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Continuous positive airway pressure facilitates spontaneous breathing in weaning chronic obstructive pulmonary disease patients by improving breathing pattern and gas exchange

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Abstract *Objective*: To elucidate the effects of continuous positive airway pressure (CPAP) on breathing pattern, gas exchange and the ability to sustain spontaneous breathing (SB) in chronic obstructive pulmonary disease (COPD) patients with dynamic hyperinflation.
Design: Prospective study with two randomised trials of SB without and with CPAP in each patient.
Setting: Medical intensive care units (ICUs) in two university hospitals.
Patients: Nine dynamically hyperinflated, intubated COPD patients recuperating from acute exacerbation.
Interventions: One SB trial with CPAP (5–7.5 cmH_2O), one without (control) in each patient .
Measurements: airway opening pressure, gas flow and thus breathing pattern, oxygen uptake, carbon dioxide excretion, arterial blood gases, dyspnoea and respiratory drive (P_{100}).
Results: With CPAP, intrinsic positive end-expiratory pressure ($PEEP_i$) fell from 11.4 to 6.3 cmH_2O ($p < 0.05$). Eight patients sustained SB with CPAP for the maximum time planned (30 min), one failed after 18 min. In contrast, only four patients successfully completed the control trial, the others failing after 5–18 min ($p < 0.05$). Dyspnoea – gauged on a visual analogue scale by five patients – was less severe or occurred later with CPAP. Breathing with CPAP tended to be slower (18.9 vs 22.2 min^{-1}, $p < 0.05$) and deeper (tidal volume 370 vs 323 ml). At the end of the control run, $PaCO_2$ was higher (60 vs 55 mmHg, $p < 0.05$) and still rising while being stable at the end of the CPAP trial.
Conclusion: CPAP helps severely ill COPD patients sustain SB. Apparently it does so by promoting slower, deeper breathing and thus facilitating carbon dioxide elimination.

Key words Chronic obstructive pulmonary disease · Dynamic hyperinflation · Spontaneous breathing · Continuous positive airway pressure · Weaning · Intrinsic positive end-expiratory pressure ($PEEP_i$)

Introduction

Discontinuation of mechanical ventilatory support represents one of the most relevant issues in the management of patients recovering from acute respiratory failure (ARF) due to an exacerbation of chronic obstructive pulmonary disease (COPD) [1]. In COPD, the respiratory system is primarily hampered by the additional elastic load associated with dynamic hyperinflation and the ensuing intrinsic positive end-expiratory pressure ($PEEP_i$) [2, 3]. Additional adverse factors include ventilation-perfusion mismatch [4] and breathing pattern abnormalities [5].

Externally applied PEEP, or continuous positive airway pressure (CPAP), can diminish the impedance imposed by $PEEP_i$. and the workload caused by it [6]. However, the potential decrease of arterial blood pressure [7] and increase in alveolar dead space due to a potential further increase in lung volume with CPAP [8] has never been ruled out in patients with COPD.

The present study was designed to determine the effect of CPAP in COPD patients unable to be weaned from mechanical ventilation despite meeting conventional criteria predictive of weaning success in a general population [9]. We hypothesised that application of CPAP would increase tolerance of spontaneous breathing (SB) without adversely affecting haemodynamics or pulmonary gas exchange.

Methods

Patients

Nine patients with severe COPD, admitted for management of ARF to the intensive care unit (ICU) of the Montreal Chest Hospital Centre or the Royal Victoria Hospital were studied. The diagnosis of COPD was made on the basis of history and physical examination and confirmed by review of pulmonary function tests in stable condition prior to or following the acute event. The primary precipitating cause of ARF was upper or lower respiratory tract infection.

Patients were entered into the study only if they exhibited a $PEEP_i$ of 5 cmH_2O or more during a brief preliminary SB trial [3]. They had to be clinically stable and their primary physician had to decide that a weaning trial was clinically indicated. At least three of the following four criteria associated with weaning success in a general population had been met during preceding weaning attempts by each patient: respiratory rate 30 bpm or less, tidal volume 250 ml or more, maximum inspiratory pressure more negative than –20 cmH_2O, and PaO_2/FIO_2 200 mmHg or more [1, 9]. They had been on mechanical ventilation via a cuffed endotracheal or tracheostomy tube for 9 ± 2.5 days (mean ± SEM). All had failed one or more weaning attempts before. Patients with severe hypertension or hypotension, myocardial ischaemia, haemodynamically significant cardiac arrhythmias, gross respiratory system pathology (e.g. pneumonectomy, persistent pneumothorax, recent pulmonary embolism) or altered mental status were excluded. The investigative protocol was approved by the respective institutional ethics committees and written informed consent was obtained in all cases. Medical management was otherwise continued as prescribed by the primary physician.

