Intensive Care Med (1995) 21:547–553

ORIGINAL

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P0.1 is a useful parameter in setting the level of pressure support ventilation

Received: 20 December 1993
Accepted: 2 June 1994

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Abstract *Objective:* The purpose of this study was to investigate whether changes in breathing pattern, neuromuscular drive (P0.1), and the work involved in breathing might help to set the individual appropriate level of pressure support ventilation (PSV) in patients with acute respiratory failure (ARF) requiring ventilatory assistance.
Design: A prospective, interventional study.
Setting: An 8-bed multidisciplinary intensive care unit (ICU).
Patients: Ten patients with ARF due to adult respiratory distress syndrome (ARDS), sepsis or airway infection were included in the study. Chronic obstructive pulmonary disease (COPD) patients with acute exacerbation were excluded. None of these patients was in the weaning process.
Interventions: We found a level of pressure support able to generate a condition of near-relaxation in each patient, as evidenced by work of breathing (WOB) values close to 0 J/l. This level was called PS100 and baseline physiological measurements, namely, breathing pattern, P0.1 and WOB were obtained. Pressure support was then reduced to 85%, 70% and 50% of the initial value and the same set of measurements was obtained.
Measurements and results: Flow ($\dot{V}$) was measured by a flow sensor (Varflex) positioned between the Y-piece of the breathing circuit and the endotracheal tube. Tidal volume was obtained by numerical integration of the flow signal. Airway pressure (P_{aw}) was sampled through a catheter attached to the flow sensor. Esophageal pressure (P_{es}) was measured with a nasogastric tube incorporating an esophageal balloon. The esophageal balloon and flow and pressure sensors were connected to a portable monitor (CP100 Bicore) that provided real-time display of flow, volume, P_{aw} and P_{es} tracings and loops of P_{es}/V, P_{aw}/V and $\dot{V}/V$ relationships. The breathing pattern was analyzed from the flow signal. Patient work of breathing (WOB) was calculated by integration of the area of the P_{es}/V loop. Respiratory drive (P0.1) was measured at the esophageal pressure change during the first 100 ms of a breath, by the quasi-occlusion technique. When pressure support was reduced, we found that the respiration rate significantly increased from PS100 to PS85, but varied negligibly with lower pressure support levels. Tidal volume behaved in a similar way, decreasing significantly from PS100 to PS85, but hardly changing at PS70 and PS50. In contrast, WOB and P0.1 increased progressively with decreasing pressure support levels. The changes in WOB were significant at each stage in the trial, whereas P0.1 increased significantly

from PS 100 at other stages. Linear regression analysis revealed a highly positive, significant correlation between WOB and P0.1 at decreasing PSV levels ($r = 0.87$), whereas the correlation between WOB and ventilatory frequency was less significant ($r = 0.53$). No other correlation was found.

Conclusions: During pressure support ventilation, P0.1 may be a more sensitive parameter than the assessment of breathing pattern in setting the optimal level of pressure support in individual patients. Although P0.1 was measured with an esophageal balloon in the present study, non-invasive techniques can also be used.

Key words Pressure support ventilation · Work of breathing · P0.1

Introduction

Inspiratory pressure support ventilation (PSV) is a mode of ventilatory assistance widely used in intensive care units (ICU). During PSV, patient/ventilator synchronization is optimized since the respiration rate is determined by the patient and each spontaneous breath is assisted by a more or less constant positive pressure. It has been suggested that PSV may be more effective than both assist/control ventilation (ACV) and synchronized intermittent mandatory ventilation (SIMV) in decreasing the ventilatory workload and in preventing respiratory muscle fatigue in patients with acute respiratory failure (ARF) [1]. Adequate PSV levels can improve pulmonary gas exchange to give a similar level to that obtained with volume-assisted modes of ventilation. PSV can be used in the early phase of ARF and during weaning from mechanical ventilation, providing more comfort for the patient and less need for sedation [2]. However, neither preset tidal volume nor minute volume is ensured during PSV. Careful monitoring is mandatory in some patients since rapid deterioration may result from inadequate assistance [3]. Any variation in total respiratory system and breathing system impedance alters patient/ventilator interaction and can excessively increase the patient's respiratory workload [4]. On the other hand, high PSV levels, able totally to unload the respiratory muscles, can induce an excessive increase in tidal volume (V_T) with an enhanced risk of pulmonary hyperinflation [5]. Conceivably, it is important to determine an individual level of pressure support for each patient, which is sufficient to maintain moderate diaphragmatic activity without inducing fatigue [6].

