Med Intensiva. 2016;40(4):250–252

medicina intensiva

www.elsevier.es/medintensiva

SCIENTIFIC LETTER

Pressure-regulated volume control versus volume control ventilation in severely obstructed patients

Control del volumen regulado por presión frente a ventilación con control del volumen en pacientes con obstrucción grave

Dear Editor,

Currently we do not know which mechanical ventilation mode is to be selected for treating severe asthma patients.[1–4]

A previous experimental study compared the efficacy of volume control ventilation (VCV), pressure control ventilation (PCV) and pressure-regulated volume control ventilation (PRVCV) in delivering the programmed tidal volume (TV) under different respiratory conditions. In this study, a series of ventilation simulations mimicking scenarios of low airway resistance, as those found in normal lungs, and of high airway resistance, as those found in obstructive conditions, were performed. The results showed that PCV and PRVCV are not appropriate ventilation modes for severely obstructed patients[5] as they could lead to hypoventilation of the patient. This result is new and has raised concern on what ventilation mode is desirable for treating severe asthma patients.

The aim of this study was to present four cases of infants with severe airway obstruction requiring mechanical ventilation to underscore the actual relevance of performing VCV versus PRVCV in the current pediatric clinical practices.

We report four cases of infants with severe airway obstruction (due to viral bronchiolitis) requiring mechanical ventilation who were unsatisfactory managed with PRVCV, and then successfully treated with VCV.

The first case involved a one-month-old infant (weight: 4.0 kg) with acute viral bronchiolitis. In the 48 h previous to PICU admission, he had been hospitalized in a regional hospital, where medical treatment failed, presenting a minimum pH of 6.86 and ultimately requiring intubation. Upon arrival, he presented with cardio-respiratory arrest that required 3 min of advanced cardio-respiratory resuscitation. The changes over time in the blood gas levels and ventilator settings are shown in Table 1.

The second case involved a four-month-old infant (weight: 4.8 kg) with respiratory syncytial virus (RSV) bronchiolitis. The results of his first blood gas analysis were: pH 6.9; $PaCO_2 = 107\ cm\ H_2O$; $PaO_2 = 220\ cm\ H_2O$; and $HCO_3 = 20.7\ mmol/L$. He required intubation and mechanical ventilation. We initially chose VCV as the ventilation mode. Peak inspiratory pressure (PIP) was very high (40 cm H_2O). Therefore, following recent recommendations,[2] we switched to PRVCV. The changes over time in the blood gas levels and ventilator settings are shown in Table 1.

The third and fourth cases involved a two-month-old infant (weight: 4.6 kg) and a one-month-old infant (weight: 4.0 kg), both with RSV bronchiolitis. The changes over time in the blood gas levels and ventilator settings are also shown in Table 1.

The existing literature on mechanical ventilation for severely obstructed patients is rather ambiguous. We have read different and often contradictory recommendations on ventilation modes. Some authors suggest using PCV[1,3]; others, VCV[4]; and still others, newer modes as PRVCV.[3] In relation to PRVCV, this technique is conceived to be the best as it is thought to assure that the patient receives the optimal TV at the lowest PIP.[5]

However, the latest trends suggest that PRVCV may be the best option, as it is thought to assure that the patient receives the desired (i.e. set) TV at the lowest peak PIP possible.[5]

As shown in our four cases of severely obstructed infants, ventilation with PRVCV has failed to ventilate the patients. We could not adequately assure normal ventilation in either patient until switching to VCV (Table 1).

In pressure control modes, if the inspiratory flow is not zero when inspiration ends, we cannot be sure that the volume of air which left the ventilator has reached the alveoli.[6,7] According to our previous experimental study,[5] this phenomenon would account for the undelivered volume that we observed with PCV and PRVCV modes. When flow was not zero, this undelivered volume is still moving inside the tubes and has not reached the alveoli. However, when the ventilator measures the volume returning back from the respiratory circuit, it did not detect any difference (i.e. it detected the sum of the volume that actually reached

2173-5727 /

Table 1 Chronological changes in blood gas levels and ventilator settings for each patient.

