

## Expiratory valves used for home devices: experimental and clinical comparison

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**ABSTRACT:** A bench study followed by a clinical trial were performed to evaluate the mechanical characteristics of five (commercially available) expiratory valves used for home ventilators, as well as the potential clinical impact of differences between these valves.

In the *in vitro* study, expiratory valve resistance was evaluated under unvarying conditions, whereas dynamic behaviour was evaluated by calculating the imposed expiratory work of breathing during a simulated breath generated by a lung model. Differences in resistance and imposed expiratory work of up to twofold and 150%, respectively, were found across valves. We then conducted a randomized crossover clinical study to compare the effects of the least resistive (Bennett) and most resistive expiratory valves (Peters) in 10 intubated patients receiving pressure support ventilation.

There were no significant differences regarding blood gases or respiratory parameters except for the oesophageal pressure-time product (PTP<sub>oes</sub>), which was significantly increased by the Peters valve (236±113 cmH<sub>2</sub>O·s·min<sup>-1</sup> versus 194±90 cmH<sub>2</sub>O·s·min<sup>-1</sup>). An analysis of individual responses found that the Peters valve induced substantial increases in intrinsic positive end-expiratory pressure (PEEP), PTP<sub>oes</sub>, and expiratory activity in those patients with the greatest ventilatory demand.

In conclusion, differences between home expiratory valve resistances may have a clinically relevant impact on the respiratory effort of patients with a high ventilatory demand.

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Over the last 10 yrs the number of patients receiving assisted ventilation at home has increased markedly [1]. As a result, there has been a resurgence of interest in the technical aspects of ventilators used to provide ventilatory support at home [2–5]. Perhaps because exhalation is usually passive, few studies have focused on the flow-impeding characteristics of external circuit components [6–8]. The expiratory valve occludes the expiratory limb of the respiratory circuit during inspiration and opens at the beginning of expiration. An ideal expiratory valve should completely obstruct air outflow during inspiration, but should offer little or no resistance to expiration in order to avoid dynamic hyperinflation or expiratory muscle recruitment.

The majority of home ventilator devices can be used with a range of expiratory valves, but criteria for rational selection of an expiratory valve are lacking since no formal evaluations of the performances of commercially available expiratory valves are available.

In the first part of this study, we examined the performance characteristics of five commercially available diaphragm or balloon expiratory valves (table 1). Data were gathered during a bench evaluation of the resistive properties of exhalation valves under quasi-static and dynamic conditions.

The purpose of the second part of the study was to evaluate the clinical relevance of the differences found between two expiratory valves during the first part of the study. We investigated critically ill patients undergoing weaning from mechanical ventilation. Each patient was ventilated with the same home pressure support device (O'Nyx; Pierre Médical, Puritan Bennett, France) during the comparison. The effects of the two valves were evaluated based on expiratory activity as assessed by gastric pressure tracing, intrinsic positive end-expiratory pressure (PEEP<sub>i</sub>), oesophageal pressure-time product (PTP<sub>oes</sub>), and respiratory pattern.

Table 1. – List of the five expiratory valves tested

Brand	Model No.	Type of valve
Peters (Bobigny, France)	40-314160	Diaphragm
Bennett (San Diego, Mexico)	T.T 11 372-00	Balloon
Pierre Médical (Verrières les Buisson, France)	EX0008	Diaphragm
Sims (Myers, USA)	003283	Balloon
Taema (Antony, France)	KY 097600	Balloon

These valves were all primarily designed for home use.

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## Methods

### Experimental *in vitro* studies

**Valves tested.** Two diaphragm and three balloon valves were investigated (table 1). The function of all of these valves is based on similar principles. In most home ventilators, gas is diverted from the inspiratory limb internal to the ventilator or from a secondary gas control module to the diaphragm or balloon of the exhalation valve, which inflates, thus occluding the exhalation port during the inspiratory phase. Since these exhalation valve charging lines are internal to the ventilator, the pressure affecting the exhalation valve is always higher than the pressure in the airway. This pressure difference is thought to minimize the risk of leakage through the exhalation valve.

**Quasi-static study.** Experimental design A (fig. 1A) was used to measure the pressure-flow relationship through the expiratory valve. Compressed air was used to deliver a predetermined steady airflow, measured by a pneumotachograph (Fleisch#2; Gould Electronic, Longjumeau, France) coupled to a second differential transducer (Validyne DP 45±2 cmH<sub>2</sub>O; Northridge, CA, USA). Pressure drop was measured using a differential pressure transducer (Validyne DP 45±2 cmH<sub>2</sub>O; Northridge, CA, USA) with one port opened to the atmosphere and the other port measuring the lateral pressure upstream from the valve. The flow values tested were in the range 0.25–1.5 L·s<sup>-1</sup>.

