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Mouth occlusion pressure ($P_{0.1}$) in acute respiratory failure

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Abstract. We studied 20 unselected patients admitted to our Intensive Care Unit (ICU) suffering from acute respiratory failure (ARF), who needed mechanical ventilatory support. In all of them we followed a prospective protocol to investigate the value of mouth occlusion pressure ($P_{0.1}$) as an indicator for weaning. Fifty-two tests were classified into three groups: a need to be reconnected to mechanical ventilation (MV), stable on intermittent mandatory ventilation (IMV), or spontaneous breathing on a T-tube (TT). The results showed that at increased values of $P_{0.1}$ there were more difficulties in weaning patients from MV. Seventy-eight percent (78%) of the occasions where weaning was successful, values of $P_{0.1}$ were $\leqslant$4.2 cm H_2O, in chronic or non-chronic patients. Eighty-nine percent (89%) of the times when $P_{0.1}$ values were higher than 4.2 cm H_2O the same patients required ventilatory support, total (MV) or partial (IMV). These differences were statistically significant ($p<0.01$). We conclude that the $P_{0.1}$ is an easily obtained non-invasive parameter, that can contribute along with other more conventional measurements to a superior indication for weaning.

Key words: Mouth occlusion pressure ($P_{0.1}$) – Acute respiratory failure – Weaning from mechanical ventilation

Mouth occlusion pressure ($P_{0.1}$) is the pressure generated at the mouth in the first 0.1 s of the inspiration made with the airway occluded at functional residual capacity (FRC).

Since its introduction by Whitelaw [1] it has gained clinical acceptance as a test for investigating the control of breathing. Its principal advantage over other traditional exploratory methods is that it is independent of compliance and resistance of the respiratory system, being measured at zero flow.

The negative pressure obtained reflects the level of stimulation of the respiratory centre, and also the functional activity of the inspiratory muscles [2]. It is therefore considered to be an index of the inspiratory neuromuscular drive [3]. Recent works show that $P_{0.1}$ is high in ARF and chronic obstructive pulmonary disease (COPD) [4, 5]. Its value tends to decrease towards normal as gas exchange improves. However, there are few observations in acutely ill patients needing mechanical ventilation, and it has not been established if its measurement in these has any practical value. With this in mind, we present this preliminary study of $P_{0.1}$ as another parameter to guide the weaning process.

Materials and methods

Twenty patients admitted to a multidisciplinary ICU, with ARF of different aetiologies, were studied prospectively. Six were females and 14 males with a mean age of 62.2 ± 11.2 years (range 40–74).

Eleven patients had COPD with acute ventilatory failure and nine had ARF with no past history of cardiopulmonary disease (non-COPD). The cause of the respiratory failure and the arterial blood gas (ABG) analysis at the time of admission ($FIO_2 = 0.21$) are shown in Table 1. All patients were intubated with oral endotracheal tubes (8.5 mm), with low-pressure cuffs and were ventilated using a Siemens-Elema servo-ventilator 900 B. Monitoring was carried out according to the general practice of the ICU. Radial or dorsalis pedis arterial lines were used to obtain arterial blood samples.

End-tidal carbon dioxide tensions ($P_{et}CO_2$) were continuously recorded with a Siemens-Elema 930 in-

Table 1. Cause of respiratory failure and arterial blood gas analysis

COPD	non-COPD
Etiology of ARF	
Bronchial infection: 4	Bilateral pneumonia: 4
Pneumonia: 3	Mendelson's syndrome: 3
Left ventricular failure: 3	Adult respiratory distress: 1
Pulmonary embolism: 1	Chest trauma: 1
Bronchospasm: 1	
Arterial blood gas analysis	
pH = 7.24 ± 0.1	pH = 7.26 ± 0.1
pCO_2 = 87 ± 13.7	pCO_2 = 55 ± 22.4
pO_2 = 40 ± 8.9	pO_2 = 50 ± 10.9
$SatO_2$ = 61 ± 12.2	$SatO_2$ = 75.5 ± 14.4
CO_3H = 33 ± 15.8	CO_3H = 21 ± 2.2

COPD: Chronic obstructive pulmonary disease; ARF: acute respiratory failure

frared analyzer and expiratory flow was measured with a Wright spirometer; FIO_2 was adjusted to 0.4.