Protocol

The subjects were on mechanical ventilation (MV) for at least 12 h prior to the study. In semi-recumbent position they underwent two consecutive SB trials, each being preceded by a 30-min period of MV in the assist/control mode with parameters set so that they would only occasionally trigger a breath. CPAP (5 cmH_2O in one patient, 7.5 cmH_2O in the other eight) was applied during one of the trials ("CPAP" trial) and set to zero in the other one ("control" trial). The order of trials was randomised; in four patients the control trial was first, in five the CPAP trial. The investigators took care that patients and treating physicians were blinded to the use of CPAP by concealing dials and displays from them and avoiding verbal clues. Trials were terminated and patients were returned to MV after a maximum of 30 min or as soon as any one of the following predetermined failure criteria were met: Respiratory rate above 35 bpm or increase of more than 15 bpm; heart rate more than 150 bpm or increase of more than 50 bpm; systolic blood pressure below 100 mmHg or above 200 mmHg or change greater than 40 mmHg; arterial oxyhaemoglobin saturation (by pulse oximetry, SPO_2) less than 90 %; marked respiratory distress; diaphoresis; agitation or altered mental status.

One ventilator (Servo 900 C, Siemens-Elema, Solna, Sweden) was used throughout the study across all modes of ventilation, including spontaneous breathing with and without CPAP, facilitating continuous data acquisition and collection of undiluted expiratory gas. Additional work imposed on the patients by the demand flow circuit did not need to be of concern with this model of ventilator [10], a notion supported by the small fluctuations of airway pressure seen during SB. Short lengths of low-compliance plastic tubing were chosen for the patient circuit, the humidifier was omitted. The FIO_2 was kept constant at or up to 10 % above the level known to provide adequate oxygenation before the study (mean 38 %, range 30–40 %).

Arterial blood pressure, electrocardiogram, and SPO_2 were monitored throughout the study period.

Data collection and analysis

Analogue signals for airway pressure (P_{aw}) and flow ($\dot{V}$) from the ventilator were filtered, digitised at a rate of 100 Hz (DT2801 A; Data Translation, Marlborough, MA), and recorded by a notebook PC. Pressure and flow transducers of the ventilator were calibrated prior to each study as recommended by the manufacturer. Tidal volume (V_T), respiratory frequency (f_r), inspiratory time (T_I), minute ventilation ($\dot{V}_E$), duty cycle (T_I/T_{TOT}), expiratory time (T_E), and mean inspiratory flow (V_T/T_I), were determined from the digitised flow signal. Analyses were performed on 10 to 15 consecutive breaths from specified times during the trials (see Results). The airway was periodically occluded at end expiration for measurement of airway occlusion pressure (P_{100}) and quasistatic $PEEP_i$ [3, 6, 11, 12].

Inspiratory and mixed expiratory gas was sampled for determination of oxygen uptake ($\dot{V}_{O_2}$), carbon dioxide excretion ($\dot{V}_{CO_2}$), and respiratory exchange ratio (R) by an open circuit indirect calorimeter (Deltatrac; Datex, Helsinki, Finland), which was calibrated as recommended by the manufacturer prior to each study. During the final minutes of the MV and SB periods, samples of arterial blood were obtained for determination of PaO_2, $PaCO_2$, and pH.

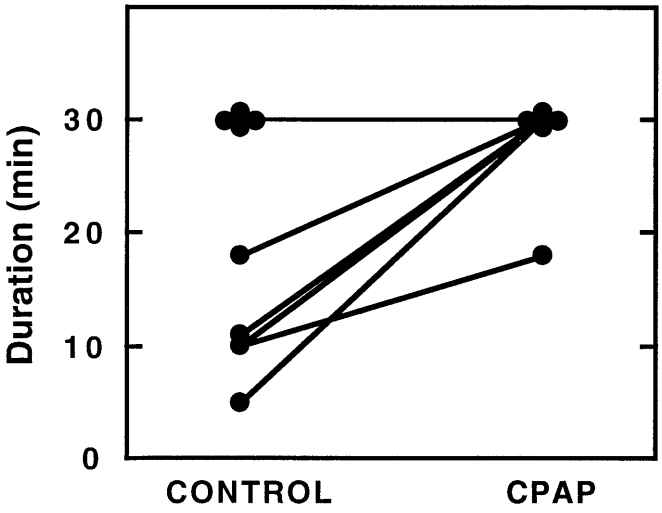

Fig. 1 Duration of spontaneous breathing during weaning trials

Alveolar ventilation ($\dot{V}_A$) was calculated from ($\dot{V}_{CO_2}$) and $PaCO_2$ according to the following equations:

