Impact of Expiratory Trigger Setting on Delayed Cycling and Inspiratory Muscle Workload

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Rationale: **During pressure-support ventilation, the ventilator cycles into expiration when inspiratory flow decreases to a given percentage of peak inspiratory flow ("expiratory trigger"). In obstructive disease, the slower rise and decrease of inspiratory flow entails delayed cycling, an increase in intrinsic positive end-expiratory pressure, and nontriggering breaths.**

Objectives: **We hypothesized that setting expiratory trigger at a higher than usual percentage of peak inspiratory flow would attenuate the adverse effects of delayed cycling.**

Methods: **Ten intubated patients with obstructive disease undergoing pressure support were studied at expiratory trigger settings of 10, 25, 50, and 70% of peak inspiratory flow.**

Measurements: **Continuous recording of diaphragmatic EMG activity with surface electrodes, and esophageal and gastric pressures with a dual-balloon nasogastric tube.**

Main Results: **Compared with expiratory trigger 10, expiratory trigger 70 reduced the magnitude of delayed cycling (0.25 ± 0.18 vs. 1.26 ± 0.72 s, $p < 0.05$), intrinsic positive end-expiratory pressure (4.8 ± 1.9 vs. 6.5 ± 2.2 cm H_2O, $p < 0.05$), nontriggering breaths (2 ± 3 vs. 9 ± 5 breaths/min, $p < 0.05$), and triggering pressure–time product (0.9 ± 0.8 vs. 2.1 ± 0.7 cm $H_2O \cdot s$, $p < 0.05$).**

Conclusions: **Setting expiratory trigger at a higher percentage of peak inspiratory flow in patients with obstructive disease during pressure support improves patient–ventilator synchrony and reduces inspiratory muscle effort. Further studies should explore whether these effects can influence patient outcome.**

Keywords: chronic obstructive pulmonary disease; cycling; expiratory trigger; mechanical ventilation; pressure support

During pressure-support ventilation, the ventilator cycles from inspiration to expiration when inspiratory flow decreases to a given percentage of peak inspiratory flow, a set-point known as "expiratory trigger" (ET) (1). On most available intensive care ventilators, ET is usually set at 25%. However, cycling should ideally coincide with the end of the patient's inspiratory effort, a condition that is not always met in patients with abnormal respiratory mechanics (2, 3). Pressurization by the ventilator beyond the end of the patient's inspiratory phase, known as delayed cycling, has been observed in the presence of obstructive airway disease (4). In these patients, delayed cycling can worsen dynamic hyperinflation and intrinsic positive end-expiratory pressure ($PEEP_i$), increase the number of nontriggering breaths and promote patient–ventilator asynchrony, increase the work of breathing (2, 5), and interfere with breathing pattern (6). Theoretically, a higher value of ET should allow better matching between the patient's and the ventilator's inspiratory-phase durations (7). However, whereas the beneficial effects of adjusting ET in patients with mainly restrictive respiratory mechanics have been documented (8, 9), so far no study has evaluated the impact of such an approach in intubated patients with obstructive airway disease.

The purpose of this study was to test the hypothesis that, in such patients, increasing the ET set-point could reduce the magnitude of $PEEP_i$, which in turn would decrease the number of nontriggering breaths as well as the level of inspiratory effort and triggering delay.

The preliminary results of this study were presented in abstract form at the annual meeting of the American Thoracic Society in May 2004 (10).

METHODS

The study was performed in the medical intensive care unit of the Geneva University Hospital. The protocol was accepted by the ethics committee of our institution. Patients meeting commonly accepted diagnostic criteria of chronic obstructive pulmonary disease (COPD) (11) and consecutively intubated and mechanically ventilated for acute respiratory failure were included. Informed consent was obtained from all patients. Patients were excluded if any of the following were present: $F_{I_{O_2}} > 0.5$ to maintain $Sp_{O_2} > 88\%$, respiratory rate > 35 breaths/min, or impaired consciousness.

Measurements were performed during the weaning period, as soon as possible after the patients were switched from controlled mechanical ventilation to pressure support, once sedation had been stopped and the patients' respiratory parameters were stable (variation of ⩽ 15% in respiratory rate and ⩽ 5% for Sp_{O_2} with $Sp_{O_2} \geqslant 88\%$, over 15 min). The ventilator used was a Galileo Gold (Hamilton Medical, Rhäzuns, Switzerland), on which ET can be adjusted from 5 to 70%. No other cycling criteria, such as time or pressure, are available on this machine (other than the opening of the expiratory valve if airway pressure reaches the set value of the high-pressure alarm). Tracheal suctioning was applied before protocol initiation. The initial ventilator settings were those made by the clinician in charge of the patient, following our usual practice guidelines: inspiratory trigger (flow trigger) set at maximum sensitivity ($0.5\ L \cdot min^{-1}$), pressure support titrated to obtain an 8 to 10 $ml \cdot kg^{-1}$ V_T, pressurization slope 200 ms, and ET at its default setting of 25%. The number of nontriggering breaths per minute was measured in the absence of external PEEP, which was then titrated by 1–cm H_2O increments until nontriggering breaths disappeared or a maximum PEEP of 10 cm H_2O was reached. If ineffective attempts persisted, pressure support was reduced (12) by 2 cm H_2O, provided V_T remained at 8 $ml \cdot kg^{-1}$ or greater. No further setting changes were made if nontriggering breaths were still present despite these measures.

