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Inspiratory effort and measurement of dynamic intrinsic PEEP in COPD patients: effects of ventilator triggering systems

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Abstract *Objective:* To investigate effects of ventilator triggering systems (pressure and flow triggering: PT and FT) on measurement of dynamic intrinsic PEEP ($PEEPi_{dyn}$) and patient-ventilator interaction in patients with chronic obstructive pulmonary disease during weaning from mechanical ventilation.
Design: Prospective study.
Setting: Medical/surgical intensive care unit of an academic hospital.
Patients and participants: 6 COPD patients with acute respiratory failure ready to wean.
Measurements: We measured flow, airway opening, esophageal and gastric pressures. Minute ventilation, breathing pattern and pressure time product (PTP) of the respiratory muscles and of the diaphragm were obtained during spontaneous ventilation through a mechanical ventilator (Puritan-Bennett 7200ae). Two triggering systems, namely PT and FT, were evaluated.
Results: The inspiratory muscles effort necessary to overcome the triggering system overestimated $PEEPi_{dyn}$ measurement of an amount equal to 49±2 and 58±3% during respectively pressure and flow triggering. FT increased tidal volume and minute ventilation and decrease PTP/b and PTP/min of the respiratory muscles and diaphragm.
Conclusions: To correctly measure $PEEPi_{dyn}$, the inspiratory effort produced to overcome PEEPi and to trigger the ventilator must be discriminated. Application of flow triggering requires less effort to initiate inspiration and provide a positive end-expiratory pressure level that is able to unload the respiratory muscles by reducing PEEPi. With flow triggering higher minute ventilation are obtained in COPD patients during the weaning phase.

Key words COPD · Acute respiratory failure · pressure time product · Intrinsic PEEP · Flow trigger

Introduction

Chronic obstructive pulmonary disease (COPD) is characterized by an increase in airway resistance and a loss of lung elastic recoil. As a result, airflow limitation develops, leading to the presence of an intrinsic positive end-expiratory pressure (PEEPi) and dynamic hyperinflation [1, 2]. When the respiratory muscles are paralyzed and the patient is on controlled mechanical ventilation (CMV), end-expiratory occlusion of the ventilator expiratory port, at the end of exhalation, provides the static measurement of PEEPi ($PEEPi_{stat}$) [3]. In patients on spontaneous breathing (SB), the deflection in intrathoracic pressure produced by the respiratory muscles during the time delay from the onset of inspiratory effort to the beginning of flow, represents the dynamic measurement of PEEPi ($PEEPi_{dyn}$) [4]. The former represents the aver-

age PEEPi level present in conditions of pulmonary time-constant inequalities [5], while the latter indicates the minimum PEEPi that has to be counterbalanced to initiate inspiratory flow. $PEEPi,_{dyn}$ therefore identifies the maximal level of externally applied PEEP/CPAP that can be used to unload the respiratory muscles without causing further dynamic hyperinflation [4, 5].

Sassoon has recently showed that, during SB through a ventilator, a substantial time delay from the onset of intrathoracic pressure deflection to the beginning of inspiratory flow is present, due to the initial effort produced by the patient to reach the trigger sensitivity initiating inspiratory flow [6, 7]. Such time delay and the relative inspiratory effort produced to open the inspiratory valve were smaller during flow triggered SB than during pressure triggered SB [6, 7].

Aim of the study was to verify whether the ventilator triggering system may interfere with the measurement of $PEEPi_{dyn}$ in COPD patients spontaneously breathing through a ventilator. Besides, effects of the two common triggering systems (pressure-triggering and flow-triggering) on patient-ventilator interaction were evaluated.

Methods

Six patients with COPD admitted to the intensive care unit of the Policlinico Hospital (University of Bari) were studied. They were nasotracheally intubated (Portex©cuffed endotracheal tube) with an inner diameter varying from 7–8 mm, and were mechanically ventilated (Puritan-Bennett 7200ae, Puritan-Bennett Corp., Carlsbad, CA) for management of acute respiratory failure (ARF). The diagnosis of COPD was confirmed by their history and physical examination as well as by previous pulmonary function tests. The precipitating causes of ARF and pertinent clinical information are provided in Table 1. Entry criteria included clinical and hemodynamic stability for the proceeding 12–24 h and a maximal inspiratory pressure (MIP) of at least $-20\ cmH_2O$. All patients were able to tolerate spontaneous breathing trials of at least 30 min duration and were in the weaning process as prescribed by their attending physician. The investigative protocol was approved by the local ethics committee, and written informed consent was obtained from each patient or next of kin. A physician not involved in the study protocol was always present to provide for patient care.