Analysis of breathing pattern has been proposed as a useful means of assessing how well the patient is responding to PSV and the occurrence of fatigue. Clinical studies have demonstrated that while tidal volume increases with rising pressure support levels, the respiration rate (f) and the ventilatory load, i.e., the patient's contribution to the total work of breathing (WOB), decrease [1, 6, 7]. However, other authors have suggested that analysis of breathing pattern may not be sufficient to detect the appearance of respiratory muscle fatigue during PSV [8, 9].

The aim of this study was to assess changes in ventilation variables, namely, breathing pattern, respiratory drive and inspiratory work of breathing, during the gradual reduction of PSV in ARF patients in order to investigate those liable to reflect the actual status of the patients more accurately in terms of inspiratory workload and, thus, the global efficiency of the ventilation.

Patients and methods

Ten patients (five male and five female) with acute respiratory failure (ARF) due to parenchymal pulmonary disease were studied. In all patients, the original cause of ARF was adult respiratory distress syndrome (ARDS), or airway infection shown by increased production of purulent sputum. The mean age was 58±16 (±SD). The mean Apache II score was 23.2±6.7 (range 13–32), on patient admission to the ICU. The Lung Injury Score (LIS) was 1.8±0.6 (range 1.0–3.2) in the acute phase of lung failure. All patients were intubated with an endotracheal or a tracheostomy tube and were ventilated by means of ACV or PSV using a Servo-C ventilator, in the days prior to the study. Patients remained in the ICU between 3 and 25 days during the study. The mean LIS was 0.9±0.3 (range 0.5–1.7) since lung damage had improved and the patients were in a clinically stable condition (no fever or sepsis, stable blood pressure and heart rate). No patients were in the weaning process. The main clinical characteristics are summarized in Table 1. Informed consent was obtained in all cases from the patient or next of kin, and the study approved by our institution's Ethics Committee.

Measurements

Flow ($\dot{V}$) was measured by a flow sensor (Varflex) positioned between the Y-piece of the breathing circuit and the endotracheal tube and tidal volume obtained by numerical integration of the flow signal. Airway pressure (P_{aw}) was measured through a catheter attached to the flow sensor and esophageal pressure (P_{es}) by a nasogastric tube incorporating an esophageal balloon. The correct position of the balloon was detected using the "occlusion test" [10]. The esophageal balloon and flow sensor were connected to a portable monitor (CP-100 cardiopulmonary monitor, BICORE monitoring system), providing real-time display of flow, volume, P_{aw} and P_{es} tracings and loops of P_{es}/V, P_{aw}/V and $\dot{V}/V$ relationships. The monitor was connected to a printer. The accuracy of the measurements provided by this monitoring system has been recently examined and found to be satisfactory [11].

Minute ventilation (VE) and breathing pattern [tidal volume (V_T), respiratory frequency (f), the duration of inspiration (Ti) and expiration (Te), and the duty cycle (Ti/TTot)] were analyzed from the flow signal. Patient WOB values were provided directly by the Bicore system, which calculates the area under the P_{es} versus lung volume curve during the negative deflection of the P_{es} tracing, i.e., the entire negative portion of P_{es} compared to the end-expiratory P_{es} value [12]. Chest-wall compliance was not measured nor taken

Table 1 Patient characteristics (*CMV* controlled mechanical ventilation, *PS* pressure support, *PEEP* positive end-expiratory pressure, *ARDS* adult respiratory distress syndrome)