Airway pressures were measured, near the entrance of the endotracheal tube, by means of connecting piece (swivel) to the pressure transducer of the ventilator (Fig. 1). Pressure, volume and flow curves were recorded on a Mingograph-34 Siemens poligraph by means of a cable connection 66989-E037E Siemens, at a speed of 50 mm/s. The internal circuit resistance was 3.04 cm/l per second at a flow of 1 l/s.

Patients were in a supine position, the airway was aspirated and no sedative muscle relaxant drugs had been given for at least 48 h prior to the study. In all patients 2–3 tests of spontaneous breathing were made at different times in the course of their illness. For this the ventilator was placed in IMV/FO position, which enables spontaneous breathing through it but without assistance.

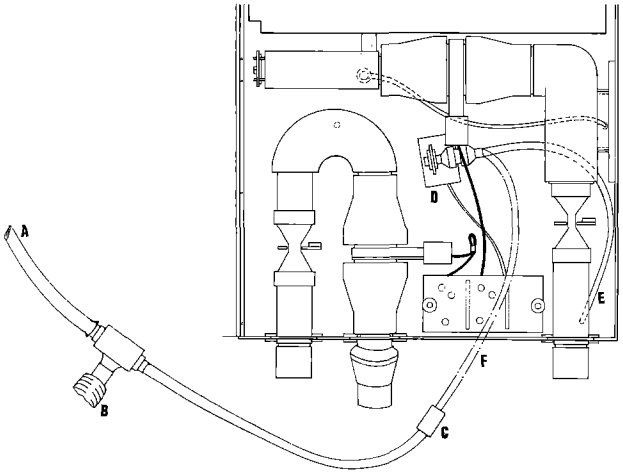


Fig. 1. Schematic illustration of the mouth occlusion pressure ($P_{0.1}$) measurement. A: Endotracheal tube. B: Ventilator connexion. C: Connexion tube to the ventilator's pressure transducer. D: Servo-Ventilator 900-B pressure transducer. E: Standard inspiratory pressure measurement. F: E-tube linked to C for $P_{0.1}$ measurement

When a steady state for minute ventilation (VE) and $P_{et}CO_2$ was obtained, we measured: (1) expired minute ventilation (VE) and respiratory rate (RR); (2) arterial blood gas tensions (ABGs); and (3) mouth occlusion pressure ($P_{0.1}$).

Occlusion was carried out by manual locking of the inspiratory valve. The valve was closed during the expiratory phase (with the expiratory valve open) and maintained closed throughout the inspiratory phase. We took an average value of three measurements of each variable. Inspiratory force was not measured because the patients were unable to cooperate.

Tests during spontaneous breathing were classified into three groups according to the subsequent course:

Mechanical ventilation group (MV)

This comprised all failed attempts at weaning. Patients were reconnected to the ventilator if signs of clinical deterioration occurred: mental changes, subjective fatigue, a respiratory rate >30 per minute, thoraco-abdominal asynchronism, tachycardia, hypotension, hypoxaemia and acidosis [6, 7].

Intermittent mandatory ventilation group (IMV)

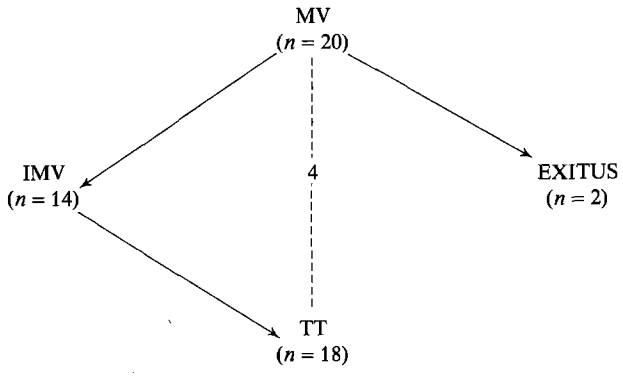
This included those tests that were followed by progressive IMV, considered as such if mandatory (obligated) and spontaneous breathing were sufficient to maintain an arterial pH >7.30 [8, 9].

Spontaneous ventilation group on T-tube (TT)

This included attempts at disconnection followed by spontaneous breathing using a T-tube in those patients in good condition without criteria for reventilation [10]. All were extubated without problem.

To calculate the sensitivity, specificity and predictive value of $P_{0.1}$ as an index of weaning, the following formulae were used [11, 12, 13]: sensitivity = TP/TP + FN, specificity = TN/TN + FP, positive predictive value = TP/TP FP, negative predictive value = TN/TN + FN. (TP: true positive, TN: true negative, FP: false positive, FN: false negative). Student's t-test for paired or unpaired data, chi-square analysis and the Pearson's correlation coefficients were used in the statistical analysis; a probability of less than 0.05 was considered as significant [14].