$$\dot{V}_A = \dot{V}_{CO_2} / F_{ACO_2}$$

$$F_{ACO_2} = P_{ACO_2} / P_{barometric}\, P_{H_2O/37°C},$$

and

$$P_{ACO_2} \approx P_{aCO_2}$$

with appropriate conversion from STPD to ATPS [13]. Absolute ($\dot{V}_D$) and fractional deadspace ventilation (V_D/V_T) were derived.

Five patients were able to estimate dyspnoea on a visual analogue scale (VAS) several times during the SB trials. The scale had no divisions and patients were instructed that the end points should denote "no shortness of breath at all" and "maximum shortness of breath imaginable", respectively [14]. Patients were kept unaware of previous responses by having them mark a fresh copy of the scale each time.

Results are reported as means ± SEM. Differences in success between conditions were tested by a McNemar test for paired samples, the differences in time sustained by a Wilcoxon test. The results of the other parameters were subjected to an ANOVA with time (MV and one or more time points during SB trials), condition (CPAP vs control, i.e. absence of CPAP), and patients as independent factors. Post hoc testing was, if appropriate, performed by paired *t*-tests of (1) CPAP versus control at equivalent points of time, with correction for multiple tests according to Bonferroni, and (2) points of time during SB versus the respective preceding MV period, with correction according to Dunett [15]. Time points during SB were not compared with each other. Differences were considered significant if p was smaller than 0.05.

Results

Four patients sustained SB with CPAP for the full 30-min period but failed the control trial after 5–18 min; the failed control trial was first for two of them and second for the other two. Four patients successfully completed both trials, one failed both after 10 (control) and 18 (CPAP) min. The difference between conditions was significant both with respect to patient number and median time of SB sustained (control 18 min, CPAP 30 min). Figure 1 shows the amount of time for which each patient tolerated SB. Weaning failure was characterised by objective and subjective signs of marked respiratory distress in all affected patients and generally accompanied by diaphoresis and/or agitation. Significant hypertension and oxygen desaturation were present in one patient.

Tables 1, 2, 3, 4and 5 and Fig. 2 show results for various time points during the SB trials and for the MV periods preceding SB. Since values for the two MV periods did not differ significantly, single values for MV are presented for reasons of clarity; they are group means of individual averages from the two MV values of the respective parameters. – The data presented for "CPAP – isotime to end of control" were taken from the points of time during the individual CPAP trials corresponding to the end times of the respective control trials.

Table 2 summarises breathing pattern data. With CPAP, tidal volumes were generally larger and respira-

Table 1 Patient characteristics

	Mean	SEM	Minimum	Maximum
Age (years)	69	4	47	86
Gender	3 f, 6 m			
FEV_1 (l)	0.63	0.08	0.35	1.10
FEV_1/FVC (%)	36.7	2.5	24	49
Days on ventilator	8.6	2.1	2	23
I. D. of ET tube (mm)			7.5	8.5
FIO_2 (%)	32	2	21	40
PaO_2 (mmHg)	83	6	68	129
$PaCO_2$ (mmHg)	53	4	34	68
pH	7.43	0.02	7.37	7.50
C_{Rs} (ml/cmH_2O)	58	8	23	98
R_{Rs} (cmH_2O/l/s)	15.6	1.4	9.2	24
$PEEP_i$ (cmH_2O)	9.10	0.80	6	14
MIP (cmH_2O)	–28	2	–40	–20

Table 2 Breathing pattern with mechanical ventilation and during the spontaneous breathing trials (*MV* ± mandatory ventilation preceding spontaneous breathing trials)

