement or no change with CPAP; the scores ranged from 0 to +5. No individual described a worsening sense of dyspnoea while on CPAP. In reference to the corresponding control period (CPAP=0), the mean dyspnoea value for CPAP 5 cmH$_2$O was +1.4 (p <0.05) improving to +2.5 (p <0.05) and +2.7 (p <0.05) for CPAP 7.5 and 10 cmH$_2$O respectively.

Discussion

The major findings of this study are that the non-invasive application of CPAP to spontaneously breathing patients in hypercapnic respiratory failure suffering an acute exacerbation of COPD results in a significant reduction in inspiratory effort and dyspnoea whilst improving the pattern of breathing. Although similar findings have been reported previously in the intubated patient recovering from an episode of respiratory failure; to our knowledge, this has not yet been described in the acutely ill nonintubated COPD patient.

Because of increased flow resistance which results in an abnormally long respiratory time constant, individuals with COPD require a prolonged expiratory phase. Often, because of an inadequate time available for expiration, these individuals fail to reach the resting volume of the respiratory system at end-expiration, i.e. at end-expiration, lung volume is often greater than the relaxed functional residual capacity. This implies that before initiating flow, a certain amount of inspiratory pressure must be developed in order to overcome the positive elastic recoil remaining. Such a threshold load or intrinsic PEEP (PEEPi) has been found to be present in the majority of severe COPD patients, both when stable [7] and in acute respiratory failure [5, 9].

In their study of 14 COPD patients in acute respiratory failure, MURCIANO et al. [9] measured an average end-expiratory tracheal occlusion pressure, i.e. static PEEPi of 8.5±3.5 cmH$_2$O, whilst PETROF et al. [5] documented an average static PEEPi of 9.8 cmH$_2$O in a cohort of COPD patients recovering from acute respiratory failure and weaning from mechanical ventilation. Although static PEEPi could not be reliably measured in these nonintubated spontaneously breathing patients, there is little reason to expect, given the severe degree of airway obstruction present in the setting of an acute exacerbation and the evidence in the study recordings of significant levels of expiratory flow at the onset of inspiratory effort, that levels of PEEPi substantially different from those reported previously were present in this study. We therefore undertook to determine whether the application of CPAP in its ability to counterbalance the PEEPi present [1, 2] on the one hand and unload the inspiratory muscles on the other [10] would reduce inspiratory effort and dyspnoea and improve the pattern of breathing in spontaneously breathing patients with severe obstructive pulmonary disease suffering an acute decompensation.

In addition to the reduction in inspiratory effort the majority of our patients improved subjectively on CPAP, albeit to different degrees. No patient reported a worsening of dyspnoea. These findings may well be due, as suggested by KILLIAN [11], to a decrease in inspiratory effort as demonstrated in the present study. Additionally, it has been suggested that dyspnoea may be heightened by dynamic airway collapse [12]. To the extent that positive pressure applied downstream from the site of flow limitation may decrease dynamic airway compression, this may have further decreased the sense of dyspnoea.

This improvement both in inspiratory effort and breathlessness is very similar to our previous findings reported by PETROF et al. [5], who examined the impact of CPAP on COPD patients during weaning from mechanical ventilation. Although both groups of patients were spontaneously breathing, those studied by PETROF et al. [5] had been intubated for variable periods of time and were no longer acutely ill. This is in contrast to the present study in which all subjects were acutely ill and breathing spontaneously through a native upper airway.

Arterial blood gases tended to remain the same or improve slightly. The trend toward a reduction in P_{a,CO_2} was probably due to the improvement in breathing pattern [13] given that minute ventilation (V'_E) remained constant. Although we cannot exclude a decrease in CO_2 production accruing to the fall in inspiratory effort, any decrease, however modest, would be of benefit.

One major concern relating to the use of extrinsic PEEP in this setting is its effect on lung volume, in that any further hyperinflation would place the inspiratory muscles at greater mechanical disadvantage. Although the ability of RIP to reliably quantify changes in end-expiratory lung volume may be limited [14], the failure of CPAP to alter the end-expiratory positions both of ribcage and abdomen in this present study would suggest that end-expiratory lung volume was not appreciably increased. In addition, end-expiratory P_{oes} fell by a mean of 0.1 cmH_2O at CPAP 5 cmH_2O whilst it increased by a mean of 1.26 cmH_2O and 1.0 cmH_2O at CPAP 7.5 and 10 cmH_2O, respectively. Assuming a normal chest wall compliance of 200 $mL \cdot cmH_2O^{-1}$, this would amount to an average increase in lung volume of approximately 200 mL. This is very similar to those values reported by PETROF et al. [5].

