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Effects of PEEP on respiratory mechanics in patients with COPD on mechanical ventilation

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Effects of PEEP on respiratory mechanics in patients with COPD on mechanical ventilation. B. van den Berg, H. Stam, J.M. Bogaard.
ABSTRACT: We studied the effects of positive end-expiratory pressure (PEEP) applied by the ventilator on respiratory mechanics in ventilated patients with chronic obstructive pulmonary disease (COPD). Airway pressures, relaxed expiratory flow-volume curves and end-expiratory volumes (EEV) were measured. In all patients investigated without PEEP applied by the ventilator, an intrinsic PEEP level (PEEPi) and a concavity in the flow-volume curve was present. Ventilator-PEEP caused a significant decrease in PEEPi in all patients ($p<0.01$). In patients in whom ventilator-PEEP exceeded PEEPi, significant increases occurred in airway pressures and EEV ($p<0.05$) and moreover the shape of the flow-volume curve was changing. In patients in whom the level of ventilator-PEEP was below the PEEPi level, no significant changes in airway pressures, EEV or flow-volume curves were found. We conclude: 1) PEEP applied by the ventilator can reduce PEEPi in ventilated patients with COPD without significant changes in airway pressures, EEV or flow-volume curves. 2) Expiratory flow-volume curves can be used to estimate the effects of ventilator-PEEP on EEV.
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In mechanically ventilated patients with Chronic Obstructive Pulmonary Disease (COPD) increased airway resistance and loss of elasticity of lung tissue have profound influences on respiratory mechanics. Considering these factors the expiration time determined by the ventilator is often too short to achieve complete expiration to the relaxed functional residual capacity (FRC) [1]. Consequently, at end-expiration a lung volume above FRC and a positive alveolar pressure is found [1, 2]. This pressure is called intrinsic Positive End-Expiratory Pressure (PEEPi) in contrast to the (extrinsic) Positive End-Expiratory Pressure applied by the ventilator [1, 3, 4]. Although PEEPi is associated with the same haemodynamic effects as PEEP applied by the ventilator, important differences between both types of PEEP exist, as the increase in patient effort to "trigger" assisted ventilator breaths due to the presence of PEEPi [1].

In order to reduce PEEPi, application of PEEP by the ventilator has been suggested [5]. The clinical benefit of application of ventilator-PEEP has been challenged in view of increases in lung volume and intrathoracic pressure [6]. Other authors stated that the effect of ventilator-PEEP on expiratory flow and end-expiratory lung volume depends upon the level of PEEP applied [1, 5, 7]. However, in these studies only lung volume changes derived from spirometry or plethysmography were reported. Studying the effects of PEEP applied by the ventilator, the end-expiratory lung volume, determining the elastic recoil of the respiratory system, is a key variable.

We studied the effects of PEEP applied by the ventilator on airway pressures, expiratory flow-volume curves and end-expiratory lung volumes in patients with COPD. For this investigation absolute lung volumes were measured. From these measurements the effects of ventilator-PEEP on expiratory flow and lung volume were examined in relation to the level of PEEPi present.

Patients and methods

Patients

Twelve patients with COPD aged 56–75 yrs were studied. Patient data are shown in table 1. Pulmonary function tests, including vital capacity (VC) and forced expiratory volume in 1 s (FEV_1), were obtained shortly before or after the period of mechanical ventilation when the patients were breathing spontaneously without an endotracheal tube. The FEV_1 is also expressed as percentage of predicted [8]. Respiratory failure was caused by acute exacerbations of chronic bronchitis in 10 patients; in the remaining two patients mechanical ventilation had been started because of pneumonia in one patient and surgery for a gastric perforation in the

