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Effect of pressure support ventilation on breathing patterns and respiratory work

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Abstract. We assessed the effect of pressure support ventilation (PSV) on breathing patterns and the work of breathing in 10 postoperative patients. Minute ventilation ($\dot{V}_E$) increased by 8% with 5 cm H_2O PSV and 10% with 10 cm H_2O PSV compared to 0 cm H_2O PSV. The increase in $\dot{V}_E$ was achieved by increased mean inspiratory flow (24% with 5 cm H_2O PSV and 67% with 10 cm H_2O PSV) and a decrease in duty cycle (13% with 5 cm H_2O PSV and 39% with 10 cm H_2O PSV). The decrease in duty cycle along with a decrease in respiratory frequency allowed a greater expiratory time including a rest period for the respiratory muscles, which might minimize the risk of muscle fatigue. Furthermore, the inspiratory work added by the ventilator was near zero with 5 cm H_2O PSV and 10 cm H_2O PSV. Oxygen consumption also decreased significantly with 5 cm H_2O PSV. We conclude that PSV improves the breathing patterns and minimizes the work of breathing spontaneously via a ventilator.

Key words: Breathing pattern – Pressure support ventilation – Work of breathing

Pressure support ventilation (PSV) has been reported to decrease the work of spontaneous breathing [1–4]. However, there are few data of the clinical setting at different levels of PSV [5]. Furthermore, PSV is designed not only to reduce the work of spontaneous breathing but also to improve synchrony between the patient and the ventilator. With PSV, the patient controls ventilatory timing and interacts with the delivered flow to determine the inspiratory flow [6]. Therefore, breathing patterns with PSV might be different from those with continuous positive airway pressure breathing. However, breathing patterns at different levels of PSV and their relationship with the work of breathing have not been investigated. We therefore studied the effect of PSV on breathing patterns and respiratory work in postoperative patients.

Materials and methods

Patients. Ten patients, 9 men and 1 woman, with a mean age of 63 years (range, 33 to 80 years) were studied. The effective compliance of the respiratory system, which was obtained by dividing tidal volume by the difference between the end-inspiratory pause pressure and the end-expiratory pressure during controlled ventilation, was 0.050 ± 0.025 L·cm H_2O^{-1}. Airway resistance, which was obtained by dividing the difference between the peak inspiratory pressure and end-inspiratory pause pressure by the inspiratory flow, was 8 ± 2 cm $H_2O \cdot L^{-1} \cdot s^{-1}$. They each required respiratory support after a major surgical procedure. They were being weaned from mechanical ventilatory support with intermittent mandatory ventilation. At the start of this study, all patients were breathing spontaneously via a demand valve of a ventilator with stable respiratory and hemodynamic conditions. Positive end-expiratory pressure (PEEP) was not used. The trigger sensitivity was set at -1 to -2 cm H_2O.

Protocol. Three levels of PSV, 0, 5 and 10 cm H_2O, were employed in random order with a ventilator (7200a, Puritan-Bennett, CA). Each mode lasted 30 min and measurements were done in the last 10 min of each period. PSV is initiated when the pressure drop meets the trigger sensitivity threshold. Flow then accelerates into the inspiratory circuit to increase the airway pressure to the preset PSV level. This inspiratory flow is regulated by the patient's demand for spontaneous breathing and the preset PSV level. With increasing levels of PSV, the inspiratory flow increases. Exhalation begins when inspiratory flow falls to 5 L·min^{-1} or below, or if airway pressure exceeds the preset PSV level by 1.5 cm H_2O. Informed consent was obtained from all patients and families.

Measurements and calculations

Airway pressure and flow were measured simultaneously at the proximal end of the endotracheal tube using a differential pressure transducer (MP-45, Validyne, CA) and a hot wire flowmeter (RM-300, Minato, Osaka, Japan). The inspired and expired gas concentrations were determined by a mass spectrometer (MGA-1100A, Perkin-Elmer, CA).

Tidal volume (V_T) and minute ventilation ($\dot{V}_E$) were obtained by integrating the flow signal. From the flow signal, respiratory frequency (f), inspiratory time (T_I) and total respiratory cycle duration (Ttot) were measured. The duty cycle, which was defined as the ratio of T_I to Ttot (T_I/Ttot), and mean inspiratory flow (V_T/T_I) were calculated.

To assess the work of spontenous breathing, the inspiratory work (W_I) added by the ventilator was measured. This superimposed inspiratory work can be calculated by integration of the product $P(t) \times \dot{V}(t)$, where P(t) is the airway pressure and $\dot{V}(t)$ is the airway flow at the instant t during the inspiratory period when P(t) is negative [7].