Flow ($\dot{V}$) was measured with a heated pneumotachograph (Fleisch No. 2; Fleisch, Lausanne, Switzerland), connected to a differential pressure transducer (Validyne MP 45 ± 2 cm H_2O; Validyne Co., Northridge, CA), which was inserted between the y-piece of the ventilator circuit and the endotracheal tube. The pneumotachograph was linear over the experimental range of flow. Equipment dead space (not including the endotracheal tube) was 70 ml. Airway opening pressure (Pao) was measured proximal to the endotracheal tube with a pressure transducer (Validyne MP $45 \pm 100\ cmH_2O$). Changes in intrathoracic and abdominal pressures were evaluated by assessment of esophageal (Pes) and gastric (Pga) pressures. Pes and Pga were measured using thin latex balloon-tipped catheter systems. Both balloons were connected by polyethylene catheters (length 70 cm; internal diameter, 1.7 mm) to separate differential pressure transducers (Validyne MP $45 \pm 100\ cmH_2O$). Both balloons were 10 cm in length and 2.4 cm in circumference. The esophageal balloon was filled with 0.75 ml of air and correctly positioned by performing an occlusion test [8]. The gastric balloon contained 1.0 ml of air. All the above variables were displayed on an eight-channel strip-chart recorder (7718A Hewlett-Packard Co., Cupertino, CA) and collected on a personal computer via a 12-bit analog-to-digital converter at a sample rate of 100 Hz. Subsequent data analysis was performed using the software package ANADAT (RHT-InfoDat, Montreal, Quebec). Tidal volume (V_T) was computed by the digital integration of the flow signal. Pes was subtracted from Pga in order to determine transdiaphragmatic (Pdi) pressure.

Experimental procedure

All patients were spontaneously breathing through the ventilator set on CPAP zero in the semirecumbent position. MIP was measured as the most negative airway pressure generated within the first 20 s of total airway occlusion at end-expiratory lung volume [9]. A one way valve was used in the expiratory limb to ensure that efforts began from a lung volume $\leq$ the equilibrium volume of the chest.

The trigger function was set by using two different mechanisms:

Pressure-triggering. Patients had to breathe against the occluded airway, producing a pre-set negative Pao value necessry to open the inspiratory demand valve and initiate the breath [10]. The threshold pressure value ranged between 0.5 and 1 cmH_2O.

Flow-triggering. Fresh gas flowed continuously within the inspiratory and expiratory circuit at a constant rate that could be set between 5 and 20 l/min. This continuous gas flow provided the base flow, which exited through the exhalation port and was measured every 20 ms. When the subjects' inspiratory flow reached a pre-set threshold value, called the flow sensitivity, the ventilator added gas to the circuit to maintain the set positive Pao level and provided the required inspired gas. The flow sensitivity could be varied between a minimum value of 1 l/min and a maximum value of no greater than half of the set base flow value [7]. In our study, the baseline flow was set at 10 l/min, while the flow sensitivity ranged between 1 and 2 l/min.

The two different triggering systems were randomly applied. Measurements were obtained after 30–40 min period of time for each experimental conditions, once a stable breathing pattern was observed. Consecutive breaths (20–30) were collected and then averaged to provide the flow, Pao, Pes, Pga and Pdi signals of the "mean representative breath" [12] (Fig. 1).

Data analysis

Inspiratory time (Ti), expiratory time (Te), and total breathing cycle time (Ttot) were determined from the flow tracing. Tidal excursions of Pes (ΔPes) and Pdi (ΔPdi) were also determined. Pressure time product per breath (PTP/b) for the inspiratory muscle ($PTP/b,_{Pes}$) and the diaphragm ($PTP/b,_{Pdi}$) were obtained by measuring the area under respectively the Pes and the Pdi signals from the onset to the end of their negative (for Pes) and positive (for Pdi) deflection [13]. For Pes signal, measurement of such areas were referred to the chest wall (CW) static recoil pressure vs time relationship. The CW static recoil pressure-time curve was extrapolated from the static volume-pressure curve of the CW of the normal subjects [14], assuming that this relationship was linear within the tidal volume range [11]. Since the static volume-pressure relationship of the abdominal wall was not measured, $PTP/b,_{Pdi}$ was calculated as referred to Pdi signal baseline. PTP per minute ($PTP/min,_{Pes}$ and $PTP/min,_{Pdi}$) were calculated as $PTP/b,_{Pes}$ and $PTP/b,_{Pdi}$ multiplied by respiratory rate.