	Blood gas levels						Ventilator settings								
	Minutes	Sample	pH	PO_2 ($cm\,H_2O$)	PCO_2 ($cm\,H_2O$)	HCO_3^- (mmol/L)	Mode	TV (ml)	RR (bpm)	PIP/P_{plat} ($cm\,H_2O$)	PEEP ($cm\,H_2O$)	*I:E*	FiO_2	T_i (s)	T_p (s)
Patient 1	0	A	6.8	78	–	–	PRVCV	25	45	32/28	11	1:2.8	0.9	0.35	
	150	A	7.05	70	130	36.6	PRVCV	35	50	32/28	12	1:2.1	1	0.35	
	420	A	7.05	68	132	37.4	PRVC	35	55	28/27	10	1:2	0.7	0.35	
	590	A	7.19	48	99	37.8	VCV	35	50	39/28	10	1:2.1	0.5	0.35	0.05
	690	A	7.38	53	64	37.9	VCV	35	50	34/27	10	1:2.1	0.5	0.35	0.05
Patient 2	0	V	7.23	62	60.4	24.8	VCV	70	45	40/26	4	1:4	0.4	0.26	0.06
	180	V	7.06	54.4	92.8	25.5	PRVCV	70	40	35/24	6	1:3	0.35	0.37	
	300	V	7.09	78.7	83.5	24.7	PRVCV	70	50	40/23	6	1:3	0.35	0.3	
	660	V	7.04	51.9	87.6	22.6	PRVCV	70	30	39/23	6	1:5	0.35	0.33	
	780	V	7.12	70.9	78.1	24.3	PRVCV	80	40	45/30	6	1:3	0.35	0.37	
	1020	V	7.17	59.6	65.7	23.3	PRVCV	80	30	40/30	6	1:3	0.35	0.5	
	1260	V	7.06	41.9	86.9	23.7	PRVCV	80	25	35/30	6	1:2	0.35	0.8	
	1440	V	7.19	67.2	76.6	28.1	PRVCV	80	35	47/28	4	1:4	0.35	0.35	
	1530	V	7.38	61.2	43.7	25.7	VCV	75	30	50/28	8	1:3	0.35	0.5	0.06
	1740	V	7.44	48.5	28.9	18.1	VCV	75	30	50/28	8	1:3	0.35	0.5	0.06
Patient 3	0	V	7.34	–	65.5	34.1	VCV	28	30	50/28	5	1:1.9	0.38	0.60	0.10
	11	V	7.24	–	79.4	33.1	PRVCV	28	30	32/28	5	1:1.9	0.38	0.70	
Patient 4	0	V	7.28	–	65	30.5	PRVC	28	40	28	7	1:3.5	0.45	0.44	–
	360	V	7.36	–	51.0	29.4	PRVC	28	45	29	8	1:3	0.4	0.44	
	1080	V	7.36	–	50	25.8	PRVC	28	45	25	8	1:3	0.4	0.44	–
	1320	V	7.49	–	34	26.9	VCV	28	45	35/24	8	1:3	0.4	0.35	0.10

A, arterial sample; V, venous sample; PO_2, oxygen partial pressure; PCO_2, CO_2 partial pressure; TV, tidal volume; RR, respiratory rate; bpm, breaths per minute; PIP, peak inspiratory pressure; P_{plat}, plateau pressure; PEEP, positive end-expiratory pressure; *I:E*, inspiratory to expiratory ratio; FiO_2, inspiratory fraction of oxygen; PRVCV, pressure-regulated volume control ventilation; VCV, volume control ventilation; T_i, inspiratory time; T_p, pause time.

the patient plus the volume that had simply remained in the tubes and could not participate in gas exchange). We observed this problem at high airway resistance (AR), a clinical scenario in which a rather long constant time (CT) was required to deliver the entire volume of gas leaving the ventilator to the alveoli.[8] At the moment the expiratory valve opens, the air that stays in the tubes ''waiting'' to be delivered returns back to the ventilator without reaching the alveoli. As reflected in our case reports, this loss in ''effective'' TV can critically compromise ventilation of these little infants. As we can see in Table 1, during the period infants were in PRVCV, PCO_2 increased causing severe hypoventilation, probably (and unnoticeably for us!!) because not all the programmed TV reached the alveoli. When the patients were switched to VCV maintaining the same TV, PCO_2 levels decrease because all set TV reached the alveoli. Therefore, if a severely obstructive patient who is being ventilated with PRVCV presents hypoventilation, we propose as the first therapeutic option to change the ventilation mode to VCV.

