Pediatr Cardiol 22:233–237, 2001
DOI: 10.1007/s002460010210

Pediatric
Cardiology

Pressure-Regulated Volume Control vs Volume Control Ventilation in Infants After Surgery for Congenital Heart Disease

K.C. Kocis,[1] M.K. Dekeon,[2] H.K. Rosen,[2] K.P. Bandy,[2] D.C. Crowley,[2] E.L. Bove,[3] T. Kulik[2]
[1]Department of Pediatrics and Surgery, University of Southern California School of Medicine, Los Angeles, CA 90027, USA
[2]Department of Pediatrics, University of Michigan Medical Center, Ann Arbor, MI 48109, USA
[3]Department of Surgery, University of Michigan Medical Center, Ann Arbor, MI 48109, USA

Abstract. The objective of this investigation was to compare how two modes of positive pressure ventilation affect cardiac output, airway pressures, oxygenation, and carbon dioxide removal in children with congenital heart disease in the immediate postoperative period. The investigation used a one group pretest–post-test study design and was performed in the pediatric cardiac intensive care unit in a university-affiliated children's hospital. Nine infants were enrolled immediately after repair of tetralogy of Fallot (2) or atrioventricular septal defects (7) with mean weight = 5.5 kg (4.2–7.3 kg). Children were admitted to the pediatric cardiothoracic intensive care unit after complete surgical repair of their cardiac defect and stabilized on a Siemen's Servo 300 ventilator in volume control mode (VCV1) (volume-targeted ventilation with a square flow wave pattern). Tidal volume was set at 15 cc/kg (total). Hemodynamic parameters, airway pressures and ventilator settings, and an arterial blood gas were measured. Patients were then changed to pressure-regulated volume control mode (PRVC) (volume-targeted ventilation with decelerating flow wave pattern) with the tidal volume set as before. Measurements were repeated after 30 minutes. Patients were then returned to volume control mode (VCV2) and final measurements made after 30 minutes. The measurements and results are as follows:

Parameter	VCV1	PRVC	VCV2
PIP (cm H_2O)	31	25*	31
P_{aw} (cm H_2O)	7.8	7.8	7.8
pH	7.46	7.47	7.47
P_aCO_2(mmHg) (kP_a)	35 (4.7)	33 (4.4)	34 (4.5)
P_aO_2(mmHg) (kP_a)	167 (22.3)	193 (25.7)	189 (25.2)
Cl(L/min/m^2)	3.6	3.5	3.6

PIP, peak inspiratory pressure; P_{aw}, mean airway pressure; CI, cardiac index.
*$p < 0.05$.

After correction of congenital heart defects in infants, mechanical ventilation using a decelerating flow wave pattern resulted in a 19% decrease in peak inspiratory pressure without affecting hemodynamics, arterial oxygenation, or carbon dioxide removal.

Key words: Congenital heart disease — Hemodynamics — Mechanical ventilation — Pressure-regulated volume control ventilation

Mechanical ventilation is essential in the perioperative period for the successful treatment of infants undergoing congenital cardiac surgery [4, 5, 7]. Mechanical ventilation supports the patient's respiratory function during much of the operative period, recovery from anesthesia, hemodynamic stabilization in the cardiac intensive care unit, and weaning to successful extubation [5, 7, 8]. Positive pressure ventilation has both positive and negative hemodynamic consequences [7, 8, 15]. High intrathoracic pressure decreases venous return to the heart while at the same time decreasing afterload to the left ventricle. These combined cardiopulmonary interactions have complex effects on cardiac output. Recent ventilator

