

Severe Head Injury Management: A Comparative Study of Pressure Controlled and Volume Controlled Ventilation

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ABSTRACT

Background: A Glasgow Coma Score (GCS) of 3-8 indicates a severe head injury. This is common after a motor vehicular accident, which is a leading cause of death in our society. It may also occur as a result of civil unrest, among other things. These patients may require ventilatory support, especially if their respiratory system is in danger of failing. **Objective:** The aim of the study was to prospectively compare which ventilation variable within the same mode of mechanical ventilation is a better option in this group of patients.

Methodology: This was a prospective study of twenty nine (29) patients from February to September 2021, admitted to the Intensive Care Unit (ICU) of 44 Nigeria Army Reference Hospital, Kaduna, with severe head injury within the first 72 hours of admission comparing two variables of the same mode of mechanical ventilatory support. **Results:** Peak airway pressure ($p < 0.15$) was found to be significantly higher during volume-controlled ventilation (VCV), while arterial oxygen tension ($p < 0.5$) was found to be significantly higher during pressure-controlled ventilation (PCV). Peak airway pressure (Paw) decreased consistently during PCV, with the percentage reduction ranging from 5% to 27%, with a mean of 13.3%. It was also discovered that during PCV, there was an increase in arterial oxygen tension as well as an improvement in arterial oxygenation in all patients (100%) compared to 9 (64.3%) patients during VCV. **Conclusion:** We concluded that PCV is as effective as VCV, but it is a better option for improving patient oxygenation in severely brain-injured patients. Though, a better option for oxygenation; but the difference is not statistically significant. Pressure-controlled ventilation variable should be used in the first 72 hours of mechanical ventilator support for patients with severe head injury.

Keywords: Arterial Oxygenation, Severe Head Injury, Pressure Controlled Ventilation.

INTRODUCTION

Mechanical ventilation is a method of providing ventilatory support to patients who have respiratory failure or are on the verge of having respiratory failure in order to improve oxygenation and expel carbon dioxide from the

body. This was accomplished through negative pressure ventilation, as seen in iron lungs. Positive ventilation, however, has become the gold standard in modern medical practice, with Volume Controlled Ventilation (VCV) as the initial mode.

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The most recent mechanical ventilators have both volume-controlled and pressure-controlled ventilation modes. In terms of improved arterial oxygenation, pressure-controlled ventilation was said to have an advantage over volume-controlled ventilation. Pressure controlled ventilation (PCV) is an alternative ventilation technique used in patients with severe respiratory failure.¹ Because of its decelerating inspiratory flow, PCV has been shown to improve arterial oxygenation and lower peak airway pressure.² Better arterial oxygenation in patients with respiratory failure is primarily due to uniform distribution of inspired gas with PCV.³

METHODOLOGY

This was a prospective non consented study. Thirty patients were recruited but 29 patients completed the study. The inclusion criteria were patients with ASA II-III, age ranges from 15 to 60 years who were admitted to the Intensive Care Unit (ICU) of the 44 Nigeria Army Reference Hospital in Kaduna between February and September 2021 after suffering a severe head injury from a car accident or an assault. Patients who were moribund, under the age of 15, or over the age of 60 were excluded from the study.

Patients were randomly assigned to one of two groups prior to intubation. With the patient breathing room air, baseline vital signs, ECG, and arterial blood-gas samples were obtained.

All patients were premedicated with 0.2mg glycopyrrolate and preoxygenated for 5 minutes. These patients were already unconscious, no induction agents were administered; however, intubation was facilitated with an intravenous injection of 1mg/kg suxamethonium.