Table 1. – Patient data

Patient no	Age yrs	Sex M/F	BH m	BW kg	VC *l*	FEV_1 *l*	FEV_1 %N	Cause of Resp. failure	Concomitant disease
1	75	F	1.54	48	1.19	0.39	22	exacerbation	
2	75	M	1.73	54	2.35	1.55	53	exacerbation	
3	56	M	1.78	70	4.10	1.03	29	exacerbation	
4	73	M	1.78	82	3.00	0.80	26	exacerbation	
5	72	M	1.80	91	1.50	0.64	24	exacerbation	TBC in past
6	63	F	1.67	78	2.58	0.69	29	exacerbation	
7	71	M	1.77	108	2.20	1.10	36	exacerbation	
8	69	F	1.65	55	2.55	1.02	46	pneumonia	
9	59	M	1.72	105	1.89	0.76	24	exacerbation	
10	72	M	1.60	63	n.d.	n.d.		gastric perforation	
11	71	F	1.60	67	1.32	0.49	25	exacerbation	thoracoplasty
12	64	F	1.58	47	1.66	0.38	19	exacerbation	

BH= body height; BW= body weight; VC= vital capacity; FEV_1= forced expiratory volume in 1 sec; FEV_1 %N= FEV_1 expressed as percentage of normal value; TBC= tuberculosis; n.d.= not done.

other patient. The 10 patients admitted for acute exacerbation were studied within a week after starting mechanical ventilation; the two patients admitted for other causes were studied after 14 days of ventilatory support when they had recovered from the acute event.

Before the study all patients were alert and haemodynamically stable. They were ventilated through a cuffed endotracheal tube size 7–9 (inner diameter: 7.0–9.0 mm) with the exception of patients no. 10 and 11 who were ventilated through cuffed tracheostomy tubes no. 6 and 8 (inner diameter: 7.0 and 8.5 mm respectively).

The aim of the study was explained to the patients and informed consent was obtained. During the study the patients were sedated with midazolam and paralyzed with pancuronium.

Methods

During the study the patients were ventilated in the supine position with a Siemens Servo 900C ventilator (Siemens-Elema, Solna, Sweden). The volume-controlled mode was used with an average tidal volume of 0.65 *l* and a respiratory rate of 10–12 min^{-1}. The inspiratory:expiratory ratio was set as follows: the inflation amounted to 25%, the inflation hold 10% and the expiration 65% of the cycle. This mode of ventilation was chosen to ensure an equal time for expiration in all patients. Airway pressures were measured at the endotracheal tube with a Validyne pressure transducer and recorded in time on a Servogor recorder (type 460, Brown Boveri Company, Rotterdam, Holland). To obtain the pressures during airway occlusion the end-inspiratory and end-expiratory hold buttons of the Servo 900C were used. The end-inspiratory pressure was defined as the pressure measured at 1.5 s after occlusion of the airway. End-expiratory pressure was measured using an expiration hold of 4 s. This end-expiratory pressure is called total PEEP (PEEPtot). Extrinsic PEEP (PEEPe) is defined as the pressure at the endotracheal tube during the last second of expiration. The difference between the pressures at the endotracheal tube during the expiration hold and during the last second of expiration is called PEEPi [9]. The compressible volume of the ventilatory tubings was not taken into account. For the estimation of the total compliance of the respiratory system, expiratory tidal volume was measured using the Silverman flow-transducer of the Servo 900C ventilator calibrated with a wet spirometer. The total static compliance was calculated by dividing expiratory tidal volume by the driving pressure, the difference between end-inspiratory and end-expiratory pressure (PEEPtot).

The end-expiratory lung volume (EEV) was obtained by using a closed circuit helium-dilution technique comparable with a system described previously [10]. The system used consists of a rolling seal spirometer, the back compartment of which was connected to the ventilator and the front compartment to the patient. In this way a rebreathing system was established operated by the ventilator without changing the ventilator mode. The CO_2 excreted by the patient was absorbed by sodalime in the spirometer. Moreover the rebreathing volume was kept constant by automatic supplemention of O_2 to compensate for the O_2 uptake by the patient. This O_2 stabilization allows long equilibration times of 8–10 minutes obligatory in patients with severe unequal ventilation.

Relaxed expiratory flow-volume curves were obtained using a heated pneumotachograph (Lilly) at the expiratory port of the ventilator. Volume displacement was obtained by integration of the flow signal (Jaeger, Würzburg, F.R.G.). The flow-volume curve was recorded on an X-Y recorder (X-Y 733, Brown Boveri Company, Rotterdam, Holland).