Oxygen consumption ($\dot{V}O_2$) was calculated on a breath by breath basis using the following equation:

$$\dot{V}O_2 = (1-F_{IO_2})^{-1} \times (F_{IO_2} \cdot \dot{V}_E - F_{EO_2} \cdot \dot{V}_E - F_{IO_2} \cdot \int F_{ECO_2}(t)\dot{V}_E(t)\,dt)$$

where F_{IO_2} and F_{EO_2} are inspired and expired oxygen fractions, respectively, and $F_{ECO_2}(t)$ is the expired carbon dioxide fraction at the instant t.

Arterial blood samples were obtained via a catheter inserted in a radial artery and analyzed with a blood gas analyzer (ABL4, Radiometer, Copenhagen).

All data are presented as mean ±SD. Statistical analysis was done by one-way analysis of variance. The alpha level was set at 0.05.

Results

The mean values of ventilatory parameters and blood gas tensions measured in this study are listed in Table 1.

$\dot{V}_E$ increased by 8% with 5 cm H_2O PSV and 10% with 10 cm H_2O PSV, when compared with 0 cm H_2O PSV. V_T also increased markedly by 16% with 5 cm H_2O PSV and 31% with 10 cm H_2O PSV. The large increase in V_T was associated with a decrease in f. V_T/T_I increased by 24% with 5 cm H_2O PSV and 67% with 10 cm H_2O PSV. On the contrary, T_I/Ttot decreased by 13% with 5 cm H_2O PSV and 39% with 10 cm H_2O PSV (Fig. 1). The mean value of W_I changed from 350 $g \cdot cm \cdot breath^{-1}$ without PSV to 6 and 5 $g \cdot cm \cdot breath^{-1}$ with 5 cm H_2O PSV and 10 cm H_2O PSV, respectively (Fig. 2). $\dot{V}O_2$ decreased significantly by 7% with 5 cm H_2O PSV. With 10 cm H_2O PSV, the decrease in $\dot{V}O_2$ was not significant. PaO_2 did not change significantly with PSV. $PaCO_2$ decreased significantly with PSV, according to $\dot{V}_E$ increase.

Discussion

The principal observations in this study were that PSV increased mean inspiratory flow and decreased both duty cycle and f, which allowed for a longer expiratory time including rest period. PSV also reduced the work of breathing estimated as measured by W_I and the change in $\dot{V}O_2$.

With increasing levels of PSV, $\dot{V}_E$ and V_T increased with a decrease in f. Ershowsy et al. [6] also reported a decrease in f and an increase in V_T during PSV in ventilator dependent patients. However, they did not study the effects of PSV on T_I/Ttot and expiratory time. $\dot{V}_E$ was analyzed in terms of V_T/T_I and T_I/Ttot as shown in the following equation [8]: $\dot{V}_E = V_T \times f = V_T/T_I \times T_I/Ttot \times 60$. The schematic breathing patterns are represented in Fig. 3. Increased $\dot{V}_E$ was achieved only by increased V_T/T_I. T_I/Ttot, however, decreased with PSV. This decrease allows the diaphragm a longer resting period between contractions, which might prolong the endurance time and minimize the risk of diaphragmatic fatigue [9, 10]. Brochard et al. [2] also reported that 10 cm H_2O PSV reduced the transdiaphragmatic pressure, which was another important factor of diaphragmatic fatigue. Furthermore, a longer expiratory time with PSV made it easier for the patient to synchronize with the ventilator. In the patients studied, auto-PEEP was not apparent from the recording of flow and airway pressure curves. However, in patients in whom auto-PEEP is present, the inspiratory muscles have to sustain supplementary inspiratory work to overcome auto-PEEP [11]. Thus, a longer expiratory time with PSV might have an advantage in the prevention or reduction of auto-PEEP in such patients.

Table 1. Ventilatory parameters and blood gas tensions during pressure support ventilation

	PSV 0	PSV 5	PSV 10
$\dot{V}_E$ ($L \cdot min^{-1} \cdot m^{-2}$)	4.186± 0.760	4.537± 0.662*	4.590± 0.575*
V_T (ml)	396 ±146	459 ±171*	520 ±200*
f (min^{-1})	19.6 ± 7.1	18.5 ± 6.6	16.2 ± 4.9*
V_T/T_I ($ml \cdot s^{-1}$)	321 ± 90	399 ±107*	536 ±100*
T_I/Ttot	0.38 ± 0.08	0.33± 0.05*	0.24 ± 0.03*
W_I ($g \cdot cm \cdot breath^{-1}$)	350 ±394	6 ± 4*	5 ± 3*
$\dot{V}O_2$ ($ml \cdot min^{-1} \cdot m^{-2}$)	126 ± 19	117 ± 17*	119 ± 16
PaO_2 (mmHg)	140 ± 24	140 ± 28	139 ± 31
$PaCO_2$ (mmHg)	42 ± 6	40 ± 5*	39 ± 5*

