

# Effect of Patient-triggered Ventilation on Respiratory Workload in Infants after Cardiac Surgery

Muneyuki Takeuchi, M.D.,\* Hideaki Imanaka, M.D.,\* Hiroshi Miyano, M.D.,\* Keiji Kumon, M.D.,† Masaji Nishimura, M.D.‡

**Background:** Patient-triggered ventilation (PTV) is commonly used in adults to avoid dyssynchrony between patient and ventilator. However, few investigations have examined the effects of PTV in infants. Our objective was to determine if pressure-control PTV reduces infants' respiratory workloads in proportion to the level of pressure control. We also explored which level of pressure control provided respiratory workloads similar to those after the extubation of the trachea.

**Methods:** When seven post-cardiac surgery infants, aged 1 to 11 months, were to be weaned with the pressure-control PTV, we randomly applied five levels of pressure control: 0, 4, 8, 12, and 16 cm H<sub>2</sub>O. All patients were ventilated with assist-control mode, triggering sensitivity of 1 l/min, and positive end-expiratory pressure of 3 cm H<sub>2</sub>O. After establishing steady state conditions at each level of pressure control, arterial blood gases were analyzed and esophageal pressure (Pes), airway pressure, and airflow were measured. Inspiratory work of breathing (WOB) was calculated using a Campbell diagram. A modified pressure-time product (PTPmod) and the negative deflection of Pes were calculated from the Pes tracing below the baseline. The measurement was repeated after extubation.

**Results:** Pressure-control PTV supported every spontaneous breath. By decreasing the level of pressure control, respiratory rate increased, tidal volume decreased, and as a result, minute ventilation and arterial carbon dioxide partial pressure were maintained stable. The WOB, PTPmod, and negative deflection of Pes increased as pressure control level was decreased. The WOB and PTPmod at 4 cm H<sub>2</sub>O pressure control and 0 cm H<sub>2</sub>O pressure control and after extubation were significantly greater than those at the pressure control of 16, 12, and 8 cm H<sub>2</sub>O ( $P < 0.05$ ). The WOB and PTPmod were almost equivalent after extubation and at 4 cm H<sub>2</sub>O pressure control.

**Conclusions:** Work of breathing and PTPmod were changed according to the pressure control level in post-cardiac surgery infants. PTV may be feasible in infants as well as in adults. (Key words: Endotracheal tube; pressure support ventilation; pressure-time product.)

PATIENT-triggered ventilation (PTV), including pressure support ventilation (PSV), is commonly used in adults because patient-ventilator synchrony enhances patient

acceptance of mechanical ventilation and decreases the work of breathing (WOB).<sup>1-3</sup> Recently, PTV using pressure-limited ventilation was applied to infants and children.<sup>4-7</sup> For infants, PTV is usually applied as continuous-flow, time- or patient-cycled, pressure-limited ventilation, which is similar to pressure-control ventilation in adults. During PTV, the ventilator is triggered by the inspiratory effort,<sup>6</sup> which improves patient breathing patterns.<sup>7-11</sup> However, only a few investigators have reported the effect of the pressure-control PTV on the WOB of infants.

A number of studies have suggested that PSV can be used to counteract the WOB imposed by endotracheal tubes and ventilator circuits.<sup>13,14</sup> The resistance posed by the endotracheal tube varies according to the diameter and flow<sup>13</sup>; therefore, the level of pressure necessary to counteract pressure decreases caused by the endotracheal tube varies from patient to patient.<sup>14</sup> It is unlikely that adult settings will be the best for infants intubated with narrow endotracheal tubes. The WOB decreases as the level of PSV increases in adults.<sup>14,15</sup> When adult patients are weaned from the ventilator during PSV, the level of PSV is commonly decreased gradually according to tolerance by the patient. Extubation can be performed when PSV has been decreased to 5-7 cm H<sub>2</sub>O.<sup>16,17</sup> For infants, however, no study has demonstrated that reducing the level of pressure-control PTV results in increased respiratory workloads or has defined the level of pressure control at which the endotracheal tube can be removed.

## Subjects and Methods

The study was approved by the institutional ethics committee, and informed consent was obtained from the parents of each patient.

### Patients

Seven infants aged 1 to 11 months who had undergone cardiac surgery to repair congenital heart disease (table 1) were enrolled in the study. Body weight ranged from 3.11 to 8.98 kg (average, 6.18 kg). Enrollment criteria were as follows: (1) infants with body weight less than 10 kg; (2) corrective surgery for cardiac anomaly such as ventricular septal defect; (3) stable hemodynamics; and (4) leakage around the endotracheal tube less than 5% of the inspired tidal volume ( $V_T$ ). We excluded candidates if they had chronic lung disease, central nervous system disorders, postoperative phrenic nerve damage, or any metabolic disorder. All patients were maintained in the

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\* Staff Intensivist, † Director, Surgical Intensive Care Unit, National Cardiovascular Center. ‡ Associate Professor, Intensive Care Unit, Osaka University Hospital.

