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Inspiratory effort and occlusion pressure in triggered mechanical ventilation*

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Abstract. We have studied eleven patients ventilated in the assisted mode during recovery from acute respiratory failure. We have measured the effort required to trigger the pressure demand valve for 3 different ventilators, and have measured the occlusion pressure as an index of neuromuscular inspiratory drive. We found a delay in the opening of the demand valve, as previously described by other authors. We also found a close correlation between the effort required to open the demand valve and the occlusion pressure. We conclude that the inspiratory effort required to open the demand valve, in the assist mode, is greater than the preset trigger level and that it is well correlated with the neuromuscular inspiratory drive. This inspiratory effort against the closed demand valve, allows the measurement of the occlusion pressure.

Key words: Inspiratory effort – Mechanical ventilation – Occlusion pressure

The inspiratory effort made by a patient to increase pulmonary volume during inspiration is due to contraction of the inspiratory muscles [1], whose magnitude depends on the intensity of stimulation applied to the inspiratory motor neurons by the respiratory center and the chemo- and mechanoreceptors [2]. Patients with acute respiratory disease have increased inspiratory drive [3]. The tracheal pressure during the first 100 ms of an occluded inspiration ($P_{0.1}$) is an useful index of the neuromuscular inspiratory drive [4].

It has been shown that there is a delay greater than 0.1 s between the beginning of the inspiratory effort and the onset of inspiratory flow during assisted mechanical ventilation [5], and that the negative pressure generated in the circuit before the opening of the demand valve may be higher than the preset trigger level [6].

The purpose of this study was to demonstrate that the pressure which is developed by the patient to begin the inspiration when he is connected to a ventilator in the assist mode, is not fixed but that it is related to the occlusion pressure. Additionally, the magnitude of the negative pressure created in the airway in the first 100 ms of the inspiration against the closed demand valve would permit us to calculate the occlusion pressure in patients ventilated in the assisted mode.

Material and methods

We studied eleven mechanically ventilated patients (Table 1) in the assist mode without PEEP. The hygroscopic humidifier was withdrawn during the measurements in order to avoid additional dead space. Inspiratory and expiratory flow was measured with a heated no. 2 Fleisch pneumotachograph which was

Table 1. Clinical data

Patient	Age	Sex	FiO_2	Disease
1	73	M	0.4	COPD; Abdominal surgery
2	69	M	0.4	COPD; Flail chest
3	53	M	0.5	Alcoholism; Pneumonia
4	64	M	0.4	COPD; Neurosurgery
5	74	M	0.3	COPD; Neurosurgery
6	63	F	0.4	COPD; Cardiac surgery
7	69	F	0.3	COPD; Pulmonary infection
8	59	F	0.3	Mitral valve replacement; Stroke
9	61	M	0.3	COPD; Respiratory arrest
10	54	M	0.4	Acute cardiogenic pulmonary edema
11	82	M	0.3	COPD; Renal failure; Seizures

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placed between the proximal end of the endotracheal tube and the Y piece of the ventilator. The airway pressure (P_{aw}) was measured at the proximal end of endotracheal tube with a HP 270 transducer. The tidal volume was obtained by integration of the flow signal with a respiratory integrator HP 8815. The flow, P_{aw} and volume signals were registered with a four channel recorder (HP 7554 B) at a 50 mm/s paper speed. At the beginning of the study we measured the occlusion pressure ($P_{0.1}$) in each patient while they were temporarily disconnected from the ventilator, as previously described [4]. All patients were consecutively ventilated by 3 different ventilators: Siemens Servo Ventilator 900 C, CPU-1 Ohmeda (Ohio CPU-1 Ventilator in the USA) [7] and Pulmosystem S-11 (Air Shields Oracle Volume Ventilator in the USA), in random order. The trigger was adjusted at the maximum sensitivity level (0 cm H_2O for Servo C and CPU-1, and 0.5 cm H_2O for Pulmosystem).

The beginning of inspiration was taken as the point where the end expiratory pressure shows a steep