Values are mean ± SD; PSV 0, 5, 10 = pressure support ventilation at 0, 5, 10 cm H_2O; *$p < 0.05$ compared with PSV 0

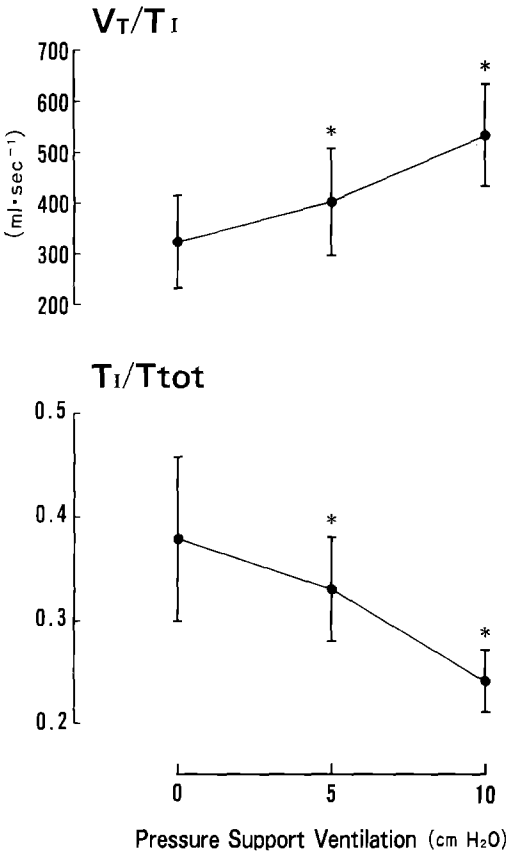

Fig. 1. Duty cycle ($T_I/Ttot$) and mean inspiratory flow (V_T/T_I) during pressure support ventilation (*PSV*). With increasing levels of PSV, V_T/T_I increased with a decrease in $T_I/Ttot$. Data are mean ±SD. *$p<0.05$ compared with 0 cm H_2O PSV

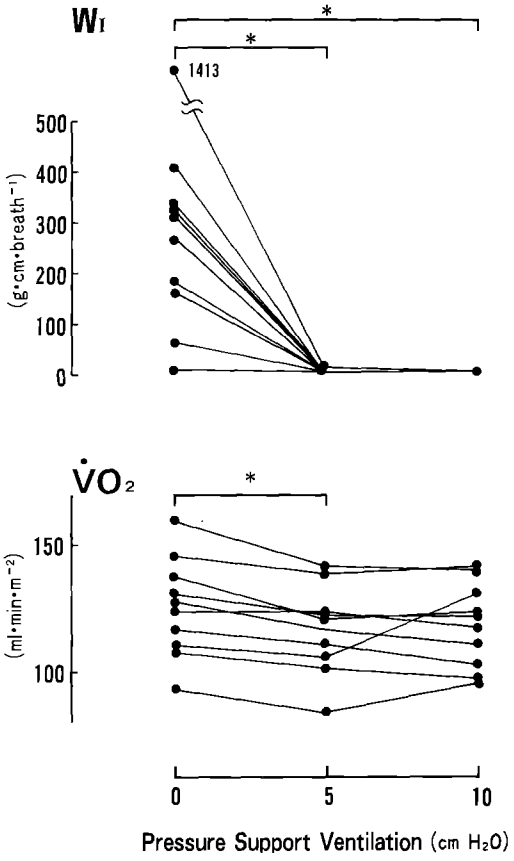

Fig. 2. The individual values of the inspiratory work added by the ventilator (W_I), and oxygen consumption ($\dot{V}O_2$) during pressure support ventilation

MacIntyre [1] proposed that PSV might decrease the work of breathing in a mechanical ventilatory system model simulating patients requiring intermittent mandatory ventilation. Fiastro et al. [3] also demonstrated that PSV could be used to compensate for the inspiratory work due to artificial airway resistances in 4 normal subjects, but they did not report its use in the clinical setting. Our study demonstrated that the inspiratory work added by the ventilator was near zero with PSV at levels of both 5 cm H_2O and 10 cm H_2O. Therefore, the problems of inspiratory work superimposed by the ventilator should be ameliorated by PSV.