	MV	Control minute 5	CPAP minute 5	Control end	CPAP isotime to end of control	CPAP end
V_T (ml)	505 ± 19	309 ± 19[d]	357 ± 19[d]	323 ± 16[d]	363 ± 17[a,d]	370 ± 16[d]
RR (bpm)	16.0 ± 1.7	21.6 ± 1.8	18.7 ± 1.7	22.2 ± 1.6[c]	19.0 ± 1.3[a]	18.9 ± 1.3[a]
$\dot{V}_E$ (l/min)	7.96 ± 0.81	6.58 ± 0.59	6.54 ± 0.60	7.06 ± 0.47	6.85 ± 0.49	6.93 ± 0.48
T_I (s)	1.10 ± 0.11	0.93 ± 0.07	1.06 ± 0.09	0.91 ± 0.06	1.04 ± 0.08	1.04 ± 0.08
T_i/T_{TOT}	27 ± 2	33 ± 3	32 ± 2	33 ± 2	32 ± 2	31 ± 2
TE (s)	2.98 ± 0.27	2.01 ± 0.25[c]	2.51 ± 0.46	1.94 ± 0.24[c]	2.24 ± 0.20	2.28 ± 0.22
V_T/T_i (ml/s)	497 ± 55	349 ± 34	361 ± 40	371 ± 32	372 ± 40	378 ± 38

Table shows means ± SEM
[a] $p < 0.05$ vs control; [b] $p < 0.01$ vs control; [c] $p < 0.05$ vs MV; [d] $p < 0.01$ vs MV

Table 3 Arterial blood gases with mechanical ventilation and during the spontaneous breathing trials (*MV* ± mandatory ventilation preceding spontaneous breathing trials)

	MV	Control end	CPAP end
PaO_2 (mmHg)	98 ± 7	89 ± 6	99 ± 5
$PaCO_2$ (mmHg)	50 ± 2	60 ± 2[d]	55 ± 2[a,d]
pH	7.44 ± 0.02	7.37 ± 0.02[d]	7.40 ± 0.02[b,d]
$AADO_2$ (mmHg)	120 ± 10	109 ± 6	112 ± 7

Table shows means ± SEM
[a] $p < 0.05$ vs control; [b] $p < 0.01$ vs control; [c] $p < 0.05$ vs MV; [d] $p < 0.01$ vs MV

Table 4 Alveolar and physiological deadspace ventilation with mechanical ventilation and during the spontaneous breathing trials (*MV* ± mandatory ventilation preceding spontaneous breathing trials)

	MV	Control end	CPAP end
VA (ml)	208 ± 14	120 ± 15[d]	151 ± 11[a,d]
$\dot{V}_A$ (l/min)	3.20 ± 0.28	2.55 ± 0.25	2.80 ± 0.23
V_D (ml)	297 ± 22	204 ± 11[d]	219 ± 18[d]
$\dot{V}_D$ (l/min)	4.76 ± 0.73	4.51 ± 0.38	4.13 ± 0.45
V_D/V_T	0.58 ± 0.03	0.64 ± 0.03	0.59 ± 0.04

Table shows means ± SEM
[a] $p < 0.05$ vs control; [b] $p < 0.01$ vs control; [c] $p < 0.05$ vs MV; [d] $p < 0.01$ vs MV

tory rates lower, while minute ventilation and T_I/T_{TOT} showed no significant differences. Throughout the CPAP trial, P_{100} tended to be lower than during the control run. Periodically measured values were lumped for the beginning and end of each trial, i.e. the first and last 10 min if the trial lasted longer than 20 min, or the first and second half if it was shorter; all values in the respective periods were averaged without prior selection. P_{100} at the beginning of the control and CPAP trials amounted to 4.4 ± 0.8 cmH_2O and 2.8 ± 0.6 cmH_2O, respectively ($p = 0.051$), those at the end of the trials to 5.0 ± 0.6 for control and 3.8 ± 0.6 cmH_2O with CPAP ($p < 0.04$).

In three patients $PEEP_i$ could not be reliably determined because the time between inspiratory efforts was too short for a clear pressure plateau during end-expiratory occlusions. In the other six, the end expiratory plateau pressure, i.e. total PEEP ($PEEP_{tot}$), was 11.6 ± 2.9 cmH_2O during the control trial and 13.9 ± 1.2 cmH_2O during the CPAP trial (n.s.). Accordingly, $PEEP_i$, the difference between externally applied CPAP and $PEEP_{tot}$, was significantly higher during the control trial (11.4 ± 2.8 cmH_2O) than with CPAP (6.3 ± 1.4 cmH_2O).

The results of the arterial blood gas analyses are summarised in Table 3. PaO_2 tended to be higher with CPAP compared to the control trial, $PaCO_2$ was significantly lower and pH higher.

