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Comparison of the Effect of Two Variable Intraoperative Ventilation Modes on Pulmonary Compliance and Gas Exchange in Patients Undergoing Laparoscopic Abdominal Surgery

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ABSTRACT

Background: Pulmonary atelectasis is a common complication post-laparoscopic surgery due to carbon dioxide insufflation, which reduces lung compliance. Volume-controlled ventilation (VCV) is standard but increases airway pressures. Pressure-controlled volume-guaranteed ventilation (PCV-VG) may reduce stress but its benefits over VCV are debated.

Aim: To compare VCV and PCV-VG regarding pulmonary compliance and gas exchange throughout laparoscopic abdominal surgery.

Methods: A randomized controlled trial of 60 adult cases having elective laparoscopic surgery. Patients have been separated into two groups (30 each): VCV and PCV-VG. Outcomes measured included respiratory compliance, peak pressure, heart rate, mean arterial pressure (MAP), SpO_2, and adverse events.

Results: The study included 60 participants (mean age 40.27±11.48 years, 1kg/m²). Surgery duration was 2.82±0.32 hours. Group B (PCV-VG) showed better SpO_2 and PaO_2, while Group A (VCV) had higher tidal volumes, peak pressures, and lower respiratory compliance. Respiratory compliance was significantly better in Group B ($p<0.0001$).

Conclusion: PCV-VG outperformed VCV in pulmonary mechanics and gas exchange, with better oxygenation, lower peak pressures, and improved respiratory compliance. VCV was associated with higher airway pressures, increasing risks of barotrauma and atelectasis.

Keywords: *Intraoperative ventilation, Pulmonary compliance, Gas exchange, Laparoscopic abdominal surgery, Ventilation modes*

1. INTRODUCTION

Pulmonary atelectasis is a common complication following laparoscopic abdominal surgery, often due to the use of carbon dioxide insufflation for pneumoperitoneum. This procedure raises intra-abdominal pressure, causing an upward shift in the diaphragm and reducing lung compliance (Umano et al., 2021). As the diaphragm shifts, lung atelectasis can occur, lowering the functional residual capacity of the lungs. The perfusion of non-ventilated alveoli significantly affects blood oxygenation, and the pneumoperitoneum further exacerbates atelectasis (Gamal et al., 2023). This leads to ventilation-perfusion mismatch and increases the likelihood of pulmonary complications during the operation. Nevertheless, there is no clear association between oxygenation and atelectasis, as measured by PaO2 or shunt calculations, which cannot accurately assess the extent of lung collapse (Zeng et al., 2022).

During general anesthesia, the most frequently utilized ventilation mode is volume-controlled ventilation, where the anesthetist controls tidal volume, respiratory rate, and the I/E ratio to maintain fixed minute ventilation (Mubark et al., 2023). While effective, this mode may result in elevated airway pressures due to changes in resistance and compliance, making the monitoring of pulmonary compliance and airway resistance essential (Civraz et al., 2023). An alternative to VCV is pressure-controlled volume-guaranteed ventilation (PCV-VG), where the anesthetist controls peak airway pressure, respiratory rate, and the I/E ratio. The tidal volume may vary in PCV-VG, and attention should be paid to this. PCV-VG aims to reduce airway pressures and systemic stress during laparoscopic surgery, but its respiratory advantages over VCV remain debated (Schick et al., 2021).

Previous studies have compared VCV and PCV-VG regarding respiratory and hemodynamic parameters, but results have been inconclusive (Taha et al., 2023). Although volume control ventilation is traditionally advocated to prevent intraoperative atelectasis, PCV-VG may provide lower airway pressures and reduce systemic stress, making it a potential alternative for laparoscopic procedures (Ewees et al., 2020).

