EFFECT OF NASAL-CPAP ON PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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ABSTRACT

Patients with chronic obstructive pulmonary disease [COPD] breath at large lung volumes because of dynamic hyperinflation. Their end-tidal lung volumes will then be much above the equilibrium position of the respiratory system and the elastic recoil pressure would be above zero at end-tidal exhalation. This auto or intrinsic positive end-expiratory pressure [auto-PEEP] contributes to the elastic work of inspiration and the sensation of dyspnoea. The purpose of this study was to offset the auto-PEEP in patients with exacerbated chronic airflow obstruction by applying continuous positive airway pressure via the nose [nasal-CPAP].

Nine out of 14 patients experienced alleviation of dyspnoea while on nasal-CPAP [4 to 8 cmH_2O]. These 9 patients had significantly more severe hyperinflation than the 5 patients who did not respond positively to nasal-CPAP. While there is a complex relationship between intrinsic and extrinsically applied PEEP in patients with COPD, the result of this study is consistent with the notion that CPAP may alleviate dyspnoea by reducing auto-PEEP, improving lung mechanics and unloading the inspiratory muscles. Nasal-CPAP may have a potential therapeutic role in exacerbations of COPD.

Keywords: Hyperinflation, dyspnoea, positive end expiratory pressure, chronic obstructive pulmonary disease.

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INTRODUCTION

Patients with severe chronic obstructive pulmonary disease [COPD] tend to breath at high lung volumes. Their lung volumes at the end of expiration during quiet tidal breathing [FRC - functional residual capacity] may be much above the equilibrium position of the respiratory system [RS] as a result of dynamic hyperinflation. In the normal subject, at FRC, the lung and chestwall static recoil pressures act in equal and opposite directions and the net RS recoil pressures is zero. The RS is then in its mechanically neutral position [Fig 1]. Patients with severe COPD and hyperinflation have greatly increased FRC. The elastic recoil pressure of the RS will then be much above zero at FRC. This finite positive recoil force exerted by the RS at FRC as a result of dynamic hyperinflation was described in patients with severe COPD during mechanical ventilatory support by two groups of investigators in 1982 [1,2]. It is called the auto-positive end expiratory pressure ["auto-PEEP"] or intrinsic-PEEP. This "auto-PEEP" is the threshold load which must be overcome by the inspiratory muscles before any airflow occurs during each tidal inspiration. This phenomenon is enhanced during exacerbation of disease because of further air trapping and contributes to the sensation of dyspnoea and the increased elastic

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Fig 1

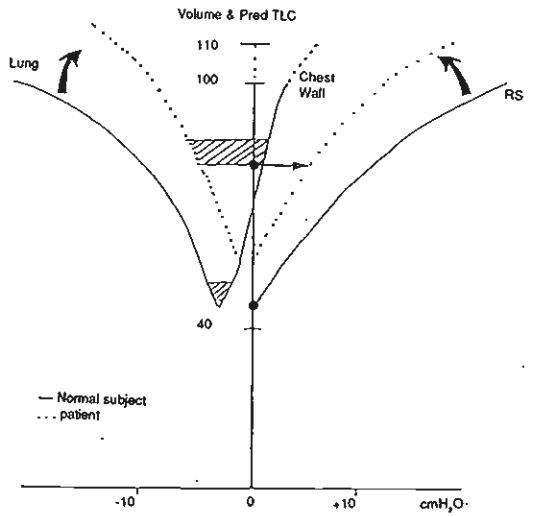

Fig 1. This schematic diagram illustrates the effect of hyperinflation on the static recoil properties of the respiratory system[RS]. The RS recoil pressure = [chestwall recoil - lung recoil]. The static pressure [horizontal axis] - volume [vertical axis] curves of the lung, chestwall and RS of a normal subject are represented by solid lines. The patient with COPD [dotted line] has reduced lung recoil, normal chestwall recoil and breathes at a higher volume [upward shift of both "lung" and "RS" recoil curves - curved solid arrows]. In the normal subject at FRC [lower •], the lung and chestwall recoil forces are equal and opposite thus the RS recoil is zero. By contrast, the patient with COPD has a much larger FRC [higher •] where the RS recoil is no longer zero but has a finite positive value [arrow]. This positive RS recoil at FRC is the "auto-PEEP". The expenditure of inspiratory elastic work [shaded area] is also increased. Further discussion in the text, references 23 and 24.

work of breathing [1-3].

