

I. Gültuna  
P.E.M. Huygen  
C. Ince  
H. Strijdhorst  
J.M. Bogaard  
H.A. Bruining

## Clinical evaluation of diminished early expiratory flow (DEEF) ventilation in mechanically ventilated COPD patients

Received: 3 June 1994  
Accepted: 18 September 1995

**Abstract Objective:** To evaluate the cardiopulmonary effects, especially the end-expiratory lung volume (EEV) and ventilation inhomogeneity during diminished early expiratory flow ventilation (DEEF), which resembles pursed-lips breathing, with the conventional intermittent positive pressure ventilation (IPPV) in postoperative mechanically ventilated patients with chronic obstructive pulmonary disease (COPD).

**Design:** A prospective study measuring cardiopulmonary parameters during IPPV, DEEF, and positive end-expiratory pressure (PEEP) as a control mode. In the PEEP mode, PEEP values were chosen such that the mean airway pressure during a breath cycle was equal to that during the DEEF mode, which was higher than the conventional IPPV mode.

**Setting:** Surgical intensive care unit of a university hospital.

**Patients:** 20 postoperative mechanically ventilated COPD patients who were optimally pretreated and had normal blood oxygenation.

**Interventions:** Measurements were started in the IPPV (IPPV<sub>1</sub>) mode, continued in a randomized order with DEEF or PEEP, and completed with a second IPPV (IPPV<sub>2</sub>) mode, with 1 h equilibration time in each mode before each measurement.

**Measurements and results:** A multi-breath indicator gas wash-out test was used to calculate the EEV and ventilation inhomogeneity. There was a 9% increase ( $p < 0.05$ ) in the mean EEV during both the DEEF and PEEP mode compared to IPPV. No significant changes in the ventilation inhomogeneity and deadspace fractions or the hemodynamic parameters were found during the different ventilatory modes.

**Conclusions:** There was no improvement in pulmonary and hemodynamic parameters during the DEEF mode in comparison to the IPPV mode. The small increase in EEV during DEEF was probably caused by the slightly higher mean expiratory pressures as in the PEEP mode. However, this had no effect on the hemodynamic parameters. As we could not observe any improvement with the DEEF ventilation in our optimally pretreated postoperative COPD patients, we do not advise applying this therapy in this group of patients, since this mode of ventilation may cause barotrauma if not monitored adequately.

**Key words** Diminished early expiratory flow · End-expiratory lung volume · COPD · Wash-out test · Pursed lips breathing · Ventilation inhomogeneity

P.E.M. Huygen · H. Strijdhorst  
H.A. Bruining  
Intensive Care Unit,  
Department of Surgery,  
University Hospital of Rotterdam,  
Rotterdam, The Netherlands

I. Gültuna (✉)  
Department of Anaesthesiology,  
University Hospital of Rotterdam,  
Dr. Molewaterplein 40,  
3015 GD Rotterdam, The Netherlands

C. Ince  
Department of Anaesthesiology,  
University Hospital AMC,  
Amsterdam, The Netherlands

J.M. Bogaard  
Department of Pulmonary Disease,  
University Hospital of Rotterdam,  
Rotterdam, The Netherlands

## Introduction

Clinical observation shows that many patients with chronic obstructive pulmonary disease (COPD), especially those with emphysema and decreased elastic recoil of the lungs, expire through pursed lips, particularly during episodes of dyspnea or increased O<sub>2</sub> consumption [1–5]. This mechanism stimulated Barach to develop the continuous positive pressure breathing (CPPB) technique to treat COPD patients [6]. He showed roentgenographically that the diameter of the branches of the bronchial tree increased when CPPB was applied in patients suffering from obstructive lung disease [7]. CPPB, later described by Gregory et al. as CPAP, is the application of continuous positive airway pressure during spontaneous breathing [8]. This mode was used to prevent premature airway closure and improve gas exchange in COPD patients. CPAP used in conjunction with mechanical ventilation is termed positive end-expiratory pressure (PEEP) and has been shown to decrease bronchoconstriction in a number of COPD patients. PEEP improves arterial oxygenation but has a number of detrimental effects, such as hyperinflation and increase in airway pressures, resulting in cardiovascular depression [9].

Diminished early expiratory flow (DEEF) is a ventilation mode in which the expiratory flow is limited to a maximum and can be compared with pursed-lips expiration during spontaneous breathing. DEEF ventilation, providing a more gradual decrease in airway pressures, may prevent premature airway collapse, decrease ventilation inhomogeneity, and improve gas exchange.