Sex	Age year	Diagnosis	CMV duration (days)	PaO_2 mmHg (kPa)	$PaCO_2$ mmHg (kPa)	FiO_2 %	PS+PEEP cmH_2O	pH	Bicarbonate mmol/l
M	66	Pneumonia	8	137 (18.2)	39.2 (5.2)	40	9+5	7.41	24
F	76	Sepsis	42	101 (13.4)	30.9 (4.1)	34	20+6	7.47	23.1
F	70	Bilateral pneumonia	32	96 (12.8)	31.6 (4.2)	40	20+4	7.43	21.3
M	64	ARDS	15	66 (8.8)	47.4 (6.3)	50	19+3	7.44	33
F	70	Sepsis	7	121 (16.1)	47.1 (6.3)	50	14+4	7.33	25.3
M	68	Pneumonia	18	106 (14.1)	43 (5.7)	40	18+5	7.46	31.6
F	17	ARDS	15	60 (8)	50 (6.6)	50	23+4	7.36	28.5
F	54	Pulmonary contusion	17	90 (12)	54 (7.2)	30	18+5	7.43	36.2
M	61	Cardiogenic pulmonary edema	18	86 (11.4)	35.7 (4.7)	30	18+5	7.44	24.5
M	61	Pulmonary contusion	16	100 (13.3)	36.6 (4.8)	30	23+5	7.45	25.7

into account since the patients were actively breathing throughout the study. Respiratory drive (P0.1) was measured during the first 100 ms of inspiratory effort against the occluded valve on the P_{es} tracing, using the quasi-occlusion technique [13]. A clear deflection on P_{es} indicated the beginning of inspiratory effort. The presence of intrinsic positive end-expiratory pressure (iPEEP) was investigated by the "zero-flow" method, whereby iPEEP is equal to the absolute change in P_{es} from the onset of inspiratory effort to the onset of inspiratory flow [14].

Procedure

We found a level of pressure support able to generate a condition of near-relaxation in each patient, evidenced by P0.1 values $<1.5\ cmH_2O$ and WOB values close to 0 J/l. We called this level PS(100), and baseline physiological measurements, namely, breathing pattern, P0.1 and WOB were obtained as mean values of ten consecutive breaths, after a stable condition had been observed. Pressure support was then reduced to 85%, 70%, and 50% of PS(100). A moderate level of PEEP was set by the ventilator in all patients. In this study, the PEEP level was included in the total PSV level. PEEP and PSV were reduced by approximately the same percentage, and PSV was always taken as inclusive of PEEP. Each level was maintained for at least 30 min and the physiological measurements were then repeated. The rapid shallow breathing index (f/V_T) was also calculated at each PSV level.

The trigger sensitivity of the Servo-C inspiratory valve was set to its minimum level between 0 and 1 cmH_2O. All four pressure support levels were studied the same day, and no sedatives were administered. FiO_2 was constant for each patient during the study.

Data, presented as mean±SD, were analyzed by two-way analysis of variance (ANOVA) and the differences between groups were studied using Student's *t*-test with Bonferroni's correction for multiple comparisons. Linear regression analysis, using the least-square method, was used to calculate the correlation coefficient.

Results

No patients had iPEEP at the beginning of the study, shown by the expiratory flow value of 0 at the end of expiration and the negative deflection of the P_{es} tracing coinciding with the start of inspiratory flow. A mean PS(100) value of 22.8±5 (±SD) cmH_2O (PS 18.2+PEEP 4.6) was obtained. There was no correlation between the PS(100) level and severity index (PaO_2 or A-aDO_2). When pressure support and PEEP were reduced, the respiratory rate significantly increased from PS(100) to PS(85) and varied negligibly between this level and PS(75) and PS(50). Tidal volume significantly decreased from PS(100) to PS(85), but hardly changed at PS(70) and PS(50) (Table 2) (Figs. 1, 2). The rapid shal-