Goal of research to compare between Volume-Controlled and Pressure-Controlled Volume-Guaranteed Ventilation upon Pulmonary Compliance and Gas Exchange throughout laparoscopic abdominal surgery

2. Patients and Methods

The research was a prospective randomized controlled trial carried out at the Anesthesia, Critical Care, and Pain Management Department of Qena University Hospital. It aimed to compare the effects of two various ventilation methods during laparoscopic abdominal surgery. The sample size was determined using a sample size equation, which calculated 30 patients per group. Group I consisted of 30 patients who were anesthetized and ventilated using volume control ventilation (VCV), while group II, also with 30 patients, received pressure control volume guaranteed ventilation (PCV-VG).

The research involved adult cases aged 18–70 years who had an ASA physical status classification of I–II and required general anesthesia with endotracheal intubation and mechanical ventilation for elective laparoscopic surgery. Cases were excluded if they refused to participate, had an ASA classification higher than II, a body mass index over 30 kilogram per square meter, a history of thoracic operation, severe obstructive pulmonary disease, diaphragmatic paralysis, nervous system disease, or had undergone mechanical ventilation within the past month.

For all patients' General anesthesia (GA) was administered. Following identification, each case was connected to monitoring apparatuses, involving an electrocardiograph, pulse oximetry for oxygen saturation (SpO_2) and capnography for carbon dioxide (CO_2). GA induction involved propofol (2 milligram per kilogram), fentanyl (1–2 microgram per kilogram), and morphine (5 milligram), while tracheal intubation was facilitated with atracurium (0.5 milligram per kilogram). Patients were then assigned to either volume control ventilation (VCV) with a tidal volume of 8–10 milligram per kilogram predicted body weight (PBW), a respiratory rate of 12 cycles per minute, an inspiratory-to-expiratory ratio of 1:2, and zero end-expiratory pressure, or pressure control ventilation with volume guarantee (PCV-VG) to maintain similar tidal volumes and respiratory settings.

Anesthesia maintenance involved sevoflurane (1.5–2.5 vol%) in a 50% oxygen/air mixture at a flow rate of 3 liter per minute, keeping blood pressure within 20% of baseline. Ventilation was managed to preserve end-tidal carbon dioxide between thirty and forty millimeters of mercury. CO_2 pneumoperitoneum was induced to an intra-abdominal pressure of 12 ± 2 mmHg. If peak airway pressure exceeded 40 mmHg or arterial desaturation ($SpO_2 \leq 92\%$) occurred, FiO_2 was increased, and the procedure was halted. Intravenous fluids were administered at 5 milliliters per kilograms per hour throughout anesthesia induction, with maintenance fluids of crystalloid solutions at 8–10 mL/kg per hour. Colloid or blood transfusions were given to replace intraoperative blood loss as needed. After surgery, patients were extubated in the operating room, and oxygen was provided in the recovery room at 6 L/min, along with postoperative analgesia upon request.

Respiratory system compliance, peak pressure and tidal volume were measured at four key points: 5 minutes after tracheal intubation, 10 minutes after pneumoperitoneum, just before desufflation, and after desufflation and before emergence. Heart rate, mean arterial pressure, and SpO_2 were documented at similar intervals, including 1 hour after emergence in the post-anesthesia care unit (PACU). Any adverse events, including barotrauma, oxygen desaturation (SpO_2 <90%), and ICU admissions, were noted. Patients were discharged when deemed appropriate by the surgeon, who was blinded to group assignment.

Data were gathered and organized for subsequent statistical evaluation. The analysis was carried out using SPSS software (version 26). For qualitative variables, the data were expressed as percentages and frequencies, with comparisons made utilizing the chi-square test. Quantitative data were summarized as means with standard deviations (SD) and compared utilizing the student's t-test. Additionally, regression and correlation analyses were conducted as needed. Statistical significance was set at a p-value of less than 0.05.

This study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained at qena University Hospital.

All participants provided informed consent prior to participation in the study. Confidentiality of participant data was maintained throughout the research process, and all collected data were used solely for research purposes.