This paper evaluates the effect of DEEF ventilation on gas exchange, end-expiratory lung volume (EEV), ventilation inhomogeneity, and hemodynamic parameters in postoperative COPD patients who were optimally treated for impaired lung function. Multi-breath indicator gas wash-out tests were performed for the measurement of EEV and a new index of ventilation inhomogeneity, which we developed [10, 11]. We compared the gas exchange and hemodynamic parameters during DEEF with the PEEP mode with the same mean airway pressures in order to differentiate changes caused by the higher mean airway pressure or by the ventilation mode itself. The aim of our study was to investigate whether the following would be achieved with DEEF ventilation: (a) more homogeneous ventilation resulting in a decreased pulmonary shunt and a higher partial pressure of oxygen in arterial blood (PaO<sub>2</sub>); (b) increase in alveolar ventilation resulting in a decrease in the physiological deadspace and partial pressure of carbon dioxide in arterial blood (PaCO<sub>2</sub>); (c) prevention of premature airway collapse by shifting the equal pressure point more to the larynx without increasing EEV; and (d) fewer cardiovascular side effects in comparison to the PEEP mode.

## Materials and methods

### Patient selection

In this study we selected 20 COPD patients (mean age 71 ± 7 years) who underwent a major elective abdominal operation (Table 1). The operation included a central vascular reconstruction for an obstructive

**Table 1** The sex, age, and preoperative lung function parameters of the COPD patients (*FEV<sub>1</sub>* forced expiratory in 1 s, *VC* vital capacity, *FRC* functional residual capacity, *EEV* end-expiratory lung volume)

No.	Sex	Age (years)	FEV <sub>1</sub> (l), ref (%) <sup>a</sup>	FEV <sub>1</sub> /VC (%) <sup>a</sup>	FRC (l), ref (%) <sup>a</sup>	EEV (l) <sup>b</sup>	EEV/FRC (%) <sup>c</sup>
1	F	74	0.79 (36)	41	3.90 (105)	2.74	70
2	M	67	1.50 (49)	46	4.16 (113)	2.53	61
3	M	74	1.50 (50)	50	5.58 (150)	3.00	54
4	M	71	1.76 (62)	57	5.24 (139)	2.92	56
5	M	71	1.86 (64)	47	4.36 (120)	1.55	36
6	M	70	1.66 (50)	67	2.96 (86)	1.40	47
7	M	62	1.37 (45)	45	4.08 (116)	1.86	46
8	M	62	1.61 (48)	63	4.35 (117)	2.98	69
9	M	62	1.51 (50)	76	3.14 (107)	1.55	49
10	M	77	1.22 (38)	49	3.21 (95)	1.49	46
11	M	57	2.22 (71)	66	5.35 (120)	2.80	52
12	M	65	1.69 (54)	50	5.04 (138)	3.63	52
13	M	63	1.28 (38)	55	3.28 (97)	1.91	58
14	M	81	2.02 (68)	80	4.05 (110)	1.59	39
15	M	79	2.27 (71)	69	4.11 (115)	1.44	35
16	M	77	1.58 (58)	59	5.25 (119)	1.85	35
17	F	79	1.06 (61)	74	3.55 (102)	2.07	58
18	M	73	1.13 (40)	44	5.28 (143)	1.75	33
19	F	66	1.44 (62)	54	4.26 (153)	3.35	78
20	F	79	1.06 (61)	64	2.78 (105)	1.65	59
Mean		71	1.50 (54)	60	4.20 (118)	2.2	52
±SD		±7	±0.4 (±11)	±14	±0.9 (±18)	±0.7	±12

<sup>a</sup> Data from the preoperative lung function test

<sup>b</sup> EEV measured during the DEEF mode by the multi-breath indicator gas wash-out test