Figure 2 shows the data on oxygen uptake and carbon dioxide excretion. After initiation of SB both ($\dot{V}_{O_2}$) and ($\dot{V}_{CO_2}$) decreased temporarily. The decrease in ($\dot{V}_{CO_2}$) was more pronounced, so R decreased, as well. Throughout the CPAP trial both ($\dot{V}_{O_2}$) and ($\dot{V}_{CO_2}$) tended to be lower than under control conditions. With CPAP, R returned to baseline significantly faster.

Table 4 shows alveolar and physiological deadspace ventilation at the end of the SB trials and of the MV period. Alveolar ventilation per tidal breath was significantly larger (26 %) during CPAP compared to the control trial. Deadspace ventilation per tidal breath was not different between conditions. Owing to the slower respiratory frequency with CPAP, the difference in ($\dot{V}_A$) was not significant.

Table 5 contains the haemodynamic data. With institution of SB an immediate and persistent increase in both BP and HR occurred. The HR was significantly faster in the control trial than in the CPAP run. BP was higher than during MV only during the control trial.

Figure 3 depicts the dyspnoea ratings of the five patients who were able to provide their estimates during

Table 5 Haemodynamic parameters with mechanical ventilation and during the spontaneous breathing trials (*MV* ± mandatory ventilation preceding spontaneous breathing trials)

	MV	Control minute 5	CPAP minute 5	Control end	CPAP isotime to end of Control	CPAP end
Systolic BP (mmHg)	133 ± 6	150 ± 5[c]	150 ± 5	154 ± 4[c]	141 ± 5[a]	143 ± 6
Diastolic BP (mmHg)	65 ± 3	71 ± 3	71 ± 3	73 ± 3[c]	70 ± 3	70 ± 3
Heart rate (bpm)	84 ± 7	91 ± 6[c]	88 ± 6	93 ± 6[c]	87 ± 6[b]	88 ± 6[a]

Table shows means ± SEM
[a] $p < 0.05$ vs control; [b] $p < 0.01$ vs control; [c] $p < 0.05$ vs MV; [d] $p < 0.01$ vs MV

both trials. The figure shows the first and last of the ratings plotted against the beginning and end times of each individual trial. At the beginning of trials the patients tended to be either equally or less dyspnoeic with CPAP than without ($p = 0.068$, Wilcoxon test). In three patients dyspnoea was maximal or submaximal at the end of both trials, which, however, occurred later with CPAP. The other two patients tolerated SB for the full 30 min under both conditions. By this time they were less dyspnoeic with CPAP.

Discussion

Continuous positive airway pressure or PEEP have been shown to facilitate spontaneous or assisted breathing in dynamically hyperinflated COPD patients under various circumstances, e.g. acute respiratory failure [16], assisted ventilation [17, 18, 19], exercise [20], sleep [21] and weaning from mechanical ventilation [6, 22]. The present study extends those findings by relating data on breathing pattern and tolerance of SB to those on gas exchange. More successful SB and the tendency towards slower and deeper breathing with CPAP went along with significantly lower arterial PCO_2 and indicators of reduced energy metabolism and improved alveolar ventilation.

Recent studies have addressed the reasons for and the clinical presentation of weaning failure [23, 24, 25]: spontaneous breathing apparently is impossible whenever the respiratory muscles are confronted with loads exceeding their capability. In COPD, $PEEP_i$ constitutes a major part of the load. And finally, the failing respiratory system tends to resort to rapid shallow breathing. It is still unclear why this breathing pattern develops: it might decrease the work per breath, but it hampers CO_2 elimination and does not alleviate the workload per time and, thus, is not an ultimately successful strategy.

The increase in inspiratory workload in COPD through dynamic hyperinflation and its reduction by CPAP have been quantified before [2, 6]. In the present study measurement of work was avoided in order to optimise patient tolerance. Indirect estimates are, however, feasible: CPAP reduced $PEEP_i$ by 5.1 cmH_2O. This translates into a 0.51 J/l reduction of inspiratory work per volume, which is equivalent to that seen in the other studies. While this estimate seems reasonable, it has to be interpreted with caution: measurements of $PEEP_i$ in active patients may yield values artificially elevated by expiratory muscle activity and, thus, may overestimate the degree of dynamic hyperinflation. Detection and quantification of this effect would have required measuring gastric pressure, an invasive procedure we wanted to avoid, as stated above.