3. Results

Table (1): Comparative analysis among the studied groups with regard to demographic data:

	Group (A) (Number = 30)	Group (B) (Number = 30)	P. Value
Age (Years)	39.1 ± 10.67	41.43 ± 12.12	0.3706
Gender			
Male	14 (46.67%)	8 (26.67%)	0.1116
Female	16 (53.33%)	22 (73.33%)	
ASA			
I	26 (86.67%)	21 (70%)	0.1212
II	4 (13.33%)	9 (30%)	
BMI (Kg/m2)	28.1 ± 1.9	28.6 ± 2.03	0.3055
Operative duration (hours)	2.96 ± 0.31	2.88 ± 0.26	0.1103

The mean age and gender distribution between groups were comparable (p = 0.3706 and p = 0.1116, respectively). ASA classification and BMI were also similar (p = 0.1212 and p = 0.3055), operative duration was longer in Group A (2.96 ± 0.31 hours vs. (2.88 ± 0.26) hours with insignificant variance, (p = 0.1103).

Table (2): Comparative analysis between the studied groups regarding hemodynamics:

	Group (A) (Number = 30)	Group (B) (Number = 30)	P. Value
HR (beat/min)			
• **5 min after intubation**	92 ± 8.09	90.83 ± 8.58	0.6273
• **Just before the de-sufflation**	90.7 ± 5.44	90.37 ± 9.06	0.8659
• **After the de-sufflation**	89.27 ± 3.82	88.53 ± 7.61	0.9703
• **Before emergence from anesthesia**	93.37 ± 5.07	92.1 ± 8.46	0.4563
• **1 h after the emergence from anesthesia**	95.3 ± 7.6	92.97 ± 7.89	0.2446
MAP (mmHg)			
• **5 min after intubation**	92.77 ± 8.28	91.37 ± 9.45	0.6947
• **Just before the de-sufflation**	82.6 ± 6.15	80.3 ± 7.24	0.2269
• **After the de-sufflation**	76.27 ± 4.64	76.43 ± 5.48	0.6834
• **Before emergence from anesthesia**	82.87 ± 3.9	83.37 ± 4.55	0.5043
• **1 h after the emergence from anesthesia**	88.47 ± 4.65	87.8 ± 4.48	0.5751

The mean heart rate (HR) 5 minutes after intubation was 92 ± 8.09 bpm in Group A and 90.83 ± 8.58 bpm in Group B (p = 0.6273). Just before de-sufflation, HR was 90.7 ± 5.44 bpm in Group A and 90.37 ± 9.06 bpm in Group B (p = 0.8659). After de-sufflation, the HR was 89.27 ± 3.82 bpm in Group A and 88.53 ± 7.61 bpm in Group B (p = 0.9703). Before emergence from anesthesia, the HR was 93.3 ± 5.07 bpm in Group A compared to 92.1 ± 8.46 bpm in Group B (p = 0.4563). One hour after emergence, HR was 95.3 ± 7.6 bpm in Group A and 92.97 ± 7.89 bpm in Group B (p = 0.2446). None of these differences were statistically significant.

Regarding mean arterial pressure (MAP), 5 minutes after intubation, the MAP was 92.77 ± 8.28 millimeters of mercury in Group A and 91.37 ± 9.45 millimeters of mercury in Group B (p = 0.6947). Just before de-sufflation, it was 82.6 ± 6.15 millimeters of mercury in Group A and 80.3 ± 7.24 millimeters of mercury in Group B (p = 0.2269). After de-sufflation, MAP measured 76.27 ± 4.64 millimeters of mercury in Group A and 76.43 ± 5.48 mmHg in Group B (p = 0.6834). Before emergence from anesthesia, MAP was 82.87 ± 3.9 mmHg in Group A and 83.37 ± 4.55 millimeters of mercury in Group B (p = 0.5043). One hour after emergence, MAP was 88.47 ± 4.65 millimeters of mercury in Group A and 87.8 ± 4.48 millimeters of mercury in Group B (p = 0.5751). All MAP comparisons also showed a statistically insignificant variance

Table (3): Comparison among the studied groups with regard to findings of arterial blood gas analysis:

	Group (A) (Number = 30)	Group (B) (Number= 30)	P. Value
SpO2 (%)			
• **5 min after intubation**	98.1 ± 0.82	98.3 ± 0.5	0.21
• **Before the de-sufflation**	97.5 ± 1.09	98.2 ± 0.79	0.013
• **After the de-sufflation**	98 ± 0.86	98.37 ± 0.48	0.0961
• **Before emergence from anesthesia**	98 ± 0.77	98 ± 0.73	0.99
• **1 h after the emergence from anesthesia**	98.07 ± 0.77	98.57 ± 0.5	0.0113
PaO2 (mmHg)			
• **5 min after intubation**	214.07 ± 19.39	220.53 ± 22.64	0.2401
• **Before the de-sufflation**	207.03 ± 16.72	214.5 ± 19.97	0.1216
• **After the de-sufflation**	188.5 ± 13.54	199.37 ± 17.28	0.0088
• **Before emergence from anesthesia**	188.33 ± 12.94	193.43 ± 36.42	0.4728
• **1 h after the emergence from anesthesia**	100.2 ± 5.97	103.07 ± 6.5	0.0801
PaCo2 (mmHg)			
• **5 min after intubation**	37.6 ± 3.09	36.17 ± 3.08	0.0805
• **Before the de-sufflation**	34.5 ± 3.02	34.33 ± 2.94	0.7774
• **After the de-sufflation**	36.37 ± 3.18	36.07 ± 3	0.7548
• **Before emergence from anesthesia**	35.83 ± 2.31	35.67 ± 2.12	0.7482
• **1 h after the emergence from anesthesia**	37.17 ± 2.02	38 ± 2.02	0.1084

The mean SpO_2 (%) 5 minutes after intubation was 98.1 ± 0.82 in Group A and 98.3 ± 0.5 in Group B (p = 0.21). Before de-sufflation, SpO_2 was significantly higher in Group B (98.2 ± 0.79) compared to Group A (97.5 ± 1.09), with a statistically significant variance (p = 0.013). After de-sufflation, SpO_2 was 98 ± 0.86 in Group A and 98.37 ± 0.48 in Group B (p = 0.0961). An insignificant variance was noted before emergence from anesthesia, with values of 98 ± 0.77 in Group A and 98 ± 0.73 in Group B (p = 0.99). However, one hour after emergence, SpO_2 remained significantly greater in Group B (98.57 ± 0.5) than Group A (98.07 ± 0.77), with a p-value of 0. 0113.

As for PaO_2 (mmHg), Group B had higher values than Group A at all- time points, although statistical significance was only reached after de-sufflation. Specifically, PaO_2 was 214.07 ± 19.39 in Group A and 220.53 ± 22.64 in Group B 5 minutes after intubation (p = 0.2401), and 207.03 ± 16.72 in Group A vs. 214.5 ± 19.97 in Group B before de-sufflation (p = 0.1216). After de-sufflation, PaO_2 was significantly higher in Group B (199.37 ± 17.28) compared to Group A (188.5 ± 13.54), with a p-value of 0.0088. An insignificant variance was found before emergence (p = 0.4728) or 1 hour after emergence (p = 0.0801).

Regarding $PaCO_2$ (mmHg), a statistically insignificant variances were found among the groups at any measured time point. Five minutes after intubation, $PaCO_2$ was 37.6 ± 3.09 in Group A and 36.17 ± 3.08 in Group B (p = 0.0805). Before de-sufflation, values were 34.5 ± 3.02 and 34.33 ± 2.94, respectively (p = 0.7774). After de-sufflation, $PaCO_2$ was 36.37 ± 3.18 in Group A vs 36.07 ± 3 in Group B (p = 0.7548). Before emergence from anesthesia, Group A had a mean of 35.83 ± 2.31 and Group B 35.67 ± 2.12 (p = 0.7482). One hour after emergence, values were 37.17 ± 2.02 and 38 ± 2.02 in Group A and B, correspondingly (p = 0.1084).