From the flow-volume curve the peak expiratory flow ($\dot{V}peak$), the expiratory flows at 50 and 25% of expiration volume ($\dot{V}_{50}$, $\dot{V}_{25}$) and the end-expiratory flow at the moment the valve of the ventilator closed ($\dot{V}_{endex}$) were measured.

The resistance at end-expiration was estimated as the quotient of driving pressure and simultaneous flow. In this case PEEPi was considered as driving pressure. In order to estimate the resistance of the equipment, the pressure-flow relationship of the endotracheal tubes, ventilator tubings and pneumotachograph was obtained [11]. The relationship was curvilinear described by a power function of the form: $P = a\dot{V}^b$, where a and b were constants. Results for endotracheal tubes of representative sizes are shown in fig. 1. For the study, the flow resistance of the equipment was subtracted from the resistance of the total respiratory system.

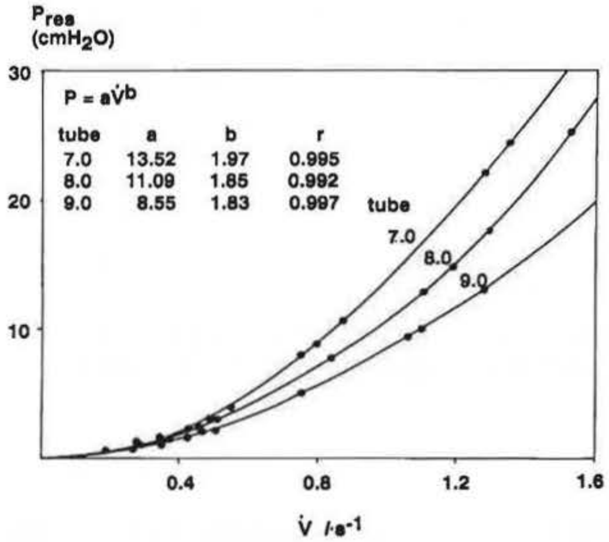

Fig. 1. – The relationship between resistive pressure (P_{res}) an airflow ($\dot{V}$) for endotracheal tubes and ventilator tubings is shown. Also indicated are the results obtained by fitting measurements to a power function of the form $P_{res} = a\dot{V}^b$, where a and b are constants (r = correlation coefficients).

All measurements were performed in duplicate at two end-expiratory pressures applied by the ventilator: zero end-expiratory pressure and a low PEEP level. This level of ventilator-PEEP was not adjusted to any criterium but was chosen by chance. Afterwards the level of ventilator-PEEP was defined as the difference between the two PEEPe levels obtained at both ventilator settings. The level of ventilator-PEEP varied from 3.0 to 9.3 cm H_2O with an average of 5.8 cm H_2O. For reasons of simplicity we will refer to 6 cm H_2O ventilator-PEEP in the following text. The scissor type expiration valve from the Siemens Servo 900C ventilator was used as PEEP valve [12]. No randomization was applied. The measurements at the different ventilator settings were obtained at least five minutes after the ventilator-PEEP level had been changed.

The measurements obtained without ventilator-PEEP was compared with those at the applied ventilator-PEEP. From the flow-volume curves isovolume flows obtained at the two ventilator-PEEP levels were compared. From the curves obtained without ventilator-PEEP the expiratory flow after exhalation of 15% of expiration volume was measured. At the applied ventilator-PEEP level the expiratory flow at the corresponding expiration volume was obtained, taken into account the increase in EEV associated with the PEEP applied by the ventilator. For statistical analysis the Wilcoxon matched pairs test was used. P-values less than 0.05 were considered statistically significant. Correlations were examined by linear regression analysis [13].