Table 2. Group distribution



MV: Spontaneous breathing tests in mechanical ventilation group; IMV: Idem in intermittent mandatory ventilation group; TT: Idem in T-tube piece group

Table 3. Mouth occlusion pressure values ($P_{0.1}$) and weaning

$P_{0.1}$	Weaning			
	TP	TN	FP	FN
≤4.2 cm H_2O	14	34	0	4

TP: true positive; TN: true negative; FP: false positive; FN: false negative

Table 4. Sensibility, specificity and predictive value of $P_{0.1}$ for weaning

	Sensibility	Specificity	PV(+)	PV(−)
$P_{0.1}$ ≤4.2 cm H_2O	78% (14/18)	100% (34/34)	100% (14/14)	89% (34/38)

PV(+): positive predictive value; PV(−): negative predictive value

Results

Group distribution

In the 20 patients, at least one premature attempt was made to dispense with ventilatory support. Two patients died so they could not pass any other assessment of weaning. In 14 patients, IMV was possible for variable periods, all going into a T-tube eventually. Four patients were placed directly into a T-tube without IMV. Therefore, a total of 52 tests were performed: 20 in the MV group, 14 the IMV group, and 18 in the T-tube group (Table 2).

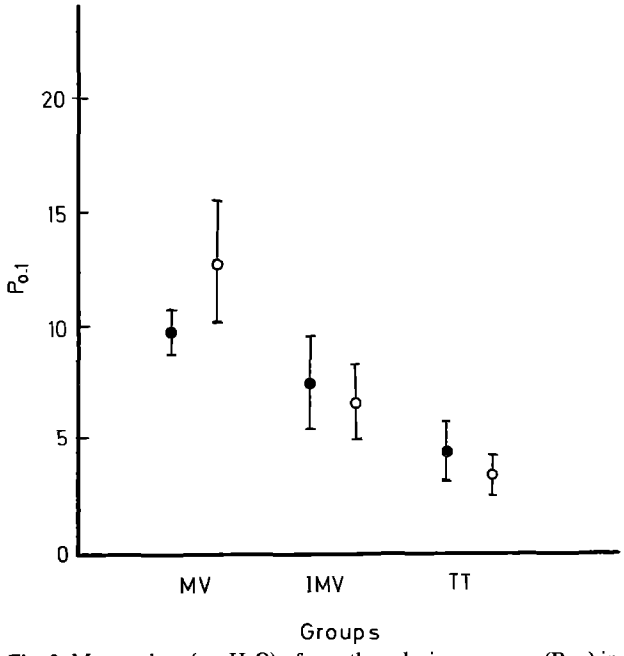


Fig. 2. Mean values (cm H_2O) of mouth occlusion pressure ($P_{0.1}$) in the three groups. ● COPD patients; ○: non-COPD patients

Mouth occlusion pressure ($P_{0.1}$)

The $P_{0.1}$ values were always above normal (1 − 2 cm H_2O in steady state) [15, 16].

We obtained the highest pressure in the MV group. The mean $P_{0.1}$ value in the non-COPD was 12.6 ± 2.7 cm H_2O which was higher than in COPD patients whose mean value was 9.8 ± 1.9 cm H_2O, but the differences were not statistically significant.

The subjects that were able to maintain spontaneous ventilation through a T-tube had the lowest $P_{0.1}$ in comparison with the previous groups ($p < 0.01$). The measurements were similar in the COPD and the non-COPD types: 4.2 ± 1.8 cm H_2 and 3.4 ± 1.1 cm H_2O respectively. These findings are shown in Figure 2. 78% (14/18) of the patients who were able to be weaned from the ventilator had mean values of $P_{0.1} \leq 4.2$ cm H_2O, and were similar in both chronic and non-chronic patients. Four patients (22%) were weaned with increased values of $P_{0.1}$. In 89% (34/38) of the times when values of $P_{0.1}$ were above 4.2 cm H_2O, the same patients required total (MV) or partial (IMV) ventilatory support.

At this level of $P_{0.1}$ (4.2 cm H_2O), sensitivity, specificity and predictive values with regard to successful weaning were: sensitivity = 78%, specificity = 100%, positive predictive value = 100%, negative predictive value = 89%. We consider as true positive those patients with $P_{0.1} \leq 4.2$ cm H_2O achieving weaning and extubation (Tables 3 and 4).

