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Airway occlusion pressure at 0.1 s (P0.1) after extubation: an early indicator of postextubation hypercapnic respiratory insufficiency

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Abstract *Objective:* To examine variables associated with postextubation respiratory distress in chronic obstructive pulmonary disease (COPD) patients.
Design: Prospective, clinical investigation.
Setting: Intensive care unit of a university hospital.
Patients: Forty COPD patients, considered ready for extubation.
Measurements and main results: We recorded, from the digital display of a standard ventilator, breathing frequency (f), tidal volume (VT) and f/VT for the respiratory pattern, airway occlusion pressure at 0.1 s (P0.1) for the respiratory drive and measured blood gases : i) before extubation, following 30 min of a 6 cm H_2O pressure support (PS) ventilation trial, ii) 1 h after extubation, at the 30th min of a face mask 4 cm H_2O PS ventilation trial. According to the weaning outcome, the patients were divided into two groups : respiratory distress, and non-respiratory distress within 72 h of the discontinuation of mechanical ventilation. The respiratory distress was defined as the combination of f more than 25 breaths/min, an increase in $PaCO_2$ of at least 20 % compared with the value measured after extubation, and pH lower than 7.35. We determined whether those patients who developed respiratory distress after extubation differed from those who did not. Respiratory pattern data and arterial blood gases recorded, either before or after extubation, and P0.1 recorded before extubation, were inadequate to differentiate the two groups. Only P0.1 recorded 1 h after the discontinuation of mechanical ventilation differentiated the patients who developed respiratory distress from those who did not (4.2 ± 0.9 vs 1.8 ± 0.8, $p < 0.01$).
Conclusions: P0.1 recorded after extubation may be a good indicator of postextubation respiratory distress. Measuring P0.1 and/or the analysis of the evolution of this parameter could facilitate decisions during the period following extubation.

Key words Airway occlusion pressure · Chronic obstructive pulmonary disease · Extubation · Non-invasive ventilation · Respiratory insufficiency

Introduction

Patients with chronic obstructive pulmonary disease (COPD) who have been intubated and mechanically ventilated for respiratory failure may prove difficult to wean [1–3]. Re-intubation exposes the patient to several complications. Indeed, intubation and mechanical ventilation may increase the morbidity and mortality of patients in the intensive care unit (ICU), especially if the duration of the ventilation is prolonged [4–6]. Non-inva-

sive pressure support ventilation (NIPSV) may render re-intubation unnecessary after weaning from mechanical ventilation [7, 8], particularly in COPD patients [9].

In order to initiate NIPSV early enough, it would be useful to have indices which are both easy to measure and predictive of postextubation respiratory distress. The rapid shallow breathing index [10] and airway occlusion pressure at 0.1 s (P0.1) [3, 11, 12] are good predictors of weaning outcome.

Our hypothesis was that one of these parameters, or both, measured shortly after extubation, i.e. 1 h after extubation, could predict postextubation respiratory distress. Respiratory distress was defined as the combination of f of more than 25 breaths/min, an increase in $PaCO_2$ of at least 20% compared with the value measured after extubation, and pH lower than 7.35. Thus, the objective of our work was to assess if the respiratory pattern, P0.1 for respiratory drive and blood gases, recorded 1 h after extubation, could predict respiratory distress after the discontinuation of mechanical ventilation. We also measured the above parameters before extubation in order to find out if, pre-extubation, our indices were indicative of weaning success.

Materials and methods

The experimental protocol was approved by the institutional review board of the hospital and all patients gave their informed consent prior to participation.

Patient selection

Forty COPD patients were prospectively studied at the moment they were considered ready for extubation by their primary physicians. The patients were intubated for acute exacerbations of COPD and were weaned with pressure support (PS) ventilation. The criteria for initiating weaning with PS ventilation were similar for all the patients, i.e., vital capacity more than 10 ml/kg, arterial oxygen saturation higher than 90% at fraction of inspired oxygen (FIO_2) 0.4, maximal inspiratory pressure more than 25 cm H_2O. The procedure was the same for all patients, and the decision to perform extubation was made by the patient's attending physician according to the usual criteria used in our I C U, i.e., the patient's being able to tolerate spontaneous breathing with 6 cm H_2O of PS over a 6-h period. No patients received benzodiazepines or narcotics in the 48 h before the study.