**Dynamic study.** In experimental design B (fig. 1B), the expiratory valves were evaluated during simulated ventilation with pressure support ventilation. The home pressure

device used for this study was the O'Nyx (Pierre Médical, Puritan Bennett, France), whose performances have been found to be similar to intensive care unit devices [5]. Inspiratory pressure level was set at 10 cmH<sub>2</sub>O, positive end-expiratory pressure (PEEP) at its lowest level (*i.e.* <0.5 cmH<sub>2</sub>O), and the rate of pressure support insufflation at the highest possible value. Pressure support was stopped during the decelerating phase at 25% of the peak inspiratory flow and exhalation was totally passive.

To simulate repeated inspiratory efforts, the pressure support ventilator (PSV) device was connected to a lung model (fig. 1B). The lung model was a two-chamber Michigan test lung. One chamber was connected to and powered by a CPU ventilator (Ohmeda, Maurepas, France) (driving chamber), whereas the other chamber (PSV pressurized chamber) was connected to the PSV ventilator under test. The CPU ventilator was set to ensure a respiratory frequency of 15 cycles·min<sup>-1</sup>, an inspiratory time of 1.15 s and a constant inspiratory flow of 30 L·min<sup>-1</sup>. The two chambers were physically connected by a small metal component that allowed the driving chamber to lift the PSV pressurized chamber, mimicking the patient's contribution to inspiration. Thus, generation of positive pressure in the driving chamber by the CPU ventilator lowered the pressure in the PSV pressurized chamber to subatmospheric levels, just as negative alveolar pressures are produced *in vivo* when the inspiratory muscles contract. This effect was detected by the triggering system of the PSVs under test. Because the metal component was not secured to the PSV pressurized chamber, the latter, once effectively pressurized, could rise above the driving chamber. The compliance of both chambers was set at 82 mL·cm H<sub>2</sub>O<sup>-1</sup>. PEEP was applied to the driving chamber at a level that ensured synchrony of motion of the two chambers at

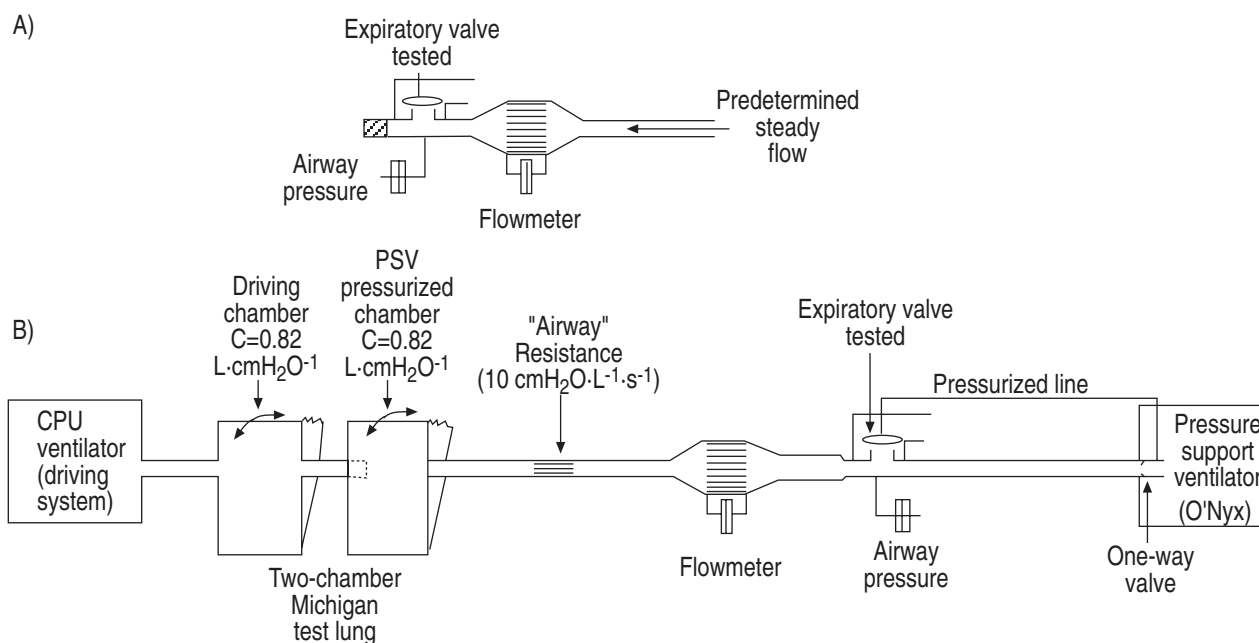


Fig. 1. — Experimental set up: A) in the quasi-static condition; and B) in the dynamic condition. In the quasi-static condition, a variable flow was adjusted and pressure and flow were measured at the extremity of the expiratory valve circuit. In the dynamic condition, the circuit of the expiratory valve was connected to a two-chamber Michigan test lung. One chamber (driving chamber) of the test lung was attached to and powered by a ventilator; the other chamber was connected to a pressure-support ventilator (PSV pressurized chamber). The two chambers were physically connected by a small metal component that allowed the driving chamber to lift the PSV pressurized. Because the metal component was not secured to the PSV pressurized chamber, the latter could expand freely as dictated by the pressure support level and could deflate freely during expiration. Pressure and flow were measured at the extremity of the ventilator circuit. CPU ventilator: Ohmeda, Mauvepas, France.

onset of inspiration. A resistance of 10 cmH<sub>2</sub>O at 1 L·s<sup>-1</sup> was used to connect the device being studied to the lung model. Under these conditions, tidal volume in the pressurized chamber was 770 mL.