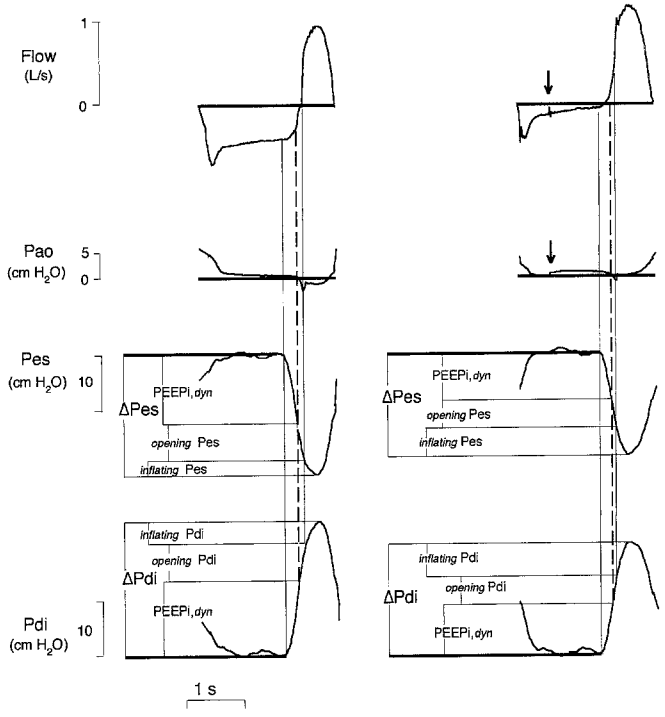

Fig. 1 Representative averaged breaths obtained during flow triggered (*right*) and pressure triggered (*left*) spontaneous ventilation. From top to bottom flow, airway opening pressure (*Pao*) esophageal pressure (*Pes*) and transdiaphragmatic (*Pdi*) signals are shown. *Horizontal solid lines* represent the zero reference for the flow, Pao Pes and Pdi signals. *Vertical solid lines* identify on the Pes and Pdi signal the inspiratory effort produced during the triggering phase *Dotted vertical line* divides the Pes triggering between intrinsic positive end-expiratory pressure ($PEEPi_{,dyn}$) and inspiratory effort to initiate flow from the ventilator (opening Pes and Pdi). The remaining portion of inspiratory deflection (for Pes signal) and inflection (for Pdi signal) represents the amount of effort available to overcome patient's resistance and elastance (inflating Pes and Pdi). During flow-triggering (*right*), *arrows* indicate the positive end-expiratory pressure present in coincidence of the beginning of base flow. See text for further details

Basing on Sassoon's [7] and Giuliani's [15] analysis, on the average tidal swings in Pes and Pdi were identified: (1) $PEEPi_{dyn}$: It was measured as the portion on Pes and Pdi signals encompassed between the onset of the negative deflection (for Pes) and positive inflection (for Pdi) to the point corresponding to the first negative Pao value (respectively $PEEPi_{,Pes}$ and $PEEPi_{,Pdi}$). The corresponding area represented the PTP of the respiratory muscles (PTP-$PEEPi_{,Pes}$) and of the diaphragm (PTP-$PEEPi_{Pdi}$) produced to overcome $PEEPi_{dyn}$ (Fig. 1); (2) opening -Pes and opening -Pdi: The amount of inspiratory muscle effort that had to be produced to activate the trigger mechanism initiating inspiration was identified as the Pes and Pdi portions encompassed between the points corresponding to the first negative Pao value and to the maximum negative Pao values (Fig. 1). PTP-$opening_{,Pes}$ and PTP-$opening_{,Pdi}$ were defined as the corresponding subtended areas; (3) inflating -Pes, and inflating -Pdi: They were identified as the remaining portions of Pes and Pdi and corresponded to the effort produced to inflate the lung overcoming patient's elastance and resistance (Fig. 1). The corresponding PTP-$inflating_{,Pes}$ and PTP-$inflating_{,Pdi}$ were calculated as: PTP-$inflating_{,Pes}$ = PTP/$b_{,Pes}$ − (PTP-$PEEPi_{,Pes}$ + PTP-$opening_{,Pes}$); PTP-$inflating_{,Pdi}$ = PTP/$b_{,Pdi}$ − (PTP-$PEEPi_{,Pdi}$ + PTP-$opening_{,Pdi}$). Pes and Pdi integrals were calculated by referring the different portions of ΔPes and ΔPdi to respectively the CW static recoil pressure-time curve [11, 14] and to the baseline Pdi signal [4].