Protocol

Throughout the study ventilation was provided with a EVITA-ventilator (Dräger, Lübeck, Germany). Each patient was placed in a semi-recumbent position which was maintained during the study. A physician not involved in the procedure was always present to take care of the patient. The physician was unaware of the purpose of the study.

The existence of dynamic hyperinflation leading to intrinsic positive end expiratory pressure (PEEPi) is a factor to be considered in patients with chronic obstructive pulmonary affections [13, 14]. The measurements obtained for P0.1 are more difficult to interpret in COPD patients who present PEEPi [15]. Our aim in this study was to employ methods which are both non-invasive and easy-to-use at the bedside. We have no non-invasive method of measuring PEEPi in patients breathing spontaneously; for this reason we measured PEEPi during a brief trial of controlled mechanical ventilation (the ventilator setting was similar for all patients) to ensure there were no significant differences in this variable between patients who developed postextubation respiratory distress and those who did not. When considered ready for extubation, the patient was returned to controlled mechanical ventilation for 30 min. Then a tidal volume (VT) of 7–8 ml/kg was delivered at a constant inspiratory flow rate of 70 l/min with a respiratory rate of 18 breaths/min; the ratio of inspiratory time over total respiratory cycle time was set at 0.35. After 30 min of controlled ventilation PEEPi was measured under conditions of passive inflation, by performing an end-expiratory occlusion of the expiratory line, and obtained from the ventilator digital display. As many as five measurements of PEEPi were obtained, but only the first three measurements were averaged and used for data analysis, unless the values fell outside the standard error twice, then the fourth and/or fifth measurements were included. Measurements were separated by an interval of not less than 15 s.

After PEEPi had been recorded, the patients were ventilated with PS ventilation with the following settings: 6 cm H_2O of PS, zero end-expiratory pressure, trigger at the maximum sensitivity value and FIO_2 unchanged. After 30 min of PS ventilation we recorded VT, breathing frequency (f), inspiratory time (Ti), and P0.1 from the ventilator digital display, and we also measured arterial blood gases. An average of 10 PS ventilation cycles were analyzed for variables of the respiratory pattern. We used the P0.1 measurement technique developed as an integrated function of the ventilator Evita. After activation of the P0.1 function in the respirators' software menu the inspiratory valve remains closed for the 104 ms after the onset of the triggered inspiration. The P0.1 value is then calculated from the pressure drop in the system within these 104 ms, recorded by the in-built pressure transducers of the respirator. In order to avoid artefacts, the airway pressure must drop 0.5 cm H_2O to define the onset of inspiration. The time of 104 ms is due to the respirators' sampling frequency of 125 Hz. As many as five measurements of P0.1 were obtained, but only the first three measurements were averaged and used for data analysis, unless the values fell outside the standard error twice, then the fourth and/or fifth measurements were included [12]. Measurements were separated by an interval of not less than 15 s. The following derived variables were also calculated: index of rapid, shallow breathing (f/VT), the product of P0.1 and f/VT $P0.1 \times f/VT$, and effective inspiratory system impedance (P0.1/VT per Ti).

After the 6 cm H_2O PS trial, the patients were returned to controlled mechanical ventilation for 30 min, and then extubated. Patients received a small oxygen flow adjusted to maintain an oxygen saturation greater than 90%, as measured by a bedside pulse oximeter (Oxisensor, Nellcor, Hayward, Calif.). Just after extubation, inhaled corticosteroids were given to all the patients, and all patients were treated with similar nursing and respiratory care.

One hour after extubation, NIPSV was initiated in all the patients. NIPSV was delivered to the patient through a full-face mask (La Cigogne, Pessac, France). The mask was adjusted and connected to the ventilator set in the PS mode with the following settings: 4 cm H_2O of PS, zero end-expiratory pressure, trigger at the maximum sensitivity value and FIO_2 similar to that used before extubation. The face mask was carefully positioned in order to avoid gas leaks around the mask. At the 30th min of a face mask 4 cm H_2O PS ventilation trial, we recorded the same variables as those measured during the 6 cm H_2O PS trial performed before extubation.

The diagnosis of postextubation hypercapnic respiratory insufficiency was established if, within 72 h of the discontinuation of mechanical ventilation, the patients presented with respiratory distress which was defined as the combination of f more than 25 breaths/min, an increase in $PaCO_2$ of at least 20% compared with the value measured after extubation during spontaneous breathing with supplemental O_2 and pH lower than 7.35. The patients who exhibited respiratory distress were ventilated with NIPSV as previously described [9, 16] in order to avoid re-intubation.