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Address reprint requests to Dr. Imanaka: Surgical Intensive Care Unit, National Cardiovascular Center, 5-7-1 Fujishiro-dai, Suita, Osaka 565-8565, Japan. Address electronic mail to: imanakah@hsp.ncvc.go.jp. Individual article reprints may be purchased through the Journal Web site, www.anesthesiology.org.

**Table 1. Patient Profiles**

No.	Age (months)	Height (cm)	BW (kg)	Gender	Diagnosis	Operation	ETT Size (mm ID)	Length of MV (h)
1	8	65.5	5.78	M	VSD	VSD closure	4.0	5
2	10	69.0	6.85	F	VSD	VSD closure	4.5	5
3	4	58.7	5.32	M	VSD	VSD closure	4.0	6
4	1	54.1	3.90	M	VSD, ASD	VSD, ASD closure	4.0	8
5	11	70.4	8.98	F	VSD, MR	VSD closure, MVP	4.0	4
6	3	56.5	3.11	M	VSD	VSD closure	3.5	5
7	4	66.5	6.30	M	VSD	VSD closure	4.0	4
Mean	5.9	62.6	6.18					5.3

BW = body weight; ETT = endotracheal tube; MV = mechanical ventilation; VSD = ventricular septal defect; ASD = atrial septal defect; MR = mitral regurgitation; MVP = mitral valve plasty.

supine position during the time that measurements were taken. No sedatives or opioids were administered during the measurement, although fentanyl (23–39  $\mu\text{g}/\text{kg}$  in total) and midazolam (0.48–1.04  $\text{mg}/\text{kg}$ ) had been administered during the surgery.

#### Measurements

A pneumotachometer (model 4500; range, 0 to 35 l/min; Hans-Rudolph Inc., Kansas City, MO) was placed at the proximal end of the endotracheal tube. The pressure differential across the pneumotachometer was measured with a differential pressure transducer (TP-602T,  $\pm 5$   $\text{cm H}_2\text{O}$ ; Nihon Kohden, Tokyo, Japan), amplified (AR-601G, Nihon Kohden), and converted to flow. Volume was calculated from the flow using data acquisition software (Windaq; Dataq Instruments Inc., Akron, OH). Intrapleural pressure was estimated from esophageal pressure ( $P_{\text{es}}$ ). An esophageal balloon (6 French; Bicore, Irvine, CA) was introduced transnasally and positioned in the lower third of the esophagus. The balloon was inflated with 0.2 ml of air at the start of each measurement. The position of the esophageal balloon was adjusted using an occlusion technique when the patients regained spontaneous breathing.<sup>18</sup> We compared the maximal deflection in the  $P_{\text{es}}$  with the maximal deflection in the airway pressure while the infants made respiratory effort against an occlusion of the airway opening. When the ratio of the  $P_{\text{es}}$  to the airway pressure was maximal ( $> 0.95$ ), we secured the position of the balloon. The  $P_{\text{es}}$  and airway pressure ( $P_{\text{ao}}$ ) at the proximal end of the endotracheal tube were measured by differential pressure transducers (TP-603T,  $\pm 50$   $\text{cm H}_2\text{O}$ ; Nihon Kohden) and amplified (AR-601G). Respiratory inductive plethysmography (SY07 Respirace Plus; NIMS, Miami Beach, FL) was used to estimate inspiratory time ( $T_I$ ) and asynchrony between rib cage and abdomen. A rib-cage band was positioned at the nipple line and an abdomen band at 0.5 cm below the umbilicus. Baseline calibrations for respiratory inductive plethysmography were made using the qualitative diagnostic calibration procedure.<sup>19</sup> Maximum compartment amplitude (MCA) was calculated as the sum of the absolute value from

trough to peak of the rib-cage and abdomen compartments, regardless of their timing in relation to the sum signal.<sup>12,19</sup> When the motions of rib cage and abdomen are in phase, the ratio of  $\text{MCA}/V_T$  is equivalent to 1.0, where  $V_T$  is calculated from summed signal of rib cage and abdomen. When out of phase, the ratio of  $\text{MCA}/V_T$  exceeds 1.0. The airway and esophageal pressure transducers were simultaneously calibrated at 20  $\text{cm H}_2\text{O}$  using a water manometer. Flow was calibrated at 10 l/min using a calibrated oxygen flowmeter (P/N 9220; Bird Corp., Palm Springs, CA) with the gas mixture of identical oxygen concentration for the patient. Volume was calibrated with a 50-ml calibration syringe.