The failure to substantially increase lung volume is consistent with previous investigations in COPD patients [1, 2, 15, 16], in which, in the presence of expiratory flow limitation, end-expiratory lung volume remained constant until the amount of applied PEEP approached the level of PEEPi present. For example, PETROF et al. [5] demonstrated that lung volume remained constant at CPAP of 5 cmH_2O in the absence of expiratory muscle recruitment, whilst at higher levels the degree of hyperinflation was less than would have been predicted by the passive inflation of the respiratory system. Indeed, KIMBALL et al. [17] had shown much the same phenomenon, in that the isovolume pressure-flow curve of a severely obstructed patient was independent of the pressure placed at the airway opening.

Although we believe that the absence of further hyperinflation in our subjects was due to the presence of expiratory flow limitation in the tidal volume range, other possible explanations remain to be considered. Others have reported that in both normals [18, 19] and asthmatics [6] any increase in end-expiratory lung volume accruing to the application of positive-pressure breathing appears to be actively resisted through the recruitment of abdominal expiratory muscles. However, as evidenced by the expiratory gastric pressure tracings, none of the subjects in the present study augmented his/her expiratory muscle recruitment at 5 cmH_2O, whilst the majority failed to do so even at 7.5 cmH_2O. That is, although these patients actively recruited expiratory muscles at baseline, as has been reported previously [20, 21], they failed to increase that recruitment to any appreciable extent even at the higher levels of CPAP.

It should be noted, in this regard, that methodological differences may account for the failure of the patients in this present study to recruit expiratory muscles. Both the patients in the present study and those of PETROF et al. were studied in semirecumbent position, in which

expiratory muscle recruitment has been found to occur far less frequently as compared to the seated position in which both MARTIN et al. [6] and BISHOP et al. [19] studied their subjects.

Alternatively, this lack of increase in lung volume could be due to CPAP-induced changes in expiratory braking mechanisms, such as glottic narrowing and persistent inspiratory muscle activity during expiration. Whilst we cannot comment on the effect of CPAP on glottic narrowing, inspiratory muscle activity during expiration has been shown not to be increased in individuals with COPD [22] in contrast to subjects with asthma [6].

These findings are in marked contrast to those reported by TUXEN [23] and are most likely due to a fundamental difference in patient population. The latter study documented a progressive increase in lung volume and intrathoracic pressure as increasing levels of extrinsic PEEP were applied to six mechanically-ventilated patients with severe airflow obstruction and hypercapnic respiratory failure. Although detailed patient characteristics are not provided, those patients were not flow-limited during tidal breathing. This may have been due to a lesser degree of airway obstruction or to the presence of asthma, a condition in which flow limitation has generally not been observed [15]. The absence of expiratory flow limitation in severe asthma may be due to the lack of reduced lung recoil and increased airway collapsibility that characterize COPD [16].

It is becoming increasingly clear that the therapeutic approach to the severely ill COPD patient with an acute exacerbation should not be limited to conventional pharmacological treatment. In this regard, BROCHARD et al. [24] have recently reported their experience in treating such patients with noninvasive inspiratory assistance by means of a face-mask. They found that, in comparison with a historical control group, the noninvasively ventilated subjects required intubation and conventional mechanical ventilation significantly less often. In addition, the period of time requiring ventilatory assistance and the number of ICU days were significantly shortened. We cannot, at this time, comment on whether CPAP or noninvasive mechanical ventilation is superior or whether certain clinical conditions favour the use of one versus the other. It is likely, however, that both forms of ventilatory support can complement one another in providing optimal reduction in inspiratory effort.

In summary, we have found that the application of CPAP to spontaneously breathing patients suffering an acute exacerbation of COPD decreases inspiratory effort and dyspnoea while improving the pattern of breathing. Whilst it was not the purpose of this study to evaluate whether or not the application of CPAP in this setting could obviate the need for mechanical ventilation, such potential clearly exists and warrants further study.

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