Results

The pressure- and volume-related measurements of the individual patients obtained without and with ventilator-PEEP are shown in table 2. The measurements obtained without ventilator-PEEP revealed a PEEPi ranging from 2.0 to 14.1 cmH_2O. Although no PEEP was applied by the ventilator, all patients showed a very low PEEPe varying from 0.4 cmH_2O to 1.3 cm H_2O. The patients were divided into two groups. In the patients no. 1–6 a PEEPi level was obtained below the value of ventilator-PEEP applied in the second part of the study (the low PEEPi group). The PEEPi level of the patients no. 7–12 exceeded the value of ventilator-PEEP used later in the study (the high PEEPi group). PEEPi decreased significantly ($p<0.05$) in both patient groups comparing the two ventilator settings without and with ventilator-PEEP; however, a significant increase in total PEEP ($P<0.05$) was only found in the patients with a low PEEPi. Accordingly significant increases in end-inspiratory pressure ($P_{endinsp}$) and end-expiratory volume (EEV) ($p<0.05$) were only found in the patients of the low PEEPi group; in the patients of the high PEEPi group no significant changes were found in $P_{endinsp}$, PEEPtot or EEV comparing the measurements obtained without and with ventilator-PEEP. When the change of EEV was compared with the change in PEEPtot for the 12 patients, a positive correlation was found which proved to be significant ($r=0.76$ $p=0.004$).

The total compliance did not change at 6 cm H_2O ventilator-PEEP in either patient group: for the 12 patients the means and standard deviations amounted to 0.073 ± 0.023 $l\cdot cmH_2O^{-1}$ for the values obtained without ventilator-PEEP and 0.072 ± 0.022 $l\cdot cmH_2O^{-1}$ at 6 cmH_2O^{-1} ventilator-PEEP.

The effects of ventilator-PEEP on the relaxed expiratory flow-volume relationships of patients no. 3 and 12 are shown in fig. 2. The right-sided curves represent the expiratory flow-volume relationships obtained without ventilator-PEEP. A concavity in the flow-volume curves is noted suggesting dynamic airway compression at lower lung volumes. Expiratory flow is still present at the moment the expiration valve of the ventilator closes. At this point an EEV of 3.44 *l* and 4.40 *l* respectively is shown on the right side of the curves. The left-sided curves represent the flow-volume relationships at 6 cm H_2O ventilator-PEEP. The shift to the left with respect to the curve obtained without ventilator-PEEP is caused by increases in EEV of 0.50 and 0.12 *l* respectively for patients no. 3 and 12 associated with the application of ventilator-PEEP. In patient no. 3 application of 6.9 cm H_2O of PEEP by the ventilator leads to an alteration in the shape of the flow-volume curve: the bi-phasic curve has been altered in a

Table 2. – Effects of ventilator-PEEP on respiratory mechanics

Patient no	Ventilator-PEEP = 0 cmH_2O P_{endins} cmH_2O	PEEPi cmH_2O	PEEPe cmH_2O	PEEPtot cmH_2O	EEV l	Ventilator-PEEP = 5.8 cmH_2O P_{endins} cmH_2O	PEEPi cmH_2O	PEEPe cmH_2O	PEEPtot cmH_2O	EEV l
1	9.9	2.0	0.5	2.5	1.68	15.5	0.7	6.7	7.4	2.05
2	11.0	3.0	0.5	3.5	2.22	14.1	2.0	3.5	5.5	2.56
3	13.3	4.0	0.8	4.8	3.44	16.4	1.4	6.9	8.3	3.98
4	12.1	5.0	0.5	5.5	3.01	16.2	4.7	5.5	10.2	3.41
5	20.6	5.9	0.4	6.3	1.18	23.4	2.6	6.4	9.0	1.53
6	16.8	6.7	1.3	8.0	2.41	20.6	1.2	10.6	11.8	2.96
mean	14.0	4.4	0.7	5.1	2.32	17.7*	2.1*	6.6	8.7*	2.75*
SD	3.7	1.6	0.3	1.8	0.76	3.2	2.0	5.5	2.0	0.82
7	13.0	6.3	0.5	6.8	2.79	15.4	5.0	4.3	9.3	3.06
8	22.3	9.3	0.4	9.7	2.51	23.7	6.1	5.6	11.7	2.42
9	19.0	9.8	0.6	10.4	1.95	17.3	1.9	8.2	10.1	2.05
10	20.6	11.0	0.5	11.5	3.06	21.2	6.7	5.6	12.3	3.03
11	28.5	12.4	0.7	13.1	1.07	28.5	6.2	7.0	13.2	1.04
12	23.7	14.1	0.8	14.9	4.40	26.2	9.2	6.8	16.0	4.52
mean	21.2	10.5	0.6	11.1	2.63	22.1	5.9*	6.3	12.1	2.69
SD	4.7	2.5	0.1	2.6	1.02	4.6	2.2	1.2	2.2	1.07

P_{endins}= end-inspiratory pressure; PEEP;= intrinsic PEEP; PEEPe= extrinsic PEEP; PEEPtot= total PEEP; EEV= end-expiratory volume; SD= standard deviation. Wilcoxon matched pairs test: significant difference:*=0.05>p>0.01.