Correspondence to: K.C. Kocis, Chief, Division of Pediatric Critical Care Medicine, Department of Pediatrics, Burnett Womack Building, No. 7220, University of North Carolina School of Medicine, Chapel Hill, NC 27599-7220, USA

technology [5, 8, 10, 11, 17] allows for the rapid delivery of small tidal volumes at high respiratory rates and short inspiratory times with improved patient ventilator synchrony in multiple modes. The modes differ in their flow characteristics and mechanisms for triggering, limiting, and cycling the ventilator. On the Siemen's 300 Servo ventilator (Siemens Elma, Stockholm, Sweden), pressure-regulated volume control (PRVC) is patient or time triggered, volume targeted, time cycled, with a decelerating flow pattern. In contrast, volume control (VC) ventilation is patient or time triggered, volume targeted, time cycled, with a constant flow pattern. There are studies in adults with respiratory distress syndrome comparing these newer modes of ventilation [1–3, 12, 13, 16] but none have examined patients with cardiac disease or infants. The purpose of this study was to compare the effects that two modes of ventilation, specifically PRVC and VC, have on the cardiorespiratory systems of infants immediately after cardiac surgery. We hypothesized that (1) the PRVC mode of ventilation with its decelerating flow pattern would result in a significant decrease in airway pressures in these children; (2) the decrease in airway pressure may result in an improvement in the hemodynamic status, and (3) there would be an improvement in P_aO_2 and/or CO_2 removal.

Materials and Methods

Patients

The patient population consisted of nine consecutive infants with tetralogy of Fallot (2) or atrioventricular septal defects (7) who underwent complete surgical repair at C.S. Mott Children's Hospital at the University of Michigan. These two diagnostic groups were chosen due to the relative homogeneity of patients within the group and need for mechanical ventilation beyond the operative period (12–24 hours). The patients' mean age was 5.7 months (3–13 months) and weight was 5.5 kg (4.2–7.3 kg). Patients were intubated by pediatric cardiac anesthesiologists prior to operation. At the conclusion of the operative procedure, a thermodilution catheter (Baxter Healthcare Corp., Deerfield, IL, USA) was placed directly into the pulmonary artery for measurement of pulmonary artery pressure and cardiac output. Patients were paralyzed and stabilized (approximately 1 hour) in the pediatric cardiac intensive care unit prior to initiation of the study. Stabilized patients did not require manipulation of inotropic agents or volume infusion and did not have any significant fluctuations in heart rate, arterial blood pressure, or oxygen saturation. This protocol was approved by the Institutional Review Board and informed consent was obtained. This protocol complies with principles established in Helsinki.

Mechanical Ventilation

All patients returned from the operating room and were placed in VC mode (time triggered, volume targeted, time cycled, with constant flow) (Siemens Servo 300). There was no significant leak present around the endotracheal tube (inspiratory and expiratory tidal volumes were nearly identical). Expiratory tidal volume was set at approximately 15 cc/kg total. Positive end expiratory pressure was held constant at 2 or 3 cm H_2O. The inspiratory time was set at 0.50–0.70 seconds. Respiratory rate was set at 16–40 breaths/minute and FIO_2 at 0.4–1 in order to obtain a P_aCO_2 of 25–45 mmHg and oxygen saturation > 95%. This variability in FIO_2 and respiratory rate was due to some infants requiring hyperventilation and hyperoxygenation due to postoperative pulmonary artery hypertension. The peak inspiratory pressure and mean airway pressure were measured near the expiratory port on the ventilator using the manufacturer's manometer. All ventilators were calibrated prior to study using the manufacturer's suggested guidelines. pH, P_aCO_2, and P_aO_2 were measured from an arterial blood gas and bicarbonate, base excess, and oxygen saturation calculated (Radiometer, Cleveland, OH, USA).

Hemodynamic Measurements

All pulmonary artery catheter measurements were made in triplicate using iced (0°C) saline at end expiration.

Study Protocol

After stabilization in VC mode, hemodynamic parameters, airway pressures and ventilator settings, and an arterial blood gas were obtained. Patients were then changed to PRVC mode. The total tidal volume, positive end expiratory pressure, inspiratory time, and respiratory rate were set as before. After 30 minutes, measurements were repeated. Finally, patients were changed back to VC ventilation with the original ventilator settings, and 30 minutes later measurements were repeated.