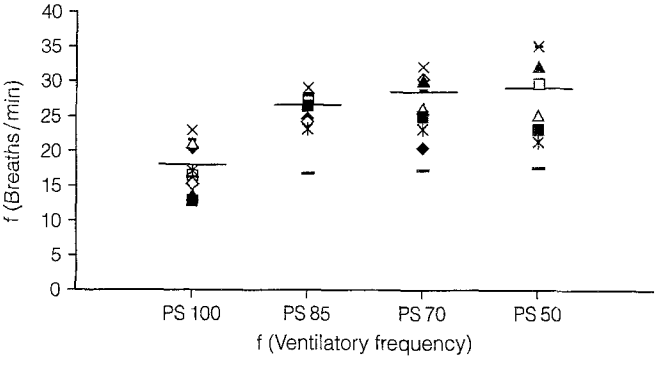

Fig. 1 Changes in respiratory rate [individual values and mean (*lines*)] at decreasing inspiratory PSV levels

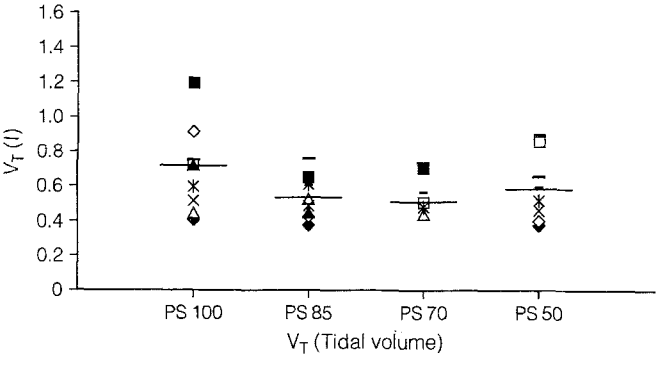

Fig. 2 Changes in tidal volume [individual values and mean (*lines*)] at decreasing PSV levels

Table 2 Breathing pattern (*f* Ventilatory frequency, *VE* minute volume, V_T tidal volume, *Ti* inspiratory time, *Te* expiratory time, f/V_T rapid shallow breathing index, *ANOVA* two-way analysis of variance)

	f br/min	VE l/min	V_T l	Ti (s)	Te (s)	f/V_T
PS 100 (PS+PEEP) (18.2+4.6 cmH_2O)	17.4±3.6	11.8±2.5	0.68±0.26	1.16±0.07	2.28±0.10	25.9±12.2
PS 85 (15.5+3.9 cmH_2O)	25.8±2.5	13.5±2.2	0.51±0.08	0.88±0.03	1.44±0.06	41 ±13.9
PS 70 (12.7+3.2 cmH_2O)	27.2+4.3	13.3±2.5	0.48±0.08	0.86±0.03	1.34±0.05	45.4±14.6
PS 50 (9.1+2.3 cmH_2O)	27.6±5.7	15 ±5.1	0.54±0.19	0.86±0.04	1.31±0.06	47.7±19.6
ANOVA	$p<0.005$	NS	NS	NS	NS	$p<0.05$
Student's *t*-test						
PS 100 v PS 85	$p<0.005$	$p<0.005$	NS	$p<0.05$	$p<0.05$	NS
PS 100 v PS 70	$p<0.005$	$p<0.05$	$p<0.05$	NS	NS	$p<0.05$
PS 100 v PS 50	$p<0.01$	NS	NS	NS	NS	$p<0.05$
PS 85 v PS 70	NS	NS	NS	NS	NS	NS
PS 85 v PS 50	NS	NS	NS	NS	NS	NS
PS 70 v PS 50	NS	NS	NS	NS	NS	NS

low breathing index (f/V_T) paralleled the behavior of its determinants at each stage. WOB and P0.1 increased progressively at decreasing pressure levels. The increase in WOB was statistically significant at each stage in PS+PEEP reduction, except from PS(85) to PS(70), whereas P0.1 increased significantly from PS(100) during the other stages, but not from PS(85) to PS(70), or from PS(70) to PS(50) (Table 3) (Figs. 3, 4). More interestingly, linear regression analysis revealed a highly positive, significant correlation between WOB and P0.1 at decreasing PSV levels ($r = 0.87$, $p<0.001$), whereas the relationship between WOB and *f* was still significant ($r = 0.53$) with a lower correlation coefficient (Figs. 5, 6). No other significant correlation was found.