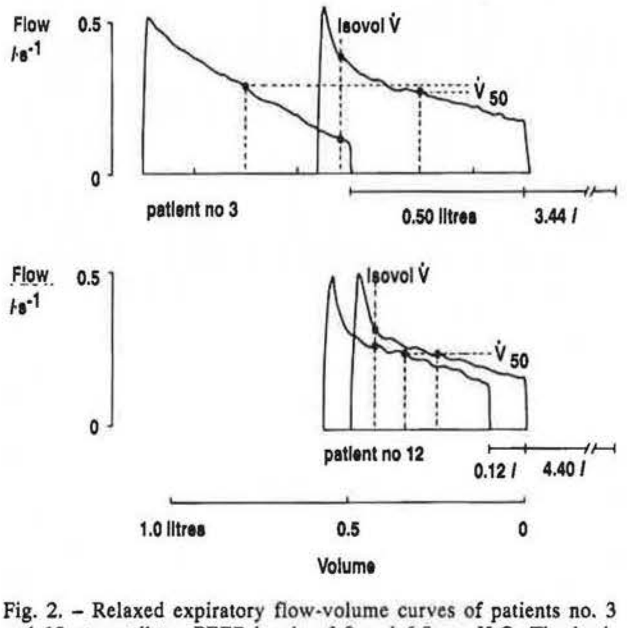

Fig. 2. – Relaxed expiratory flow-volume curves of patients no. 3 and 12 at ventilator-PEEP levels of 0 and 6.8 cm H_2O. The horizontal scale relates to both changes in EEV and volume changes in the flow-volume curves; $\dot{V}_{50}$= expiratory flow at 50% of expiration volume; Isovol $\dot{V}$= isovolume flow; EEV= end-expiratory volume.

mono-phasic curve. In the flow-volume curves of patient no. 12 no change in the shape of the curve is found comparing zero with 6.8 cm H_2O of ventilator PEEP. The isovolume expiratory flows are also indicated in the figure. The isovolume expiratory flow of patient no. 3 was reduced from 0.35 $l \cdot s^{-1}$ to 0.13 $l \cdot s^{-1}$ at 6.9 cmH_2O ventilator-PEEP; the isovolume flows of patient no. 10 were 0.28 $l \cdot s^{-1}$ and 0.24 $l \cdot s^{-1}$ at zero and 6.8 cm H_2O ventilator-PEEP.

The expiratory flows measured from the flow-volume curves and the resistances calculated from driving pressure and flow are shown in table 3. In the patient group with low PEEPi significant increases in $\dot{V}_{50}$ and $\dot{V}_{25}$ were found associated with a significant decrease in $\dot{V}_{endex}$ comparing the measurements obtained with and without ventilator-PEEP (p<0.05). In the patient group with high PEEPi no significant changes were found in expiratory flows. The levels of isovolume flows obtained without ventilator-PEEP were not different for the two patient groups; a significant decrease in isovolume flow was only found in the low PEEPi group comparing zero with 6 cm H_2O of ventilator-PEEP. No significant changes were found in $\dot{V}$peak in either patient group.

However, the resistance at end-expiration was significantly reduced for both patient groups comparing the two ventilator settings.

Significant correlations were found between the changes in EEV and expiratory flows. For the 12 patients the change in EEV was positively correlated with the change in $\dot{V}_{50}$ (r=0.65 p=0.02) and negatively correlated with the change in isovolume expiratory flow (r=-0.81 p=0.002).