Results are expressed as mean ± standard error of the mean (SEM). Differences between flow and pressure-triggering were evaluated by using the paired t-test. Statistical significance was defined as a p-value < 0.05.

Results

Causes of ARF, sex, age, days of mechanical ventilation and respiratory parameters obtained on the day of the study in individual patients are shown in Table 1. PaO_2, $PaCO_2$ and pH values immediately before the study and during SB are also indicated.

ΔPes during pressure triggered-SB amounted to 18.54 ± 4.17 cmH_2O (Fig. 2). According to Petrof's defi-

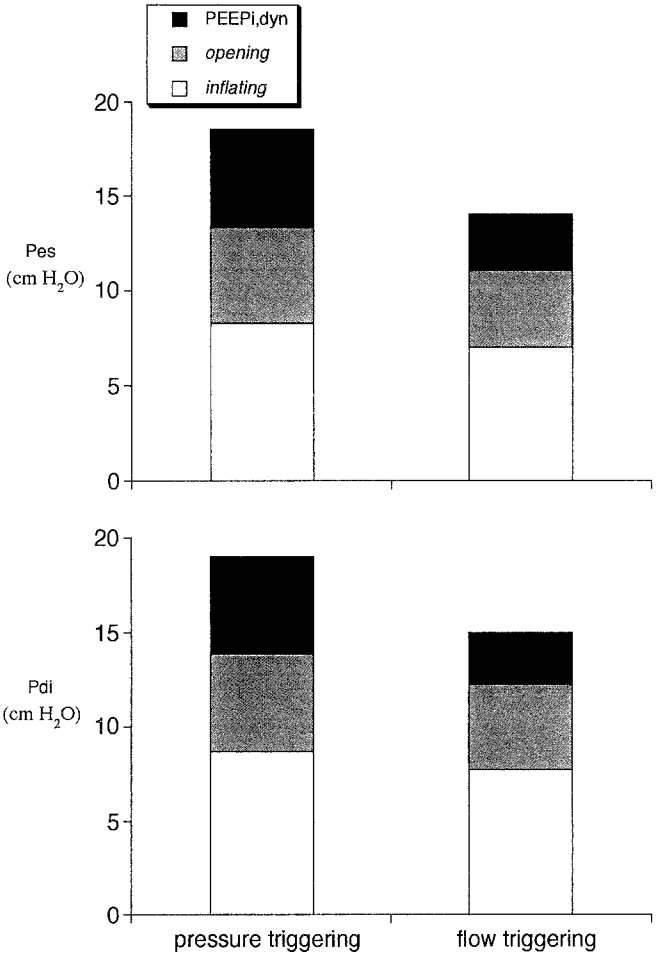

Fig. 2 Comparison between effects of Pressure-triggering and Flow-triggering on dynamic intrinsic positive end-expiratory pressure (*PEEPi, dyn*) and opening and inflating effort of the respiratory muscles (*Pes*) and of the diaphragm (*Pdi*). See text for further details

Table 1 Patients characteristics (Definition of abbreviations: *AVF* acute respiratory failure; *FIO_2* fraction of inspired oxygen mechanical ventilation, *MIP* maximal inspiratory pressure)

Patients no.	Sex	Age (years)	Cause of ARF	FIO_2	PaO_2[a] (mmHg)	$PaCO_2$[a] (mmHg)	pH[a]	MIP (cmH_2O)	Baseline FEV_1/FVC (l)	Days[b]
1	M	67	Exacerbation COPD	0.5	97	48	7.45	−37	0.71/2.11	8
2	M	63	Bronchopneumonia	0.5	92	45	7.41	−38	0.60/2.01	8
3	M	55	Exacerbation COPD	0.4	108	44	7.46	−42	0.31/1.55	11
4	F	58	Exacerbation COPD	0.5	105	45	7.41	−52	0.91/1.69	7
5	M	51	Heart failure	0.5	81	48	7.40	−37	0.95/1.91	9
6	M	62	Exacerbation COPD	0.5	106	47	7.40	−40	0.51/1.41	6