Thereafter, ET was sequentially and randomly set at 10, 50 and 70%, for 15-min periods each. ET 10, which should be expected to favor delayed cycling, and is therefore unlikely to be used by clinicians, might seem irrelevent. However, it was tested because some intensive care ventilators have a default and unmodifiable ET in that range. All other settings were maintained at their original level.

Measurement Techniques

Patients were equipped with a nasogastric tube with esophageal and gastric balloons (Guenard ch 16 nasogastric tube; Marquat Génie Biomédical, Boissy-Saint-Léger, France), the correct position of which was verified by the occlusion method (13). Esophageal (Pes) and gastric pressures (Pga) were continuously recorded by differential pressure transducers (Biopac Systems, Inc., Goleta, CA), digitized at a sampling

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rate of 1,000 Hz, and the transdiaphragmatic pressure (Pdi) curve obtained by subtracting the Pes signal from the Pga signal.

Diaphragmatic EMG activity (EMGd) was recorded with surface electrodes placed bilaterally over the costal margin, one reference electrode being placed over the sternum (14). The analog signal was first filtered and digitized at a sampling rate of 1,000 Hz by means of the Biopac EMG module, then rectified and integrated.

Measured and Computed Variables

Data were continuously recorded during the last 5 min at each ET setting and stored for subsequent analysis (AcqKnowledge software; Biopac Systems, Inc.). Then, a 1-min period devoid of artifacts (cough, esophageal spasm, patient movement) was selected from the 5-min tracing. For each variable, the reported results are the average of all breaths during that 1-min period. The following parameters were determined:

1. Ventilatory profile. Patient inspiratory effort duration (ti), measured as the interval between the initial positive deflection of Pdi and the initial rapid decrease of electrical activity on the processed EMGd signal (7, 15). The choice of using Pdi to determine the onset of ti was dictated by the difficulty with which EMGd reflects the initiation of inspiratory effort, due to the potential interferences from background noise and/or neuromuscular uncoupling. On the other hand, accuracy is not lost with regard to determining the end of patient inspiratory effort; respiratory rate indicated by the ventilator (RR_{vent}); patient respiratory rate (RR_{pat}); ineffective (nontriggering) inspiratory efforts: $IE_{ineff} = RR_{pat} - RR_{vent}$; V_T and $\dot{V}_E$: obtained by analysis and integration of the $\dot{V}$ tracing; trigger delay (td): the time interval between the initial Pdi deflection and the start of pressurization by the ventilator; ventilator duty cycle ($Vent_{Ti/Ttot}$), where Ti is the duration of inspiratory flow, Ttot that of inspiratory and expiratory flows; delayed cycling: expressed as ti_{excess}, which is the difference between the end of patient inspiratory effort and the duration of pressurization by the ventilator; premature cycling: interruption of pressurization by the ventilator before the end of patient inspiratory effort. Occurrence of premature cycling at each ET level was quantified as the number of events over the 1-min evaluation period. A representative tracing of the recordings from one patient is shown in Figure 1.
2. Respiratory mechanics. Expiratory time constant (RC_e): slope of the expiratory flow–volume curve (16); peak expiratory airway resistance (Rexp): from the equation Rexp = (Pes − Paw)/peak expiratory flow, where Paw = airway pressure; $PEEP_i$: the difference between the value of Pes at the start of inspiration and that when instantaneous flow ($\dot{V}$) reaches 0. The value of $PEEP_i$ was corrected for expiratory muscle activity (17).
3. Inspiratory workload. Due to the presence of numerous nontriggering breaths despite the initial ventilator setting optimization, only those indices independent of flow were used. Pdi = Pga − Pes; Pdi–time product (PTPdi) = area under the Pdi tracing during patient ti (as defined above, i.e., the interval between the initial Pdi deflection and the initial rapid decrease of electrical activity on the processed EMGd signal); triggering pressure–time product (PTPt), which estimates the magnitude of inspiratory effort required to trigger the ventilator: area under the Pes tracing between the initial Pes deflection and the onset of pressurization by the ventilator.

Arterial blood gases were measured immediately before protocol initiation and at the end of the ET modification sequence. Finally, systemic pressure, measured by an indwelling radial artery catheter, and pulse oxymetry were monitored continuously during the protocol.