The auto-PEEP may be offset by either applying positive pressure ["push"] to the airways during breathing in the form of CPAP or alternatively by applying a negative pressure ["suck"] around the chestwall. Both these techniques have been used safely in small numbers of patients with COPD[4-7]. Recent studies on the effect of either airway CPAP or negative chestwall pressures in severe COPD have shown reduction of ventilatory muscle activity and improve muscle performance [6,7]. Smith and Marini reported that PEEP applied during mechanical ventilation may reduce airway resistance and the mechanical work of breathing in patients with severe COPD [8]. This reduced work load on the diaphragm may account for the observation by O'Donnell et al that CPAP may increase the endurance and reduce the dyspnoea index during exercise in patients with COPD[9].

Most investigators have applied CPAP through the mouth while the natural route of inspiratory airflow during tidal breathing is the nose. Moreover the experience of applying CPAP via the nose has been safe and effective in patients with obstructive sleep apnoea [10,11]. The nasal route may be a more convenient route especially for acutely dyspnoeic patients. The purpose of this study was therefore to examine the effect of continuous positive airway pressure through the nose [nasal CPAP] on the perception of dyspnoea in patients with severe COPD during exacerbation of disease.

PATIENTS AND METHODS

We studied 14 in-patients, aged mean[SD] 66[8] yrs. There were 3 women and 11 men. They had severe COPD with mean(SD) FEV_1 of 31(7)% predicted and total lung capacity [TLC] of 110(20)% predicted. Flow-volume curve measurements during tidal breathing and maximal forced expiration show that all 14 patients were flow limited during tidal expiration [12]. These measurements were recorded on the Gould 2000 dry spirometer during clinically stable periods. All subjects had been heavy current or ex-cigarette smokers. They were studied during recovery from acute exacerbation of symptoms. Patients with complications such as pneumonia, pneumothorax and other concomitant illnesses were excluded.

Nasal-CPAP was applied to the patient while in the sitting position, during tidal breathing of room air via a fitting nose mask [Respironics Inc.] and t-piece. Airway pressure was monitored by a tap on the mask and regulated with a PEEP valve [11].

The breathing pattern was monitored *qualitatively* using inductance plethysmography [Respitrace] during application of CPAP. The sum of ribcage and abdominal wall signal amplitudes reflect that of the tidal volume while baseline changes follow changes in FRC [13]. Since no formal calibration against a known volume was carried out the assessment of any change in FRC must be subjective. However the Respitrace recordings were displayed in real time on the oscilloscope screen and hard copies recorded on paper.

The CPAP was stepped up by 2-3 cmH_2O increments until just over 10 cm H_20. Patients indicated using hand signals if they felt that their breathing was 'lighter', 'heavier' or 'the same/uncertain' compared to breathing without the mask, at each level of CPAP which was maintained for about 15 min. They were also asked if the difference was perceived during inspiration or expiration.

No more precise assessment of dyspnoea indices was possible because of the range of ethnic and dialect groups among the patients.

RESULTS

The sensation of dyspnoea was reduced by low to moderate levels of nasal-CPAP [4 to 8 cm H_20] in 9 out of 14 patients. All 9 patients reported that they felt 'lighter' during inspiration and 'the same' during expiration when breathing nasal-CPAP. In the remaining 5 patients nasal-CPAP either did not have any perceptable effect on dyspnoea or it increased the sensation of respiratory effort [during inspiration, expiration or both.]

These 9 patients had similar FEV_1 but significantly greater TLC [$p<0.05$] than the 5 patients who did not perceive reduction of dyspnoea on CPAP [Fig 2 & Table 1]. This suggests that while the 9 patients who felt less dyspnoeic on nasal-CPAP had the same degree of airflow obstruction [measured by FEV_1] as those 5 patients who did not respond positively to CPAP, they were significantly more hyperinflated [and therefore probably had a greater degree of auto-PEEP].