When mechanical ventilators (i.e. pressure generators, in a physical sense) are used for patients, as the air goes into the respiratory system causing the alveolar pressure to increase, the pressure gradient between the generator and the patient's airway decreases, and so the inspiratory flow. In clinical situations with increased airway resistance (i.e. a prolonged CT), such as that occurring in asthma exacerbations or in our four pediatric patients with infectious bronchiolitis and hypercapnic respiratory failure,[9] this ''decelerating flow'' phenomena causes hypoventilation if you do not assure enough time to complete the inspiration. In addition, severely obstructed patients require a prolonged expiratory time to avoid air-trapping, leading to I:E of 1:1 to optimize the ventilation.

Using high-pressure generators (i.e. VCV mode), the pressure gradient do not practically decrease during the time the inspiration lasts (in VCV the inspiration finishes when the set TV is delivered) and consequently, the delivered TV is barely affected even if the patient's airway resistance increases (e.g. from a bronchospasm). Indeed, high-pressure generators are characterized by their constant inspiratory gas flow, which is resilient to major changes in lung or AR.[10] As we have said before, in severely obstructed patients you have to prolong the expiratory time to avoid air-trapping. But, because of its physical nature, high-pressure generators are able to deliver the entire set TV in a short inspiratory period of time (<1 CT), enabling these patients to be correctly managed at low I:E ratios (e.g. 1:3–1:5) with VCV.

In summary, severe obstructed patients have to be ventilated with high-pressure generators, i.e. VCV mode, in order to avoid hypoventilation and air-trapping.

In the mechanical ventilation of a situation of severely increased AR, VCV should be the first choice among modes, as it provides better ventilation of the patient and avoids air-trapping when used at low I:E ratios.

Financial disclosure

No financial support. Authors have no commercial associations or sources of support to declare.

Conflict of interest

Theauthors declare that they have no conflict of interest.

References

1. Medoff BD. Invasive and noninvasive ventilation in patients with asthma. Respir Care. 2008;53:740–8.
2. Shankar V, Churchwell KB, Deshpande JK. Isoflurane therapy for severe refractory status asthmaticus in children. Intensive Care Med. 2006;32:927–33.
3. Stather DR, Stewart TE. Clinical review: mechanical ventilation in severe asthma. Crit Care. 2005;9:581–7.
4. Oddo M, Feihl F, Schaller M-D, Perret C. Management of mechanical ventilation in acute severe asthma: practical aspects. Intensive Care Med. 2006;32:501–10.
5. Medina A, Modesto-Alapont V, Lobete C, Vidal-Micó S, Alvarez-Caro F, Pons-Odena M, et al. Is pressure-regulated volume control mode appropriate for severely obstructed patients? J Crit Care. 2014;29:1041.
6. Heulitt MJ, Thurman TL, Holt SJ, Jo CH, Simpson P. Reliabilityof displayed tidal volume in infants and children during dual-controlled ventilation. Pediatr Crit Care Med. 2009;10:661–7.
7. Marik PE, Krikorian J. Pressure-controlled ventilation in ARDS: a practical approach. Chest. 1997;112:1102–6.
8. Nève V, Leclerc F, Noizet O, Vernoux S, Leteurtre S, Forget P, et al. Influence of respiratory system impedance on volume and pressure delivered at the Y piece in ventilated infants. Pediatr Crit Care Med. 2003;4:418–25.
9. Mayordomo-Colunga J, Medina A, Rey C, Díaz JJ, Concha A, Los Arcos M, et al. Predictive factors of non-invasive ventilation failure in critically ill children: a prospective epidemiological study. Intensive Care Med. 2009;35:527–36.
10. Young JD, Sykes MK. Assisted ventilation. 1. Artificial ventilation: history, equipment and techniques. Thorax. 1990;45:753–8.

A. Medina[a], V. Modesto-Alapont[b], P. del Villar Guerra[c,*], M.R. Redal[b], A.M. Cambra[b], C. Rey[a], L. Perez-Baena[d]

[a] *Pediatric Intensive Care Unit, Hospital Universitario Central de Asturias, University of Oviedo, Oviedo, Spain*
[b] *Pediatric Intensive Care Unit, Hospital Universitario La Fe, Valencia, Spain*
[c] *Department of Pediatrics, Hospital Universitario Río Hortega, Valladolid, Spain*
[d] *Department of Pediatrics, Hospital Universitario Nuestra Señora de Candelaria, Santa Cruz de Tenerife, Spain*

* Corresponding author.
E-mail address: pablitovg@hotmail.com
(P. del Villar Guerra).