Data from the 1-min period were averaged, and are expressed as mean ± SD. Comparisons were made with an analysis of variance on ranks for repeated measurements and Dunn's test. A p value of less than 0.05 was considered significant.

RESULTS

Ten patients were included in the study, whose main clinical characteristics are summarized in Table 1. All patients had severe obstructive mechanics, as reflected by the results of pulmonary function tests and expiratory time constants. Peak expiratory resistance was 30 ± 12 cm $H_2O \cdot l^{-1} \cdot s^{-1}$. Patients were ventilated with 14.3 ± 3 cm H_2O of pressure support, which resulted in a V_T of 9 ± 2 ml/kg (mean ± SD). Baseline Pa_{O_2}/FI_{O_2} and Pa_{CO_2} were 230 ± 15 and 50 ± 4 mm Hg, respectively (mean ± SD). All patients exhibited signs of poor patient–ventilator synchrony, as shown by the presence of nontriggering breaths, increased trigger delay, and delayed cycling (Table 2).

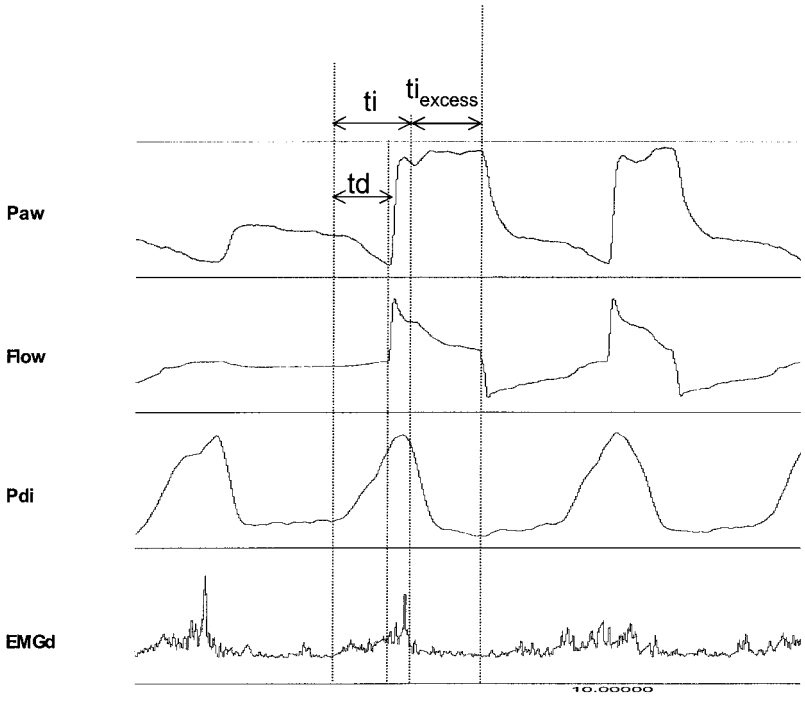

Figure 1. Representative tracing in one patient of airway pressure (Paw), Flow, transdiaphragmatic pressure (Pdi), and rectified diaphragmatic EMG recording (EMGd) showing pertinent measured intervals: triggering delay (td), duration of inspiratory effort (ti), duration of pressurization by the ventilator extending beyond that of inspiratory effort (ti_{excess}).

TABLE 1. MAIN CHARACTERISTICS OF THE PATIENTS

Patient No.	Age (*yr*)	M/F	BW (*kg*)	$FEV_{1.0}$ (*% predicted*)	FI_{O_2}	RC (*s*)	PS Level (*cm H_2O*)	PEEPe	ICU Days	Cause of ARF
1	77	M	55	26	0.25	3.6	11	7	3	Pneumonia
2	57	M	65	30	0.25	2.6	14	7	5	Undetermined
3	65	M	47	29	0.30	2.4	12	10	4	Acute bronchitis
4	77	M	60	34	0.40	1.8	13	6	7	Acute bronchitis
5	78	M	67	30	0.25	2.1	14	5	10	BZP overdose
6	71	M	60	29	0.40	2.1	12	5	3	LVF
7	77	F	56	38	0.40	1.9	15	10	5	Undetermined
8	75	F	57	32	0.40	2.1	12	5	4	Acute bronchitis
9	60	M	68	35	0.35	2.0	18	8	6	Acute bronchitis
10	50	F	50	32	0.35	2.0	22	10	9	Pneumothorax
Mean (SD)	68.7 (9.6)	58.5 (6.6)	31.5 (3.5)	0.34 (0.10)	2.0 (0.8)	14.3 (3.2)	7.3 (2.0)	5.6		

Definition of abbreviations: ARF = acute respiratory failure; BW = bodyweight; BZP = benzodiazepine; $FEV_{1.0}$ = last documented value of 1-s FEV, expressed as % of predicted value; ICU days = elapsed time in the intensive care unit on the day the protocol was performed; LVF = left ventricular failure; M/F = male/female; PS level = level of pressure support above PEEPe; PEEPe = level of externally applied positive end-expiratory pressure; RC = time constant of the respiratory system.