To measure the expiratory work imposed by the expiratory valve (WEV), a Fleisch#2 pneumotachograph was inserted between the lung model and the circuit of the tested device, and airway pressure ( $P_{aw}$ ) was measured at the distal end of the circuit using a differential pressure transducer (Validyne MP45 ± 70 cmH<sub>2</sub>O, Northridge, CA). Signals were digitized at 128 Hz and sampled using an analogic/numeric system (MP100; Biopac Systems, Goleta, USA). WEV was calculated based on the area between the recorded volume-pressure curve during expiration and the same reference pressure corresponding to the end-expiratory pressure (fig. 2):

$$W_{EV} = \frac{1}{2} \int_{end\ tI}^{end\ tE} P_{aw} \cdot V' dt$$

where  $V'$  is the flow given by the pneumotachograph, and  $tI$  and  $tE$  are the inspiratory and expiratory times, respectively.

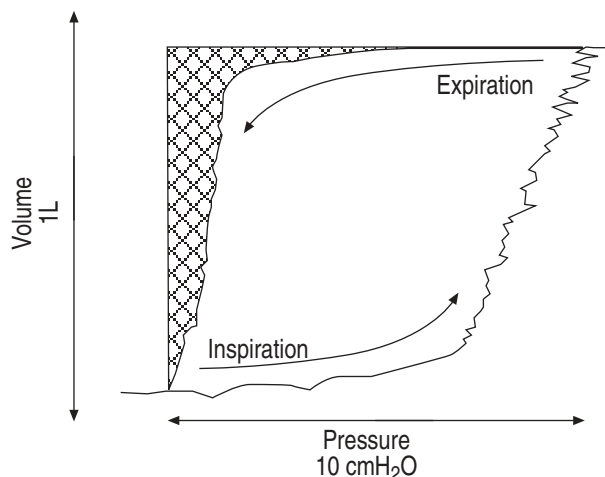


Fig. 2. – Volume-pressure curve obtained in the dynamic condition (fig. 1b). : expiratory work.

**Statistics.** Means and standard deviations were determined from a total of 15 valves for each valve model. Comparisons were made using paired Wilcoxon's tests. The level of significance was set at 5%.

#### Clinical study

**Valves tested.** Two valves were tested: 1) the valve that was least resistive at 0.5 and 1 L·s<sup>-1</sup> in the quasi-static study and generated the least expiratory work of breathing in the dynamic study (Ref TT 11 372-00; Bennett, San Diego, Mexico); and 2) the valve that was most resistive at 0.5 and 1 L·s<sup>-1</sup> in the quasi-static study and generated the most expiratory work of breathing in the dynamic study (Ref 40-314160, Peters, Bobigny, France).

**Patients.** This study was approved by the Research Ethics Committee of our institution. All patients gave informed consent to participation in the study. Ten patients aged 36–72 yrs were included. Their main clinical and respiratory features are listed in table 2. At the time of the study, all 10 patients were intubated or tracheotomized, and none could breathe spontaneously for more than 15 min. Despite differences in the causes of respiratory failure, all patients satisfied the following criteria: 1) respiratory frequency ranging 15–35 breaths·min<sup>-1</sup> without PEEP and with an inspiratory pressure support (IPS) level under 20 cmH<sub>2</sub>O; 2) arterial oxygen tension ( $P_{a,O_2}$ ) above 9.3 kPa (70 mmHg) with an inspired oxygen fraction ( $F_{I,O_2}$ ) under 50%; and 3) haemodynamic stability (without vasopressive drugs) and apyrexia.

**Methods.** An arterial line was inserted before measurements were started, to allow repeated arterial blood gas analysis. Blood gases were measured using an ABL 30 analyser (Radiometer, Copenhagen, Denmark).

Flows were measured using a Fleisch #2 pneumotachograph connected to a differential pressure transducer (Validyne MP 45±5 cmH<sub>2</sub>O, Northridge, CA, USA). The flow signal was integrated to yield volume. Respiratory frequency and minute ventilation were calculated from the flow signal.  $P_{aw}$  was measured at the proximal end of the

Table 2. – Characteristics of the 10 patients studied

Patient No.	Sex M/F	Age yrs	Duration of ventilation days	IPS level cmH <sub>2</sub> O	Diagnosis at admission	Outcome
1	F	36	8	15	Myasthenia	Survived
2	M	64	15	22	COPD	Survived
3	M	59	41	20	Pneumonia Pneumonia Pancreatitis	Tracheotomy Survived
4	M	53	2	20	Pneumonia	Survived
5	F	72	11	14	Lymphoma	Survived
6	M	69	2	15	Abdominal surgery	Survived
7	M	60	11	12	COPD	Survived
8	F	47	10	20	Brain infarction Leukaemia Pneumonia	Tracheotomy Survived
9	M	52	13	12	Liver transplant	Survived
10	M	63	10	20	COPD Pneumonia	Survived