<sup>c</sup> Ratio of postoperative EEV and preoperative FRC

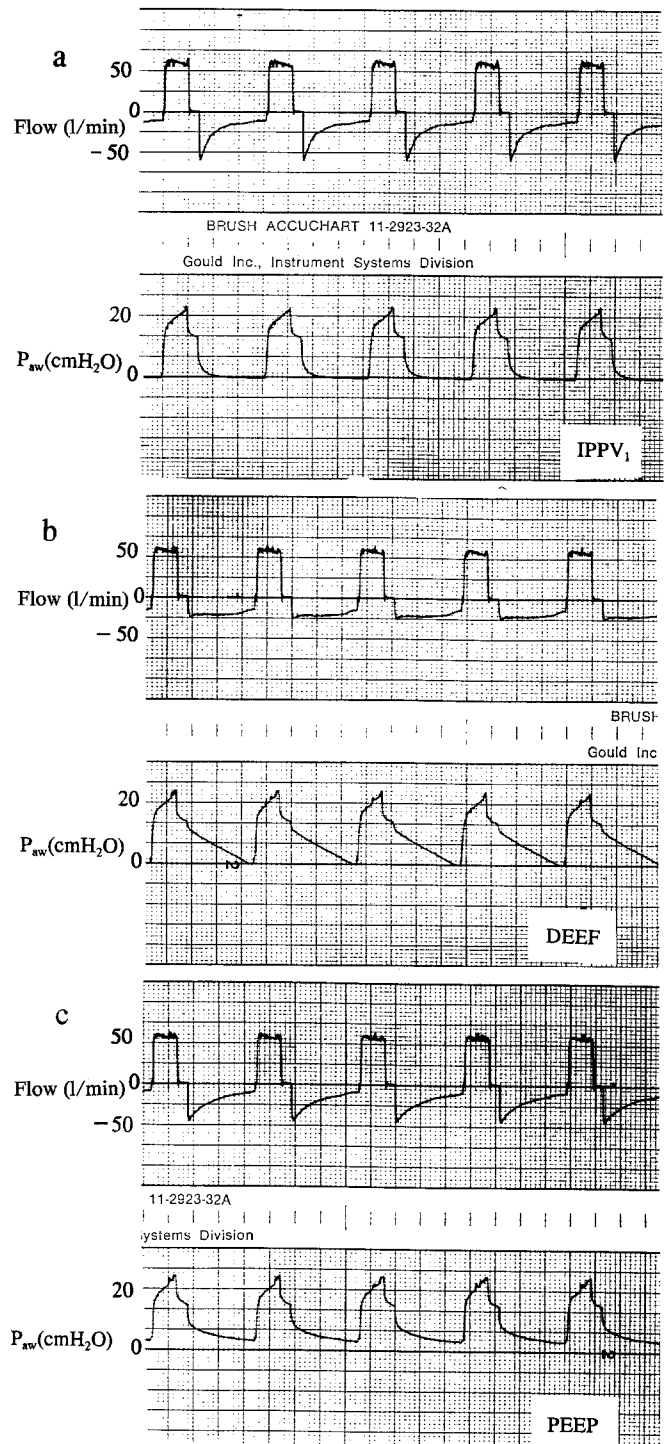
tion or aneurysm, or an esophageal resection followed by a reconstruction for a malignancy without a thoracotomy. The diagnosis of COPD was based on the history, physical examination, chest X-ray, and the lung function test according to the ATS criteria [12]. All patients had poor exercise tolerance combined with a chronic cough and were, or had been, heavy smokers. All patients were seen preoperatively by a pulmonologist and all were prescribed steroids and theophylline intravenously and nebulized salbutamol. Preoperatively, the patients underwent a lung function test at the lung function laboratory of the pulmonary diseases department. In clinical practice airway obstruction has usually been assessed by measuring the forced expiratory volume in 1 s vital capacity ratio  $FEV_1/VC$ , and any COPD patients who fulfilled the clinical criteria of a  $FEV_1/VC$  value less than 80% of reference under optimal pulmonary treatment were entered in to the study. The preoperative mean functional residual capacity, measured by a closed circuit helium wash-in test, in these patients was 118% of the normal expected value, showing slight hyperinflation, as expected in COPD patients. Preoperatively a pulmonary artery catheter (Swan-Ganz) was introduced in all patients for hemodynamic measurements using the internal jugular vein. All patients gave informed consent before they were included in the study, and the study was approved by the hospital ethical committee. The following criteria were used to exclude patients from the study: (1) thorax or ventilator system leakage; (2) acute respiratory distress syndrome ( $PEEP > 5$  cmH<sub>2</sub>O, fractional inspired oxygen ( $FIO_2$ )  $> 60\%$  and/or diffuse shaded chest X-ray); (3) postoperative bleeding (transfusions); (4) hemodynamic instability (vasopressors); (5) sepsis or renal insufficiency; and (6) restlessness (fighting the ventilator).

### Protocol

All patients arrived on the intensive care unit directly from surgery, sedated and paralysed, and were ventilated in the intermittent positive pressure ventilation mode (IPPV<sub>1</sub>). All patients were ventilated with zero end-expiratory pressure, a tidal volume range of 7–10 ml/kg, a frequency of 10–12, an inspiratory/expiratory (I/E) ratio of 1:2, a constant inspiratory flow rate, and a constant  $FIO_2$  of 40%. We waited 1 h before starting measurements to achieve a steady state and continued ventilation with the PEEP or DEEF mode in a randomized order. All patients were ventilated with the same ventilator (Servo 900-B, Siemens Elema, Sweden) and the same PEEP valve. The DEEF mode was installed with the maximum expiratory flow knob (Servo 900-B), where the flow was limited until the expiratory pressure just reached zero before the next inspiration started. The flow, measured by the heated pneumotachograph, and the airway pressure, measured by the ventilator manometer, were recorded on a printer (Fig. 1). From the pressure signal we calculated the mean airway pressure with an integrator built by our department. A small amount of PEEP was applied as a control mode to obtain the same mean airway pressure as in the DEEF mode, thus equalizing the higher mean expiratory airway pressures that occurred in the DEEF mode (Table 2). After the PEEP and DEEF modes, a control IPPV mode (IPPV<sub>2</sub>) eliminated time effects. In all the modes the ventilatory parameters (frequency, I/E ratio, inspiratory flow rate,  $FIO_2$ ) were kept constant.