Table 3. – Effects of ventilator-PEEP on respiratory mechanics

Patient no	Ventilator-PEEP= 0 cmH_2O $\dot{V}_{peak}$ $l \cdot s^{-1}$	$\dot{V}_{50}$ $l \cdot s^{-1}$	$\dot{V}_{25}$ $l \cdot s^{-1}$	$\dot{V}_{endex}$ $l \cdot s^{-1}$	Isovol $V l \cdot s^{-1}$	R_{endex} $cmH_2O l \cdot s^{-1}$	Mean ventilator-PEEP= 5.8 cmH_2O $\dot{V}_{peak}$ $l \cdot s^{-1}$	$\dot{V}_{50}$ $l \cdot s^{-1}$	$\dot{V}_{25}$ $l \cdot s^{-1}$	$\dot{V}_{endex}$ $l \cdot s^{-1}$	Isovol $\dot{V} l \cdot s^{-1}$	R_{endex} $cmH_2O l \cdot s^{-1}$
1	0.47	0.20	0.14	0.08	0.37	26.6	0.59	0.29	0.14	0.04	0.11	13.2
2	0.52	0.17	0.12	0.09	0.31	34.9	0.42	0.19	0.14	0.09	0.18	17.9
3	0.56	0.26	0.20	0.17	0.35	23.2	0.51	0.29	0.20	0.12	0.13	6.4
4	0.46	0.17	0.14	0.12	0.26	42.6	0.38	0.18	0.14	0.12	0.19	35.6
5	0.88	0.29	0.19	0.12	0.59	47.3	0.76	0.32	0.20	0.11	0.25	19.0
6	0.74	0.29	0.20	0.16	0.42	42.0	0.73	0.36	0.24	0.11	0.15	4.5
mean	0.61	0.23	0.16	0.12	0.38	36.1	0.57	0.27*	0.18*	0.10*	0.17*	16.1*
SD	0.16	0.05	0.03	0.03	0.10	8.7	0.14	0.07	0.04	0.03	0.05	10.2
7	0.53	0.18	0.13	0.11	0.29	59.7	0.41	0.18	0.14	0.11	0.19	42.6
8	0.54	0.18	0.13	0.10	0.36	90.1	0.66	0.18	0.13	0.09	0.38	65.4
9	0.68	0.28	0.20	0.14	0.46	66.5	0.65	0.31	0.21	0.13	0.44	8.8
10	0.65	0.17	0.12	0.08	0.28	139.4	0.60	0.17	0.12	0.09	0.27	68.3
11	0.75	0.33	0.21	0.14	0.58	81.2	0.71	0.33	0.21	0.13	0.62	41.0
12	0.55	0.20	0.17	0.15	0.28	89.5	0.50	0.17	0.14	0.12	0.18	70.1
mean	0.62	0.22	0.16	0.12	0.37	87.8	0.59	0.22	0.16	0.11	0.35	49.4*
SD	0.08	0.06	0.04	0.02	0.11	25.7	0.10	0.07	0.04	0.02	0.16	21.6

$\dot{V}$peak= peak expiratory flow; $\dot{V}_{50}$, $\dot{V}_{20}$= expiratory flow at 50 and 25% of expiration volume; $\dot{V}_{endex}$= expiratory flow at end-expiration; Isovol $\dot{V}$= isovol flow; R_{endex}= trans pulmonary resistance calculated from PEEPi and $\dot{V}_{endex}$. SD= standard deviation. Wilcoxon matched pairs test: significant difference: *= 0.05>p>0.01

Discussion

The occurrence of PEEPi is frequently encountered in patients with COPD who are mechanically ventilated. However, PEEPi can originate from different pathophysiological mechanisms [14].

In our study the PEEPi found was associated with dynamic airway compression, considering the patients were paralyzed and the tidal volume and respiratory rate were low, making other types of PEEPi unlikely. However, the presence of expiratory flow limitation was not confirmed in our study. Flow limitation can only be estimated using the interrupter technique during relaxed expiration [7, 11]. The presence of expiratory flow limitation is suggested when application of ventilator-PEEP is associated with an unchanged expiratory flow at the same lung volume [7, 11]. This was also demonstrated in our study in the patients with a PEEPi-level higher than the applied ventilator-PEEP.

PEEPi was measured at the endotracheal tube using an airway occlusion at end-expiration. Using this technique we could also demonstrate that during expiration the airway pressure did not fall to zero, although no ventilator-PEEP was applied. This PEEPe was present in all patients, caused by the ventilator tubings in the presence of an end-expiratory flow.