Statistical Analyses

Statistical comparisons between the different modes of ventilation were made using the analysis of variance procedure with post hoc pairwise comparisons (Tukey) using commercially available PC software (SYSTAT Inc., Evanston, IL, USA). Significant results were reported if $p < 0.05$. All values are reported as mean ± standard deviation.

Results

The hemodynamic data, airway and ventilator parameters, and arterial blood gas results are summarized in Table 1. There were no changes in any hemodynamic or blood gas parameters when comparing the two modes of ventilation. All airway pressures remained unchanged except for the peak inspiratory pressure, which decreased by 19% in PRVC mode when compared to either trial of VC ventilation. No patients required a change in inotropic agents or volume infusion during the study period.

Discussion

Mechanical ventilation is essential for the care of infants and children undergoing cardiac surgery [4, 7]. Ventilator technology has improved dramatically in recent years, particularly for mechanically ventilated infants

Table 1. Hemodynamic, respiratory, and arterial blood gas measurements during study periods (mean ± standard deviation).

Parameter	VCV1	PRVC	VCV2
Hemodynamic parameters			
Heart rate (bpm)	166 ± 26	167 ± 27	165 ± 26
Blood pressure, systolic (mmHg)	85 ± 19	88 ± 19	85 ± 14
Blood pressure, diastolic (mmHg)	48 ± 11	48 ± 10	49 ± 8
Blood pressure, mean (mmHg)	63 ± 14	65 ± 13	61 ± 10
Pulmonary artery pressure, systolic (mmHg)	33 ± 7	34 ± 8	35 ± 7
Pulmonary artery pressure, diasystolic (mmHg)	18 ± 7	16 ± 9	18 ± 7
Pulmonary artery pressure, mean (mmHg)	24 ± 6	25 ± 7	25 ± 7
Central venous pressure (mmHg)	9 ± 4	7 ± 4	8 ± 4
Cardiac index (L/min/m^2)	3.6 ± 1	3.5 ± 0.9	3.6 ± 0.9
Stroke volume (ml)	24 ± 9	23 ± 7	24 ± 7
Pulmonary vascular resistance (dynes sec/cm^5)	383 ± 166	419 ± 176	391 ± 160
Systemic vascular resistance (dynes sec/cm^5)	1353 ± 484	1398 ± 563	1322 ± 469
Airway and ventilator parameters			
Total tidal volume (ml)	88 ± 20	88 ± 20	88 ± 20
Corrected tidal volume (ml/kg)	16 ± 1	16 ± 1	16 ± 1
Positive end expiratory pressure (cm H_2O)	3 ± 1	3 ± 1	3 ± 1
Peak inspiratory pressure (cm H_2O)	31 ± 4	25 ± 4*	31 ± 5
Respiratory rate (breaths/minute)	24 ± 7	24 ± 7	24 ± 7
Inspiratory time (seconds)	0.65 ± 0.07	0.65 ± 0.07	0.65 ± 0.07
Fractional inspired oxygen concentration	0.83 ± 0.21	0.83 ± 0.21	0.83 ± 0.21
Mean airway pressure (cm H_2O)	7.8 ± 1.4	7.8 ± 1.5	7.8 ± 1.6
Alveolar–arterial oxygen gradient	364 ± 217	339 ± 215	343 ± 213
Oxygenation index	6 ± 5	5 ± 4	5 ± 4
Arterial blood gas measurements			
pH	7.46 ± 0.09	7.47 ± 0.09	7.47 ± 0.10
P_aCO_2 (torr) (kP_a)	35 ± 6 (4.7 ± 0.8)	33 ± 6 (4.4 ± 0.8)	34 ± 7 (4.5 ± 0.9)
P_aO_2 (torr) (kP_a)	167 ± 103 (22.3 ± 13.7)	193 ± 100 (25.7 ± 13.3)	189 ± 102 (25.2 ± 13.6)
Oxygen saturation	98 ± 2	99 ± 1.5	99 ± 2
Bicarbonate (meq/dl)	24 ± 3	24 ± 3	25 ± 2
Base excess	0.8 ± 2.4	1.7 ± 3.6	1.7 ± 4.1