### Measurements

Pulmonary parameters were calculated by a multi-breath indicator gas wash-out test that we have previously described [10, 11]. A small fraction of a poorly soluble indicator gas ( $SF_6$ ) was added to the inspiratory gas fraction of the Servo ventilator with a low pressure system by an indicator gas injector developed by our depart-



**Fig. 1a–c** The pressure and flow signals IPPV, DEEF, and PEEP ventilatory modes are shown. **a** In IPPV there is a sudden decrease of airway pressure during the expiration to zero. Note that the end-expiratory flow is not zero. **b** In the DEEF mode there is a more gradual decrease in airway pressure, reaching zero at the end of expiration and resulting in a lower initial expiratory flow pattern. **c** In PEEP there is the same expiratory airway pressure pattern as in IPPV, but the pressure will be still positive at the end of expiration. The flow pattern is comparable to IPPV; however, the initial part of the expiratory flow is retarded by the PEEP valve

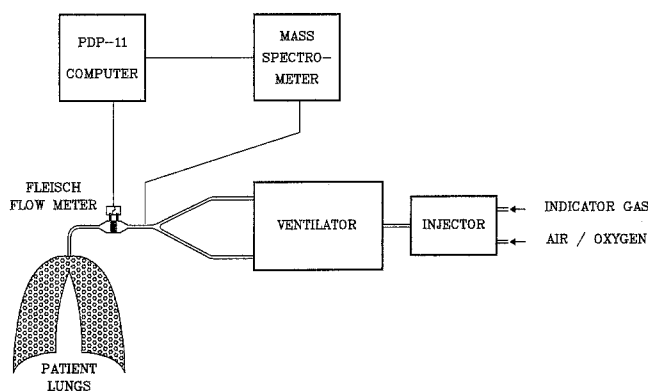
**Table 2** Respiratory parameters of COPD patients during the different ventilatory modes

No.	IPPV <sup>a</sup>	PEEP <sup>a</sup>	DEEF <sup>a</sup>	Max. exp. flow <sup>b</sup>
1	26/13/0	28/16/5	26/14/0	20
2	18/10/0	19/11/3	18/10/0	18
3	25/16/0	26/17/3	25/17/0	12
4	26/17/0	28/19/3	26/17/0	15
5	18/10/0	20/12/4	20/12/0	12
6	22/14/0	23/15/3	22/14/0	10
7	28/17/0	30/19/4	28/18/0	13
8	25/17/0	27/19/3	27/19/0	15
9	30/22/0	32/23/3	32/22/0	12
10	26/19/0	28/20/3	27/19/0	10
11	23/15/0	24/16/3	25/17/0	10
12	24/14/0	26/15/3	25/15/0	10
13	20/12/0	22/14/4	22/14/0	10
14	18/11/0	20/12/3	19/11/0	15
15	22/15/0	23/16/4	22/15/0	11
16	25/14/0	27/16/4	25/15/0	10
17	18/11/0	20/12/3	19/11/0	9
18	26/16/0	27/18/3	26/16/0	10
19	24/15/0	26/17/3	24/16/0	9
20	21/14/0	23/16/3	22/14/0	10
Mean	23.3/14.6/0	25.0/16.1/3.4	24.0/15.3/0	12.1
±SD	3.5/3.0/-	3.6/3.0/0.6	3.5/3.0/-	3.2

<sup>a</sup> Peak, plateau, and end-expiratory pressures respectively (cmH<sub>2</sub>O)