Table (4): Comparative analysis among the studied groups with regard to pulmonary mechanics:

	Group (A) (Number = 30)	Group (B) (Number = 30)	P. Value
Tidal volume			
• **5 min after intubation**	445.24 ± 15.47	433.27 ± 13.41	0.0052
• **10 min after pneumoperitoneum**	461.24 ± 14.43	444.82 ± 14.73	0.0001
• **Before the de-sufflation**	439.43 ± 16.19	424.22 ± 15.54	0.0018
• **After the de-sufflation**	456.72 ± 17.35	437.35 ± 15.98	0.0002

• **Before emergence from anesthesia**	432.1 ± 18.95	411.97 ± 16.04	0.0003
Peak pressure (cmH2O)			
• **5 min after intubation**	14.37 ± 1.78	11.63 ± 2.39	<0.0001
• **10 min after pneumoperitoneum**	30.3 ± 2.18	13.83 ± 1.46	<0.0001
• **Before the de-sufflation**	28.83 ± 2.28	13.4 ± 1.47	<0.0001
• **After the de-sufflation**	17.27 ± 2.78	11 ± 1.48	<0.0001
• **Before emergence from anesthesia**	13 ± 2.11	7.67 ± 1.19	<0.0001
Respiratory system compliance (mL/cmH2O)			
• **5 min after intubation**	32.61 ± 3.97	43.71 ± 5.82	<0.0001
• **10 min after pneumoperitoneum**	14.19 ± 1.64	32.86 ± 2.47	<0.0001
• **Before the de-sufflation**	12 ± 1.5	31.13 ± 2.52	<0.0001
• **After the de-sufflation**	28.73 ± 4.39	37.94 ± 4.61	<0.0001
• **Before emergence from anesthesia**	40.2 ± 5.71	46.8 ± 6.13	0.0005

The tidal volume was significantly greater in Group A at all time points. Five minutes after intubation, it was 445.24 ± 15.47 mL in Group A compared to 433.27 ± 13.41 mL in Group B ($p = 0.0052$). Ten minutes after pneumoperitoneum, tidal volume remained significantly higher in Group A (461.24 ± 14.43 mL) than in Group B (444.82 ± 14.73 mL; $p = 0.0001$). Similarly, values before de-sufflation were 439.43 ± 16.19 mL in Group A and 424.22 ± 15.54 mL in Group B ($p = 0.0018$), after de-sufflation were 486.72 ± 17.35 mL vs 437.35 ± 15.98 mL ($p = 0.0002$), and before emergence were 432.1 ± 18.95 mL vs 411.97 ± 16.04 mL ($p = 0.0003$).Regarding peak airway pressure (cmH_2O), Group A consistently showed significantly higher values than Group B at all measurement points. Five minutes after intubation, peak pressure was 14.87 ± 1.78 in Group A vs 11.63 ± 2.39 in Group B ($p < 0.0001$). At 10 minutes after pneumoperitoneum, it was 30.3 ± 2.18 vs. 13.83 ± 1.46 ($p < 0.0001$), before de-sufflation was 28.83 ± 2.28 vs. 13.54 ± 1.47 ($p < 0.0001$), after de-sufflation was 17.27 ± 2.78 vs. 11 ± 1.48 ($p < 0.0001$), and before emergence was 13 ± 2.11 vs. 7.67 ± 1.19 ($p < 0.0001$).

Conversely, respiratory system compliance (mL/cmH_2O) was significantly greater in Group B at all-time points. Five minutes after intubation, Group B recorded 43.71 ± 5.82 vs. 32.61 ± 3.97 in Group A ($p < 0.0001$). Ten minutes after pneumoperitoneum, values were 32.86 ± 2.47 vs. 14.19 ± 1.64 ($p < 0.0001$), before de-sufflation were 31.13 ± 2.52 vs. 12 ± 1.5 ($p < 0.0001$), after de-sufflation were 37.94 ± 4.61 vs. 28.73 ± 4.39 ($p < 0.0001$), and before emergence were 46.8 ± 6.13 vs. 40.2 ± 5.71 ($p = 0.0005$).