M: male; F: female; IPS: inspiratory pressure support; COPD: chronic obstructive pulmonary disease.

endotracheal tube using a differential pressure transducer (Validyne MP 45±70 cmH<sub>2</sub>O, Northridge, CA, USA). Gastric pressure ( $P_{ga}$ ) and oesophageal pressure ( $P_{oes}$ ) were measured using a probe with two piezo-electric sensors inserted through a nostril and advanced until the distal sensor was in the stomach and the proximal one was in the lower third of oesophagus (probe and amplifier Gaeltec S7B/2; Gaeltec Ltd. Dunvegan, Isle of Skye, UK). Correct positioning of the transducers was verified by observing  $P_{ga}$  fluctuations produced by applying gentle pressure to the subject's gastric area and by looking for a sharp increase in  $P_{oes}$  on the  $P_{oes}$  tracing when an oesophageal contraction was induced by asking the subject to drink water. In addition, the oesophageal balloon was positioned at the appropriate site after a pressure manoeuvre consisting of an occlusion test [9].

All signals were digitized at 128 Hz and sampled for subsequent analysis using an analogic/numeric acquisition system (MP100; Biopac System, Goleta, USA).

Expiratory muscle activity was evaluated as, described previously, from the changes in  $P_{ga}$  [10, 11]. The direct mechanical effect of expiratory contraction of the abdominal muscles is an increase in  $P_{ga}$  during the expiratory phase of the breathing cycle. Therefore, we measured from the  $P_{ga}$  trace the decrease from the maximal end-expiratory level to the minimal value ( $\Delta P_{ga}$ ).

The  $PTP_{oes}$  was measured as the area enclosed within  $P_{oes}$  and the chest wall static recoil pressure ( $P_{stw}$ )-time curve over  $tI$ , taking into account the PEEP<sub>i</sub> or, if  $\Delta P_{ga}$  was positive, the corrected PEEP<sub>i</sub> (corrected PEEP<sub>i</sub> = PEEP<sub>i</sub> -  $\Delta P_{ga}$ ), as in previous studies [11, 12]. PEEP<sub>i</sub> was defined as the  $P_{oes}$  decrease required to bring the expiratory flow abruptly to zero. The  $P_{stw}$ -time curve was extrapolated to the predicted value of chest wall compliance ( $C_{stw}$ ), which theoretically contributes about 4% of the predicted value of vital capacity per centimetre of water. Thus, the slope of the  $P_{stw}$ -time relationship was:

$$(\Delta VT/C_{stw})/\Delta tI$$

where  $\Delta VT$  is change in tidal volume and  $\Delta tI$  is change in inspiratory time.

$PTP_{oes}$  was multiplied by the respiratory frequency and expressed in cmH<sub>2</sub>O·s·min<sup>-1</sup>.

After elimination of abnormal cycles characterized by coughing or excessively small tidal volumes, the mean value for at least 10 breaths was determined for each parameter.

**Protocol.** Measurements with the two valves were performed in the morning of the same day, and lasted 3–4 h. All patients were studied in a semirecumbent position. Endotracheal suctioning was performed before each study

period. Settings of pressure support ventilation were strictly identical with the two valves. The home pressure device used for this study was the O'Nyx (Pierre Médical, Puritan Bennett, France), whose performances have been found to be similar to those of intensive care unit devices. Because the O'Nyx device does not allow  $F_{I,O_2}$  to be set, all patients were ventilated with room air, and an additional flow of oxygen was added to the ventilatory circuit if necessary and kept constant throughout the study.

The two valves were tested in random order. Patients were ventilated with each valve for 20 min. Respiratory parameters were recorded during the last 5 min of each test period. Arterial samples for blood gas analysis were collected at the end of each period.

**Statistics.** Data are given as means±SD. Comparisons were made using paired Wilcoxon's tests. The level of significance was set at 5%.

## Results

### Experimental in vitro study

The pressure-flow relationships obtained under quasi-static conditions with each expiratory valve are presented in table 3. When flow was set at 0.5 L·s<sup>-1</sup>, pressure varied across valves, from 1.27–2.03 cmH<sub>2</sub>O. When flow was set at 1 L·s<sup>-1</sup>, greater than twofold pressure increases were seen across valves. Under dynamic conditions, expiratory work of breathing varied across valves, from 0.10 J·cycle<sup>-1</sup> for the Bennett expiratory valve to 0.16 J·cycle<sup>-1</sup> for the Peters expiratory valve. The dynamic expiratory pressure-volume relationship of these two valves are presented in figure 3. Interestingly, we found a highly significant correlation between imposed expiratory work of breathing in the dynamic study and pressure values observed at 1 L·s<sup>-1</sup> in the static study ( $r=0.92$ ,  $p<0.03$ ) (fig. 4).