<sup>b</sup> Maximal expiratory flow limitation during the DEEF mode (l/min)

ment [13]. When the difference between the inspiratory and expiratory fractions of the indicator gas was smaller than 2%, a wash-out test was started by stopping the injection of the indicator gas. The wash-out measurements continued for 5 min. The indicator and metabolic gas fractions were continuously measured with a mass spectrometer (Airspec, MGA 3000, Case Scientific, Biggin Hill, UK) at a rate of 200 ml/min by means of a teflon capillary. Respiratory flow was measured by a heated pneumotachograph (Fleisch-2, Gould Godard, The Netherlands) connected to a differential pressure transducer (Validyne MP-45-14-871, Northridge, Calif., USA), which was placed between the Y-piece and the patient. The signals from the mass spectrometer and the pneumotachograph were sampled at a frequency of 100 Hz by the computer (PDP-11, DEC, Maynard, Mass., USA) (Fig. 2). From these data the EEV, ventilation inhomogeneity, and Bohr deadspace were calculated off-line. The EEV extrapolation is based on the relation between the net amount of indicator gas washed out from the lung and the alveolar dilution caused by this indicator gas transport. The alveolar dilution of the indicator gas is estimated as the difference between the indicator gas fraction before the start of the wash-out and the end-expiratory gas fraction [13]. The ventilation inhomogeneity, measured as the volumes regression index, is based on the discrepancy between the end-expiratory and the mean alveolar indicator gas fraction in the course of the wash-out procedure. A value up to 0.6–0.7 was measured in healthy patients with no obstructive disease and higher values (>1.0) were significant in patients with obstruction [10]. The Bohr dead space is calculated by the difference between the mean expired and the end-expiratory gas fraction, where the end-expiratory fraction is assumed to be the mean alveolar gas fraction [14]. Three wash-out tests were performed for each patient in each ventilatory mode to determine reproducibility. The



**Fig. 2** The measurement set-up for the respiratory parameters. The indicator gas is injected into the bellows of the ventilator. Respiratory flow is measured by the Fleisch flowmeter and the gas fractions are sampled by the mass spectrometer. The respiratory parameters are off-line calculated by the computer

mean of these three wash-out tests for each parameter was taken to evaluate the different ventilatory modes.

Arterial blood gases were obtained by a radial artery catheter during each ventilatory mode and measured by a blood gas analyser (ABL-3 Radiometer, Copenhagen, Denmark). Hemodynamic parameters [(pulmonary artery pressure (PAP), pulmonary capillary wedge pressure (PCWP), central venous pressure (CVP)] and mixed venous blood gases were obtained by the pulmonary artery catheter. The cardiac output was measured using the pulmonary artery catheter with the thermodilution method by injection of 10 ml saline at ambient temperature with a pistol, which had a constant injection time [15]. The pistol was synchronized with the ventilator and the average of three measurements in the different phases of a ventilatory cycle were taken (0%, 33%, 66%) to estimate a mean cardiac output. Averaging three measurements spread equally over the ventilatory cycle has been proven to estimate a reliable mean cardiac output in mechanically ventilated patients [16].

#### Statistical analysis

The lung function parameters, blood gases, and hemodynamic parameters during IPPV<sub>1</sub> were compared with the DEEF, PEEP, and IPPV<sub>2</sub> parameters with the Wilcoxon rank test. When  $p < 0.05$ , the value was considered to be significant. The comparison between IPPV<sub>1</sub> and IPPV<sub>2</sub> was done to evaluate the time effect of the measured parameters. All values are shown as mean ± SD.

## Results

In our study the maximum expiratory flow during the DEEF mode was  $12.1 \pm 3.2$  l/min (range 9–20 l/min), which created the same mean expiratory pressures equivalent to  $3.4 \pm 0.6$  cmH<sub>2</sub>O (range 3–5 cmH<sub>2</sub>O) of PEEP (Table 2). The results of the cardiopulmonary parameters in the different ventilatory modes are given in Table 3. The mean EEV calculated with IPPV<sub>1</sub> was  $2.2 \pm 0.7$  l, with DEEF  $2.4 \pm 0.8$  l, with PEEP  $2.4 \pm 0.7$  l, and with IPPV<sub>2</sub>  $2.2 \pm 0.7$  l. There was an increase of 9% with

**Table 3** The pulmonary and hemodynamic parameters measured during the different ventilatory modes (mean  $\pm$  SD,  $n = 20$ ) ( $V_D/V_T$  volume dead space/tidal volume,  $SaO_2$  arterial oxygen saturation,  $SvO_2$  mixed venous oxygen saturation,  $MAP$  mean arterial pressure,  $MPAP$  mean pulmonary arterial pressure)