#### Study Protocol

We used V.I.P. Bird ventilators (Bird Corp.) with continuous-flow time-cycled pressure-limit ventilation. Ventilatory settings were as follows: assist-control mode, positive end-expiratory pressure, 3  $\text{cm H}_2\text{O}$ ; pressure control ventilation, 0–16  $\text{cm H}_2\text{O}$ ; continuous flow, 20 l/min; and triggering sensitivity, 1.0 l/min. Inspired oxygen fraction was adjusted to maintain an arterial oxygen pressure greater than 100 mmHg.

Baseline data were obtained when the patients recovered spontaneous breathing in the surgical intensive care unit and satisfied our weaning criteria: ratio of arterial oxygen pressure to inspired oxygen fraction greater than 200;  $p\text{H}$  greater than 7.30;  $V_T$  greater than 5 ml/kg; and respiratory rate less than 50 breaths/min at a backup ventilatory rate of 6 breaths/min and pressure control of 7  $\text{cm H}_2\text{O}$ . Then we measured compliance of the respiratory system ( $C_{\text{RS}}$ ) and chest wall ( $C_{\text{CW}}$ ). After hyper-ventilating the patients for 2–3 min to lessen their inspiratory efforts, we switched ventilation settings to  $T_I$  of 1.5–2 s, respiratory rate of 10 breaths/min, and pressure control of 16  $\text{cm H}_2\text{O}$ . Conditions of zero gas flow to permit measurement of static compliance were confirmed on a computer display for data acquisition. For each patient we evaluated the duration in which the dynamic inspiratory flow was sustained, and it was used

as a later setting of  $T_I$ . Compliance was calculated using the following formulas:<sup>20</sup>

$$C_{RS} = V_T / (\text{end inspiratory Pao} - \text{end expiratory Pao}) \quad (1)$$

$$C_{CW} = V_T / (\text{end inspiratory Pes} - \text{end expiratory Pes}) \quad (2)$$

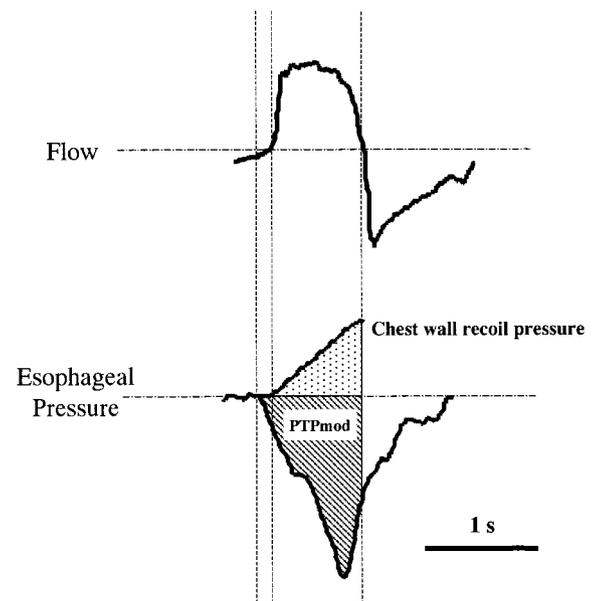
Measurements were repeated five times and averaged. Five levels of pressure control (0, 4, 8, 12, and 16 cm H<sub>2</sub>O) were then applied in random order with assist-control mode; positive end-expiratory pressure, 3 cm H<sub>2</sub>O; continuous flow, 20 l/min; and triggering sensitivity, 1.0 l/min. After establishing steady state conditions (approximately 15 min), the airflow, airway pressure, esophageal pressure, rib-cage, and abdominal signals of inductive plethysmography were recorded. All signals were digitized and recorded at a sampling rate of 100 Hz/channel (Windaq) during the last 2 min of each setting. Arterial blood samples were obtained *via* a catheter inserted into the radial artery and were analyzed with a calibrated blood gas analyzer (ABL 505; Radiometer, Copenhagen, Denmark).

All subjects were successfully extubated 90 min after the completion of all measurements. After extubation we waited for at least 60 min, confirmed that they were breathing quietly, and repeated the measurement of Pes and rib-cage and abdomen signals of inductive plethysmography, and arterial blood gas analysis. We did not measure the flow after extubation directly because it was likely that stimuli resulting from fitting masks to awake infants would alter their inspiratory patterns. Instead, we computed the volume using respiratory inductive plethysmography signals.