Statistics

The patients were divided into two groups: postextubation hypercapnic respiratory insufficiency (distress group), and non-postextubation hypercapnic respiratory insufficiency (non-distress group). We determined whether those patients who developed respiratory distress after extubation differed from those who did not. Baseline data, PEEPi and the mean duration of mechanical ventilation were compared between the two groups, using an unpaired *t*-test and a chi-square test with Yates' correction. We used Mann-Whitney U test for the following quantitative variables, recorded before and after extubation : f, f/VT, $PaCO_2$, PaO_2/FIO_2, pH and P0.1, and for the derived variables P0.1 × f/VT, and P0.1/VT per Ti. For each group of patients we also compared the variables measured before and after extubation using Wilcoxon test for paired samples. A value of p less than 0.05 was considered as significant.

Results

Thirteen (32%) of the 40 patients presented postextubation hypercapnic respiratory insufficiency (distress group), with f of 30 ± 4 breath/min, $PaCO_2$ of 74 ± 6 mm Hg, PaO_2/FIO_2 of 225 ± 43 and pH of 7.32 ± 0.04. The time between extubation and respiratory distress was 16 ± 14 h. NIPSV was initiated as soon as the diagnosis of postextubation hypercapnic respiratory insufficiency was established. Three of the 13 patients failed with NIPSV and required endotracheal intubation.

Table 1 Anthropometric and steady-state lung function data of patients (Distress = postextubation hypercapnic respiratory insufficiency)

	Group distress (n = 13)	p	Group non-distress (n = 27)
Age (year)	69 ± 8	NS	68 ± 10
FEV1 (% predicted)[a]	33 ± 10	NS	35 ± 8
FEV1/VC (% predicted)[a]	52 ± 9	NS	54 ± 8
PaO_2 (mm Hg)[b]	58 ± 8	NS	59 ± 9
$PaCO_2$ (mm Hg)[b]	60 ± 7	NS	59 ± 6
Bicarbonate (mmol/l)	30 ± 3	NS	30 ± 4

Values are mean ± SD

[a] Functional steady-state characteristics were obtained from previous spirometric tests in 8 of the patients in the distress group, and in 18 of those in the non-distress group. Reliable pulmonary-function data were obtained within 2 months after inclusion in 4 of the patients in the distress group, and in 9 of those in the non-distress group

[b] Data recorded on air in spontaneous breathing

The anthropometric and steady-state lung function data of the patients are shown in Table 1. The patients in the two groups had respiratory disease of similar severity, i.e. the functional steady-state characteristics were similar in the two groups. The same was true for the severity of acute decompensation, i.e., there were also no significant differences in the new Simplified Acute Physiologic Score (SAPS II) [17] (26 ± 6 vs 25 ± 7) and mean duration of intubation and ventilatory assistance (11 ± 4 days vs 10 ± 5 days) between the distress group and the non-distress group. At the time of inclusion, there was no difference in medical treatment between the two groups of patients; distress and non-distress groups, respectively: corticosteroids 7 (54%) vs 13 (48%), antibiotics 10 (77%) vs 19 (70%), sympathomimetic agents 8 (62%) vs 14 (52%), diuretics 6 (46%)