	IPPV <sub>1</sub>	DEEF	PEEP	IPPV <sub>2</sub>
EEV (l)	2.2 $\pm$ 0.7	2.4 $\pm$ 0.8 <sup>a</sup>	2.4 $\pm$ 0.7 <sup>a</sup>	2.2 $\pm$ 0.7
S <sup>b</sup>	1.2 $\pm$ 0.2	1.1 $\pm$ 0.2	1.2 $\pm$ 0.2	1.2 $\pm$ 0.2
$V_D/V_T$ (%)	40 $\pm$ 4	40 $\pm$ 4	40 $\pm$ 3	40 $\pm$ 5
PaO <sub>2</sub> (kPa)	13.9 $\pm$ 4	14.1 $\pm$ 3	15.0 $\pm$ 3	14.8 $\pm$ 3
PaCO <sub>2</sub> (kPa)	4.8 $\pm$ 0.8	4.7 $\pm$ 0.8	4.9 $\pm$ 0.8	5.0 $\pm$ 0.8
SaO <sub>2</sub> (%)	97 $\pm$ 2	98 $\pm$ 1	98 $\pm$ 2	97 $\pm$ 2
SvO <sub>2</sub> (%)	70 $\pm$ 5	69 $\pm$ 4	72 $\pm$ 4	71 $\pm$ 3
Shunt (%)	17 $\pm$ 5	16 $\pm$ 4	16 $\pm$ 4	19 $\pm$ 5
MAP (mmHg)	89 $\pm$ 15	85 $\pm$ 14	85 $\pm$ 15	86 $\pm$ 16
MPAP (mmHg)	21 $\pm$ 7	21 $\pm$ 6	20 $\pm$ 7	21 $\pm$ 7
CVP (mmHg)	9 $\pm$ 4	9 $\pm$ 3	10 $\pm$ 4	9 $\pm$ 4
PCWP (mmHg)	9 $\pm$ 4	9 $\pm$ 5	10 $\pm$ 4	9 $\pm$ 4
Cardiac output (l/min)	4.8 $\pm$ 1	4.7 $\pm$ 1	4.7 $\pm$ 1	5.0 $\pm$ 1

<sup>a</sup> Significantly different from IPPV<sub>1</sub> and IPPV<sub>2</sub> ( $p < 0.05$ ); all other changes were insignificant

<sup>b</sup> (S) index of ventilation inhomogeneity

DEEF and PEEP in the mean EEV in comparison with the IPPV mode ( $p < 0.05$ ). There was a good correlation between the increase in EEV and the FEV<sub>1</sub>/VC ratio. We observed a smaller increase in EEV in patients with a lower FEV<sub>1</sub>/VC ratio. The ventilation inhomogeneity as measured by the volumes regression index (S) showed a small insignificant decrease during the DEEF mode. There was a small insignificant increase in peak and plateau airway pressures during DEEF, with a range of 1–2 cmH<sub>2</sub>O (Table 2).

The PaO<sub>2</sub> during PEEP and IPPV<sub>2</sub> was higher than in the IPPV<sub>1</sub> and DEEF mode, and a small increase in arterial oxygen saturation was measured during DEEF and PEEP modes. A small decrease was measured in mean arterial pressure, cardiac output, and shunt fraction during DEEF and PEEP. However, these changes in the different ventilatory modes were not significant.

## Discussion

Pursed-lips breathing is based on the generation of a longer-lasting positive mouth pressure during expiration that is reflected to the airways. This may have a beneficial effect on the pattern of lung emptying in some COPD patients, especially those with emphysema who have decreased elastic recoil of the lungs. It has been shown that positive expiratory pressure during tidal breathing with pursed lips may increase tidal volume and decrease respiratory rate, resulting in improved oxygenation [2, 3, 17].

Since expiratory obstruction is a basic problem in asthma and emphysema, it is logical to prevent any maneuver that would further increase the resistance to airflow. However, it has been observed clinically that emphysema patients expire through pursed lips, especially during increased activity [5].

A comparable effect can be obtained by the application of expiratory resistance that causes a more gradual

decrease in the longitudinal pressure gradient over the airways, reducing air trapping, increasing tidal volume, and decreasing respiratory frequency during spontaneous breathing. The lower pressure gradient over the airways and the lower initial flow during expiration will shift the equal pressure point (EPP) – the point where the intrabronchial pressure equals the pleural pressure – more to the mouth [18]. In healthy subjects EPP is localized in the large airways close to the larynx consisting of cartilaginous tissue that keeps the airways open during the whole of expiration [19]. However, in COPD patients, especially emphysema patients due to loss of lung elasticity, EPP is localized more peripherally to the alveoli in the smaller, less rigid airways, and dynamic compression will cause airway compression, especially in the lower zones during end-expiration [19]. The collapsed airways will not participate in the ventilation that will increase the shunt fraction and decrease the PaO<sub>2</sub>. Pursed-lips expiration or expiratory retard in emphysema patients will shift the EPP more to the mouth, increase the tidal volume, decrease the respiratory rate, and a more homogeneous ventilation will be expected, resulting in an improved gas exchange. One of the main goals in the treatment of COPD patients is to improve gas exchange by increasing the tidal volume and reducing the ventilation inhomogeneity [2].