### Clinical study

The main characteristics of the 10 patients are presented in table 2. Nine patients required addition of oxygen (2–4 L·min<sup>-1</sup>). No significant differences were observed between the periods of ventilation with the Peters and the Bennett valves, in terms of arterial blood gases, respiratory parameters or PEEP<sub>i</sub> (table 4).  $PTP_{oes}$  increased significantly with the Peters valve (table 4). An analysis of individual responses showed that the three patients (patients 3, 7 and 10) with the highest minute ventilations (above 15 L·min<sup>-1</sup>) and the shortest expiratory times exhibited substantial increases in both PEEP<sub>i</sub> and  $PTP_{oes}$  with the Peters

Table 3. – Expiratory flow-pressure relationships through valves

Flow L·s <sup>-1</sup>	Bennett	Peters	Taema	Sims	Pierre M.
0.25	0.88±0.99	0.80±0.10	0.87±0.27	0.64±0.18*	1.18±0.20*
0.50	1.29±0.11	1.90±0.19*	1.49±0.35	1.27±0.24	2.03±0.23*
1.00	1.87±0.24	4.44±0.44*	2.61±0.50*	2.98±0.26*	3.41±0.25*
1.50	2.4±0.33	7.44±0.69*	3.73±0.95	5.25±0.35*	4.52±0.30*

\*:  $p<0.05$  versus Bennett (Wilcoxon test).

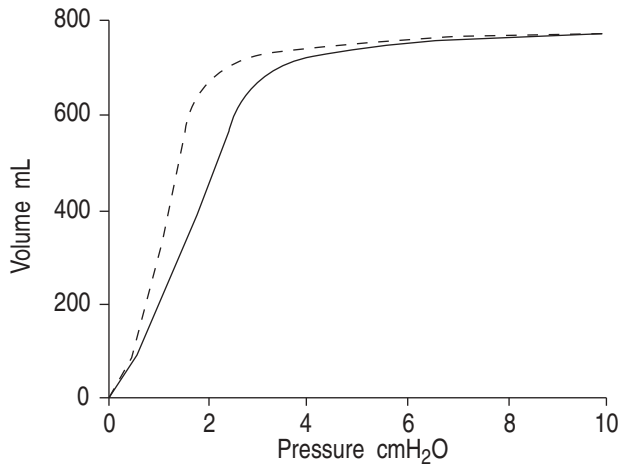


Fig. 3. - Illustration of the pressure volume relationship during expiration in the dynamic condition with the least (Bennett; - - -) and the most (Peters; —) resistive expiratory valve.

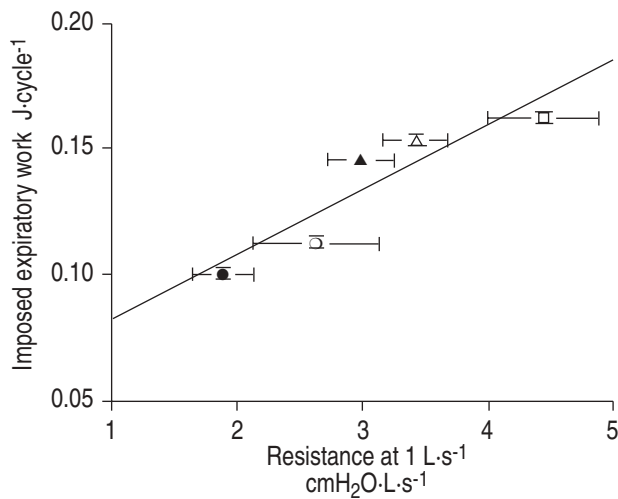


Fig. 4. - Plot of the imposed expiratory work of breathing obtained in the dynamic condition as a function of the expiratory resistance obtained in the quasi-static condition at 1 L·s<sup>-1</sup> for the five expiratory valves tested. A significant correlation was observed ( $p < 0.03$ ;  $r = 0.92$ ). ●: Bennett; ○: Taema; ▲: Sims; □: Pierre; ◻: Peters.

Table 4. - Mean values ( $\pm$ SD) of breathing pattern, arterial blood gases and oesophageal pressure-time product with the Bennett and the Peters expiratory valves

	Bennett	Peters
$V_T$ mL	553 $\pm$ 99	557 $\pm$ 93
$V'_E$ L·min <sup>-1</sup>	13.7 $\pm$ 3.4	13.7 $\pm$ 3.5
$f_R$ breaths·min <sup>-1</sup>	25.6 $\pm$ 7.7	25.5 $\pm$ 8.1
PEEPi cmH <sub>2</sub> O	2.0 $\pm$ 2.4	2.8 $\pm$ 3.5
PTP <sub>oes</sub> cmH <sub>2</sub> O·s·min <sup>-1</sup>	194 $\pm$ 90	236 $\pm$ 113*
Arterial pH	7.38 $\pm$ 0.10	7.38 $\pm$ 0.10
$P_{a,O_2}$ mmHg	136 $\pm$ 54	137 $\pm$ 63
$P_{a,CO_2}$ mmHg	42 $\pm$ 11	42 $\pm$ 11

\*:  $p < 0.05$  versus Bennett.  $V_T$ : tidal volume;  $V'_E$ : minute ventilation;  $f_R$ : respiratory frequency; PEEPi: intrinsic positive end-expiratory pressure; PTP<sub>oes</sub>: oesophageal pressure-time product;  $P_{a,O_2}$ : arterial oxygen tension;  $P_{a,CO_2}$ : arterial carbon dioxide tension; 1 mmHg=0.133 kPa.