We investigated the effects of a low ventilator-PEEP on PEEPi, end-expiratory lung volumes and expiratory flow-volume curves. The clinical benefit of application of ventilator-PEEP to mechanically ventilated patients in whom PEEPi is found, remains to be determined [5, 6, 7]. Increase in the pressure downstream of the critical site of flow limitation (the "equal pressure point") is considered not to influence the expiratory flow until the applied ventilator-PEEP exceeds the level of the extramural pressure surrounding the airway [14]. If the ventilator-PEEP exceeds this critical pressure, an increase in end-expiratory lung volume will occur associated with a reduction in expiratory flow at the same lung volume. The hyperinflation causes the elastic recoil to increase and the equal pressure point will be displaced downstream.

Application of ventilator-PEEP in this study caused a significant decrease in PEEPi in all patients. However, the effects of ventilator-PEEP on airway pressures, end-expiratory volumes and flow-volume curves varied among the individual patients. Data analysis could discern two groups of patients with different responses to the application of ventilator-PEEP. The difference between the two groups depended upon the relationship between PEEPi and ventilator-PEEP. In the patients in whom the PEEP applied by the ventilator exceeded PEEPi, significant increases in airway pressures and EEV were found associated with the application of ventilator-PEEP. In this group of patients ventilator-PEEP is considered to exceed the critical pressure: significant increases in both end-inspiratory pressure and total PEEP were also found. In the patients in whom PEEPi exceeded ventilator-PEEP, the changes in airway pressures and EEV were not significant.

In most investigations concerning PEEPi and ventilator-PEEP only volume changes derived from spirometry on respiratory inductive plethysmography are reported [6, 15]. In contrast, we measured end-expiratory lung volumes, assuming that in the process of flow limitation lung volume is a key variable. We found that the level of PEEPi did not correlate with the magnitude of EEV, despite the presence of hyperinflation. Relatively low end-expiratory volumes were found in patients suffering from obesity, and in those with a history of tuberculosis and thoracoplasty. However, a reduction in FRC is well-established when supine position is compared with seated position [16]. In this study the loss of tonic activity of the inspiratory muscles may have contributed to this decrease.

Previous investigations have claimed detrimental increases in lung volumes and airway pressures associated with ventilator-PEEP [6]. In our study a group of patients could be discerned in whom application of PEEP by the ventilator was not associated with significant increases in EEV or airway pressures. This contradiction can be explained if we assume that in the previous investigation the PEEP applied by the ventilator surpassed the PEEPi in all patients studied [6].

We also studied PEEPi and ventilator-PEEP in relation to relaxed flow-volume curves. Relaxed expiratory flow-volume curves have been suggested to determine the presence of PEEPi [1]. In patients who can not expire to their elastic equilibrium volume, the flow-volume relationship does not pass through zero but has an intercept on the flow axis *i.e.* an end-expiratory flow associated with PEEPi [11]. Moreover, in all patients studied, a concavity in the expiratory flow-volume relationship was found consistent with dynamic airway compression. In previous studies the concavity in the flow-volume curve has been encountered in ventilated patients with COPD but not in patients ventilated for other forms of respiratory failure [7, 11, 17].

In order to estimate the shape of the flow-volume relationship the peakflow, the $\dot{V}_{50}$, the $\dot{V}_{25}$, the flows at 50% respectively 25% of expiration volume and the $\dot{V}_{endex}$, the flow at the moment the valve of the ventilator closed was measured. By measuring these flows the expiratory flow-volume relationship is considered as 'free floating'; in order to examine flow-volume curves as volume-based, *i.e.* in relation to the end-expiratory lung volume, isovolume expiratory flows were obtained.

The effect of ventilator-PEEP on the expiratory flow-volume curves differed between the two patient groups. In the patients in whom ventilator-PEEP exceeded the critical pressure, the shape of the flow-volume curve was changing as indicated by slight but significant increases in $\dot{V}_{50}$ and $\dot{V}_{25}$ and decreases in $\dot{V}_{endex}$. The changing pattern indicated a marked decrease or disappearance of expiratory flow limitation.