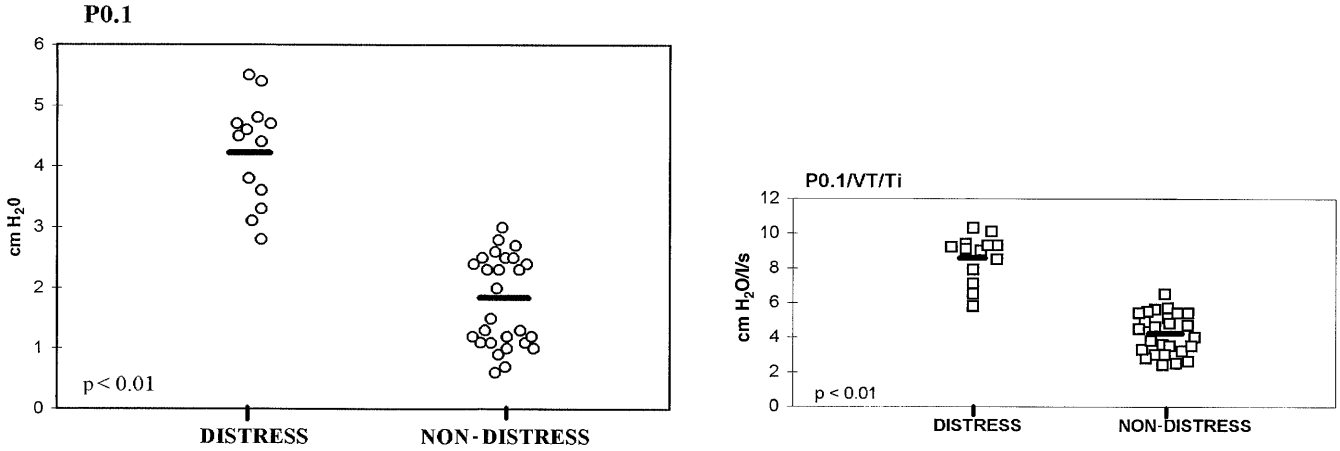

Fig. 1 Individual values of airway occlusion pressure at 0.1 s (P0.1) (*left panel)* and effective inspiratory system impedance (P0.1/VT/Ti) (*right panel*), recorded 1 h after extubation, for distress group patients and non-distress group patients

Table 2 Breathing pattern, arterial blood gases, airway occlusion pressure at 0.1 s (P0.1), and derived variables, before extubation and 1 h after extubation, in all patients (*f* breathing frequency). Values are mean ± SD and (median)

Pressure support (PS) Trial	Variables derived variables	Group distress ($n = 13$)	Group non-distress ($n = 27$)	p
Before extubation	f (breaths/min)	25 ± 5 (24)	24 ± 5 (24)	NS
PS = 6 cm H_2O	f/VT (breaths/min/l)[a]	66 ± 28 (69)	60 ± 2 (51)	NS
	$PaCO_2$ (mmHg)	60 ± 7 (61)	58 ± 6 (59)	NS
	PaO_2/FIO_2	241 ± 56 (231)	248 ± 62 (239)	NS
	pH	7.41 ± 0.02 (7.41)	7.42 ± 0.02 (7.41)	NS
	P0.1 (cm H_2O)	2.9 ± 0.7 (2.9)	2.4 ± 0.9 (2.4)	NS
	P0.1 *f/VT (cm H_2O breaths/min/l)[b]	206 ± 115 (209)	158 ± 110 (132)	NS
	P0.1/VT/Ti (cm H_2O/L/s)[c]	6.1 ± 1.3 (6.2)	5.2 ± 1.2 (5.4)	NS
After extubation	f (breaths/min)	27 ± 4 (26)	23 ± 5 (24)	NS
PS = 4 cm H_2O	f/VT (breaths/min/l)[a]	69 ± 19 (62)	60 ± 21 (52)	NS
	$PaCO_2$ (mmHg)	62 ± 6 (61)	57 ± 7 (58)	NS
	PaO_2/FIO_2	252 ± 61 (246)	257 ± 63 (254)	NS
	pH	7.40 ± 0.03 (7.40)	7.42 ± 0.02 (7.41)	NS
	P0.1 (cm H_2O)	4.2 ± 0.9 (4.5)	1.8 ± 0.8 (1.5)	$p < 0.01$
	P0.1 * f/VT (cm H_2O breaths/min/l)[b]	302 ± 124 (262)	117 ± 78 (79)	NS
	P0.1/VT/Ti (cm H_2O/L/s)[c]	8.6 ± 1.4 (9.0)	4.3 ± 1.2 (4.5)	$p < 0.01$

[a] Index of rapid, shallow breathing
[b] product of P0.1 and f/VT;
[c] Effective inspiratory system impedance

vs 14 (52 %). After the discontinuation of mechanical ventilation, inhaled corticosteroids were given to all patients.