There are no systematic studies on the effect of CPAP on expiratory muscle activity in patients with dynamic hyperinflation based on expiratory flow limitation. Petrof and co-workers, studying a group of weaning COPD patients [6], found phasic swings of gastric pressure to be absent in most of them and not changing with the application of CPAP. Healthy dogs increase expiratory muscle activity in proportion to increasing CPAP, apparently defending lung volume against the elevation by the external pressure [26]. So it seems reasonable to assume that overestimation of dynamic hyperinflation due to expiratory muscle activity was more pronounced during the CPAP trial, if different at all. In this case the true differences in $PEEP_i$ as well as work between the SB trials would actually have been larger than the ones measured or deducted, respectively.

Work also can be quantified by studying the energy metabolism. Differences in oxygen consumption between states of SB and MV have been used to gauge the "oxygen cost of breathing" [27]. It, however, may also reflect other components changing with the level of ventilatory support, like the degree of stress and agitation associated with – especially laboured – breathing [28]. Changes of ($\dot{V}_{O_2}$) are inversely related to changes in ventilatory support [29, 30, 31]. Accordingly, the trend towards a lower ($\dot{V}_{O_2}$) with CPAP in our study can be interpreted as a beneficial reduction of energy metabolism through muscle unloading as well as reduction of agitation and stress.

The alleviation of hypercapnia by CPAP most probably was not only effected through a reduction of energy requirements, but also through more efficient ventilation. Practically the entire increase in V_T associated with the application of CPAP was translated into an increase in tidal alveolar ventilation. The underlying

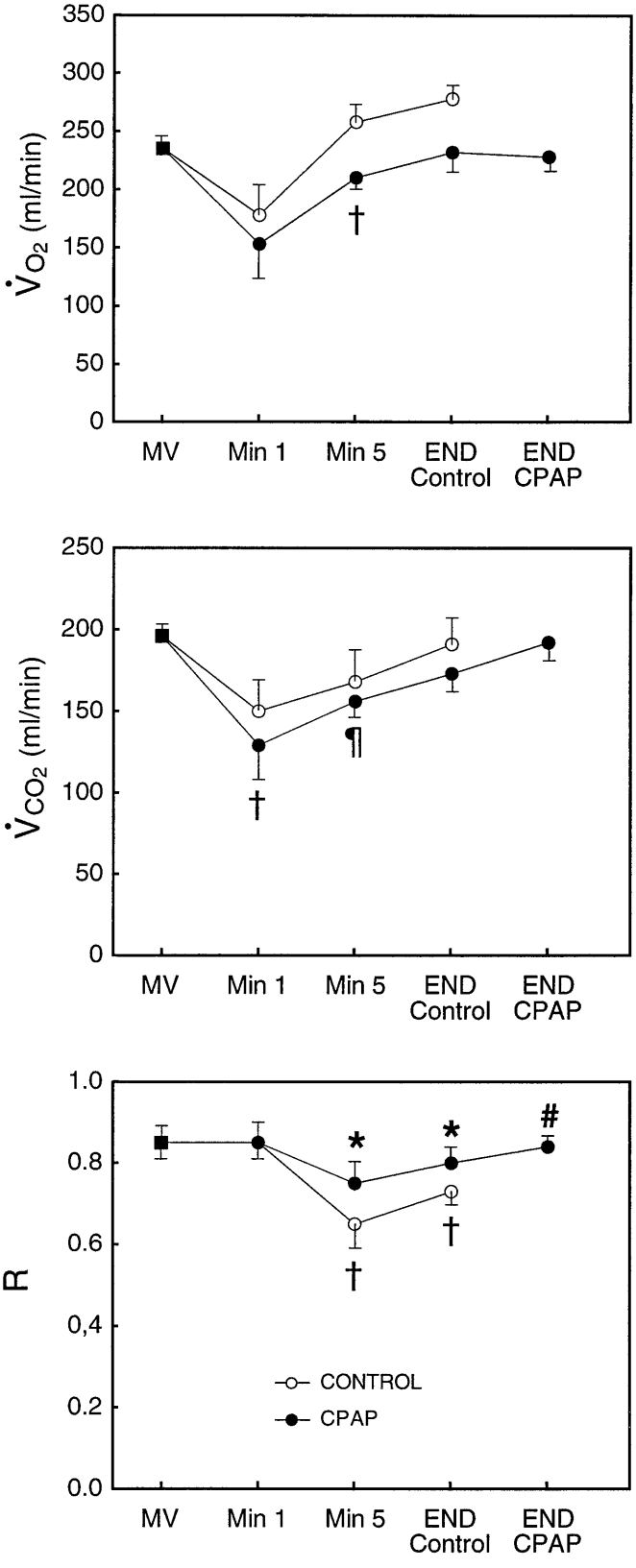

Fig. 2 Oxygen uptake and carbon dioxide excretion during weaning trials. * $p < 0.05$ vs control, † $p < 0.05$ vs MV, # $p < 0.01$ vs control, ¶ $p < 0.01$ vs MV (*MV* mandatory ventilation preceding spontaneous breathing trials)