#### Data Analysis

Because the backup respiratory rate was set as low as 6 breaths/min, all breaths were assisted breaths. The onset of inspiration was defined as the point at which the Pes started to decrease. Intrinsic positive end-expiratory pressure was defined, if any, as the difference between this initial Pes level and the zero-flow point.<sup>21</sup> The end of inspiration was determined in two ways: (1) as the zero crossing of the inspiratory flow during mechanical ventilation (fig. 1), or (2) as the peak of inductive plethysmograph value when the patients were extubated. We confirmed that the values of each definition of  $T_I$  were equivalent during mechanical ventilation (precision and bias,  $0.00 \pm 0.05$  s). The  $T_I$ , ratio of inspiratory time to total respiratory cycle time ( $T_I/T_T$ ), respiratory rate, and mean inspiratory flow were calculated using the flow signal or inductive plethysmography. The  $V_T$  and minute ventilation were obtained from the expiratory flow.

Inspiratory WOB performed by the patient was computed from the curve of Pes versus  $V_T$  as previously



**Fig. 1.** Flow (inspiration upward) and esophageal pressure (Pes) tracing in a patient. Recoil pressure of the chest wall was calculated from chest wall compliance and lung volume. Pressure-time product (PTP) was calculated using the integral of the difference between Pes and the chest wall recoil pressure from the onset of the rapid decrease in Pes to the transition from inspiratory to expiratory flow. Modified pressure-time product (PTPmod) was calculated using the area of the Pes below the baseline value during the inspiration. The first vertical broken line shows when Pes started to decrease. The second and third vertical broken lines show when there was zero flow.

described.<sup>11</sup> The WOB per breath was calculated from Campbell diagram by computing the area enclosed between the recorded Pes- $V_T$  curve during inspiration on the one hand, and the static chest wall compliance curve on the other. The WOB was expressed both as per liter of ventilation (J/l) and as power normalized by body weight ( $J \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ ). We also used the pressure-time product (PTP) of esophageal pressure to estimate the inspiratory muscle load, because PTP is regarded as an index of oxygen cost of breathing of the respiratory muscles as well as WOB.<sup>22,23</sup> The PTP was calculated as the area subtended by the esophageal pressure tracing and the chest wall static recoil pressure for inspiratory time (fig. 1).<sup>23</sup> The chest wall static recoil pressure curve was obtained from the  $C_{CW}$  and volume. We also defined a modified esophageal pressure-time product (PTPmod) as the area of Pes-time tracing below the baseline during inspiration (fig. 1). After extubation, PTP was not obtained because of the lack of the flow information; instead, we used PTPmod for comparison. Both PTP and PTPmod were expressed as values for 1 min. Negative deflection of esophageal pressure ( $\Delta\text{Pes}$ ) was also measured as the maximal negative excursion from the baseline over breath. After extubation, values for  $V_T$ , minute ventilation, WOB, and  $\text{MCA}/V_T$  were calculated from volume obtained by the respiratory inductive plethys-

**Table 2. Baseline Respiratory Parameters**

No.	BW (kg)	C <sub>RS</sub> (ml · cm H <sub>2</sub> O <sup>-1</sup> · kg <sup>-1</sup> )	C <sub>CW</sub> (ml · cm H <sub>2</sub> O <sup>-1</sup> · kg <sup>-1</sup> )	FiO <sub>2</sub>	pH	Paco <sub>2</sub> (mmHg)	Pao <sub>2</sub> (mmHg)	T <sub>I</sub> (s)	RR (breaths/min)	V <sub>T</sub> (ml/kg)	MV (l · min <sup>-1</sup> · kg <sup>-1</sup> )
1	5.78	0.99	5.88	0.4	7.40	33.6	107	0.75 ± 0.05	33	11.6 ± 0.7	0.38
2	6.85	1.04	3.36	0.4	7.39	41.8	182	0.86 ± 0.02	28	8.5 ± 0.7	0.24
3	5.32	0.58	1.41	0.5	7.40	43.7	200	0.57 ± 0.04	41	7.9 ± 0.4	0.32
4	3.90	0.82	6.67	0.5	7.42	41.8	183	0.54 ± 0.02	41	7.4 ± 0.3	0.30
5	8.98	0.99	3.45	0.4	7.41	39.3	199	0.87 ± 0.02	28	8.0 ± 0.2	0.23
6	3.11	0.96	3.86	0.4	7.38	43.5	173	0.69 ± 0.04	35	10.0 ± 0.3	0.35
7	6.30	0.87	3.17	0.4	7.44	41.8	145	0.86 ± 0.05	29	7.6 ± 0.5	0.22
Mean	6.18	0.89	3.97	0.43	7.40	40.8	170	0.73	34	8.7	0.29

Baseline Parameters were evaluated when the patients satisfied extubation criteria.

BW = body weight; C<sub>RS</sub> = compliance of the respiratory system; C<sub>CW</sub> = compliance of the chest wall; FiO<sub>2</sub> = inspired oxygen fraction; Paco<sub>2</sub> = arterial carbon dioxide tension; Pao<sub>2</sub> = arterial oxygen tension; T<sub>I</sub> = inspiratory time; RR = respiratory rate; V<sub>T</sub> = tidal volume; MV = minute ventilation.

mography. We confirmed that the values of V<sub>T</sub> were equivalent during mechanical ventilation (precision and bias, 0.2 ± 2.6 ml). Ten consecutive breaths were used for data analysis.