Although DEEF is comparable with pursed-lips expiration, there are some physiological differences. The DEEF mode is applied during controlled mechanical ventilation, whereas pursed-lips expiration is applied during spontaneous breathing. Both create a longer positive expiratory pressure to prevent premature airway collapse. With pursed lips, the respiratory rate is decreased, resulting in prolonged expiration and an increase in the tidal volume. However, in the controlled DEEF mode both the respiratory rate and the tidal volume are kept constant. This could be the reason that this mode had hardly any effect on the blood gases, ventilation inhomogeneity, and shunt fraction. If we compare DEEF with IPPV, in both

modes the inspiratory peak and plateau pressures are almost the same (Table 2), but the expiratory pressure patterns differ. The conventional IPPV mode shows a sudden airway pressure drop and the pressure will become zero in an earlier stage of the expiration. Although the end-expiratory pressure becomes zero during the DEEF mode (Fig. 1), the mean expiratory pressure will be higher than in the IPPV mode. An increase in the EEV would theoretically increase the intrathoracic pressure, increasing the CVP and decreasing cardiac output [9]. In our study we found only a small, insignificant decrease in cardiac output and no changes in CVP and PCWP. Although not measured in this study, intrinsic PEEP ( $PEEP_i$ ) may have been present, with positive end-expiratory alveolar pressures caused by airway closure, compression, or bronchoconstriction in our COPD patients [20–22]. We believe, however, that our measurements reliably reflected the difference in the EEVs between the three different ventilatory modes. We found a good correlation between the change in EEV and the preoperative  $FEV_1/VC$  and observed a smaller increase in EEV in patients who had more obstruction. Falke et al. could not find any effect up to 5 cmH<sub>2</sub>O of PEEP on cardiac output in patients with acute respiratory failure [23]. We found little change in the hemodynamic parameters with DEEF and PEEP increasing the intrathoracic pressure only with 3 to 5 cmH<sub>2</sub>O. We used the PEEP mode equivalent to the mean airway pressure as in the DEEF mode with the same frequency to eliminate the higher airway pressure effects. The increase in the EEV during DEEF and PEEP could be explained by the increased mean airway pressures and absence of marked intrinsic PEEP. We measured the EEV using a multi-breath indicator gas wash-out test. With the indicator gas wash-out test we might have underestimated the actual EEV, as only the adequately ventilated parts of the lung are measured. This is especially true in COPD patients, who may have air-trapping. For the best EEV estimation an extrapolation is made to reduce the inaccuracy of the ventilation inhomogeneity [10]. This shows that the effect of inhomogeneous ventilation is included in the estimation of the lung volume. With the DEEF mode we would expect an improvement in the ventilation

inhomogeneity with a more gradual emptying of the lung but only observed a small, insignificant decrease in the ventilation inhomogeneity during DEEF. There was no essential change in the dead space fraction during DEEF and PEEP. This may be explained by the small difference in the mean airway pressures in these modes of ventilation. It can be seen from Table 3 that the postoperative PaO<sub>2</sub> values of the COPD patients were acceptable, indicating that the patients were adequately pretreated. The lowest PaO<sub>2</sub> value was 9.5 kPa and the mean PaO<sub>2</sub> reached 14–15 kPa with an FIO<sub>2</sub> of 40%, which supplied adequate oxygenation. This may be the reason that no significant differences were observed in blood gases in the different ventilatory modes. The PaCO<sub>2</sub> values did not change in the different ventilatory modes, indicating unchanged alveolar ventilation. A change in the amount of airway closure should certainly have influenced the blood gases and shunt fraction, which is not the case. The small increase in EEV did not affect the blood gases, perhaps due to the minimal changes in ventilation–perfusion inhomogeneity and adequate oxygenation. An essential difference between pursed-lips breathing and the DEEF mode was that respiratory frequency and tidal volume were kept constant during DEEF, in order to compare it with the IPPV and PEEP mode. However, an important effect of pursed-lips breathing is a decrease in respiratory rate, providing a longer expiration time for more efficient ventilation [17], which could explain the marginal effects of DEEF on the pulmonary as well as the hemodynamic parameters under these conditions. As the DEEF mode was effective in some individual COPD patients, there was no improvement in our 20 optimally pretreated COPD patients as a group. However, our results cannot be extrapolated to COPD patients who have hypoxia, air-trapping, and consequently intrinsic PEEP during an acute exacerbation. Perhaps in such patients DEEF could be an effective treatment mode but it should always be carefully monitored as excessive DEEF can cause hyperinflation resulting in barotrauma.