mechanism could, on the one hand, be the mere reduction of the relative weight of anatomical deadspace accompanying any increase in V_T or, on the other, the improvement of ventilation-perfusion match, which is frequently disturbed in patients with severe COPD [4]. Our data do not permit differentiation between these two. Studies comparing mechanical ventilation to SB in COPD patients show the different tidal volumes to be the main factor influencing gas exchange [32, 33]; effects of PEEP or CPAP were not addressed. This was undertaken in another study [34] showing that under controlled ventilation moderate levels of PEEP (50% of PEEPi) effect improvements of ventilation-perfusion matching along with a slight increase in PO_2 and a decrease in PCO_2. Equivalent studies on CPAP during otherwise unassisted SB are still lacking.

The metabolic data give another clue to the sufficiency of SB during the two trials: instituting SB after MV in both trials meant a sudden decrease in ventilation. For physiological reasons gas exchange with the environment had to decrease transiently, not reflecting energy metabolism in this unsteady state. Due to the relatively large body stores for CO_2, CO_2 excretion reached a steady state more slowly than O_2 uptake, accordingly R transiently decreased as well [35]. The return of R to baseline can be interpreted as gas exchange having reached a new steady state. This indeed was the case by the end of the CPAP trial. In contrast, R was lower throughout the control run and still significantly below baseline at its end. Apparently, CO_2 excretion and $PaCO_2$ still were rising, suggesting that the $PaCO_2$ measured at the end of the control trial was still rising as well, and so the actual hypercapnia about to develop was underestimated.

If the CPAP or PEEP applied to dynamically hyperinflated COPD patients is kept below a "critical" level it does not augment hyperinflation. Due to inhomogeneities of the obstruction, and thus of regional $PEEP_i$ [6], and to other circumstances [36] this "critical" pressure is usually lower than the average $PEEP_i$ measured by end-expiratory occlusion. If CPAP of PEEP is increased above this pressure, negative side effects of additional hyperinflation on respiration and circulation [7, 8] have to be anticipated. Thus, ideally the expiratory pressures should be individually titrated to the patients' mechanics.

Intrinsic positive end-expiratory pressure monitoring is feasible: quasistatic measurements can be performed with a minimum of inexpensive equipment, if an appropriate function is not incorporated in the ventilator