Viale et al. [4] demonstrated that $\dot{V}O_2$ was decreased with 15 cm H_2O PSV when compared with continuous positive airway pressure breathing in 7 postoperative patients, but they did not report $\dot{V}O_2$ at lower levels of PSV usually used in patients during the weaning process. Kanak et al. [12] described one patient who demonstrated a marked decrease in $\dot{V}O_2$ when PSV was used. In our study, $\dot{V}O_2$ was decreased with 5 cm H_2O PSV. Our result reflects the reduction of external inspiratory work. With 10 cm H_2O PSV, $\dot{V}O_2$ showed a tendency to decrease, but this change

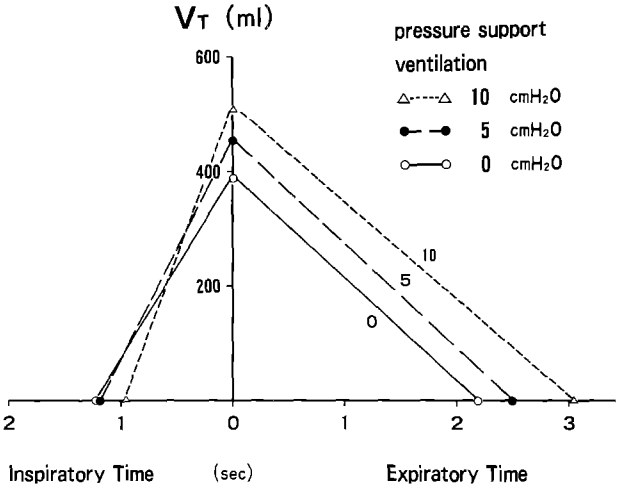

Fig. 3. Schematic spirogram. *Points* on the vertical axis are mean tidal volume (V_T). The horizontal axis represents mean inspiratory and expiratory time. *Slopes of lines* to the left of the vertical axis give mean inspiratory flow

was not significant. The absence of significance might be due to the limitation of resolution of our method.

We emphasize that we measured only the added work by the ventilator, which could be a small factor of the total work of breathing. Although the low levels of PSV can eliminate the added work and decrease $\dot{V}O_2$, such low levels of PSV cannot suppress completely the total work of breathing. Our results may not apply to patients with severe respiratory failure. Such patients may need higher levels of PSV. More studies are needed before these results can be applied to patients with severe respiratory failure using higher levels of PSV.

In conclusion, PSV increased mean inspiratory flow with a decrease in the duty cycle and minimized the work of breathing spontaneously via a ventilator.

References

1. MacIntyre NR (1986) Respiratory function during pressure support ventilation. Chest 89:677
2. Brochard L, Pluskwa F, Lemaire F (1987) Improved efficacy of spontaneous breathing with inspiratory pressure support. Am Rev Respir Dis 136:411
3. Fiastro JF, Habib M, Quan SF (1988) Pressure support compensation for inspiratory work due to endotracheal tubes and demand continuous positive airway pressure. Chest 93:499
4. Viale JP, Annat GJ, Bouffard YM, Delafosse BX, Bertrand OM, Motin JP (1988) Oxygen cost of breathing in postoperative patients: pressure support ventilation vs. continuous positive airway pressure. Chest 93:506
5. MacIntyre NR (1987) Pressure support ventilation: effects on ventilatory reflexes and ventilatory-muscle workloads. Respir Care 32:447
6. Ershowsky P, Krieger B (1987) Changes in breathing pattern during pressure support ventilation. Respir Care 32:1011
7. Viale JP, Annat G, Bertrand O, Godard J, Montin J (1985) Additional inspiratory work in intubated patients breathing with continuous positive airway pressure systems. Anesthesiology 63:536
8. Milic-Emili J, Grunstein MM (1976) Drive and timing components of ventilation. Chest 70:131
9. Bellemare F, Grassino A (1982) Effect of pressure and timing of contraction of human diaphragm fatigue. J Appl Physiol 53:1190
10. Clanton TL, Dixon GF, Drake J, Gradek E (1985) Effects of breathing pattern on inspiratory muscle endurance in humans. J Appl Physiol 59:1834
11. Possi A, Gotterried SB, Zocchi L, Higgs BD, Lennox S, Calverley PMA, Begin P, Grassino A, Milic-Emili J (1985) Measurement of static compliance of the total respiratory system in patients with acute respiratory failure during mechanical ventilation. Am Rev Respir Dis 131:672
12. Kanak R, Fahey P, Vanderwarf C (1985) Oxygen cost of breathing. Changes dependent upon mode of mechanical ventilation. Chest 87:126

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