Although in the former group the expiratory flows increased due to application of PEEP by the ventilator, these changes in flow should be considered in relation to total lung volume. In contrast with the increase in $\dot{V}_{50}$ and $\dot{V}_{25}$, the isovolume flows were significantly decreased due to the increase in end-expiratory lung volume. In the patient group with the high PEEPi level, in whom ventilator-PEEP did not increase EEV, the shape of the expiratory flow-volume curve remained unchanged as did the isovolume flow comparing the two ventilator settings.

Considering these expiratory flows the resistance of the endotracheal tubes and ventilator tubings have to be taken into account. In order to calculate the transpulmonary resistance at end-expiration the flow-resistance of the equipment was estimated and subtracted from the total resistance.

In both patient groups the R_{endex}, the transpulmonary resistance calculated at end-expiration from driving pressure and flow was significantly reduced. In the patient group in whom the EEV was unchanged, the decrease in resistance can be elucidated by the hypothesis that during flow limitation the driving pressure can be diminished without a change in expiratory flow [7]. It has to be underlined that the decrease in resistance should be considered mainly as reduction of energy dissipation within the flow-limiting segment of the airways.

Considering these results, two major issues should be taken into account. PEEP was applied by means of the scissor type PEEP device of the Siemens Servo ventilator. Although this PEEP device has been considered to exert a low resistance during passive expiration, the effects of ventilator-PEEP on the expiratory flow-volume relationship could be related to the device used [12].

Secondly the flow-volume relationship was studied under muscle paralysis. However, we have reasons to believe that the results of our study can be extrapolated to clinical circumstances without muscle relaxation. We could reproduce the results in patients without muscle paralysis during controlled mechanical ventilation. Moreover relaxed flow-volume curves have been obtained in previous investigations in patients without muscle paralysis [7, 11]. Recently, a paper has been published on the effects of continuous positive airway pressure showing the effects of ventilator-PEEP on flow-volume curves during spontaneous breathing [18].

In mechanically ventilated patients with COPD application of PEEP by the ventilator in order to reduce PEEPi has been challenged in view of increasing hyperinflation. This study suggests that ventilator-PEEP can reduce PEEPi without increasing EEV and this could be identified from the effect of ventilator-PEEP on the expiratory flow-volume curve. The concavity of the flow-volume curve remained unchanged when a ventilator-PEEP level was applied which did not increase EEV. However, when a level of ventilator-PEEP did increase EEV, a change in the shape of the expiratory flow-volume curve was found.

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Effets de la pression positive en fin d'expiration (PEEP) sur la mécanique respiratoire, chez les patients atteints de BPCO sous ventilation mécanique. B. van den Berg, H. Stam, J.M. Bogaard.

RÉSUMÉ: Nous avons étudié les effets de la PEEP appliquée par le ventilateur sur la mécanique respiratoire chez des sujets atteints de bronchopneumopathie chronique obstructive sous ventilation. Les pression des voies aériennes, les courbes, de débit-volume expiratoire relaxé, et les volumes à la fin de l'expiration, ont été mesurés. Chez tous les patients investigués sans application de PEEP par le ventilateur, un niveau intrinsèque de PEEP (PEEPi) et une concavité dans la courbes débit-volume étaient présents. La PEEP appliquée au ventilateur entraîne une diminution significative de PEEPi chez tous les patients ($p<0.01$). Chez les patients chez lesquels la PEEP appliquée au ventilateur était supérieure à PEEPi, des augmentations significatives apparaissent dans les pressions des voies aériennes et dans les volumes de fin d'expiration ($p<0.05$), et de plus, la forme de la courbe débit-volume se modifie. Chez les patients chez lesquels le niveau de PEEP au ventilateur était inférieur au niveau de PEEPi, aucune modification significative n'a été découverte en ce qui concerne les pressions de voies aériennes, les volumes en fin d'expiration, ou les courbes débit-volume. Nous concluons que: 1) PEEP appliquée par le ventilateur peut réduire PEEPi chez les sujets ventilés atteints de BPCO, sans modification significative des pressions des voies aériennes, des volumes de fin d'expiration, ou des courbes débit-volume; 2) les courbes de débit expiratoire/volume peuvent être utilisées pour estimer les effets de la PEEP appliquée au ventilateur sur les volumes de fin d'expiration.

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