[a] Data obtained during spontaneous breathing
[b] Days on mechanical ventilation

nitions [4], $PEEPi_{dyn}$ was equal to $10.29 \pm 1.22\ cmH_2O$. However, if the inspiratory effort necessary to overcome the pressure-trigger sensitivity was taken into account, $PEEPi_{dyn}$ amounted only to $5.18 \pm 1.66\ cmH_2O$ (Table 2) being the difference ($5.11 \pm 1.08\ cmH_2O$) equal to opening Pes (Fig. 2). During flow triggered-SB, ΔPes amounted to $14.06 \pm 4.45\ cmH_2O$ ($p < 0.001$ versus pressure triggered-SB). Also in this case, because of a relevant opening Pes value (equal to $4.08 \pm 2.01\ cmH_2O$), $PEEPi_{dyn}$, measured according to Petrof's and co-workers [4], could be overestimated up to $7.07 \pm 1.88\ cmH_2O$ (Fig. 2) being the true $PEEPi_{dyn}$ equal to $2.99 \pm 1.80\ cmH_2O$ (Table 2). Similar results were obtained considering the tidal swing in transdiaphragmatic pressure (Fig. 2). Mean breath during pressure triggering (right) and flow triggering (left) SB in a representative patient are shown in Fig. 1. In such patient the portion of pressure used to inflate the lung was larger with flow triggering than with pressure triggering (respectively 37 and 13% of the total ΔPes). Similar results were obtained considering ΔPdi signal. On average, with pressure triggering the inflation pressure represented the $43 \pm 2\%$ of the total ΔPes. Similar findings were observed on ΔPdi.

Table 2 Ventilatory pattern during spontaneous ventilation through pressure and flow ventilator triggering system (Definition of abbreviations: *V_T* tidal volume, *Ti* inspiratory time, *Te* expiratory time, *Ti/Tot* ratio of inspiratory time to total breath cycle duration, *Fr* respiratory frequency, *V_E* minute ventilation, *V_T/Ti* mean inspiratory flow, *$PEEPi_{dyn}$* dynamic intrinsic positive end-expiratory pressure measured from esophageal (*Pes*) and transdiaphragmatic (*Pdi*) pressure)

	Pressure triggering	Flow triggering
V_T (l)	0.42 ± 0.01	0.52 ± 0.03*
Ti (s)	0.61 ± 0.04	0.59 ± 0.07
Te (s)	2.57 ± 0.25	2.54 ± 0.26
Ti/Ttot	0.19 ± 0.05	0.19 ± 0.02
Fr (s^{-1})	19.00 ± 1.52	19.31 ± 1.76
V_E (l/min)	8.46 ± 0.81	10.09 ± 0.88*
V_T/Ti (l/s)	0.65 ± 0.10	0.88 ± 0.06*
Peak flow (l/s)	0.67 ± 0.17	0.77 ± 0.17*
$PEEPi_{dyn}$, Pes (cmH_2O)	5.18 ± 1.66	2.99 ± 1.80*
$PEEPi_{dyn}$, Pdi (cmH_2O)	5.13 ± 2.02	2.82 ± 2.26*

*$p < 0.001$ pressure triggering versus flow triggering

PTP/$B_{,Pes}$, PTP/$b_{,Pdi}$, PTP/$min_{,Pes}$ and PTP/$min_{,Pdi}$ during pressure triggered SB amounted to 14.85 ± 2.19, $14.96 \pm 1.14\ cmH_2O \cdot s$, 282.15 ± 18.10 and $284.24 \pm 19.12\ cmH_2O \cdot s/min$ respectively. A significant ($p < 0.001$) reduction of such indexes of oxygen utilization by the contracting respiratory muscles [13] was observed during flow triggered SB, amounting PTP/$b_{,Pes}$ and PTP/$b_{,Pdi}$ to 7.55 ± 2.21 and $7.70 \pm 3.31 cmH_2O \cdot sec$ and PTP/$min_{,Pes}$ and PTP/$min_{,Pdi}$ to 145.79 ± 15.52 and $148.61 \pm 18.25\ cmH_2O \cdot sec/min$. In Table 3, PTP-PEEPi, PTP-opening, and PTP-inflating values for the respiratory muscles and the diaphragm during pressure triggered and flow triggered SB are shown. During pressure triggered SB, the inspiratory effort of the respiratory muscles to overcome PEEPi and to trigger the ventilator represented the 15.26 ± 2.09 and $17.03 \pm 2.07\%$ of the total muscle effort. Flow triggering significantly ($p < 0.001$) reduced PTP-$PEEPi_{,Pes}$ and PTP-$opening_{,Pes}$ amounting respectively to the 6.22 ± 2.13 and $11.90 \pm 2.22\%$ of the total inspiratory effort. The absolute value of PTP-$inflating_{,Pes}$ during flow triggering was significantly lower than during pressure triggering. However, the relative amount of inspiratory effort available to inflate the lung overcoming patient's elastance and resistance was significantly ($p < 0.001$) larger during flow triggering than during pressure triggering (72.05 ± 2.11 and $81.85 \pm 2.15\%$ respectively). Similar results were obtained considering Pdi instead of Pes (Table 3).