The influence of modifying the level of ET on the patients' ventilatory pattern and patient–ventilator synchrony are shown in Table 2. Predictably, increasing ET reduced the duration of inspiration and increased that of expiration, as reflected by the decrease in $Vent_{Ti/Tot}$, and ti_{excess}. Of note, RR_{pat} and inspiratory effort duration remained unchanged. A representative tracing of ventilatory pattern changes at ET 10, 25, and 70 in one patient is shown in Figure 2. At ET 70, nontriggering breaths were absent in seven patients (Figure 3). V_T was lower at ET 50 and ET 70 than at ET 25, but minute volume remained unchanged (Table 2). $PEEP_i$, Td, and PTPt decreased as ET was increased (Tables 2 and 3). PTPdi of all breaths was unchanged over the range of ET 10 to ET 70 (Table 3). At ET 10, however, both Pdi and PTPdi were higher than at all other ET settings (Table 3). Individual patient tracings are shown in Figures 3 through 6. Nontriggering breaths had lower Pdi and PTPdi than triggering breaths at all ET levels (Table 3). No signs of premature cycling were documented at the ET 50 and ET 70 settings. Pa_{O_2}/FI_{O_2} and Pa_{CO_2} after the last ET change were 236 ± 12 and 49 ± 5 mm Hg, respectively (mean ± SD).

DISCUSSION

To summarize, the results of this study show that, in intubated patients with COPD undergoing pressure-support ventilation and presenting signs of poor synchrony with the ventilator, increasing the ET setting to greater than 25% of peak inspiratory flow had the following consequences:

1. There was a decrease in the duration of pressurization by the ventilator and a decrease in delayed cycling without the occurrence of premature cycling.
2. There was a reduction in $PEEP_i$.
3. There was improved triggering of the ventilator, in the form of a reduction in trigger delay, magnitude of inspiratory effort to trigger the ventilator, and number of nontriggering breaths. This improved patient–ventilator synchrony occurred without changes in patient inspiratory and expiratory time or increase in the metabolic cost of breathing (as shown by the absence of change in PTPdi) or alveolar ventilation (as shown by the absence of change in minute volume and Pa_{CO_2}).

TABLE 2. PATIENTS' RESPIRATORY PATTERN AND PATIENT–VENTILATOR SYNCHRONY AT VARIOUS EXPIRATORY TRIGGER SETTINGS

	ET 10	ET 25	ET 50	ET 70
RR_{vent}, breaths/min	14.8 ± 4.8	16.2 ± 3.9*	18.6 ± 3.9†	22.2 ± 5.2‡
RR_{pat}, breaths/min	23.7 ± 6.1	23 ± 5.2	23.8 ± 5.7	24.4 ± 6.3
IE_{ineff}, breaths/min	9.1 ± 5.5	6.9 ± 4.4*	4.9 ± 5.1†	2.1 ± 3‡
V_T, L	0.620 ± 0.2	0.611 ± 0.25	0.55 ± 0.25†	0.46 ± 0.19‡
$\dot{V}_E$, L/min	8.8 ± 3	9.4 ± 3.5	10.5 ± 4.5*	9.7 ± 3.1
ti, s	0.73 ± 0.25	0.72 ± 0.25	0.70 ± 0.23	0.68 ± 0.23
te, s	1.82 ± 0.71	1.87 ± 0.41	1.82 ± 0.3	1.77 ± 0.5
ti_{excess}, s	1.26 ± 0.72	0.87 ± 0.37*	0.51 ± 0.22†	0.25 ± 0.18‡
td, s	0.53 ± 0.28	0.48 ± 0.24*	0.42 ± 0.19†	0.37 ± 0.20‡
Ti/Ttot, %	34 ± 7	29.2 ± 6.7*	25.8 ± 7†	22 ± 6.4‡
$PEEP_i$, cm H_2O	6.5 ± 2.2	5.4 ± 2*	4.7 ± 2†	4.8 ± 1.9†

Definition of abbreviations: ET = expiratory trigger; IE_{ineff} = ineffective inspiratory efforts ($IE_{ineff} = RR_{pat} - RR_{vent}$); RR_{pat} = patient respiratory rate; RR_{vent} = respiratory rate indicated by the ventilator; td = trigger delay (time between onset of ti and start of pressurization by the ventilator); te = neural expiratory duration (interval between the end of ti and the onset of the next ti); ti = duration of patient inspiratory effort; ti_{excess} = duration of pressurization by the ventilator extending beyond the end of ti; Ti/Ttot = ventilator duty cycle.

Values expressed as mean ± SD.

* $p < 0.05$ versus ET setting (ET) 10.

† $p < 0.05$ versus ETS 10 and 25.

‡ $p < 0.05$ versus ETS 10, 25, and 50.