4. Discussion

Heart rate (HR) and mean arterial pressure remained comparable between Group A and Group B at all measured time points, with no statistically significant differences observed, these findings indicate that both groups maintained stable hemodynamic responses throughout the perioperative period, with no significant fluctuations, suggesting effective anesthetic management and similar compensatory mechanisms in both groups (Zhou et al., 2024). Wang et al. (2015) also reported no significant differences in HR or MAP between ventilation groups, supporting our results. Yılmaz et al. (2022) also reported that two ventilation modes have similar effect on HR and MAP in patients undergoing gynecological laparoscopic surgery with exaggerated Trendelenburg positioning (Al-Aziz et al., 2024).

SpO_2 levels were significantly greater in Group B in comparison with Group A before de-sufflation, and 1 hour after emergence, with no significant differences observed at 5 minutes after intubation, after de-sufflation or before emergence. PaO_2 was significantly higher in Group B after de-sufflation, but no significant differences were found at other time points. $PaCO_2$ remained comparable between groups. These findings are in line with Assad and Khalil (2016), who reported a significant decline in PaO_2 in both groups during later time points due to pneumoperitoneum, though SpO_2 remained stable, These outcomes are in accordance with Li et al. (2020), who reported PC-VGV significantly improved the PaO2 ratio compared to VCV indicating better oxygenation while $PaCO_2$ levels remained comparable between two groups, however Zhu et al. (2022) showed that $PaCO_2$ was significantly reduced in the PCV-VG group, however, there were an insignificant variances in PaO_2 ratio between two groups during pulmonary resection in pediatric patients requiring one-lung ventilation (OLV) , there outcomes wasn't consistent with the outcomes of our research that is probably due to type of patient , a pediatric case, and type of surgery, pulmonary resection, and its physiological consequences (Taha et al., 2025).

Our study observed that tidal volume was significantly greater in Group A at multiple time points, including 5 minutes following intubation, 10 minutes after pneumoperitoneum, before de-sufflation. Peak pressure was greater in Group A at all-time points. Respiratory system compliance was significantly greater in Group B at all-time points, particularly after pneumoperitoneum. These results highlight the impact of different ventilatory strategies on lung mechanics, with Group A showing greater airway resistance and alveolar strain, while Group B had improved lung elasticity and reduced restrictive effects. Suleiman et al. (2022) supported these findings, emphasizing that Group B's improved compliance minimized lung stress. Wang et al. (2015) also noted that PCV was related to lower peak airway pressure and higher compliance, consistent with our study's results for Group B, also in accordance with our research Toker et al. (2019) reported that tidal volume was significantly greater in the VCV group compared to the PCV-VG group, but PCV-VG showed lower peak inspiratory pressures and improved dynamic lung compliance, however Deng et al. (2023) reported Tidal volume remained comparable between groups without significant difference, which not consistent with our result, but in line with our result Group B's improved compliance minimized air way pressure, these findings suggest that PC-VG ventilation may offer mechanical advantages over VCV by reducing airway pressures and improving lung compliance during laparoscopy. Kim et al. (2016) The results showed that PCV was related to significantly lower peak inspiratory pressure, these results were consistent with our study regarding peak pressure, however Lung compliance and tidal volume didn't vary significantly among two groups which is not consistent with our study throughout one-lung ventilation (OLV) in adult thoracic surgery.

5. Conclusion

While both volume-controlled ventilation and pressure-controlled volume-guaranteed ventilation (PCV-VG) maintained hemodynamic stability during laparoscopic abdominal surgery, PCV-VG outperformed VCV in pulmonary mechanics and gas exchange. The PCV-VG group had reduced peak airway pressures, higher dynamic compliance, and better oxygenation indices (SpO_2 and PaO_2), particularly after pneumoperitoneum, indicating improved alveolar recruitment and reduced ventilator-induced lung stress. VCV, while providing higher tidal volumes, was associated with higher airway pressures, increasing the risk of barotrauma and atelectasis under laparoscopic conditions.

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