VCV, volume control ventilation; PRVC, pressure-regulated volume control.
*$p < 0.05$: VCV1 and VCV2 vs PRVC.

and children [5, 8, 11, 17]. Current technology allows for the synchronized delivery of small tidal breaths at rapid rates with improved monitoring and safety. Different modes of ventilation have also been developed which vary the triggering, limiting, and cycling of the ventilator. Demand valves now exist that flow trigger with response times as low as 10 msec [6, 10] and these have allowed synchronized ventilation to occur in infants and children with greatly reduced work of breathing [5]. Pressure-limited ventilation was the mainstay in pediatric critical care in the past due to the inability of ventilators to deliver small tidal volumes consistently. Pressure-limited ventilation is particularly problematic in critically ill children with congenital cardiac disease due to the inability to deliver a constant minute ventilation in patients undergoing rapid changes in lung compliance [4]. This limitation has been overcome by the newer generation ventilators. Volume-targeted modes of ventilation now exist which vary in their flow characteristics. On the Servo 300 ventilator, VC ventilation is patient or time triggered, volume targeted, time cycled, with constant flow pattern. In contrast, PRVC is patient or time triggered, volume targeted, time cycled, with decelerating flow pattern. We believe that this study is the first to compare these two modes of volume-targeted ventilation in infants and children with minimal lung dysfunction immediately after cardiac surgery.

Several reports in the adult literature compare the different flow patterns (i.e., decelerating vs constant) in either volume- or pressure-targeted mode [1–3, 12, 13, 16]. In these studies, hemodynamics, respiratory mechanics, and arterial blood gas results were obtained in patients with severe lung injury. The studies differed in design and results but can be summarized as follows. Davis et al. [3] compared a volume-targeted mode with decelerating flow to a pressure-targeted mode with decelerating flow and a volume-targeted mode with constant flow. There were no differences in results when comparing either decelerating flow mode of ventilation to each other. There was a significant decrease in mean airway pressure and P_aO_2 and an increase in peak inspiratory pressure in the constant flow mode. No hemodynamic changes occurred in any mode of ventilation. Munoz et al. [13] found no differences in respiratory mechanics when comparing a volume-targeted to a pressure-targeted mode of ventilation, both with decelerating flow. Al-Saady and Bennett [2] compared a constant to decelerating flow pattern in a volume-targeted mode of ventilation. They found that when using decelerating flow, there was a decrease in peak inspiratory pressure, total respiratory resistance, work of inspiration, ratio of dead space to tidal volume, and alveolar–arterial gradient for oxygen. There was an increase in total static and dynamic compliances and P_aO_2. There were no hemodynamic changes. Rappaport et al. [16] found that patients ventilated in a pressure-targeted mode of ventilation with decelerating flow had a decrease in peak inspiratory pressure and length of intubation and an increase in static compliance when compared to a volume-targeted mode with constant flow.

In an animal model of acute respiratory distress syndrome, Markstrom et al. [12] found a decreased peak inspiratory pressure and an increased mean airway pressure and CO_2 removal in piglets ventilated in PRVC mode rather than VC mode. There were no differences in end inspiratory occlusion pressure, end inspiratory lung volume, static compliance, hemodynamics, or oxygen delivery.