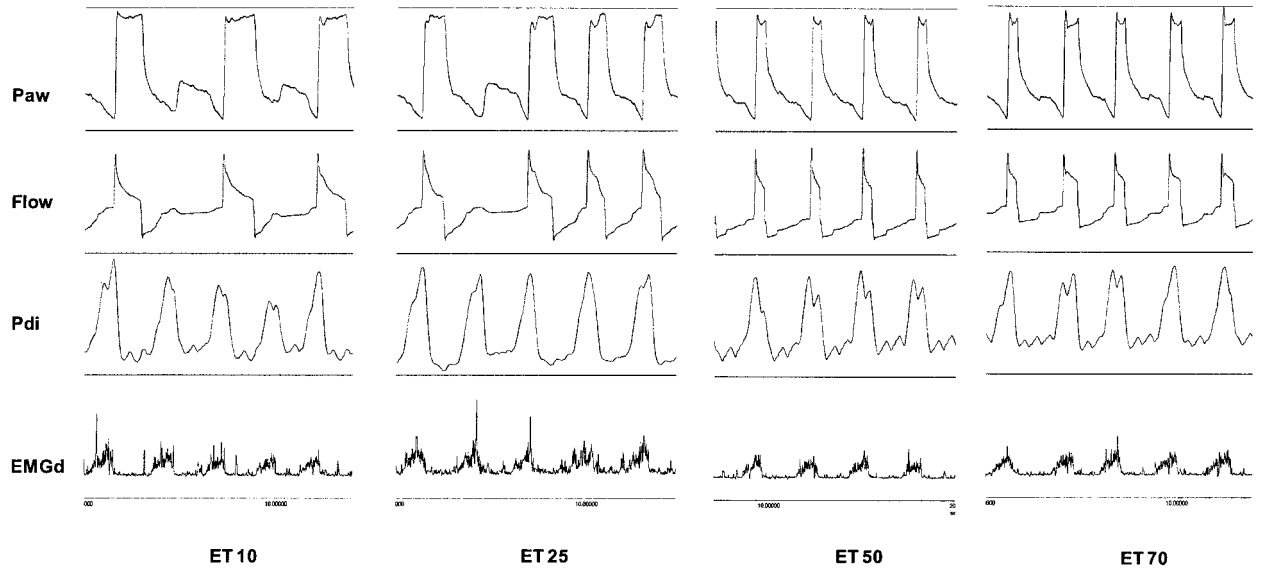

Figure 2. Representative tracing in one patient of Paw, Flow, Pdi, and rectified EMGd changes over several breaths at expiratory trigger (ET) settings of 10, 25, and 70% of peak inspiratory flow.

4. Finally, decreasing the ET setting to less than 25% led to a worsening of parameters reflecting patient–ventilator asynchrony.

Before discussing these results further, some limitations of the study should be addressed. First, our study was conducted in a small group of patients. Nonetheless, as reflected by the individual tracings, there was a very similar interpatient response to the changes in ET, suggesting that our patients were representative of at least a certain category of intubated patients with COPD. Second, patients had severe obstructive mechanics, as shown by their pulmonary function tests and respiratory system time constants, and presented signs of poor synchrony with the ventilator. Therefore, our results might not apply to patients with less severe obstruction and no signs of asynchrony. Third, the duration of inspiratory effort is difficult to determine in obstructive disease with dynamic hyperinflation, due to a variable degree of neuromuscular uncoupling. We used the EMGd signal, a validated approach (15), but one which carries limitations as to the quality of the tracing due to artifacts stemming from cardiac electrical activity background noise, or expiratory muscle activity. However, cardiac artifact and expiratory muscle activation (appearing as a secondary burst of electrical activity occurring immediately after diaphragmatic activation) are easy to identify and exclude from ti measurement. Background noise, on the other hand, can impede proper EMGd interpretation, thus requiring analysis of the Pdi tracing to determine ti. Fourth, quantifying the magnitude of inspiratory effort can prove very difficult, as the work of breathing cannot be interpreted properly in the presence of nontriggering breaths. Indeed, in the classical approach, only triggering breaths are used to compute the work of breathing, even though nontriggering breaths also require energy. For these reasons, only those indices of inspiratory workload that are independent of flow (thereby including nontriggering breaths) have been used in the present study. Finally, patients remained at each ET setting for only 15 min, and steady-state conditions might not have been achieved. However, the duration was in line with that of two recent similar studies on the effects of cycling criteria modifications (8, 9). Nonetheless, this short time frame still leaves open the question of whether the observed effects are long lasting, and whether any additional effects might be observed over a longer time period.