Discussion

The results of this study show that changes on P0.1, measured with the quasi-occlusion technique, can be a reliable parameter in setting the level of pressure assistance during inspiratory PSV. Indeed, changes in P0.1 paralleled changes in WOB at different levels of PSV, from full to partial support.

Table 3 Respiratory drive (P0.1) and work of breathing (WOB)

	P0.1 cmH_2O	WOB Joules/l
PS 100	0.8±0.5	0.04±0.01
PS 85	2 ±1.2	0.39±0.19
PS 70	3.2±1.4	0.76±0.42
PS 50	4.2±2.7	1.17±0.54
ANOVA Student's *t*-test	$p<0.005$	$p<0.001$
PS 100 v PS 85	$p<0.05$	$p<0.005$
PS 100 v PS 70	$p<0.05$	$p<0.01$
PS 100 v PS 50	$p<0.05$	$p<0.005$
PS 85 v PS 70	NS	NS
PS 85 v PS 50	NS	$p<0.05$
PS 70 v PS 50	NS	$p<0.05$

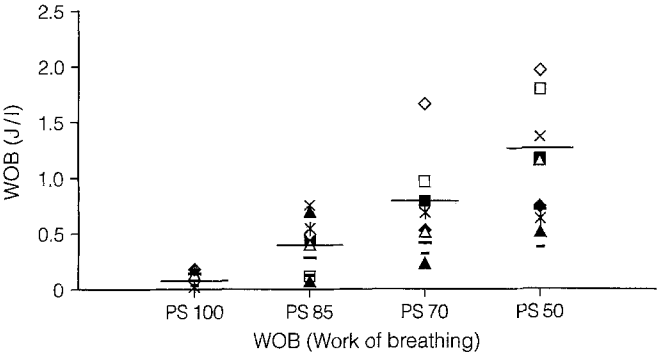

Fig. 3 Changes in inspiratory work of breathing [individual values and mean (*lines*)] at decreasing PSV levels

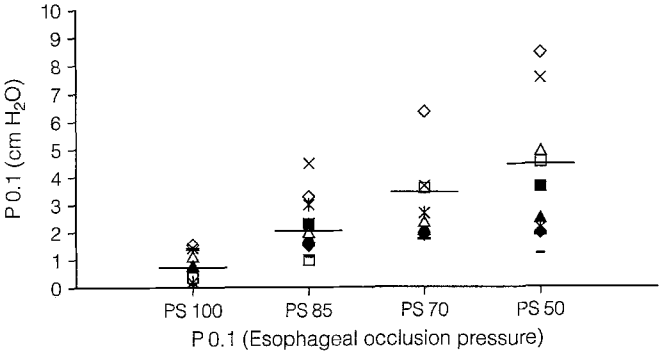

Fig. 4 Changes in P0.1 esophageal occlusion pressure [individual values and mean (*lines*] at decreasing) PSV levels

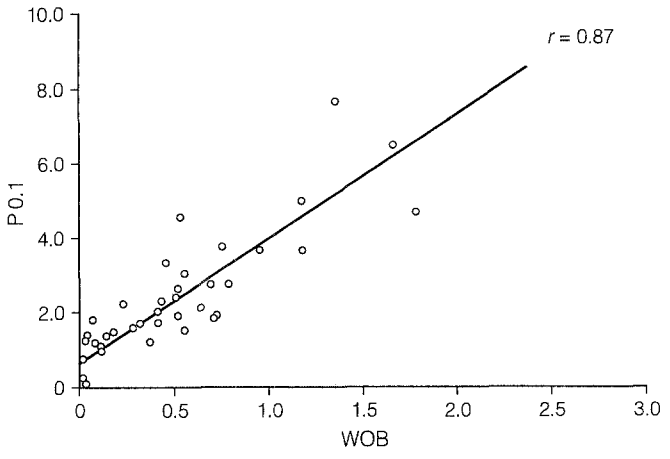

Fig. 5 Relationship between changes in inspiratory work of breathing and P0.1 at decreasing PSV levels. The regression line is drawn by means of the least-square method