Similar to other studies [2, 3, 16], we found a 19% decrease in peak inspiratory pressure, but unlike the previous studies we did not find any changes in mean airway pressure, P_aO_2, alveor–arterial oxygen gradient, or CO_2 removal. Airway resistance, static compliance, work of inspiration, and dead space were not measured in this study. Also, this was an acute study, so a decrease in duration of intubation could not be assessed. The differences found in this study can be explained by differences in patients sampled in each study. This study examined infants in the immediate postoperative period. Unlike other studies, these patients did not have severe lung disease, although lung injury does occur following cardiopulmonary bypass [4, 7]. The mean airway pressure measured in this study was relatively low and did not differ in the two modes of ventilation. Oxygenation and alveolar–arterial oxygen gradient were not different in either mode of ventilation. As in previous studies, no hemodynamic differences were noted when comparing PRVC to VC modes of ventilation.

When infants and children return from the operating room following cardiac surgery, we choose to mechanically ventilate these patients in PRVC mode. This allows us to deliver a relatively constant minute ventilation to the patients with the lowest peak inspiratory pressure. It is postulated that lowering the peak inspiratory pressure minimizes lung injury that occurs with positive pressure ventilation [9, 14]. Although not addressed in this study, we hope that ventilating infants and children with decelerating flows will shorten their duration of intubation, as noted by Abraham and Yoshihara [1]. These benefits of PRVC ventilation lead us to recommend this mode of ventilation in all infants and children who require full mechanical ventilatory support in the immediate postoperative period. Once children are stabilized and the decision to begin weaning toward removal from mechanical ventilation has been made, alternative modes of ventilation should then be employed.

Several limitations of this study need to be mentioned. This study examined a small number of patients and therefore did not have a large degree of power to expose small differences in the variables measured. Our inability to detect differences in oxygenation may represent a Type II error. Due to the small number of patients, we were unable to identify any differences in results between subgroups of children with different ages, diagnoses, or clinical presentations (i.e., pulmonary hypertension). Finally, we did not measure plateau pressures or intrinsic PEEP (positive end expiratory pressure) in this study. Other authors [12] discussed the limitations of measuring peak inspiratory pressure rather than plateau pressure. Our experience with similar patients has revealed that no appreciable intrinsic PEEP can be measured.

Conclusions

In summary, infants undergoing cardiac surgery can be mechanically ventilated using a volume-targeted mode of ventilation with decelerating flow wave pattern (PRVC) which results in a 19% decrease in peak inspiratory pressure without affecting hemodynamics, arterial oxygenation, or carbon dioxide removal.

References

1. Abraham E, Yoshihara G (1990) Cardiorespiratory effects of pressure controlled ventilation in severe respiratory failure. *Chest 98:*1445–1449.
2. Al-Saady N, Bennett E (1985) Decelerating inspiratory flow waveform improves lung mechanics and gas exchange in patients on intermittent positive pressure ventilation. *Intensive Care Med 11:*68–75
3. Davis K Jr, Branson RD, Campbell RS, Porembka DT (1996) Comparison of volume control and pressure control ventilation: is flow waveform the difference? *J Trauma 41:*808–814
4. DiCarlo JV, Raphaely RC, Steven JM, Norwood WI, Costarino AT (1992) Pulmonary mechanics in infants after cardiac surgery. *Crit Care Med 20:*22–27
5. Elixson EM, Myrer ML, Horn MH (1997) Current trends in ventilation of the pediatric patient. *Crit Care Nursing Q 20:*1–13
6. el Khatib MF, Chatburn RL, Potts DL, Blumer JL, Smith PG