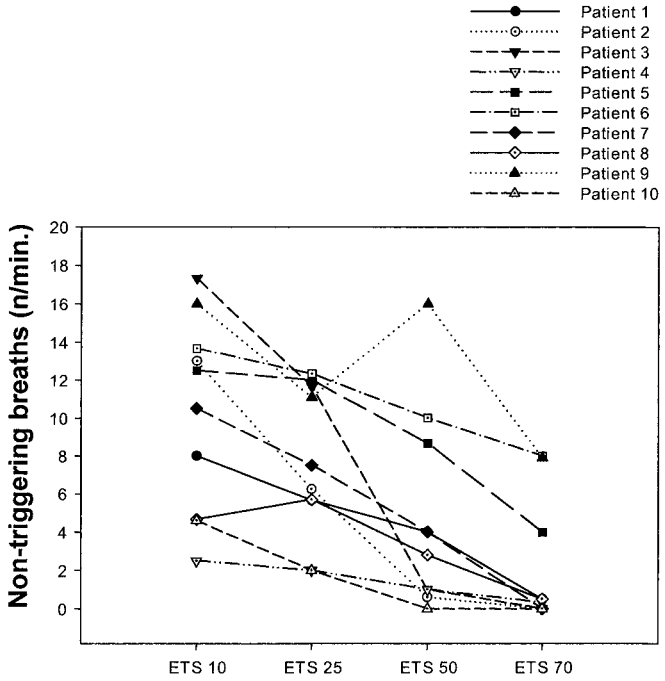

Figure 3. Individual variations in the number of nontriggering breaths at each of the four ET settings (10, 25, 50, and 70% of peak inspiratory flow rate).

Effects of Increasing ET

As predicted, increasing ET from 10 to 70% of peak inspiratory flow led to a decrease in the duration of inspiration and an increase in that of expiration, the earlier interruption of pressurization leading to a reduction of V_T. $PEEP_i$ was also reduced, likely as a consequence of both these mechanisms. In turn, the reduction in $PEEP_i$ induced less effort to trigger the ventilator,

TABLE 3. PATIENTS' INSPIRATORY EFFORT

	ET 10	ET 25	ET 50	ET 70
Pdi, cm H_2O				
All	6.8 (4.2–8.8)	5.5 (3.9–8.1)	6.6 (4.8–7.6)	6.6 (4.4–8.4)
Triggering*	7.6 (5.9–9.5)	6.4 (4.2–8.2)§	6.5 (4.6–7.7)§	6.6 (4.4–8.4)§
Nontriggering†	4.1 (3.4–6.5)‡	4 (2.8–5.5)‡	4.5 (3.7–5)‡	6.5 (4.5–8)¶
PTPdi, cm $H_2O \cdot s$				
All	3.9 (2.2–6.19)	3.8 (1.4–7.4)	4 (2.3–5.2)	4.4 (2.2–7.1)
Triggering	6 (3–7)	6.4 (1.1–7.7)	4.5 (2.6–6.2)§‖	4.4 (2.2–7.1)§‖
Nontriggering	2.5 (1.3–4.4)‡	1.8 (1.09–4.1)†	2 (1.6–2.5)‡	2.1 (1.4–2.9)¶
PTPt, cm $H_2O \cdot s$				
Triggering	2.09 (1.3–2.8)	2.1 (0.5–3)	1.5 (0.9–1.9)§‖	0.9 (0.7–1.8) §‖

Definition of abbreviations: ET = expiratory trigger; Pdi = transdiaphragmatic pressure; PTPdi = transdiaphragmatic pressure-time product; PTPt = triggering pressure-time product.

Values expressed as median (25–75% confidence interval). Analysis of variance on ranks.

* Inspiratory efforts succeeding triggering the ventilator.

† Inspiratory efforts not succeeding in triggering of the ventilator.

‡ $p < 0.05$ versus triggering breath at same ET.

§ $p < 0.05$ versus ET setting (ETS) 0.10.

‖ $p < 0.05$ versus ETS 0.25.

¶ Data from three patients only (the seven other patients had no ineffective inspiratory attempts).

a lower trigger delay, and a decrease in the number of nontriggering breaths. The ET 70 level provided the best results, in line with mathematical predictions for the degree of airway obstruction witnessed in our patients (3, 7). Of note, at that setting, the magnitude of delayed cycling was 0.25 s (Table 3), which is the optimal range of 0.2 to 0.4 s that one would expect. Indeed, cycling should not occur immediately on cessation of neural inspiratory effort, but slightly after that, due to persistent inspiratory muscle activity after neural ti ceases (18). Nonetheless, ET 70 should not be considered as an optimal setting in all patients with obstructive disease, nor should one infer that, in obstructive disease, the higher the ET the better. Indeed, increasing ET above a certain level carries the risk of an excessive reduction in VT stemming from premature cycling. Illustrating this point, Tokioka and colleagues (8) observed premature cycling in four of eight patients with acute lung injury when ET was increased to 45%. Of interest, expiratory time (te) remained unchanged at the various ET settings in our study, as opposed to the findings of two studies in which te increased in the presence of delayed cycling (6, 19). However, Kondili (19) studied patients with acute lung injury, COPD being an exclusion criteria, which makes any comparison speculative. Younes and colleagues (6) studied a heterogeneous group of 50 patients, 45 of whom exhibited an increase in te in the presence of delayed cycling. The five patients in whom te did not increase had COPD, which is in line with our findings, suggesting that, in these patients, modifying the ventilator's inspiratory duration has little effect on neural te. Increasing te