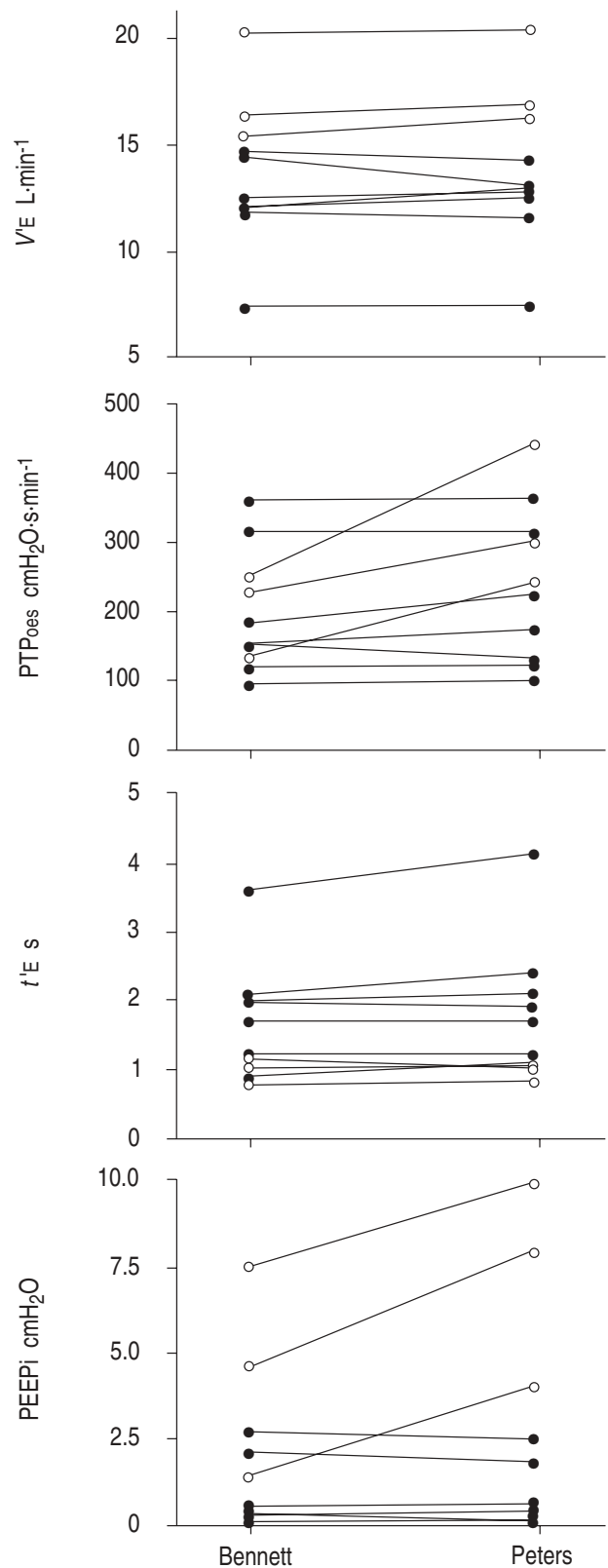


Fig. 5. - Individual values of minute ventilation ( $V'_E$ ), expiratory time ( $t'_E$ ), oesophageal pressure-time product (PTP<sub>oes</sub>) and intrinsic positive end-expiratory pressure (PEEPi) obtained in nine patients with the least (Bennett) and the most (Peters) resistive expiratory valve. Open circles represent the three patients who presented an increase in PEEPi and PTP<sub>oes</sub> with the Peters expiratory valve.