The data regarding P0.1, f, f/VT, $PaCO_2$, PaO_2/FIO_2 and pH of the two groups of patients are listed in Table 2. Among all the variables measured, both before and after extubation, only P0.1 recorded after 30 min of face mask 4 cm H_20 PS ventilation enabled us to distinguish between the two groups of patients. Breathing frequency measured independently of the mask ventilation, before the onset of the face mask 4 cm H_2O PS ventilation trial, was 25 ± 4 breath/min and 24 ± 5 breath/min in the distress and non-distress groups, respectively. Figure 1 shows individual values of P0.1, recorded 1 h after extubation, for distress group patients and non-distress group patients. The data regarding the derived variables P0.1 × f/VT and P0.1/VT per Ti, recorded before and after extubation, in the two groups of patients are listed in Table 2. Figure 1 also shows individual values of P0.1/VT per Ti, recorded 1 h after extubation, for distress group patients and non-distress group patients. With regard to the comparison of the variables measured before and after extubation, there was a significant difference ($p < 0.01$) only in the measurements for P0.1 and P0.1/VT per Ti recorded in the distress group.

There were no significant differences in PEEPi during controlled mechanical ventilation between the distress group and the non-distress group (6 ± 3 cm H_2O vs 5 ± 3 cm H_2O). On the other hand, P0.1/VT per Ti after 30 min of face mask PS ventilation was significantly higher (8.6 ± 1.4 vs 4.3 ± 1.2, $p < 0.01$) in the distress group than in the non-distress group.

Discussion

Only P0.1 recorded 1 h after the discontinuation of mechanical ventilation followed by 30 min of 4 cm H_2O PS ventilation, was significantly different between the patients with and without respiratory distress (4.2 ± 0.9 vs 1.8 ± 0.8, $p < 0.01$). The derived variable P0.1/VT per Ti, measured under the same conditions, was also adequate to differentiate the patients with and without respiratory distress. The P0.1 values found in the present study are lower than those reported by Murciano et al. (18), who measured P0.1 during spontaneous breathing before extubation. This may be explained partly by the fact that, in the present study, P0.1 was measured during PS, which may reduce the inspiratory drive.

Many authors [3, 11, 12, 18, 19] have studied breathing pattern, arterial blood gases and respiratory drive for prediction of successful weaning in COPD patients. These studies have shown that conventional weaning criteria are often inadequate in predicting successful weaning, while P0.1 is a good weaning predictor. In these studies, weaning failure was defined either as the inability to perform extubation, or as the need for the re-institution of endotracheal intubation within 24–48 h of the discontinuation of mechanical ventilation. In contrast to these studies, and taking into account the possibility of using NIPSV in order to prevent re-intubation [7–9], we have looked for indices capable of providing an early indication of postextubation respiratory distress. The patients in the present study were comparable to those studied by Meduri et al. [7], who had "hyper-

capnic respiratory insufficiency", defined as the combination of a moderate to severe dyspnea, f of more than 25/min, pH lower than 7.35 and $PaCO_2$ of greater than 6.0 kPa. Those patients had not reached the subjective and/or objective criteria for re-intubation at the time of entering the study, and NIPSV was indicated early on to prevent reintubation. This strategy may appear premature in some patients, but the patient's condition may deteriorate rapidly, with the risk of diminished tolerance of NIPSV. This explains why the 35 % rate of postextubation respiratory distress is substantially higher than the re-intubation rate reported in other series, usually around 10–20 %.

The idea of studying breathing pattern, arterial blood gases and respiratory drive in the period following extubation, in order to obtain indications related to extubation outcome, is another important original aspect of our study. Our data confirm the advantage of taking these measurements after extubation, since P0.1 recorded after extubation was the only variable which enabled us to note a significant difference between the patients who developed respiratory distress and those who did not.

In our study, the analysis of breathing pattern did not predict postextubation respiratory distress, and this is in line with previous reports [20–22], which have suggested that breathing pattern analysis may not be sufficient to predict weaning outcome. As Sassoon et al. [12] showed, patients can adopt different breathing stategies depending on their ventilatory capabilities, loads and/or the levels of discomfort induced. Some patients with a high ventilatory demand will have an increase in P0.1 and choose to adopt a breathing pattern that is probably more economical by reducing VT with consequent high f/VT. Inversely, some patients with a relatively good ventilatory reserve may increase P0.1 and maintain a low f/VT, the latter by maintaining a relatively high or adequate VT. On the other hand, some patients may not be able to increase P0.1. As some patients with a high P0.1, other patients with a low P0.1 may also choose to adopt a rapid shallow breathing pattern. Nevertheless, before extubation, we did not measure f/VT with the reference method, that is to have the patient breathing spontaneously on a T-tube [10]. Similarly, the 30-min period of mask ventilation could have influenced the breathing patterns of the patients. Nevertheless, f did not change significantly between the period preceding the mask ventilation and the 30th min of the face mask 4 cm H_2O PS ventilation trial; distress and non-distress groups, respectively: 25 ± 4 breath/min vs 27 ± 4 breath/min, and 24 ± 5 breath/min vs 23 ± 5 breath/min. Consequently, it is unlikely that mask ventilation by itself influenced these values.