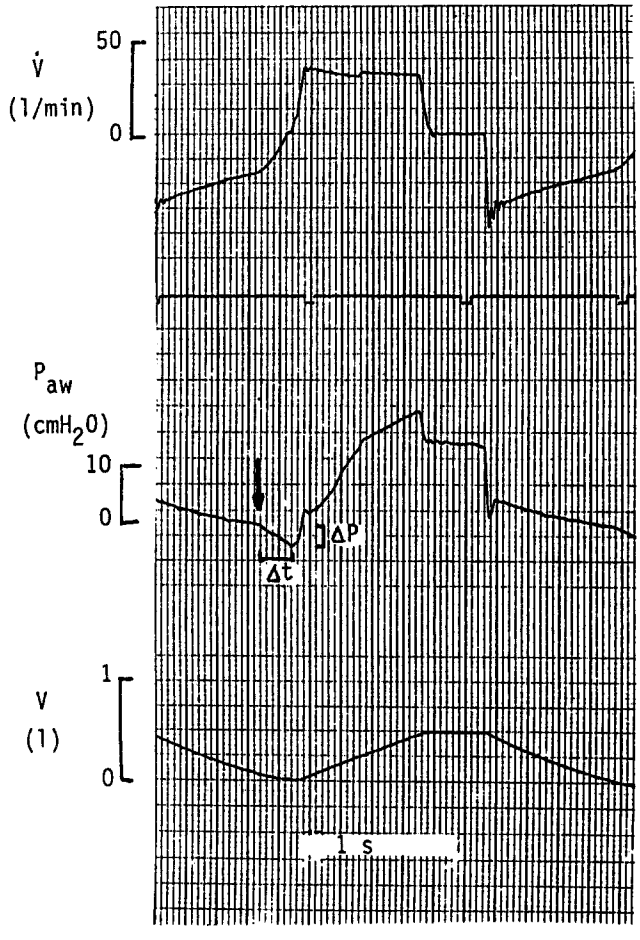

Fig. 1. Flow, volume and P_{aw} recordings during an assisted cycle. We can see Δt and ΔP_{aw} from the beginning of inspiration to the opening of the demand valve

negative deflection in P_{aw} as previously described by other authors [8]. The recording of P_{aw} showed a constant downward slope until the inspiratory valve opened and then the pre-set tidal volume was delivered (see Fig. 1). We measured the pressure gradient (ΔP_{aw}) and the time delay (Δt) in the P_{aw} channel until the valve opened. These values were calculated as the arithmetic mean of the measurements in five consecutive cycles. A simple linear regression analysis with the 95% confidence intervals was performed to assess the correlation between variables ($P_{0.1}$, Δt, ΔP_{aw}) and ANOVA test for differences between ventilators.

Results

The results of $P_{0.1}$, ΔP_{aw} and Δt for each ventilator can be seen in Table 2. We did not find any statistically significant correlation between Δt and ΔP_{aw}, or between Δt and $P_{0.1}$. However, the correlation between $P_{0.1}$ and ΔP_{aw} was highly significant ($p<0.001$) with $r = 0.85$, $y = -1.01 + 1.12x$, with a 95% confidence interval from 1.14 to 1.10 for the slope and from 0.15 to -2.17 for the intercept (Fig. 2).

Additionally, we detected a close correlation when values for $P_{0.1}$ and ΔP_{aw} obtained with each ventilator were analyzed separately: $y = -2.07 + 1.49x$, $r = 0.91$ (Servo C), $y = 0.03 + 0.96x$, $r = 0.90$ (CPU-1) and $y = -0.97 + 0.90x$, $r = 0.93$ (Pulmosystem), but the differences between the regression lines are not statistically significant (F: 1.31, p: NS).

Discussion

In this study we have found a close correlation between the respiratory drive ($P_{0.1}$) and the inspiratory effort to trigger the ventilator (ΔP_{aw}). This was the case for all ventilators studied, namely that the greater the magnitude of the occlusion pressure the greater the negative pressure in the airway before the opening of the demand valve.

The use of demand valves to synchronize ventilation forces the patient to make an inspiratory effort so as to create a depression in the airway pressure, which is detected by the trigger mechanism of the ventilator. A fixed value of the trigger sensitivity level is given by the ventilator manufacturers. Using an artificial pulmonary model, Christopher et al. [6] recently showed that the ventilator manometer measuring the internal machine pressures significantly underestimated the amount of negative pressure required to open the demand valve. This error was not due to erroneous measuring of the ventilator manometer, as when it was placed next to the endotracheal tube, the pressure was

Table 2. Results; ($P_{0.1}$ and P_{aw} in cm H_2O; Δt in s)

Patients	$P_{0.1}$	Servo C		CPU-1		Pulmosystem	
		Δt	ΔP_{aw}	Δt	ΔP_{aw}	Δt	ΔP_{aw}
1	6.5	0.15	5.7	0.14	6.3	0.18	4.5
2	4.5	0.13	3.2	0.22	5.25	0.31	4.25
3	5.5	0.18	7	0.20	6.9	0.21	3.85
4	8.25	0.16	12	0.15	7.15	0.20	6.9
5	2	0.22	2.9	0.21	1.8	0.15	1.5
6	3	0.14	2	0.16	2.7	0.20	2.16
7	4.25	0.19	4	0.19	3.4	0.27	1.95
8	3	0.14	2.1	0.14	2.2	0.17	1.25
9	4	0.17	4.2	0.21	2.9	0.32	2.15
10	3.4	0.13	2.5	0.17	3.3	0.20	1.8
11	3.1	0.13	2.6	0.16	3.8	0.20	1.8