Table 5. Expired minute ventilation and respiratory rate in the different groups

	COPD		non-COPD	
	VE	RR	VE	RR
A MV	7.9±3.1[a]	31±11.2	13.8±3.9[a]	40±8.5[a]
B IMV	10.2±3.2	29±7.7	12.5±2.3	35±2.1
C TT	9.5±1.7[a]	27±9.4	12.2±1.4[a]	31±6.6[a]

COPD: chronic obstructive pulmonary disease; VE: expired minute ventilation; RR: respiratory rate; MV: mechanical ventilation group; IMV: intermittent ventilation mandatory group; TT: T-tube piece group. [a] = $p < 0.01$

Minute ventilation (VE) and respiratory rate (RR)

There were no marked changes in VE from one group to another. The general tendency was to increase VE in COPD patients and to decrease it in non-COPD patients, as they had less dependency on ventilatory support. If chronic patients developed ARF they had lower VE than non-COPD in the MV and TT groups ($p<0.01$). In the IMV group there were no significant differences between both types of patients (Table 5). The non-COPD decreased their respiratory rate from 39.6±8.5 breaths/min when ventilatory support was indispensable to 31.2±6.6 breaths/min during spontaneous breathing ($p<0.05$). There was no significant correlation between VE, RR and $P_{0.1}$.

Arterial blood gases (ABG)

In the early phase of acute ventilatory failure all the patients were acidotic with pH values of 7.31±0.07 in COPD and 7.32±0.09 in non-COPD (Table 6).

The arterial pO_2 and oxygen saturation showed no significant differences between the groups. The COPD patients progressively decreased their arterial pCO_2 from 71.7±14.7 mm Hg in MV group to 58.3±9.3 mm Hg when they belonged to the TT group ($p<0.01$).

Of the 52 attempts at spontaneous breathing there were only 7 (13%) when the pO_2 fell below 60 mm Hg ($FIO_2 = 0.4$). No significant correlations between $P_{0.1}$ and arterial pH, pCO_2 and pO_2 were found.

Discussion

Although clinical, functional and arterial blood gas criteria [17–20], have been applied to define the most appropriate time for weaning, in practice its institution is still partly empiric and, as recently confirmed [21], in some ways more of an art than a science. This explains the continuous search for measurements to make its timing more objective and scientific [22–25].

Some useful parameters for weaning, vital capacity, inspiratory force, maximal ventilation, etc, need the patients collaboration, which may not be easy to obtain. An advantage of $P_{0.1}$ is its independence of the patients motivation. It has been demonstrated that there is a difference of 150 ms between the occlusion act and the subsequent reaction. In addition, in anaesthetized animals and in the human, a close relationship has been observed between $P_{0.1}$ and the phrenic electroneurogram, diaphragmatic electromyogram and arterial pCO_2 [26–28]. Therefore, we considered it to be a good parameter for assessing the activity of the respiratory centre and the inspiratory neuromuscular drive.

In our study the $P_{0.1}$ values were high, up to 17.3 cm H_2O in a case of ARDS. We also found twofold increases in mean values compared to those previously considered as normal in patients breathing through a T-tube prior to extubation.

One explanation for this observation may be an increased neurochemical stimulation of the respiratory centre, since many of these patients were acidotic or hypoxic and chronic ones had CO_2 retention. Nevertheless, we could find no correlation between $P_{0.1}$ and arterial blood gases. Of course, hypoxaemia was relieved by the administration of oxygen-enriched air in the majority of patients and a pH of below 7.35 was only found in the MV group.

These findings are not too surprising since it has been shown that it is not necessary to have changes in ABG to effect greater stimulation of the respiratory centre. Healthy persons breathing through a resistance increase their $P_{0.1}$ or diaphragmatic electromyo-

Table 6. Arterial blood gas analysis in the different groups

	COPD				non-COPD			
	pH	pCO_2	pO_2	$SatO_2$	pH	pCO_2	pO_2	$SatO_2$
A MV	7.31±0.07	72±14.7[a]	64.5±27	83±15	7.32±0.09	38±5.6	97±30.3	95±5.1
B IMV	7.35±0.05	67±6.9	72.5±16	92±4.7	7.37±0.09	45±9.4	100±31	98±4.6
C TT	7.36±0.04	58±9.3[a]	82.2±20	93±3.9	7.36±0.08	38±5.4	93±2.4	95±3.8

COPD: chronic obstructive pulmonary disease; MV: mechanical ventilation group; IMV: intermittent mandatory ventilation group, TT: T-tube piece group. [a] = $p < 0.05$

gram without modifications of ABG [29, 30]. This points to other factors, particularly of a mechanical nature. COPD individuals have high $P_{0.1}$ values in the steady state [31 – 33]; values between 2.5 to 4 cm H_2O have been obtained in normo as well as hypercapnoea. This means that at rest there is an increased neuromuscular drive, perhaps because hyperinflation forces the muscles to work in a mechanically disadvantageous situation.