Breathing pattern during pressure and flow triggered SB are shown in Table 2. Compared to pressure triggered, flow triggered-SB significantly ($p < 0.001$) increased V_T and minute ventilation (V_E), peak and mean inspiratory flow, while Ti, Te, Ti/Ttot and respiratory rate remained unchanged.

Table 3 Effects of triggering systems on pressure time product at different phases of inspiratory effort[a] (Definition of abbreviations: *PTP* pressure time product per breath, *Pes* esophageal pressure, *Pdi* transdiaphragmatic pressure, *PEEPi* dynamic intrinsic positive end-expiratory pressure)

	PTP PEEPi, $_{Pes}$ ($cmH_2O \cdot s$)	PTP opening, $_{Pes}$ ($cmH_2O \cdot s$)	PTP inflating, $_{Pes}$ ($cmH_2O \cdot s$)
Pressure triggering	2.32±0.10	2.52±0.10	10.07±3.13
Flow triggering	0.47±0.32*	0.90±0.16*	6.18±2.33*
	PTP PEEPi, $_{Pdi}$ ($cmH_2O \cdot s$)	PTP opening, $_{Pdi}$ ($cmH_2O \cdot s$)	PTP inflating, $_{Pdi}$ ($cmH_2O \cdot s$)
Pressure triggering	2.36±0.12	2.56±0.16	10.04±3.30
Flow triggering	0.51±0.34*	0.88±0.10*	6.29±3.53*

*$p<0.001$ paired t-test pressure versus flow triggering

Discussion

Our data show that 1) ventilator trigger systems may overestimate dynamic measurements of PEEPi of an amount equal to 49±2% during pressure triggered SB, and to 58±3% during flow-triggered SB; 2) inspiratory muscle effort was lower during flow triggered-SB than during pressure triggered-SB bacause of a significant reduction in $PEEPi_{dyn}$, and in effort necessary to overcome trigger sensitivity.

Effects of triggering mechanisms on $PEEPi_{dyn}$ measurements

PEEPi is measured during CMV by means of an end-expiratory occlusion ($PEEPi_{stat}$) [2, 3]. During SB, PEEPi can also be evaluated dynamically ($PEEPi_{dyn}$) by recording the Pes deflection from the onset of inspiratory effort to the onset of flow [4].

