## Short communications



## **Pressure control ventilation in a patient with low respiratory compliance and high airway resistance**

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Pressure control ventilation (PCV) is a new mechanical ventilation modality in which a preset airway pressure is maintained over a preset inspiratory time by modulating the inspiratory flow [1]. The flow pattern of PCV, a rapidly rising and exponentially decelerating waveform, is similar to that of pressure support ventilation (PSV) [2]. If a ventilator works in cooperation with the patient's inspiratory effort, PCV is the same pressure assist mode as PSV except for the difference in inspiratory flow termination.

We report here a patient with low respiratory compliance and high airway resistance and PCV assisted his spontaneous breathing more appropriately than PSV. The effect of PCV on the patient's respiratory parameters was compared with PSV.

A 59-year-old man was admitted to ICU due to respiratory failure after three-segment resection of the liver, cholecystectomy and choledocho-jejunostomy for portal cholangioma. He had undergone thoracoplasty and lobectomy of the left lung for tuberculosis at the age of 19. His preoperative pulmonary function test demonstrated obstructive and restrictive changes, forced expiratory volume in 1s (FEV<sub>1.0</sub>) of  $0.961 \cdot s^{-1}$  [64% of forced vital capacity (FVC)] and FVC of 1.491 (45% of predicted FVC). He was reintubated followed by mechanical ventilation due to hypoxia on the 8th postoperative day. He was admitted to ICU on the 9th postoperative day. Moist rale and wheezing were detected in the right lung on admission. His chest roentgenogram demonstrated pneumonia-like consolidation in the right lung and transformation of the left thorax due to thoracoplastic surgery. Arterial blood gas analysis indi-

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cated Pao<sub>2</sub> of 152.8 mmHg, Paco<sub>2</sub> of 48.4 mmHg, pH of 7.339, and HCO<sub>3</sub><sup>-</sup> of 25.3 mmol·l<sup>-1</sup> (Fio<sub>2</sub> 0.8). The white blood cell count was  $16190 \mu l^{-1}$ . Culture study revealed *Pseudomonas aeruginosa* in his sputum.

Pneumonia progressed in spite of chest physiotherapy and the administration of ceftazidime and tobramycin. Although the patient's respiration was supported by PSV of 25 cmH<sub>2</sub>O plus a positive end-expiratory pressure (PEEP) of 5cmH<sub>2</sub>O, he manifested respiratory distress pattern. Respiratory flow (V), airway pressure (Paw), and esophageal pressure (Pes) were measured with a pulmonary monitor CP100 (BICORE, Irvine, CA, USA) to investigate the cause of insufficient support by PSV (Fig. 1A). After the inspiratory demand flow of PSV had been stopped with a ventilator, Pes still dropped and a second triggering, socalled double breathing, occurred. The inspiratory assist time appeared to be too short because V or Paw reached the PSV termination criteria too early. Airway resistance was high  $(9.8 \text{ cmH}_2\text{O}\cdot\text{s}\cdot\text{I}^{-1})$  and lung compliance was low  $(19.9 \text{ ml} \cdot \text{cmH}_2\text{O}^{-1})$  at control mechanical ventilation (tidal volume 500 ml, inspiratory flow  $401 \cdot m^{-1}$ , and end-inspiratory plateau 2.0 s). We considered that PCV would support the patient's respiration more effectively than PSV because the inspiratory time (TI) could be preselected in PCV. We compared the effects of PCV at 25 cmH<sub>2</sub>O with those of PSV at 25 cmH<sub>2</sub>O. The Newport E200 Wave (Newport, Newport Beach, CA, USA) was used as a ventilator. The PEEP level was set at  $5 \text{ cmH}_2\text{O}$  and PCV T<sub>I</sub> at 0.4, 0.6, and 0.8 s. At a TI of 0.4 s, double breathing occurred, as was the case with PSV (Fig. 1B). At a T<sub>I</sub> of 0.6s, negative deflection of the Pes was minimal (Fig. 1C). When the decelerating inspiratory flow approached zero, the patient initiated expiration. At a TI of 0.8s, Paw exceeded the preset PCV level after inspiratory flow had stopped (Fig. 1D). This phenomenon indicated that the expiratory valve of the ventilator was still closed when the patient began his expiratory effort. From these findings, we determined the optimal PCV TI to be 0.6s for this patient.