The trachea was intubated using a polyvinyl chloride (PVC) endotracheal tube of the appropriate size as predicted by the patient's little finger. The typical tracing of the end-tidal carbon dioxide analyser connected via the main stream confirmed appropriate endotracheal intubation. All patients' lungs were ventilated at a tidal volume of 10 ml/kg and a frequency of 10 cycles per minute. PCV was started with a peak airway pressure of 10

ml per kilogram body weight, and ventilation frequency was adjusted to maintain a PaCO₂ of 35 - 45mmHg during PCV. Both PCV and VCV have an inspiratory and pause time of 25% and 10%, respectively. The inspired oxygen concentration (FiO₂) was reduced to 60% after the initial 100% for the first hour of intubation. The PCV group had 15 patients complete the study, while the VCV group had 14 patients complete the study. This was due to the death of one of the VCV patients. To avoid bias, the following variables were measured and recorded at regular intervals for each group. The arterial and venous oxygen tensions and saturations (PaO₂, SaO₂, SVO₂), PaCO₂, and haemoglobin (Hb) were measured in 120 seconds using an I-stat machine equipped with a CG8+ cartridge. Temperature (°C), heart rate (HR), blood pressure (BP), and mean arterial pressure (MAP) were all recorded. The mid-chest level was used as the zero reference. During each mode of ventilation, peak airway pressure (Paw) and mean airway pressure (Paw) were measured. The obtained data were presented as mean (standard deviation) and paired. To determine the significance of the differences between the two groups, the Student's t-test, chi-square test, and ANOVA were used. *P*<0.05 was considered statistically significant.

RESULTS

Table 1 shows the patient's biodata, while Tables 2 and 3 show the mean values for ventilation, gas exchange, and haemodynamic variables. The following variables, SaO₂, PaCO₂, SVO₂,

Table 1: A summary of the patients' bio-data and arterial partial pressure of oxygen and carbon dioxide

Variables	Indices
Age (yrs.)	35.1 (22-61)
Gender (M/F)	23/6
Preoperative PaO ₂ (mmHg)	78.2±1.12
Preoperative PaCO ₂ (mmHg)	53±0.63

PaO₂: arterial oxygen tension, PaCO₂: arterial carbon dioxide tension. Total number of patients recruited (n=29).

Table 2: Post intubation dynamic variables

	Pressure Controlled		Volume Controlled		
	Ventilation (PCV) n=15		Ventilation (VCV) n=14		
Tidal volume (ml)	590 ±79		619 ± 84		
Paw (cm H ₂ O)+	19.35 ±3.31		22.3 ±4.7		<i>P</i> <0.15
PaO ₂ (mmHg)+	94.3 ±13.5		88.4 ±14.1		<i>P</i> < 0.5
SaO ₂ (%)	98.5 ±1.9		97.3 ±1.1		
PaCO ₂ (mmHg)	39.15 ± 0.07		39.28 ±0.79		
SVO ₂ (%)	83.4 ±6.71		81.9 ±8.6		

Paw: Peak airway pressure. PaO₂: arterial oxygen tension, SaO₂: arterial oxygen saturation, PaCO₂: arterial carbon dioxide tension, SVO₂: mixed venous oxygen saturation, PCV: pressure-controlled ventilation, VCV: volume-controlled ventilation.

Table 3: Dynamic variables

Variables	PCV	VCV
Temperature (°C)	36.8 ± 2.2	36.4 ± 2.8
MAP (mm Hg)	91.8 ± 11	88 ± 10
HR (beat/min)	80.4 ± 9.1	81.9 ± 9.5

HR: Heart rate, MAP: mean arterial pressure, PCV: pressure-controlled ventilation, VCV: volume-controlled ventilation.

Temperature, Hb, and MAP, did not differ significantly between ventilation modes. However, when the PCV and VCV modes were compared, there were significant differences in Paw and PaO₂.

During PCV, paw was significantly lower than during VCV (19.35 (3.31) cm H₂O vs 22.3 (4.7) cm H₂O) with a *p*-value 0.15. In comparison to VCV, Paw decreased consistently during PCV, ranging from 2.5 to 11.5 cm H₂O. The mean PaO₂ during PCV was significantly higher 94.3±13.5mmHg vs VCV 88.4±14.1mmHg (*p*<0.5). Arterial oxygenation improved in all (n=15) PCV patients and 9 (n=14) VCV patients (*p*<0.5).