(1994) Mechanical ventilators optimized for pediatric use decrease work of breathing and oxygen consumption during pressure-support ventilation. *Crit Care Med 22:*1942–1948
7. Fuhrman B (1990) *Assisted Ventilation and Cardiac Disease.* Norwalk: Appleton & Lange, Norwalk, CT, pp 969–982
8. Hubmayr R, Abel M, Rehder K (1990) Physiologic approach to mechanical ventilation. *Crit Care Med 18:*103–113
9. Kolobow T, Moretti M, Fumagalli R, et al. (1987) Severe impairment in lung function induced by high peak airway pressure during mechanical ventilation. *Am Rev Respir Dis 135:*312–315
10. Konyukov Y, Kuwayama N, Fukuoka T, et al (1996) Effects of different triggering systems and external PEEP on trigger capability of the ventilator. *Intensive Care Med 22:*363–368
11. MacIntyre NR (1996) New modes of mechanical ventilation. *Clin Chest Med 17:*411–421
12. Markstrom A, Lichtwarck-Aschoff M, Svensson B, Nordgren K, Sjostrand U (1996) Ventilation with constant versus decelerating inspiratory flow in experimentally induced acute respiratory failure. *Anesthesiology 84:*882–889
13. Munoz J, Guerrero JE, Escalante JL, Palomino R, De La Calle B (1993) Pressure-controlled ventilation versus controlled mechanical ventilation with decelerating inspiratory flow. *Crit Care Med 21:*1143–1148
14. Parker J, Hernandez L, Peevy K (1993) Mechanisms of ventilator induced lung injury. *Crit Care Med 21:*131–143
15. Pinsky M (1990) The effects of mechanical ventilation on the cardiovascular system. *Crit Care Clin 6:*663–677
16. Rappaport SH, Shpiner R, Yoshihara G, et al (1994) Randomized, prospective trial of pressure-limited versus volume-controlled ventilation in severe respiratory failure. *Crit Care Med 22:*22–32
17. Tobin MJ (1994) Mechanical ventilation. *N Engl J Med 330:*1056–1061

DOI: 10.1007/s002460010224

Around *PediHeart*: Exercise Syncope

Children are referred to pediatric cardiologists for syncope and presyncope every day. Usually their history and physical examination plus a little reassurance are all that's needed. Occasionally, the situation is a little different. One of our colleagues sought advice about a young lady who complained of presyncope during exercise. The patient had no other symptoms and no presyncope at rest or upon standing. Her resting electrocardiogram, and echocardiogram were normal. Exercise ECG using a standard Bruce Protocol led to presyncopal symptoms during maximal exercise at around 12 minutes. All measured parameters were normal except for her blood pressure. She had a resting pressure of 97/65 mmHg and a pressure at maximal exercise of 110/67 mmHg. She asked what to make of these findings?

There were two unusual features to this case. First, she complained of presyncope during exercise, which is uncommon in an otherwise healthy child. Secondly, she had a significantly blunted blood pressure response. Readers generally divided their comments between these two points. Relevant to her exercise presyncope some mentioned the typical post-exercise vasodilatory syncope commonly seen at the finish line of distance events. Several other members believed that exercise hyperventilation or "pseudo-asthma" was a possible cause for this child's exercise presyncope. They wrote that hyperventilation and hypocapnea during exercise can be associated with chest pain and dyspnea [2]. In addition, hypocapnea or the neural mediation of breathing mechanics may play a role in syncope [3] and by extension may be a factor in exercise syncope. Finally, one additional thought was that she may have a form of vertigo made worse by motion at maximal exercise. The other issue was the blunted blood pressure response. Readers pointed out that such a blunted response to exercise typically reflects left ventricular dysfunction, ischemia, or aortic outflow obstruction. However, while an increase in systolic pressure with exercise of less than 30–40 mmHg is abnormal, both *PediHeart* members and the literature [1] report the occurrence in normal children. Finally, others thought that perhaps the true peak BP was missing or inaccurate and repeating the exercise using cycle ergometry would be helpful.

Francis McCaffrey, M.D.
PediHeart Editor

References

1. Albert BA, Verill DE, Flood NL, et al. (1983) Complications of ergometer exercise in children. *Pediatr Cardiol 4*:91–96
2. Hammo A, Weinberger MM (1999) Exercise-induced hyperventilation: A pseudoasthma syndrome. *Ann Allergy Asthma Immunol 82*:574–578
3. Jordan J, Shannon JR, Diedrich A, et al. (2000) Interaction of carbon dioxide and sympathetic nervous system activity in the regulation of cerebral perfusion in humans. *Hypertension 36*:383–388