In COPD patients with expiratory flow limitation, the existence of time-constant inequalities should lead to varying levels of PEEPi among different portions of the lung, with the highest levels of PEEPi being found in the slowest-emptying alveolar units. The fall in intrathoracic pressure that must be produced to initiate inspiration should represent the lowest PEEPi found in the fastest-emptying alveolar units [5]. Under these conditions, the value of $PEEPi_{stat}$ should represent the *average* level of PEEPi, reflecting alveolar pressure readjustment of dynamic regional volume and pressure differences, while $PEEPi_{dyn}$ indicates the *lowest* regional value of PEEPi in an inhomogeneous lung and should correspond to the minimum level of applied CPAP that will unload the respiratory muscle without causing further hyperinflation (P_{crit}) [16]. In fact, in presence of expiratory flow limitation, applied CPAP higher than P_{crit} will decrease elastic work as PEEPi is counterbalanced in those units with the highest levels of PEEPi, while will cause further hyperinflation and increase in elastance and elastic work in the faster-empty units (with lower levels of PEEPi) [17]. Therefore, the absence of changes in flow-volume events and end-expiratory lung volume with applied CPAP may not entirely exclude the presence of significant regional hyperinflation and risk of barotrauma [18]. Under these circumstances it seems therefore fundamental to correctly assess $PEEPi_{dyn}$ in order to set correct values of CPAP during the weaning phase of COPD patients. Our data show that the overestimation of $PEEPi_{dyn}$ due to the effort in opening the demand valve amounted to 49±2% and 58±3% with respectively pressure and flow triggering. This overestimation was larger ($p<0.001$) with flow than with pressure triggering. This is because with flow triggering, the actual $PEEPi_{dyn}$ is a smaller fraction ($2.99 \pm 1.80\ cmH_2O$) whereas the opening effort is a larger fraction ($4.08 \pm 2.01\ cmH_2O$) of $PEEPi_{dyn}$ measured according to Petrof's method [4]. However, the degree of $PEEPi_{dyn}$ overestimation observed in our patients (all recovering an episode of ARF) may not be extrapolated to that eventually observed in patients during clear episodes of ARF whereby the actual $PEEPi_{dyn}$ value may be the largest part of the $PEEPi_{dyn}$ measured (i.e. from the onset of inspiratory effort the onset of inspiratory flow). Besides, it may also be influenced by the mode of ventilation (e.g. assist-control mode or pressure support vs CPAP) and the set trigger sensitivity. In our patients $PEEPi_{,Pes}$ and $PEEPi_{Pdi}$ were identical (Table 2). This indicate that in our COPD patients expiratory abdominal muscles recruitment was absent [19, 20].

In four of our patients we measured $PEEPi_{stat}$ during a short period of suppression of the respiratory muscle activity obtained by small doses of a short action hypnotic agent (propofol: 8–10 mg/kg) and during CMV with the ventilator setting reproducing the breathing pattern obtained during pressure triggered-SB [18]. Airway occlusion, reproducibly timed to occur coincidentally with end-expiration, was obtained by manipulation of three-way manual valve placed in the inspiratory limb of the external ventilator circuit [21]. We found that $PEEPi_{stat}$ amounted to $10.28 \pm 1.82\ cmH_2O$. Uncorrected $PEEPi_{dyn}$ (i.e. calculated including the inspiratory effort produced to trigger the ventilator) ranged between the 83 and the 63% of $PEEPi_{stat}$ while correct $PEEPi_{dyn}$ (i.e. calculated

without including $Pes_{opening}$) ranged between the 54 and the 67% of the $PEEPi_{stat}$ value. Similar results were obtained during flow triggered SB. These data show that $PEEPi_{dyn}$ measurements that do not take into account $Pes_{opening}$ may significantly overestimate the P_{crit} value, inducing the application of CPAP levels that may cause further hyperinflation. The 50% of the $PEEPi_{stat}$ value measured during CMV reproducing SB breathing pattern, may approximate the $PEEPi_{dyn}$ value, indicating the critical CPAP value above which regional hyperinflation may occur.

Effects of triggering mechanisms on inspiratory effort of COPD patient

Several studies [6, 10, 11, 15, 19] showed that ventilator systems equipped with pressure-triggering system create an inspiratory load which may compromise the weaning process. On the contrary, a relevant reduction in work of breathing has been described when demand flow systems were replaced by continuous flow systems [6, 10, 11]. In normal subjects [6] Sassoon and co-workers found that work of breathing was significantly less with flow triggered CPAP than with pressure-triggered CPAP. In patients with acute respiratory failure Mancebo [22], and Polese [23] confirmed these observations. The delivery of insufficient initial flow may result in an excessive Pao drop and can result in increased inspiratory muscles work [6, 11].

Differences between pressure and flow-triggering should be evaluated based on events during the triggering itself and after the triggering is completed [7]. Facing this assumption with the concept of the equation of motion [24], COPD patients during the triggering phase have to overcome two consecutive inspiratory load namely PEEPi and the set pressure or flow that must be attained at the onset of inspiration for the ventilator to deliver fresh gas into the inspiratory circuit. When the triggering is completed, the patient may inflate the lung overcoming his/her resistance and elastance. Our data indicate that flow-triggering is able to reduce the oxygen consumption of the diaphragm and of the respiratory muscles increasing V_E in spontaneously breathing COPD patients because of its ability to reduce 1) $PEEPi_{dyn}$, 2) inspiratory effort necessary to start inspiration through the ventilator inspiratory circuit (opening effort), and 3) inspiratory effort necessary to inflate the lung overcoming elastance and resistance (inflating effort).