DISCUSSION

VCV and PCV are ventilation variables that are commonly used in the same modes of mechanical ventilation in the intensive care unit. However, PCV is one of the most recent ventilation variables, with the benefit of improved arterial oxygenation as well as recruitment of respiratory alveoli in preventing ventilation perfusion mismatch and

atelectasis. VCV provides the safety of a pre-set tidal volume and minute ventilation, but it requires the clinician to set the inspiratory flow, flow waveform, and inspiratory time appropriately. Airway pressure rises during VCV in response to decreased compliance, increased resistance, or active exhalation, potentially increasing the risk of ventilator-induced lung injury. PCV primarily limits the maximum airway pressure delivered to the lung; however, it may cause variable tidal and minute volume. During PCV, the clinician should titrate the inspiratory pressure to the measured tidal volume, but the ventilator determines the inspiratory flow and flow waveform as it attempts to maintain a square inspiratory pressure profile. The majority of studies that compare the effects of PCV and VCV contribute little to our understanding of when and how each control variable should be used.^{1,3,5} However, the advantages of PCV and VCV can be combined in so-called dual-control modes, which are volume-targeted, pressure-limited, and time-cycled. We used the same tidal volumes (10ml/kg) to compare the effects of each variable of the same mode of ventilation on airway pressure and arterial oxygenation, because tidal volumes greater than 10ml/kg are detrimental to arterial oxygenation. A tidal volume of 10ml/kg has the least negative impact on arterial oxygenation.⁴ Peak inspiratory pressures in our study were consistently lower

during PCV. This was consistent with previous research that found that high airway pressures can cause barotrauma.⁵ Because of its decelerating flow profile; PCV has previously been linked to sustained reductions in peak inspiratory pressure.⁶ It is worth noting that the beneficial effects of reduced peak inspiratory pressures during PCV also cause a reduction in peak alveolar pressure, resulting in less alveolar over distension.⁷

Our findings show that the beneficial effects of PCV's decelerating inspiratory flow pattern on gas distribution within the lung resulted in improved arterial oxygenation. The decelerating flow of PCV maintains alveolar pressure longer than the constant flow of VCV, resulting in more alveolar recruitment and improved oxygen homogeneity.⁸ In our study, we also found that the improved PaO₂ and mean PaO₂ in all patients during PCV were significantly higher than during VCV. This is consistent with a similar study by Ahmed et al,⁹ although it was done on one lung but the overall findings is in agreement with that of this study. In yet another study by Hassan et al,¹⁰ a similar outcome was reported that an improved PaO₂ in PCV than VCV; although it was done in obese patients. The flow profile of this ventilatory mode may explain the improvement in arterial oxygenation during PCV.

One of the limitations associated with this study is that not all the patients had a normal pulmonary function from the beginning of the ventilation; some had a blunt chest injury. It has been reported that such limitations may not extrapolate to sicker patients with compromised pulmonary function.^{11, 12} In a comparative study, the minimum sample size as indicated in the literature was 52 patients per group; the limited sample size in this study is also part of the limitations as it limits the sensitivity of the study outcome^{13,14,15}

CONCLUSION

We conclude that when available on a mechanical ventilator; the used of pressure-controlled ventilation is a superior ventilation variable for providing ventilatory support in patients with

severe head injuries especially in the first 72 hours of mechanical ventilation. This is due to beneficial effects such as a decelerating flow pattern of PCV, which improved alveolar gas distribution and arterial oxygenation. It also has the advantage of maintaining constant and consistent alveolar pressure. This ventilation variable is critical for preventing barotrauma and atelectasis. It is therefore recommended that pressure-controlled ventilation variable should be used in the first 72 hours of mechanical ventilator support for patients with severe head injury.

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