Fig 2

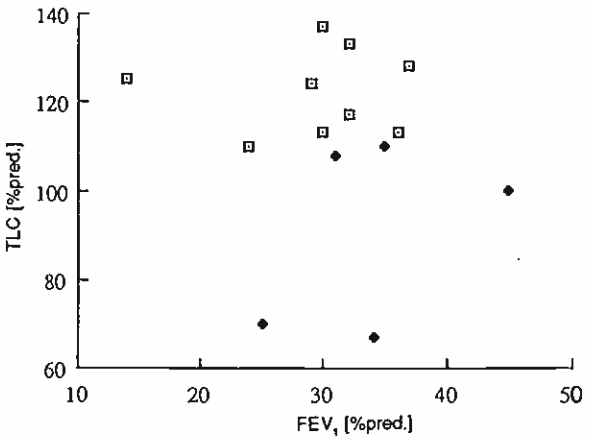

Fig 2. This figure plots the relationship between TLC [% predicted] and FEV_1 [% predicted] of the 14 patients studied. The 9 patients [open symbols] who experienced alleviation of dyspnoea on nasal-CPAP [4-6 cm H_20] had significantly higher TLC [$p < 0.05$] but similar FEV_1 when compared with the 5 patients [solid symbols] who did not.

Fig 3 shows the summed signals of the ribcage and abdominal wall motion as measured on the Respitrace in two patients with and without CPAP of 6 cm H_20. Note that the FRC position [dotted line] was not affected by CPAP breathing in both subjects. This was true of all 9 patients who felt better with CPAP.

All 14 subjects felt that breathing was 'heavier' in both inspiration and expiration when the CPAP was increased to above 10 cm H_20. This was associated with increased "FRC", larger tidal volumes and expiratory times as monitored on the Respitrace.

Fig 3

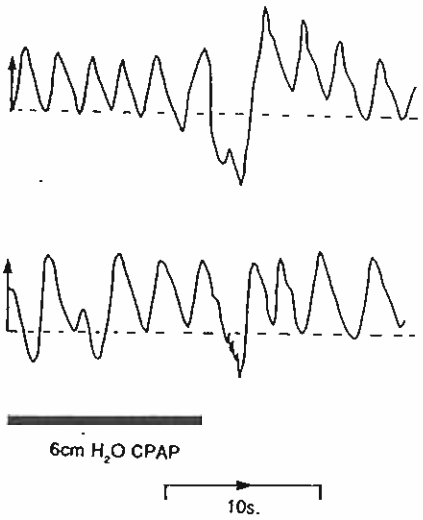

Fig 3. These are Respitrace recordings of the summed ribcage and abdominal signals in two representative patients. They are qualitatively similar to tidal volume measurements. The vertical arrows indicate tidal inspiration while the horizontal bar indicate the period during which nasal-CPAP of 6 cm H_20 was applied. Note that there were no large changes in the position at end tidal expiration ["FRC" - dotted line] when nasal-CPAP was discontinued.

Table I
Pulmonary Function Results

	Responders [n = 9]	Non-responders [n = 4]	P
FEV_1[% pred.]	36 (6)	29 (7)	0.113
TLC[% pred.]	122 (10)	82 (20)	<0.05

Values expressed as mean (SD), p values from un-paired t-tests.

It should be noted that since the Respitrace was not calibrated against a known lung volume, all observations of "FRC" level and breathing pattern must necessarily be subjective.

DISCUSSION

While the traditional approach to the treatment of patients with COPD had been directed primarily at reducing the severity of airway obstruction, more recently, research on respiratory failure in COPD had focused on the failure and fatigue of the muscles of the respiratory "pump" [14,15]. This new emphasis on the *skeletal* muscles of the *chestwall* and *diaphragm* during *inspiration* is in direct contrast to the traditional concern with the *smooth* muscles of the *airways* during *expiration*[16].

The perception of dyspnoea is highly subjective and it is difficult to precisely account for all the observations in this study without direct measurements of auto-PEEP, breathing patterns, the mechanical behaviour of the inspiratory muscles and lung. Nevertheless, the reduction of dyspnoea sensation in patients with severe COPD by nasal-CPAP may be anticipated from a reduction of the threshold pressure load to tidal inspiration. The observation that only the more severely hyperinflated patients benefitted from nasal-CPAP is also consistent with the notion that the severity of auto-PEEP may be related to the degree of hyperinflation [3].

Other important factors which may affect the dyspnoea sensation in patients with COPD during application of nasal-CPAP such as the breathing pattern, increased airflow in the nares, blood gas changes and reduced expiratory airflow will be addressed. Firstly, a change in the level, depth and timing of respiration could have affected the sensation of breathlessness. The relatively stable Respitrace recordings in Fig 3 suggest that nasal-CPAP of 6 cm H_20 did not have a major impact on breathing patterns. When the CPAP pressures exceeded 10 cm H_20 however, there was a large increase in both tidal volume and FRC accompanied by aggravation of dyspnoea. Since no calibration of the Respitrace was undertaken the changes or stability noted in "FRC" were subjective assessments only and should be interpreted as such. My purpose in monitoring the breathing level was primarily as a safety measure to ensure that the externally applied CPAP did not result in further airtrapping.