$PEEPi_{dyn}$

During flow triggered SB, a small positive end-expiratory pressure on the Paw signal was evident (Fig. 1) [7]. This is due to the pressure generated by the base flow passing through the ventilator tubing system at the end of patient's expiration when the flow triggering is activated [6]. On average this end-expiratory positive pressure amounted to $2.16 \pm 0.12\ cmH_2O$. Recent works have suggested that in COPD patients with expiratory flow limitation the use of continuous positive airway pressure (CPAP) in spontaneously breathing patients [4, 16, 20] can counterbalance and reduce the inspiratory threshold load imposed by PEEPi without causing further hyperinflation. It is therefore not surprising that the end-expiratory positive pressure present during flow triggered SB was able to partially unload the respiratory muscles and the diaphragm from the additional work imposed by PEEPi. In fact, $PEEPi_{dyn}$ measured during flow triggered SB corresponds to the difference between $PEEPi_{dyn}$ measured during pressure triggered SB minus the end-expiratory positive pressure present during flow triggered SB.

Opening effort

Using a respiratory simulator, Sassoon elegantly analysed the relationship between PTP-opening and the time delay for both pressure and flow-triggering [7]. She found that for a given time delay, initial inspiratory effort during pressure and flow-triggering was similar. However, because the total time delay with flow-triggering was relatively shorter then with pressure-triggering, PTP-opening would be less with flow than with pressure-triggering [7]. Our data confirm Sassoon's observation in a sense that time delay during pressure-triggered SB was significantly ($p < 0.01$) longer than during flow-triggered SB (0.08 ± 0.02 vs 0.04 ± 0.01 sec). Besides, we found that when our COPD patients initiated the inspiratory efforts able to reduced Pao below atmospheric pressure, the pressure trigger showed a significantly greater Pao drop as compared to the flow trigger (respectively 2.83 ± 0.40 vs $1.40 \pm 0.16\ cmH_2O$), the Pao drop exceeding in all patients the pre-set pressure sensitivity [6, 11]. As a consequence, opening -Pes and opening -Pdi were significantly smaller with flow than with pressure-triggering (Fig. 2). The reduction in PTP-opening with flow-triggering observed in our patients may indeed be attributed to a shorter time delay and to a smaller negative deflection in Pes during the triggering phase.

Inflating effort

Differences in pressure and flow-triggering on work of breathing and PTP are also related to events after the triggering has been completed [7]. This is a result of the insufficient flow delivery during pressure-triggering. On the contrary with flow-triggering, immediately after the trigger sensitivity is attained, Pao increases and is maintained above atmospheric pressure throughout inspiration, act-

ing as a small inspiratory pressure assist [6, 7]. Our data confirm these observations. In fact, we found that during flow-triggered SB, mean and peak inspiratory flow significantly increased (Table 2). It seems therefore reasonable to assume that patient's inspiratory demand could be better satisfied during flow than during pressure triggering. In our patients we found that absolute values of PTP-inflating$_{Pes}$ and PTP-inflating$_{Pdi}$ were higher during pressure than during flow triggered SB (Table 3). However, the relative amount of inspiratory effort available to inflate the lung was higher during flow than during pressure SB. In other words a larger percentage of the total inspiratory effort was available to produce tidal volume during flow triggered SB. Therefore, with flow triggering, a larger proportion of pressure generated by the respiratory muscles is used to overcome the elastic and resistive components of the respiratory system when compared to pressure triggering. This explain why tidal volume was larger with flow triggering (Table 2). These data therefore show that flow triggering reduces the relative amount of "isometric work" (i.e. inspiratory effort necessary to overcome PEEPi and to trigger the ventilator that do not produce relevant changes in lung volume, ignoring the minimal inspiratory flow that follows gas decompression) while increase the relative amount of "mechanical work" (i.e. the inspiratory effort necessary to overcome elastance and resistance of the respiratory system and that produces concomitant changes in tidal ventilation). We can hence assume that the same results may be achieved in pressure triggering systems by adding a small PEEP and a small pressure assist.

In conclusion our data show that in order to measure PEEPi$_{dyn}$ and identify the critical level of CPAP that can be applied in COPD patients without causing further hyperinflation, the inspiratory effort produced to overcome PEEPi and to trigger the ventilator must be discriminated. Application of flow triggering requires less effort to initiate inspiration and provide a positive end-expiratory pressure level that is able to unload the respiratory muscles by reducing PEEPi. Consequently, with flow triggering higher minute ventilation are obtained in COPD patients during the weaning phase.

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