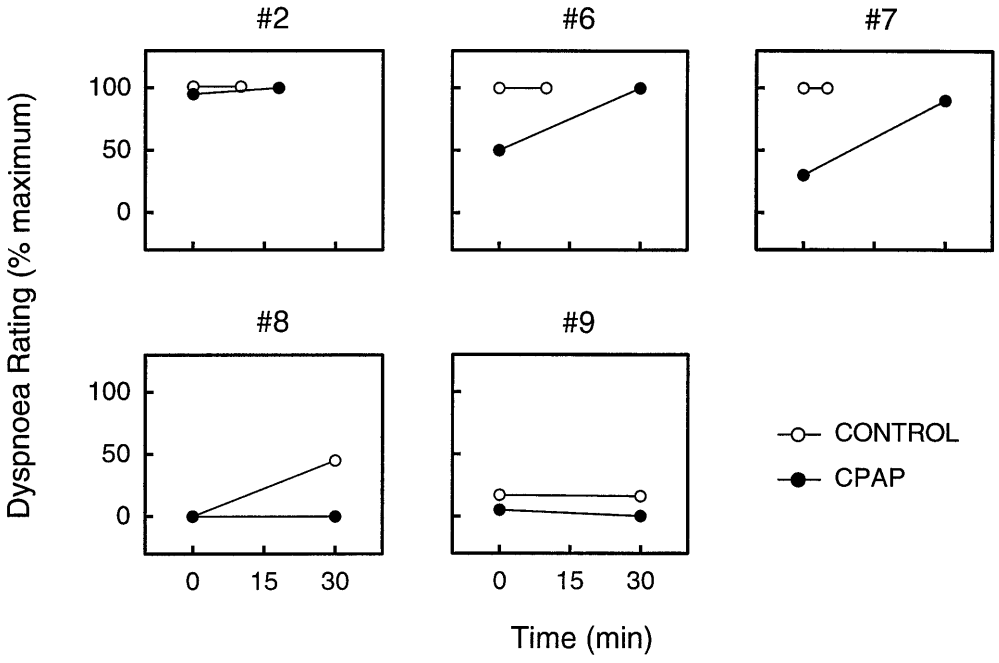

Fig. 3 Dyspnoea ratings (% of maximum) from five patients able to provide estimates. High initial dyspnoea was associated with failure to complete spontaneous breathing trial. With CPAP, dyspnoea tended to be less severe or to occur later

[37]. And the measurement of dynamic $PEEP_i$ does not require any manoeuvres at all [38]. However, occlusions necessitate operator involvement, and dynamic measurements in spontaneously breathing patients have to be derived from oesophageal pressure, which is not routinely monitored in the majority of ICU patients. Our study shows that a more pragmatic approach is feasible. With the application of a standard CPAP we had to tolerate a merely modest increase in lung volumes, as estimated by an average rise in $PEEP_{tot}$ of only 2.3 cmH_2O. Any side effects should therefore have been minimal. In fact, the haemodynamic data showed beneficial, instead of adverse, effects of CPAP on the circulation: The presumably stress-related increases in blood pressure and heart rate seen upon change from MV to SB were smaller (Table 5). Also, the data on SB duration, respiratory drive and dyspnoea suggest that the positive effects of CPAP outweighed any potentially adverse consequences.

In clinical practice SB trials during weaning frequently are performed on a T-piece rather than on a ventilator. Our decision to keep the patients on the ventilator throughout the study despite some (rather small) amount of work being imposed on them by the machine had not only technical reasons (facilitation of signal acquisition, blinding, equal amounts of imposed work during the two SB trials). We believe that SB through a ventilator – especially a modern one imposing even less additional work – has definite advantages over SB through a T-piece: Monitoring and alarms are maintained and the transition back to any form of assistance (CPAP and/or PS, etc.) will not mean reconnecting a patient to the ventilator, which for many is a discouraging experience.

How do the supportive effects of CPAP in dynamically hyperinflated COPD patients compare to those documented for pressure support (PS) ventilation? In a group of predominantly COPD patients, Sassoon and colleagues [39] found CPAP and PS, both 5 cmH_2O, to effect practically equal reductions in oesophageal and transdiaphragmatic pressure swings and pressure time integrals compared to breathing at atmospheric pressure. Apparently, both CPAP and PS can augment tidal volume and reduce respiratory drive in this setting. However, there is an important difference: With PS (like with assisted mandatory ventilation and other modes of ventilatory assist) the increase in tidal volume is likely to increase dynamic hyperinflation. Since the patient has to balance $PEEP_i$ before triggering a breath, the effective trigger threshold will be elevated. Accordingly, increasing the level of PS increases the probability of efforts too weak to trigger [19, 40, 41]. Any reduction in respiratory drive will add to this effect. In contrast, CPAP can diminish $PEEP_i$, lower the effective trigger threshold and reduce the probability of missed triggers. Thus, it seems rational to employ both modes together: CPAP should be applied at a level not causing significant further hyperinflation, and supplementary PS could provide additional inspiratory assistance, if needed. This strategy has been shown to be effective in COPD patients under various circumstances [17, 19, 40].

In summary, CPAP enabled dynamically hyperinflated COPD patients recuperating from an exacerbation to tolerate spontaneous breathing better. With CPAP, en-

ergy metabolism tended to be reduced, CO_2 elimination was more efficient, breaths tended to be deeper and slower and patients had less respiratory drive and were less dyspnoeic. These findings complement previous studies looking at the physiological basis of weaning failure and at the beneficial effects of elevated expiratory pressure in patients with dynamic hyperinflation and expiratory flow limitation. Apparently, weaning failure stands at the end of a cascade starting with a mismatch between demands on the respiratory system and its ventilation capacity, continuing with breathing pattern abnormalities and insufficient CO_2 elimination, and culminating in stress and dyspnoea. Unloading of the respiratory muscles as well as reduction of the demands on ventilation appear to be the basis for the beneficial effect of CPAP. Studies on a larger population of comparable patients should be able to show benefits in final weaning outcome.

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