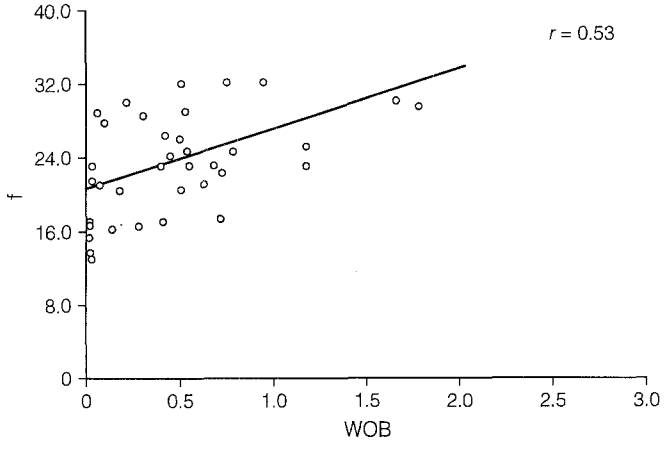

Fig. 6 Relationship between changes in inspiratory work of breathing and respiratory frequency at decreasing PSV levels

It has been widely shown that PSV reduces patient work of breathing [15–17]. WOB has been shown to have a good correlation with patient oxygen cost of breathing [6], although this could be underestimated in conditions of high inertia of the inspiratory valve or in the presence of iPEEP [18, 19]. Brochard et al. demonstrated a marked reduction in work of breathing in patients who failed to wean, when ventilated on PSV at increasing levels [6]. Kakmarek reported the same results in an experimental setting [20]. These data support the idea that PSV unloads the ventilatory muscles. In this study, we progressively decreased the level of PSV from full support (PS100) to a level at which the patients mean WOB was 1.17±0.54 J/l. This value corresponded to PS50 in our patients and was considered in the range of respiratory muscle fatigue [21]. Since the aim of mechanical ventilation, in patients with ARF is to rest the respiratory muscles and to prevent fatigue, we did not reduce the level of PSV further. In accordance with other authors, we considered the optimal level of pressure support to be the minimum pressure necessary to maintain the patient in a condition of near-relaxation or mild activation of respiratory muscles [6]. However, this level can be rapidly modified by both instantaneous variations in patient chest and lung impedance, and the extra load induced by the endotracheal tube [22, 23]. We found that there was a very wide range of individual PS100 values, depending on the differences in respiratory mechanics, the severity of illness and, presumably, the impedance of the endotracheal tubes.

Changes in breathing pattern – a simple and non-invasive procedure – has been proposed as a means of estimating how well the patient is responding to PSV, and the occurrence of respiratory muscle fatigue. Some authors have shown that increasing PSV levels either modify the breathing pattern (the respiratory rate decreases whereas tidal volume increases) or reduce work of breathing and oxygen consumption, in comparison with spontaneous breathing with continuous positive airway pressure (CPAP) and assist/control mode ventilation (ACV) [24–26].