As in previous studies [20, 22, 23], VT, f and Ti were derived from an average of the ventilator digital display. PEEPi and P0.1 were also displayed on the ventilator screen. This approach, which reflects routine monitoring practice in our ICU, was adopted to obviate the need for alternative measurements apparatus. The aim of our study was also to evaluate indicators which are easy to measure using standard measurement techniques widely available for clinical purposes. During PS ventilation it should be possible to estimate P0.1 with the simple non-invasive recording of airway pressure without specialized systems. Fernandez et al. [24] have demonstrated, in a group of acute respiratory failure patients in assist control mode on a ventilator, that the inspiratory effort against the closed demand valve of a ventilator allows the measurement of the occlusion pressure. Conti et al. [3, 25] confirmed the correlation in COPD patients spontaneously breathing during PS ventilation, and suggested that P01 against the closed demand valve could be incorporated in the ventilatory monitoring software to facilitate decision analysis during weaning. Kuhlen et al. [26] have validated the P0.1 measurement technique developed as an integrated function in the Evita ventilator by comparing it to standard P0.1 measurements in a mechanical lung model as well as in ventilated patients. For Derenne [27], this method provides easy and accurate measurements using standard respiratory equipment and makes P0.1 a clinical bedside method available on a routine basis.

This study shows that the inspiratory neuromuscular drive, as assessed by P0.1, is higher as early as at the first hour following extubation in patients in whom respiratory distress will occur, compared to those who will remain stable. However, it would be premature to rely on our results to suggest a threshold value for P0.1 which would predict the evolution of patients after discontinuation of mechanical ventilation. To our knowledge, P0.1 has not been previously studied in COPD patients just after extubation, and so a threshold value of P0.1 is not available. Furthermore, we recorded P0.1 from the ventilator digital display, after 30 min of NIPSV delivered to the patient through a full-face mask connected to the ventilator set in the PS mode, with a PS level of 4 cm H_2O. The major advantage of this technique of measurement of P0.1 is its non-invasiveness. On the other hand, one should compare it to reference P0.1 measurements, i. e., mouth occlusion pressure or esophageal occlusion pressure [15] in COPD patients with spontaneous breathing. In order better to understand what a high P0.1 as early as the first hour following extubation means, it would also be useful to study the time course of P0.1 up to the diagnosis of respiratory distress. Further clinical studies are, therefore, clearly necessary.

A high P0.1 implies an increase in inspiratory neuromuscular activity. If this increased muscular activity is sustained, it may lead to inspiratory muscle overload and to hypercapnic respiratory insufficiency. In patients who developed postextubation respiratory distress, an increase in mechanical load, as indicated by the higher

effective inspiratory system impedance might have partially contributed to the higher P0.1. The higher P0.1 may not be ascribed to an increase in chemoreceptors input, given that the $PaCO_2$ values were not significantly different between the two groups.

The existence of dynamic hyperinflation leading to PEEPi is a factor to be considered in patients with chronic obstructive pulmonary affections [13, 14]. Dynamic hyperinflation can result in a decrease in P0.1 [28]. We did not measure PEEPi after extubation, yet the PEEPi recorded before extubation, after 30 min of controlled ventilation, was not significantly different between the two groups. Furthermore, Fernandez et al. [24] and Conti et al. [3, 25] have demonstrated that the measurement of P0.1, from the inspiratory effort against the closed demand valve of a ventilator, was not influenced by the presence of PEEPi.

In conclusion, our study shows that P0.1 recorded 1 h after the discontinuation of mechanical ventilation, is higher in patients in whom respiratory distress will occur, compared to those who will remain stable. Therefore, P0.1 recorded after extubation can represent a good indicator of postextubation respiratory insufficiency. Measuring PO.1 and/or the analysis of the evolution of this parameter, could facilitate decisions during the period following extubation.

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