This does not however negate the observation that only the more hyperinflated subjects reported less dyspnoea with CPAP [Fig 2 & Table I]. The sensation of dyspnoea may be alleviated by increasing the rate of airflow at the nares without any effect on the mechanical properties of the RS [17]. This local effect may be obviated by the use of local lignocaine. While the local effect of increased nasal airflow on dyspnoea perception was not examined in this study, only the more hyperinflated patients felt less breathless while all subjects experienced increased nasal airflow during the application of nasal-CPAP. This suggest that the rate of nasal airflow was not a major determinant of dyspnoea perception in this study. The arterial blood composition was not monitored in this study. Therefore the observed variable effect of nasal-CPAP could be accounted for by different changes in blood gas tensions. The effect of nasal-CPAP however, was perceived on the very first breath after it was applied and it did not show any further enhancement with time. This immediate effect of an intervention on the first breath suggests that changes in blood gas tensions - which would require about 20 to 30 minutes to take effect in the presence of the impaired gas exchange associated with COPD - had little influence on dyspnoea sensation. This "first breath" effect of an intervention on the sensation of dyspnoea would indeed be more consistent with improvement in mechanical factors rather than changes in arterial blood tensions.

Finally, I will discuss the complex effects of positive pressure breathing on expiratory flow and load sensation. If the transpulmonary pressures during tidal expiration do not exceed the critical pressure needed to produce maximal expiratory flows, then the application of positive airway pressures would result in fall in expiratory flow rates and increased expiratory load sensation. This increase back pressure to expiratory flow and air trapping may result in the risk of barotrauma in normal subjects and some patients with stable COPD at rest [18]. By contrast, O'Donnell et al found that the application of 5 cm H_20 CPAP to patients with COPD during exercise resulted in the alleviation of dyspnoea and did not reduce volume matched expiratory flows [9]. Furthermore, Smith

and Marini showed beneficial effects of PEEP on respiratory mechanics at pressures of up to 10 cm H_20 in patients with severe COPD during mechanical ventilation[8]. The effect of positive pressure breathing on expiratory flow was minimised in this study by selecting subjects with apparent flow limitation during tidal breathing [discussed under METHODS] and by studying them during acute exacerbation of symptoms which would have resulted in further hyperinflation and generation of greater transpulmonary pressures. Moreover, the reduced effort sensation was felt during inspiration in the 9 patients who had a favourable response to 4 to 8 cm H_20 of nasal-CPAP. If the backpressure effect of CPAP had been significant at 4 to 8 cm H_20 they would have experienced an increase load during expiration instead. It is possible however that an increased sensation of expiratory load might have been an important factor among some of the 5 patients in this study who did not respond positively to nasal-CPAP of 4 to 8 cm H_20. The unfavourable effect of back pressure on expiratory flow probably accounted for the increase in FRC and dyspnoea experienced by all subjects in this study when the CPAP exceeded 10 cm H_20. These considerations remind us that CPAP is an experimental therapeutic modality which should be evaluated under carefully controlled laboratory conditions in well selected patients.

This study showed that CPAP of 4 to 8 cm H_20 may be safely, beneficially and conveniently applied via the nose to selected, symptomatic patients with severe COPD. The observation that nasal-CPAP of 4 to 8 cm H_20 reduced the sensation of dyspnoea in patients with severe COPD is similar to that observed using mouth-CPAP, inspiratory pressure support and negative pressure ventilation [5,7,8,19]. While there is a complex and incompletely defined relationship between intrinsic or "auto-PEEP" and externally applied positive pressures to the airway, nevertheless, these observations are consistent with the report by Smith and Marini that externally applied PEEP may favourably affect both lung and respiratory muscle mechanics in some patients with severe COPD [8].

Nasal-CPAP may be a potential therapeutic modality for the dyspnoea caused by severe airflow obstruction and hyperinflation [20, 21]. A recent study has suggested that nasal-CPAP may be used to rest the inspiratory muscles during sleep in patients with COPD [22]. Future studies will further define the role of this new modality in the therapy of COPD.

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