A continuously rising ventilatory frequency (to a rate exceeding 30 breaths per minute) has been suggested as a sign of ventilatory muscle decompensation [27]. Applying this concept, some new ventilators can work in a servo-controlled PSV mode, modifying support breath by breath in order to achieve the desired respiratory frequency [28] or tidal and minute volume [29]. However, McIntyre et al. found that, at low levels of inspiratory pressure support the applied pressure only partially unloads the ventilatory muscles, and continued muscle energy expenditure is still required, whereas the ventilatory pattern hardly changes [5]. Recently, other authors have found poor correlations between the traditional parameters and the actual work of breathing in patients on PSV [8, 9]. In our patients, the progressive increase in WOB with decreasing PSV was not paralleled by corresponding changes in respiratory frequency. Indeed, WOB increased at each stage in PSV reduction, whereas f, V_T, VE, Ti/TTot and f/V_T changed significantly from PS100 to PS85 without further significant changes with lower PSV levels. We found PS100 levels that were considerably lower than those observed by McIntyre [5]. This may reflect differences in patient populations or may be explained by the fact that we chose a level of pressure support able to ensure a condition of near-relaxation for each patient, as indicated in the methods, whereas McIntyre defined PSV_{max} as the level resulting in the slowest regular respiration rate.

In contrast to the breathing pattern, P0.1 increased with increasing WOB and decreasing PSV. Although the increase in P0.1 was not always significant at each stage, this was mostly the case, and the correlation between the

changes in P0.1 and in WOB was highly significant, whereas the correlation between WOB and f was less significant. P0.1 is a widely accepted measurement of neuromuscular drive and has been shown to be a reliable parameter in predicting successful weaning in patients with ARF or decompensated chronic obstructive pulmonary disease [30–32]. In patients undergoing assisted mechanical ventilation, Marini et al. found a close correlation between P0.1 and the respiratory work performed by the patient in spite of ventilatory assistance [33].

In our patients, the progressive decrease in pressure support levels resulted in a constant increase in P0.1 until a mean value of 4.2 ± 2.7 cmH_2O ($\pm$SD) at PS 50 was obtained. Values over 4 cmH_2O are generally considered a poor prognostic indicator in the assessment feasibility of weaning off mechanical ventilation [30, 34].

It has been suggested that weaning may be indicated at values of WOB < 0.75 J/l [21, 35]. From linear regression analysis of the plot of individual WOB values against P0.1 in the ten patients in the study, it was possible to obtain the P0.1 value corresponding to this WOB "threshold" level by extrapolation to the y-axis. This value was 3.2 cm H_2O.

In this study, P0.1 was measured by an invasive technique, namely, the esophageal balloon. The routine use of the esophageal balloon in mechanically ventilated patients is discouraged since it is an invasive and complicated procedure in the crowded atmosphere of the ICU. However, P0.1 has a major advantage over WOB measurement since P0.1 can be measured non-invasively using changes in mouth pressure rather than esophageal pressure. It has been shown that in patients without iPEEP and dynamic pulmonary hyperinflation P0.1 is the same whether measured at the mouth or in the esophagus [36]. A good correlation between P0.1 measured in the trachea and in the esophagus in intubated COPD patients has also been reported [37]. Furthermore, it has been shown that there is a good correlation between P0.1 measured as indicated in our work and P0.1 measured using the conventional occlusion technique. We can therefore suggest that non-invasive measurement of P0.1 at the mouth can be used to set the appropriate level of pressure support in individual patients, in the absence of significant obstructive airway disease. Currently, modern microprocessors, ventilators and monitors can provide such data for each breath, by the quasi-occlusion technique. However, it must be mentioned in this context that although P0.1 provides valuable information and is the only convenient non-invasive measure of respiratory drive, it has some limitations during mechanical ventilation. For example P0.1 may be underestimated because of gas compression between airway opening and the site of occlusion if this is performed in the ventilator. In addition P0.1 may be low with intact central drive if respiratory muscles are weak or at a geometrical disadvantage. Owing to the poorly reproducibility of P0.1 at low values (~ 1 cmH_2O), the mean for several breaths (7–10) should be calculated. Therefore, P0.1 should not be used "out of the black box," but keeping in mind the advantages and limitations [38].

In conclusion, during pressure support ventilation, we suggest that a pressure level sufficient to maintain P0.1 below 3.5 cmH_2O be set, as suggested by the correlation between P0.1 and WOB. Further work is clearly needed in this field before a specific cut-off value can be indicated as a guideline.

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