*Statistical Analysis*

Data are presented as mean ± SD. Values of ΔPes, PTP, PTPmod, and WOB are presented as median and 25–75% percentiles, because these did not seem to be normally distributed. Using nonparametric tests (Friedman test), values at different conditions were compared (pressure control of 0, 4, 8, 12, and 16 cm H<sub>2</sub>O, and after extubation). When significance was observed, multiple comparison testing of means was performed using a Wilcoxon signed rank test. Statistical significance was set at a level of P less than 0.05.

**Results**

*Respiratory Parameters*

Table 2 shows baseline parameters at 7 cm H<sub>2</sub>O pressure control when the infants were considered to be ready for extubation. Table 3 shows respiratory parameters during each ventilatory setting. As the pressure control level decreased, respiratory rate increased and V<sub>T</sub> decreased significantly (P < 0.01). Minute ventilation remained almost constant at all levels of pressure control and was identical to the value at the baseline (table 2). The T<sub>I</sub> value was almost constant at pressure control levels of 0–12 cm H<sub>2</sub>O, although it tended to be longer at a level of 16 cm H<sub>2</sub>O. The pH, arterial carbon dioxide partial pressure, arterial oxygen pressure, T<sub>I</sub>/T<sub>T</sub>, mean inspiratory flow, heart rate, and intrinsic positive end

**Table 3. Parameters at Each Ventilatory Setting**

	Inspiratory Pressure Level Greater Than PEEP					
	16 cm H <sub>2</sub> O	12 cm H <sub>2</sub> O	8 cm H <sub>2</sub> O	4 cm H <sub>2</sub> O	0 cm H <sub>2</sub> O	After Extubation
pH	7.40 ± 0.03	7.41 ± 0.01	7.40 ± 0.03	7.40 ± 0.02	7.38 ± 0.03	7.43 ± 0.02
Paco <sub>2</sub> (mmHg)	41.2 ± 4.4	40.8 ± 3.8	41.7 ± 4.9	40.8 ± 3.5	43.3 ± 4.8	38.4 ± 2.3
Pao <sub>2</sub> (mmHg)	173 ± 42	181 ± 40	178 ± 39	170 ± 33	159 ± 25	202 ± 123
Inspiratory time (s)	0.90 ± 0.15	0.76 ± 0.15*	0.75 ± 0.13*	0.72 ± 0.15*	0.75 ± 0.19	0.66 ± 0.21*
Respiratory rate (breaths/min)	24.9 ± 6.6	28.6 ± 5.7*	32.9 ± 7.5†	34.3 ± 6.9†	34.4 ± 7.9†	36.6 ± 8.2†
Tidal volume (ml/kg)	11.9 ± 2.2	10.1 ± 1.7*	9.0 ± 1.9†	8.6 ± 1.5†	8.6 ± 1.3†	8.3 ± 2.2*
Minute ventilation (ml · min <sup>-1</sup> · kg <sup>-1</sup> )	293 ± 82	286 ± 59	291 ± 69	294 ± 72	293 ± 74	303 ± 92
T <sub>I</sub> /T <sub>T</sub>	0.36 ± 0.06	0.35 ± 0.02	0.40 ± 0.02	0.40 ± 0.01	0.41 ± 0.04	0.38 ± 0.05
MIF (l/min)	4.63 ± 1.59	4.51 ± 1.18	4.09 ± 1.28	4.04 ± 0.97	3.88 ± 0.66	4.36 ± 1.41
Heart rate (beats/min)	166 ± 14	162 ± 16	163 ± 17	163 ± 14	163 ± 15	159 ± 13
PEEPi (cm H <sub>2</sub> O)	0.24 ± 0.15	0.28 ± 0.21	0.40 ± 0.21	0.41 ± 0.35	0.36 ± 0.14	
ΔPes (cm H <sub>2</sub> O)	1.44 (0.88, 2.04)	1.92 (1.25, 2.31)	3.25*† (2.56, 3.91)	6.13*†‡ (5.27, 6.87)	8.39*†‡§ (7.58, 9.14)	6.91*†‡ (6.45, 8.19)
PTP (cm H <sub>2</sub> O · s)	27.4 (19.2, 29.5)	36.9* (26.0, 46.3)	74.5*† (56.3, 82.6)	108.5*†‡ (93.8, 122.8)	149.9*†‡§ (142.2, 160.0)	94.3*†‡ (87.7, 101.4)
PTPmod (cm H <sub>2</sub> O · s)	-16.8 (-24.9, 6.8)	13.1* (-1.5, 16.1)	45.4*† (30.1, 53.0)	87.2*†‡ (66.2, 96.4)	126.5*†‡§ (118.5, 131.9)	94.3*†‡ (87.7, 101.4)
WOB (J/l)	0.17 (0.14, 0.20)	0.20 (0.15, 0.28)	0.36*† (0.26, 0.41)	0.52*†‡ (0.43, 0.53)	0.73*†‡§ (0.68, 0.78)	0.56*†‡ (0.54, 0.67)
WOB (J · min <sup>-1</sup> · kg <sup>-1</sup> )	0.04 (0.03, 0.08)	0.05 (0.04, 0.10)	0.09*† (0.06, 0.15)	0.17*†‡ (0.11, 0.20)	0.22*†‡§ (0.16, 0.29)	0.16*†‡ (0.13, 0.25)
MCA/V <sub>T</sub>	1.06 ± 0.03	1.09 ± 0.05	1.12 ± 0.07	1.14 ± 0.08*	1.21 ± 0.14†	1.21 ± 0.13†