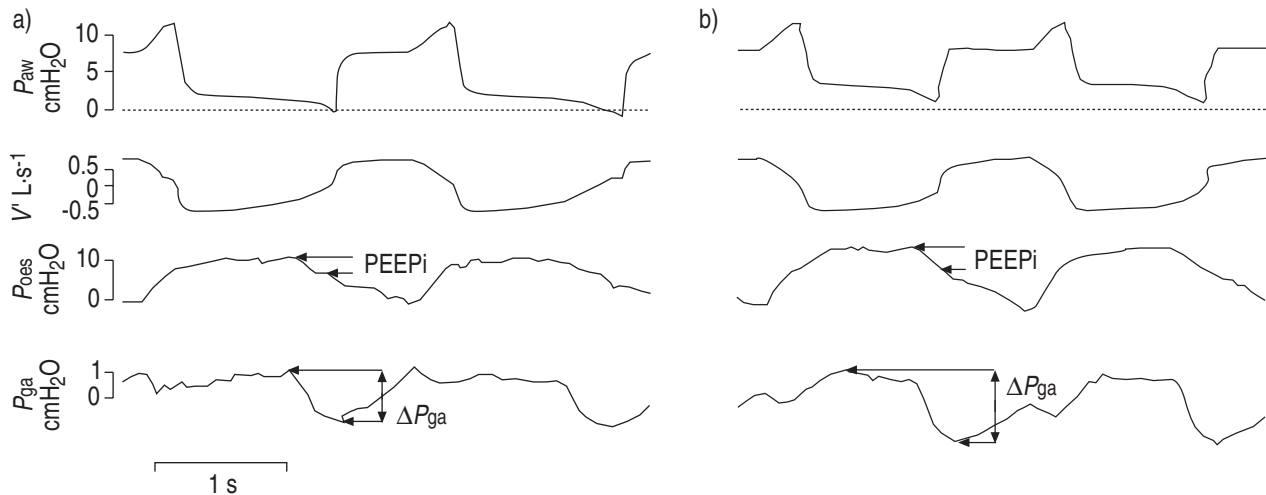


Fig. 6. – Representative recordings of airway pressure ( $P_{aw}$ ), flow ( $V$ ), oesophageal pressure ( $P_{oes}$ ) and gastric pressure ( $P_{ga}$ ) in patient 7. Tracings demonstrated increases in  $P_{aw}$  during expiration, intrinsic positive end-expiratory pressure (PEEPi), and decline in  $P_{ga}$  ( $\dot{y}P_{ga}$ ) with: a) the expiratory Peters valve as compared to b) the expiratory Bennett valve.

valve (fig. 5). In addition, two of these three patients (patients 7 and 10) demonstrated detectable expiratory activity, which increased with the Peters valve, as evidenced by an increase in  $\Delta P_{ga}$  from 1.8 to 2.5 cmH<sub>2</sub>O (patient 7; fig. 6) and from 3.7 to 4.3 cmH<sub>2</sub>O (patient 10). No expiratory activity could be detected on gastric pressure tracings in the other patients.

### Discussion

Resistance through the expiratory limb of the circuit is due primarily to the exhalation valve. Excessive resistance of the expiratory valve can impair exhalation, which can result in PEEPi. Minor fluctuations in PEEPi may have little practical relevance when expiration occurs passively and inflation is driven by the ventilator. However, during assisted ventilation, it is inspiratory flow initiation by the patient that triggers ventilator assistance. Any PEEPi induced by expiratory valve resistance is a supplemental inspiratory threshold load that must be counterbalanced before inspiratory flow can start and initiate ventilator assistance. In addition, the expiratory activity seen in some patients with acute or stable conditions prior to ventilation [10, 11] may be increased by expiratory valve resistance [13].

Among available assisted ventilation modalities, PSV has been widely used [1, 14]. More recently, PSV *via* a nasal mask has been widely advocated as home treatment for chronic respiratory failure [15–17]. This prompted us, in a recent study, [5] to investigate the performances of various home PSV devices. We found several differences between devices, including marked variation in expiratory resistance. After this study was published, several manufacturers suggested that a range of commercially available expiratory valves could be used on the same IPS devices. Therefore, we designed the present study to evaluate the specific performances of several available expiratory valves and the clinical relevance of any differences in these performances.

In an early study by MARINI *et al.* [6] of the resistance of various intensive care expiratory circuits, the diaphragm

valve, as compared to other designs such as scissor valves, was found to produce a level of resistance of similar magnitude to that of a 5 mm endotracheal tube. Such a high level of resistance may be clinically important in patients supported by mechanical ventilation during the hyperpnoeic or weaning phase of a respiratory illness. Incidentally, although remarkable technical improvements have been made on ventilators in the last 10 yrs, the Peters expiratory valve tested in the present study behaved similarly, in both the quasi-static and dynamic conditions, to the diaphragm valve tested in the study by MARINI *et al.* [6]. In general, we found marked differences across the commercially-available expiratory valves tested in our study. For example, the resistance at 1 L·s<sup>-1</sup> in the quasi-static analysis was reduced by more than 50% and the imposed expiratory work in the dynamic analysis by 30% with the Bennett expiratory valve *versus* the Peters expiratory valve. We investigated the potential clinical relevance of these differences. For the reasons explained above, we expected to find higher PEEPi, and consequently PTP<sub>oes</sub>, with the most resistive compared to the least resistive valve. However, in intubated patients being weaned from mechanical ventilation we were unable to demonstrate significant differences between these two valves regarding blood gas parameters, respiratory parameters or PEEPi. We did observe a significant increase in PTP<sub>oes</sub> with the Peters valve despite the absence of any significant change in PEEPi.

The alveolar expiratory pressure-time curve has been described mathematically by a first-order exponential function, which, at the end of expiration, equals PEEPi and can be expressed as [18, 19]:

$$PEEPi = (V_T/C)e^{-t/RC}$$

where R is resistance, C is compliance and RC is the time constant of the respiratory system. R includes the resistance of the expiratory circuit, so that an increase in expiratory valve resistance results in an increase in PEEPi through the increase in time constant.