**Acknowledgements** This study was supported by the Netherlands Asthma Foundation (grant no. 84–12).

## References

- Schmidt RW, Wasserman K, Lillington GA (1964) The effort of airflow and oral pressure on the mechanics of breathing in patients with asthma and emphysema. *Am J Respir Crit Care Med* 90:564–571
- Thoman RL, Stoker GL, Ross JC (1966) The efficacy of pursed lips breathing in patients with COPD. *Am J Respir Crit Care Med* 93:100–106
- Ingram RH, Schilder DP (1967) Effect of pursed lips expiration on the pulmonary pressure-volume relationship in obstructive lung disease. *Am J Respir Crit Care Med* 96:381–388
- Miller WF (1958) Physical therapeutic measures in the treatment of chronic bronchopulmonary disorders. *Methods for breathing training. Am J Med* 24: 929–940
- Barach AL (1973) Physiological advantages of grunting, groaning and pursed lips breathing: adaptive symptoms related to the development of continuous positive pressure breathing. *Bull NY Acad Med* 49:666–673
- Barach AL (1938–1939) Physiological methods in the diagnosis and treatment of asthma and emphysema. *Ann Intern Med* 12:454–481

7. Barach AL, Swenson P (1939) Effect of breathing gases under positive pressure on lumens of small and medium-sized bronchi. *Arch Intern Med* 63:946–948
8. Gregory GA, Kitterman JA, Phibbs RH, Tooley WH, Hamilton WK (1971) The treatment of idiopathic respiratory distress syndrome with continuous positive airway pressure. *N Engl J Med* 284:1333–1340
9. Tuxen D (1989) Detrimental effects of positive end-expiratory pressure during controlled mechanical ventilation of patients with severe airflow obstruction. *Am J Respir Crit Care Med* 140: 5–9
10. Huygen PEM, Gültuna I, Ince C, Zwart A, Bogaard JM, Feenstra BWA, Bruining HA (1993) A new ventilation inhomogeneity index from multiple breath indicator gas washout tests in mechanically ventilated patients. *Crit Care Med* 21:1149–1158
11. Huygen PEM, Feenstra BWA, Hoorn E, Jansen JRC, Zwart A (1991) PDPS: Pulmonary Data Processing System for assessment of gas exchange properties by multiple gas washout. *Comput Methods Programs Biomed* 36: 223–235
12. American Thoracic Society (1987) Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease (COPD) and asthma. *Am J Resp Crit Care Med* 136:225–243
13. Huygen PEM, Feenstra BWA, Holland WPJ, Ince C, Stam H, Bruining HA (1990) Design and validation of an indicator gas injector for multiple gas washout tests in mechanically ventilated patients. *Crit Care Med* 18:754–758
14. Zwart A, Jansen JRC, Luijendijk SCM (1982) Bohr dead space during helium washout. *Bull Eur Physiopathol Respir* 18:261–272
15. Jansen JRC, Schreuder JJ, Bogaard JM, van Rooyen W, Versprille A (1981) Thermodilution technique for measurement of cardiac output during artificial ventilation. *J Appl Physiol* 50:584–591
16. Jansen JRC, Schreuder JJ, Settels JJ, Kloek JJ, Versprille A (1990) An adequate strategy for the thermodilution technique in patients during mechanical ventilation. *Intensive Care Med* 16:422–425
17. Mueller RE, Petty TL, Filley GF (1970) Ventilation and arterial bloodgas changes induced by pursed lips breathing. *J Appl Physiol* 28:784–789
18. Mead J, Turner JM, Macklem PT, Little JB (1967) Significance of the relationship between lung recoil and maximum expiratory flow. *J Appl Physiol* 22: 95–108
19. Pride NB (1971) The assessment of airflow obstruction. Role of measurements of airway resistance and of tests of forced expiration. *Br J Dis Chest* 65:135–169
20. Pepe PE, Marini JJ (1982) Occult positive end-expiratory pressure in mechanically ventilated patients with airflow obstruction. The auto-PEEP effect. *Am J Respir Crit Care Med* 126: 166–170
21. Hubmayr RD, Gay PC, Tayyab M (1987) Respiratory system mechanics in ventilated patients: techniques and indications. *Mayo Clin Proc* 62:358–368
22. Berg vd B, Stam H, Bogaard JM (1991) Effects of PEEP on respiratory mechanics in patients with COPD on mechanical ventilation. *Eur Respir J* 4: 561–567
23. Falke KJ, Pontoppidan H, Kumar A, Leith DE, Geffin B, Laver MB (1972) Ventilation with end-expiratory pressure in acute lung disease. *J Clin Invest* 51:2315–2323