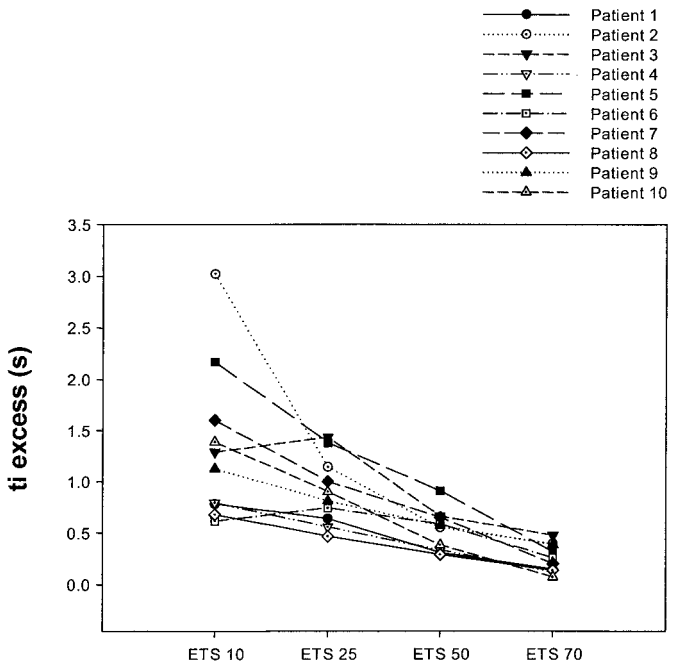

Figure 4. Individual variations in excess duration of pressurization by the ventilator relative to patient's inspiratory time (ti_{excess}) at each of the four ET settings (10, 25, 50, and 70% of peak inspiratory flow rate).

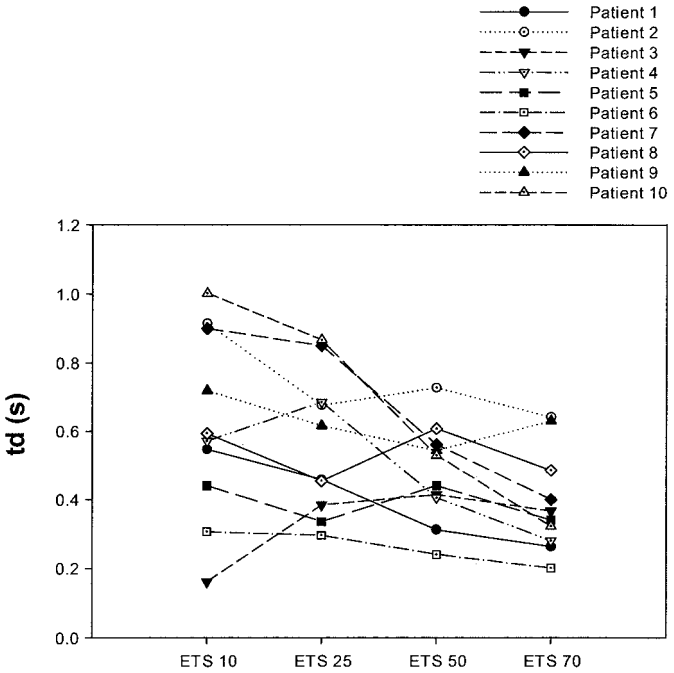

Figure 5. Individual variations in trigger delay (td) at each of the four ET settings (10, 25, 50, and 70% of peak inspiratory flow rate).

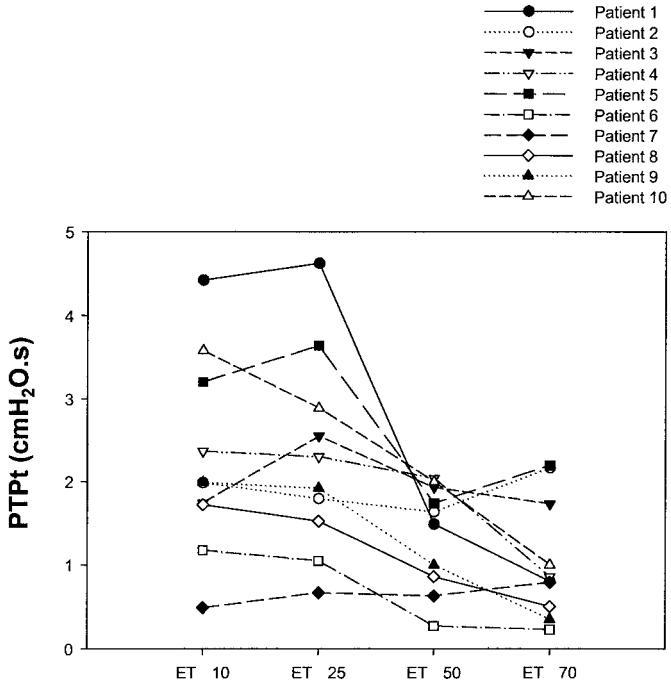

Figure 6. Individual variations in the magnitude of inspiratory effort required to trigger the ventilator, expressed as the trigger pressure-time product (PTPt) at each of the four ET settings (10, 25, 50, and 70% of peak inspiratory flow rate).