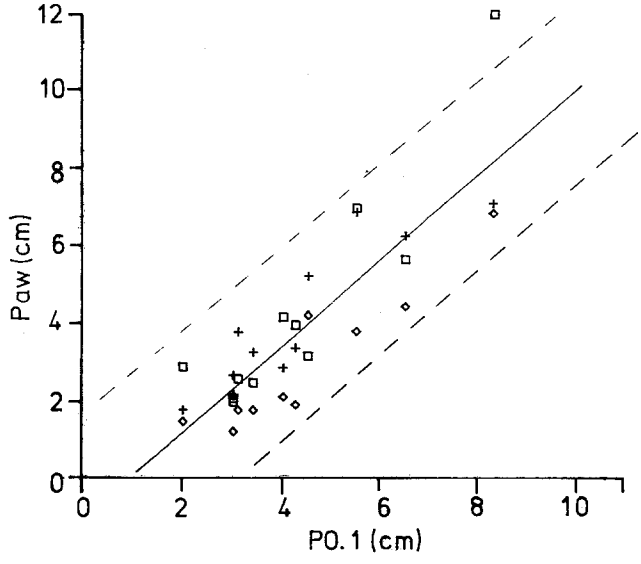

Fig. 2. Regression line between $P_{0.1}$ and ΔP_{aw} for the 3 ventilators: Servo 900 C (□), CPU-1 (+) and Pulmosystem (◇) with the 95% confidence interval for prediction (– – –)

close to that obtained with the external manometer. The authors attributed this finding to the distance between the patient and the ventilator manometer, which dampened the signal to the sensor. Furthermore, the amount of pressure necessary to trigger the ventilator has been taken as a determinant of the good performance of the demand valves, or as a factor that could increase the work of breathing [5].

We have measured in our patients the effort until the valve opens, while the inspiration is still occluded. *Sensu strictu*, in such quasi-isovolumetric conditions there is no inspiratory work [9–12], but only effort. The negative airway pressure during inspiration is created by the contraction of inspiratory muscles, mainly the diaphragm, due to the neural discharge from the respiratory centers. These centers are stimulated through the receptors by the agents and effects of respiratory disease (hypoxia, acidosis, low pulmonary volume). With these inputs the respiratory centers create an individual and characteristic ventilatory pattern for each clinical situation [3]. In patients with neuromuscular integrity, the occlusion pressure in the first 100 ms ($P_{0.1}$) reflects the inspiratory center drive [4].

The negative pressure at the beginning of inspiration and before the valve opens, is synchronous in the airway (P_{aw}) and the esophagus [3, 13]. We chose to use the P_{aw} because the deflection point due to the inspiratory valve opening is much more evident, despite the fact that this point frequently appears while there is still expiratory flow (Fig. 1). This pattern is more evident with demand valve systems than continuous flow systems [8]. Besides a greater expiratory flow at the beginning of inspiration may suggest that dynamic hyperinflation phenomena occur (intrinsic PEEP). The uniform response of our patients, despite frequent COPD, and probably intrinsic PEEP, suggests that our $P_{0.1}$ estimation from ΔP_{aw} is not influenced by the presence or not of intrinsic PEEP.

We found a time delay (Δt) from 0.13 to 0.32 s, similar to other studies [8], but failed to prove statistically significant differences between ventilators. We have not found any relation between Δt and ΔP_{aw} though the response time was always higher than 100 ms. We attribute the different values of ΔP_{aw} and $P_{0.1}$, to the distance between the endotracheal tube and the inspiratory valve, and probably to the fact that the gas inside the mechanical circuit may be decompressed at the beginning of inspiration, thus representing a quasi-isovolumetric manoeuver. The ΔP_{aw} developed by the patients ventilated in the assisted mode permits use of the change in P_{aw} to calculate the occlusion pressure ($P_{0.1}$) value as the opening of the demand valve always involved more than 100 ms in all the ventilators that we have studied. Thus, it is easy to estimate the $P_{0.1}$ by means of ΔP_{aw} at the bedside of ventilated patients in the assist mode, without the need of additional equipment for the standard measurement of $P_{0.1}$ as shown in normal subjects by Taylor et al. [14].

In conclusion, the inspiratory effort required by patients to trigger the demand valves is not fixed but is related to the output of the respiratory center, which is variably increased in patients with acute respiratory failure, and the measurement of ΔP_{aw} in ventilated patients in the assist mode allows clinical $P_{0.1}$ to be estimated.

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