Values of median, 25%, and 75% percentiles are shown for change in negative deflection of esophageal pressure (ΔPes), pressure–time product (PTP), modified PTP (PTPmod), and work of breathing (WOB).

\* P < 0.05 versus inspiratory pressure level of 16 cm H<sub>2</sub>O greater than PEEP. † P < 0.05 versus 12 cm H<sub>2</sub>O. ‡ P < 0.05 versus 8 cm H<sub>2</sub>O. § P < 0.05 versus 4 cm H<sub>2</sub>O.

PEEP = positive end-expiratory pressure; Paco<sub>2</sub> = arterial carbon dioxide tension; Pao<sub>2</sub> = arterial oxygen tension; T<sub>I</sub>/T<sub>T</sub> = ratio of inspiratory time to total respiratory cycle time; MIF = mean inspiratory flow; PEEPi = intrinsic PEEP; MCA/V<sub>T</sub> = ratio of maximum compartment amplitude to tidal volume.

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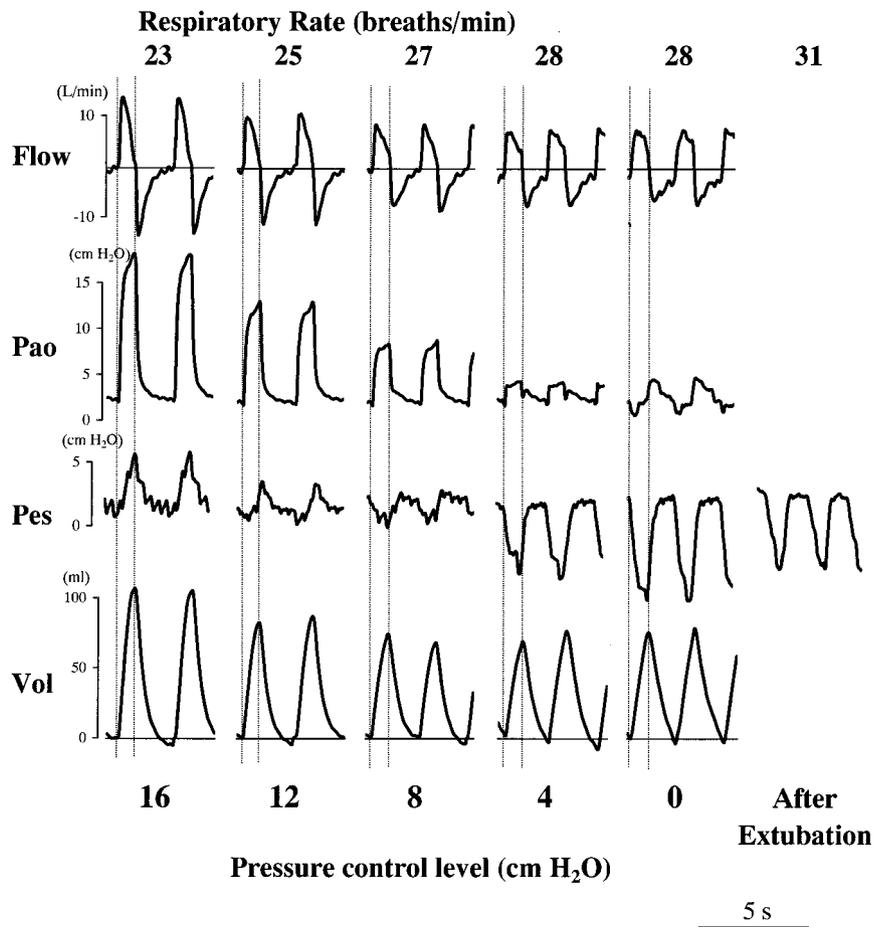


Fig. 2. Representative tracings of flow, airway pressure (Pao), esophageal pressure (Pes), and volume during each ventilatory condition: at pressure control levels of 16, 12, 8, 4, and 0 cm H<sub>2</sub>O and after extubation.

expiratory pressure did not differ significantly at any of the ventilatory settings.