is probably a strategy for minimizing dynamic hyperinflation, and in those patients in whom it increased in the two studies cited, te reached 1.3 to 1.9 s. In patients with COPD, te is already in the upper portion of this range, as seen from our results (Table 3), and therefore might have reached its upper limit before any change in the ventilator setting is applied. Furthermore, one of the mechanisms by which te increases in this situation is the Hering Breuer expiratory promoting reflex, which has been shown to be weaker in patients with COPD (20) .

In our study, VT decreased as ET was increased, but minute volume remained unchanged, due to the decrease in the number of nontriggering breaths. According to mathematical predictions, for an unvarying inspiratory time, the optimal level of ET depends on the severity of obstructive disease, the level of pressure support, and the magnitude of inspiratory effort (3, 7). Our results point to the need of increasing ET in at least some patients with obstructive disease, but not systematically in all such patients, nor to a fixed level. Indeed, such an approach could lead to premature cycling, resulting in insufficient VT, increased respiratory drive, and worsening patient–ventilator asynchrony. Of note, no patient presented signs of premature cycling, even at the ET 70 level.

The impact of increasing ET on the work of breathing remains uncertain. Indeed, no effect on the magnitude of Pdi and PTPdi was documented whatever the ET level, at least when triggering and nontriggering breaths are considered together. The only change observed was an increase in the Pdi and PTPdi of triggering breaths at ET 10, which is not surprising considering the mathematical predictions and preliminary results observed in patients with COPD (3, 7). Of note, the Pdi and PTPdi of nontriggering breaths were lower than that of triggering breaths. These findings differ from those of Leung and colleagues (12), who documented higher levels of inspiratory efforts during such breaths. The difference could stem from methodologic differences, or from the impact of changing the ET versus modifying the level of pressure support as did Leung and coworkers. Another explanation could stem from the findings of these authors that nontriggering breaths usually follow cycles with higher levels of pressure support and VT, entailing higher levels of dynamic hyperinflation (12). One possible explanation is that the latter could in turn lead to a worsening of neuromuscular uncoupling, whereby a given level of central respiratory drive results in a weaker force generation by the diaphragm (18). However, in the absence of any measurement of central respiratory drive, this explanation remains speculative. In any case, it should be noted that the absence of change in the PTPdi at a higher ET setting when all breaths are considered is most likely the result of the decrease in the number of nontriggering breaths. Indeed, these breaths are associated with a lower PTPdi, and a decrease in their number impacts on the mean value for all breaths.

Clinical Implications

All patients presented with nontriggering breaths and signs of poor patient–ventilator synchrony, despite prior optimization by the clinician of pressure support and external PEEP settings. Although no significant effect of increasing ET was observed in terms of reducing the magnitude of inspiratory efforts, clear signs (decreased level of $PEEP_i$, ventilator triggering effort, and number of nontriggering breaths) of improved patient–ventilator synchrony were documented. Of note, the reduction in pressurization time while minute volume was maintained due to a reduction in the number of nontriggering attempts supports the concept that improved synchrony results in a more efficient delivery of pressure support. These effects are likely to lead to an overall reduction in the inspiratory workload over time, and one might hypothesize that the same measurements performed over a longer period (several hours) might have yielded more clear-cut results in that respect. Nonetheless, at this stage, although it seems logical to follow the path of increasing ET in patients with obstructive disease, we still need to explore various aspects of this complex problem. Indeed, although adjusting ET could provide the clinician with an additional tool to improve patient–ventilator interactions and perhaps shorten the duration of weaning, it could add another difficult setting to titrate in a ventilator mode, which has become more complex and demanding over the years (21, 22). To justify this added complexity, we need further studies to determine which patients are more likely to benefit from increased ET, what is the desirable range of settings, and what, if any, are the consequences on patient outcome. Automatic adjustment of ET may provide part of the answer to that problem in coming years (23).

To conclude, adjusting ET to higher levels in intubated patients with COPD undergoing pressure-support ventilation led to a lower level of $PEEP_i$, a decreased number of nontriggering breaths, and a reduced magnitude of inspiratory effort to trigger the ventilator. These results should encourage further studies aimed at exploring whether such effects are long-lasting and whether they can favorably influence patient outcome.

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