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**Fig. 1.** Respiratory flow, airway pressure (*Paw*), esophageal pressure (*Pes*) during pressure support ventilation (*PSV*) (*A*), pressure control ventilation (*PCV*) (T1 0.4 s) (*B*), PCV (T1 0.6 s) (*C*), and PCV (T1 0.8 s) (*D*). With PSV, Pes still dropped and the second triggering occurred after a ventilator stopped

the inspiratory demand flow. With PCV (T1 0.6 s) negative deflection of the Pes was minimal. When decelerating inspiratory flow approached zero, expiration was initiated.  $T_{I}$ , inspiratory time

Table 1. Respiratory characteristics during PSV and PCV (T1 0.6s)

	RR (m <sup>-1</sup> )	VE (L·m <sup>−1</sup> )	Pao <sub>2</sub> ª (mmHg)	Paco <sub>2</sub> <sup>a</sup> (mmHg)	Wv (J·breath <sup>-1</sup> )	WOB (J·breath <sup>-1</sup> )	WOB $(J \cdot m^{-1})$
PSV	39.1	17.5	110.7	47.0	0.77	0.25	9.75
PCV (Ti 0.6 s)	27.8	13.7	135.6	44.6	0.89	0.06	1.67

PSV, pressure support ventilation; PCV, pressure control veatilation; RR, respiratory rate; VE, minute ventilation; Wv, work of ventilator; WOB, patient's work of breathing.

 $^{\rm a}$  Pao\_2 and Paco\_2 were measured at F10\_2 0.8.

The respiratory rate (RR) and minute ventilation  $(V_E)$  decreased during PCV (TI 0.6s) compared with those during PSV (Table 1). PCV caused higher Pao<sub>2</sub> and lower Paco<sub>2</sub> than PSV at FIo<sub>2</sub> 0.8. PCV also produced larger work of ventilator ( $W_V$ ) per breath and smaller patient's work of breathing (WOB) per breath than PSV. Because of the reduction in RR with PCV, the patient's WOB per minute during PCV was much smaller than during PSV.

We considered that the inadequate ventilatory assist of PSV was due to premature termination of inspiratory demand flow resulting in insufficient inspiratory assist time. Recently, PSV has been widely used mainly because of improvements in patient-ventilator synchrony [3,4]. PSV requires little use of sedatives and no muscle relaxants, thus keeping the cough reflex and secretion drainage intact. PSV, however, causes some problems for patients with severely impaired pulmonary function. In patients with low respiratory compliance, inspiratory flow might decrease rapidly and reach the flow termination criterion too early. The flow termination criteria are not changeable on any of the currently available ventilators. If the ventilator does not decelerate the demand flow appropriately, Paw easily exceeds the Paw termination criterion in patients with high airway resistance and low lung compliance. Because our patient showed similar findings with regard to these two parameters, PSV seemed to terminate too early and cause additional inspiratory muscle load. We therefore switched to PCV in order to increase T<sub>1</sub>.

We determined the optimal TI for PCV by measuring Pes,  $\dot{V}$ , and Paw. PCV (TI of 0.6s) reduced the WOB more efficiently than did PSV. Furthermore, PCV produced a higher Pao<sub>2</sub> and lower Paco<sub>2</sub> than did PSV. This was thought to be because PCV decreased dead space ventilation and improved the perfusion ventilation ratio since PCV ventilated alveoli with a large time constant more efficiently than PSV [5].

In conclusion, the use of PCV for a patient with low respiratory compliance and high airway resistance showed that PCV was more effective in improving WOB and alveolar gas exchange than PSV.

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