Figure 2 is a representative tracing of flow, airway pressure, esophageal pressure, and volume during the five levels of pressure control and after extubation. As pressure control level was decreased, the negative deflection in esophageal pressure increased.

#### Work of Breathing and Pressure-Time Products

Figure 3 shows WOB per liter at each level of pressure control and after extubation. Similarly, figure 4 shows the PTPmod at each ventilatory setting. As the pressure control level was decreased, both WOB and PTPmod increased ( $P < 0.01$ ). The PTPmod value after extubation was almost equivalent to the value at 4 cm H<sub>2</sub>O pressure control and significantly larger than at levels of 8, 12, and 16 cm H<sub>2</sub>O ( $P < 0.05$ ). The WOB after extubation was also almost equivalent to the value at 4 cm H<sub>2</sub>O pressure control and larger than at levels of 8, 12, and 16 cm H<sub>2</sub>O ( $P < 0.05$ ). Similar results were observed regarding PTP,  $\Delta$ Pes, and WOB ( $J \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ ) (table 3). The values of MCA/V<sub>T</sub> observed in respiratory inductive plethysmography were approximately equal to 1.0 at high levels of pressure control ventilation, whereas they increased when pressure control level was decreased or after extubation (table 3).

#### Discussion

The main findings of this study are that: (1) when the level of pressure control was decreased, tidal volume decreased, respiratory rate increased, and minute ventilation and arterial carbon dioxide partial pressure remained constant; (2) in proportion to the level of pressure control, PTV reduced WOB, PTPmod, and  $\Delta$ Pes; and (3) the WOB and PTPmod after extubation were similar to those when the pressure control level was 4 cm H<sub>2</sub>O.

Pressure support ventilation provides patient-ventilator synchrony<sup>2,15</sup> and so has been widely used as PTV in adults<sup>1,2,3,15</sup> and recently in children.<sup>7,9,12</sup> In our study, pressure-control PTV was triggered successfully by every spontaneous breath, and minute ventilation was maintained constant through all levels of pressure control. This finding correlates with previous reports.<sup>4-6</sup> PSV has been shown to decrease respiratory work in proportion to pressure support in adults.<sup>14,15</sup> This observation has been empirically extrapolated to and applied as PTV in infants. Clinicians have also been adopting a strategy of gradually decreasing the pressure control as infants are weaned from a ventilator during pressure-control PTV.<sup>24</sup> In the absence of experimental evidence to corroborate that this strategy is similarly effective for infants, we



infants from the ventilator. Providing that satisfactory clinical and gas exchange status are exhibited, it may be possible to consider extubation when the pressure control level reaches 4 cm H<sub>2</sub>O. On the other hand, the following approach may be an alternative. When pressure control can be moved below 8 cm H<sub>2</sub>O, the level of pressure could go directly to 0 cm H<sub>2</sub>O because there is no statistically significant difference in the load between 4 cm H<sub>2</sub>O, 0 cm H<sub>2</sub>O, and by extubation (figs. 3 and 4). In this case, going to 0 cm H<sub>2</sub>O pressure control would represent a mild short-term trial and make it even less likely that an infant would fail if he or she surmounted this challenge.

In conclusion, after cardiac surgery, for infants with healthy lungs, pressure-control PTV reduces WOB and PTPs in proportion to the level of pressure control. The analysis of WOB and PTP indicated that a zero pressure trial is not always necessary before extubation of the trachea. Pressure-control PTV with flow triggering is as feasible a strategy for infants as it is for adults.