With increasing lung volume the respiratory muscle fibre becomes relatively shorter and hence loses force, according to the length-tension curve. Muscle fibres are then less efficient as they work in an inadequate segment of this curve. To compensate it is logical to increase pressures above normal [34, 35].

In addition, with an increased FRC the diaphragm becomes flatter and hence develops less contractile force according to the Laplace relationship ($p = 2T/r$). To maintain an adequate ventilatory response it is necessary to increase the activity and work of the respiratory muscles to develop larger pressures [36, 37].

When acute respiratory failure supervenes with several disturbances such as bronchospasm, acute bronchitis, retained secretions, etc., all of which contribute to hyperinsufflation, it will exaggerate all these events, explaining why $P_{0.1}$ increases still further.

In ARF patients without COPD, an increased neuromuscular drive has also been described that is not entirely explained by hypoxaemia [38]. This has been observed in patients with pneumonia, and then attributed, from experimental observations, to pulmonary J-receptor stimulation modulated by the vagus nerve [39 – 41]. In these cases, the central level of stimulation could be even higher than in the COPD group of patients. In our series the patients without previous bronchial disease had an average $P_{0.1}$ superior to the chronic when studied during the early stage of ARF when it was not possible to disconnect them from the respirator. Later the discrepancy was reduced and disappeared during the IMV or T-tube course.

This study indicates that $P_{0.1}$ showed a progressive and significant fall as patients needed less ventilatory support whereas the VE and RR showed fewer changes. Only the non-COPD acute respiratory failure reduced their RR with an improvement in respiratory function. In those with COPD and respiratory failure we also found an inverse relation between $P_{0.1}$ and VE. When the $P_{0.1}$ was higher (MV group), values of VE were lower (although this was not statistically significant). This is probably due to the increased airway obstruction and more pronounced ventilatory failure.

From the above, it is inferred that in ARF there exists an augmented central response and an extremely high inspiratory muscular activity probably near maximal. In comparative terms, one must consider that in normal healthy individuals, $P_{0.1}$ values of 6 – 8 cm H_2O can only be obtained with a maximal voluntary ventilation (MVV) between 50 to 70 l/m [36]. This involves a great respiratory effort that obviously cannot be maintained for a long time, given that $P_{0.1}$ cannot increase indefinitely. If this hyperactivity is maintained, it is most probable that fatigue will ensue with ventilatory failure [42].

In our subjects we confirmed that it was uncomfortable to maintain spontaneous breathing with $P_{0.1}$ values above 4.2 cm H_2O. In 89% of the cases that we obtained this occlussion pressure value, artificial ventilation absolute (MV) or relative (IMV) was always required. Therefore, the higher the neuromuscular centre drive, the more difficult is it to be successfully weaned. Other authors have obtained similar results with different methods [43, 44].

The finding of a high $P_{0.1}$ value reflects an increased central neuronal discharge that is transmitted to the muscular fibre requiring more effort, and is impossible to maintain. Attempting to wean in these conditions would be, if not dangerous, at least useless.

Although the specificity of $P_{0.1}$ was high in this study, we have to be cautious in interpreting the results. The correlation between any isolated parameter and the possibilities of weaning cannot be absolute since there may exist many reasons for failure. It would be very unlikely that one clinical or biological index could integrate all the relevant factors. The more frequent sources of failure are: hypoxaemia, augmented ventilatory requirements, respiratory muscle weakness, and excessive respiratory work [45, 46]. Each one of these may require a different therapeutic approach. An increase in $P_{0.1}$, if a progressive event, is one measurement that would indicate the existence of a disproportionate respiratory effect.

In summary, we suggest that mouth occlusion pressure or $P_{0.1}$ is an easily obtained non-invasive parameter, that can contribute, together with the other classic and conventional measurements, to a better guideline of weaning from mechanical ventilation.

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