TUXEN and LANE [20] validated this mathematical model in mechanically ventilated patients with severe air-flow obstruction and demonstrated unequivocally that the risk of dynamic hyperventilation was greater when the volume delivered by the ventilator ( $V_T$ ) was increased and  $t_E$  reduced. Consistent with these findings, our analysis of individual responses showed that the three patients with the highest minute ventilation (above  $15 \text{ L}\cdot\text{min}^{-1}$ ) and the shortest expiratory time (fig. 5) exhibited substantial increases in PEEP<sub>i</sub> and, consequently, in PTP<sub>oes</sub>. In addition, the significant increase in PTP<sub>oes</sub> observed with the Peters valve was mainly due to these three patients. Furthermore, for two of these three patients, part of the increase in PEEP<sub>i</sub> with the Peters expiratory valve was explained by an increase in expiratory activity evidenced by an increase in  $\Delta P_{ga}$  (fig. 6).

We conclude that important differences in resistance exist between commercially-available expiratory valves for home ventilator devices. In addition, these differences may influence both inspiratory and expiratory efforts in patients with a marked ventilatory demand and/or a short expiratory time. For these reasons, as previously recommended for intensive care ventilators [6], it is imperative that greater attention be devoted to the design and selection of expiratory components of ventilator circuits.

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### References

- Hill N. Noninvasive ventilation. Does it work, for whom, and how? *Am Rev Respir Dis* 1993; 147: 1050–1055.
- Pennock B, Kaplan P, Carlin B, Sabangan J, Magovern J. Pressure support ventilation with a simplified ventilatory support system administered with a nasal mask in patients with respiratory failure. *Chest* 1991; 100: 1371–1376.
- Strumpf D, Carlisle C, Millman R, Smith K, Hill N. An evaluation of the Respironics BiPAP Bi-Level CPAP device for delivery of assisted ventilation. *Respir Care* 1990; 35: 415–422.
- Lofaso F, Brochard L, Touchard D, Hang T, Harf A, Isabey D. Evaluation of carbon dioxide rebreathing during pressure support ventilation with BiPAP devices. *Chest* 1995; 108: 772–778.
- Lofaso F, Brochard L, Hang T, Lorino H, Harf A, Isabey D. Home versus intensive-care pressure support devices, experimental and clinical comparison. *Am J Respir Crit Care Med* 1996; 153: 1591–1599.
- Marini J, Culver B, Kirk W. Flow resistance of exhalation valves and positive end-expiratory pressure devices used in mechanical ventilation. *Am Rev Respir Dis* 1985; 131: 850–854.
- Kayaleh R, Wilson A. Mechanisms of expiratory valves resistance. *Am Rev Respir Dis* 1988; 137: 1390–1394.
- Banner M, Lampotang S. Expiratory pressure valve. In: Branson R, Hess D, Chatburn R, eds. *Respiratory Care Equipment*. Philadelphia, Lippincott Company, 1995; pp. 479–488.
- Baydur A, Behrakis PK, Zin WA, Jaeger M, Milic-Emili J. A simple method for assessing the validity of the esophageal balloon technique. *Am Rev Respir Dis* 1982; 126: 788–791.
- Ninane V, Yernault JC, Troyer AD. Intrinsic PEEP in patients with chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1993; 148: 1037–1042.
- Lessard MR, Lofaso F, Brochard L. Expiratory muscle activity increases intrinsic positive end-expiratory pressure independently of dynamic hyperinflation in mechanically ventilated patients. *Am J Respir Crit Care Med* 1995; 151: 562–569.
- Yan S, Kayser B, Tobiasz M, Sliwinski P. Comparison of static and dynamic intrinsic positive end-expiratory pressure using the Campbell diagram. *Am J Respir Crit Care Med* 1996; 154: 938–944.
- Campbell E. The effects of increased resistance to expiration on the respiratory behavior of the abdominal muscles and intra-abdominal pressure. *J Physiol* 1957; 136: 556–563.
- Brochard L. Non-invasive ventilation: practical issues. *Intens Care Med* 1993; 19: 431–432.
- Nava S, Ambrosino N, Rubini F, et al. Effect of nasal pressure support ventilation and external PEEP on diaphragmatic activity in patients with severe stable COPD. *Chest* 1993; 103: 145–150.
- Carrey Z, Gottfried S, Levy R. Ventilatory muscle support in respiratory failure with nasal positive pressure ventilation. *Chest* 1990; 97: 150–158.
- Strumpf D, Millman R, Carlisle C, et al. Nocturnal positive pressure ventilation via nasal mask in patients with severe chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1991; 144: 1234–1239.
- Brody A. Mechanical compliance and resistance of the lung-thorax calculated from the flow recorded during passive expiration. *Am J Physiol* 1954; 178: 189–196.
- Ligas J, Moslehi F, Epstein M. Occult positive end-expiratory pressure with different types of mechanical ventilators. *J Crit Care* 1990; 5: 95–100.
- Tuxen D, Lane S. The effect of ventilatory pattern on hyperinflation, airway pressure and circulation in mechanical ventilation of patients with severe air-flow obstruction. *Am Rev Respir Dis* 1987; 136: 872–879.