## References

- MacIntyre NR: Respiratory function during pressure support ventilation. *Chest* 1986; 89:677-83
- Brochard L, Pluskwa F, Lemaire F: Improved efficacy of spontaneous breathing with inspiratory pressure support. *Am Rev Respir Dis* 1987; 136:411-5
- Sassoon CSH, Light RW, Lodia R, Sieck GC, Mahutte CK: Pressure-time product during continuous positive airway pressure, pressure support ventilation, and T-piece during weaning from mechanical ventilation. *Am Rev Respir Dis* 1991; 143:469-75
- Greenough A, Greenall F: Patient triggered ventilation in premature neonates. *Arch Dis Child* 1988; 63:77-8
- Greenough A, Pool J: Neonatal patient triggered ventilation. *Arch Dis Child* 1988; 63:394-7
- Hird MF, Greenough A: Patient triggered ventilation using a flow triggered system. *Arch Dis Child* 1991; 66:1140-2
- Tokioka H, Kinjo M, Hirakawa M: The effectiveness of pressure support ventilation for mechanical ventilatory support in children. *ANESTHESIOLOGY* 1993; 78:880-4
- Bernstein G, Heldt GP, Mannino FL: Increased and more consistent tidal volumes during synchronized intermittent mandatory ventilation in newborn infants. *Am J Respir Crit Care Med* 1994; 150:1444-8
- El-Khatib MF, Chatburn RL, Potts DL, Blumer JL, Smith PG: Mechanical ventilators optimized for pediatric use decrease work of breathing and oxygen consumption during pressure-support ventilation. *Crit Care Med* 1994; 22:1942-8
- Cleary JP, Bernstein G, Mannino FL, Heldt GP: Improved oxygenation during synchronized intermittent mandatory ventilation in neonates with respiratory distress syndrome: A randomized, crossover study. *J Pediatr* 1995; 126:407-11
- Jarreau PH, Moriette G, Mussat P, Mariette C, Mohanna A, Harf A, Lorino H: Patient-triggered ventilation decreases the work of breathing in neonates. *Am J Respir Crit Care Med* 1996; 153:1176-81
- Tokioka H, Nagano O, Ohta Y, Hirakawa M: Pressure support ventilation augments spontaneous breathing with improved thoracoabdominal synchrony in neonates with congenital heart disease. *Anesth Analg* 1997; 85:789-93
- Fiastro JF, Habib MP, Quan SF: Pressure support compensation for inspiratory work due to endotracheal tubes and demand continuous positive airway pressure. *Chest* 1988; 93:499-505
- Brochard L, Rua F, Lorino H, Lemaire F, Harf A: Inspiratory pressure support compensates for the additional work of breathing caused by the endotracheal tube. *ANESTHESIOLOGY* 1991; 75:739-45
- Brochard L, Harf A, Lorino H, Lemaire F: Inspiratory pressure support prevents diaphragmatic fatigue during weaning from mechanical ventilation. *Am Rev Respir Dis* 1989; 139:513-21
- Tobin MJ, Alex CG: Discontinuation of mechanical ventilation, *Principles and Practice of Mechanical Ventilation*. Edited by Tobin MJ. New York, McGraw-Hill, 1994, pp 1177-206
- Esteban A, Alía I, Gordo F, Fernández R, Solsona JF, Vallverdú I, Macías Allegue JM, Blanco J, Carriedo D, León M, de la Cal MA, Taboada F, de Velasco JG, Palazón E, Carrizosa F, Tomás R, José S, Goldwasser RS: Extubation outcome after spontaneous breathing trials with T-piece or pressure support ventilation. *Am Rev Respir Crit Care Med* 1997; 156:459-65
- Coates A, Stocks J, Gerhardt T: Esophageal manometry, *Infant Respiratory Function Testing*. Edited by Stocks J, Sly PD, Tepper RS, Morgan WJ. New York, Wiley-Liss, 1996, pp 241-58
- Adams JA: Respiratory inductive plethysmography, *Infant Respiratory Function Testing*. Edited by Stocks J, Sly PD, Tepper RS, Morgan WJ. New York, Wiley-Liss, 1996, pp 139-64
- Nunn JF: Elastic forces and lung volumes, *Nunn's Applied Respiratory Physiology*, 4th Edition. Edited by Nunn JF. Oxford, Butterworth-Heinemann, 1993, pp 36-60
- Rossi A, Polese G, Brandi G, Conti G: Intrinsic positive end-expiratory pressure (PEEPi). *Intensive Care Med* 1995; 21:522-36
- McGregor M, Becklake MR: The relationship of oxygen cost of breathing to respiratory mechanical work and respiratory force. *J Clin Invest* 1967; 46:971-80
- Sassoon CSH, Mahutte CK: Work of breathing during mechanical ventilation, *Physiological Basis of Ventilatory Support*. Edited by Marini JJ, Slutsky AS. New York, Marcel Dekker, 1998, pp 261-310
- Donn SM, Nicks JJ: Special ventilatory techniques and modalities I: Patient triggered ventilation, *Assisted Ventilation of the Neonate*, 3rd Edition. Edited by Goldsmith JP, Karotkin EH. Philadelphia, Saunders, 1996, pp 215-28
- Fletcher ME, Baraldi E, Steinbrugger B: Passive respiratory mechanics, *Infant Respiratory Function Testing*. Edited by Stocks J, Sly PD, Tepper RS, Morgan WJ. New York, Wiley-Liss, 1996, pp 283-327
- Hammer J, Numa A, Newth CJ: Albuterol responsiveness in infants with respiratory failure caused by respiratory syncytial virus infection. *J Pediatr* 1995; 127:485-90