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Original Research Article

Comparison of the effects of volume-controlled ventilation and pressure-controlled ventilation modes on hemodynamics, respiratory mechanics and blood gas parameters in patients undergoing laparoscopic cholecystectomy

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ABSTRACT

Background: This study aimed to compare the changes induced by VCV and PCV modes in hemodynamics, respiration (airway pressures, gas exchange parameters) and metabolism (acid-base balance) in patients undergoing laparoscopic cholecystectomy.
Methods: Patients were divided into two randomised groups as volume-controlled ventilation (VCV) group (VC) and pressure-controlled ventilation (PCV) group (PC). The following parameters were recorded at 3 different time points: T1: in supine position 10 minutes after induction of anaesthesia, T2: 15 minutes after CO_2 insufflation in inverted Trendelenburg position (head 30 degrees up), T3: 10 minutes after CO_2 desufflation. HR, SpO_2, SAP (systolic arterial pressure), DAP (diastolic arterial pressure), MAP (mean arterial pressure), $PetCO_2$ (end-tidal carbon dioxide pressure), P_{peak}, $P_{plateau}$, P_{mean}, Vt (tidal volume) and compliance with the available data, the cases in both groups Vd, Vd/Vt ratios and P(A-a)O_2 were calculated. Arterial blood gas parameters (pH, PaO_2, $PaCO_2$, SaO_2, P(a-et)CO_2) values were recorded.
Results: It was found that P_{peak} and $P_{plateau}$ values were significantly higher in the VC group ($p<0.05$). It was found that compliance was significantly higher in the PC group ($p<0.05$) ($p<0.01$). In the postoperative period, it was found that PaO_2 values were significantly higher in the PC group compared to the VC group ($p<0.05$). It was found that the P(A-a)O_2 values of the PC group were significantly higher than those of the VC group during the desufflation phase ($p<0.05$).
Conclusions: We think that PCV mode can be a good alternative for the prevention and correction of physiopathological changes due to laparoscopic surgery.

Keywords: Volume-controlled ventilation, Pressure-controlled ventilation, Laparoscopic cholecystectomy

INTRODUCTION

Because of the short hospital stay, small incision area, less postoperative pain, early mobilisation, less degradation in pulmonary functions in the postoperative period and less scarring at the incision site, laparoscopic surgery has become a preferred method in recent years.[1]

Cardiac, pulmonary and metabolic physiopathological changes caused by laparoscopic surgical intervention under general anaesthesia may be related to mechanical

and neurohumoral causes. It is stated that these changes are related to pneumoperitoneum.[2,3] The length of pneumoperitoneum, the rise of intra-abdominal pressure (IAP), underlying diseases, patient position and mechanical ventilation mode were all related to $PaCO_2$ changes.[2-4]

With a square-wave flow pattern, VCV is the traditional ventilation mode used during anaesthesia. PCV is an alternative mode of ventilation claimed to improve gas exchange in hypoxic patients (primarily adult respiratory syndrome) in intensive care units.[5] The pressure difference between the alveoli and the proximal airways is highest during the start of inspiration in PCV. As a result, during the early inspiratory phase, most of the tidal volume fills the alveoli, making it more straightforward for the unstable alveoli to remain open.[6] It is also stated that PCV delivers better oxygenation than VCV with the same tidal volume and inspiratory time because it has a higher mean airway pressure.[7,8] Ventilation with the PCV mode regulates the distribution of tidal volume from the short-term constant alveoli to the long-term constant alveoli while keeping the inspiratory pressure stable. Thus, it increases arterial oxygenation by reducing ventilation heterogeneity.[6]

Pneumoperitoneum results in increased IAP, decreased lung volume and functional residual capacity (FRC), decreased pulmonary compliance, but high airway resistance, pressure on the diaphragm, atelectasis of the basal parts of the lung, redistribution of hydrostatic forces and impaired ventilation-perfusion in laparoscopic cholecystectomy surgery.[9,10]

This study aimed to compare the changes induced by VCV and PCV modes in hemodynamics, respiration (airway pressures, gas exchange parameters) and metabolism (acid-base balance) in patients undergoing laparoscopic cholecystectomy.

METHODS

Our research involved 50 patients scheduled for laparoscopic cholecystectomy at the Haseki training and research hospital of the Ministry of Health (MOH). The study comprised patients in the ASA I-II group between 18 and 65 and were scheduled for laparoscopic cholecystectomy. Those who developed complications during surgery, ASA>II, body mass index (BMI) >35 kg/m^2, previously known cardiopulmonary disease, less than 18 years of age and older than 65 years, patients with the hepatorenal disorder, neuromuscular disease, Reynaud's disease, Buerger's disease, hypotensive patients, those who had previous thoracic surgery and those who were harmful to the modified Allen test were excluded from the study.

Patients brought to the operating table were monitored and vascular access was established with a 22-G intravenous cannula. The 0.9% NaCl infusion was started at a rate of 8 ml/kg/hour. HR (heart rate), SAP, DAP, MAP and SpO_2 (peripheral oxygen saturation) were recorded as baseline values. Under local anaesthetic, the non-dominant hand was used to perform radial artery cannulation with a 20-G IV cannula. A preoperative arterial blood gas sample was obtained and analysed (ABL 800 Flex, Radiometer Medical ApS, Denmark).

Thiopental 7 mg/kg for induction of anaesthesia, 2 µg/kg fentanyl and 0.1 mg/kg vecuronium for neuromuscular blockade were administered to patients who had received premedication (0.025 mg/kg IV midazolam). Direct laryngoscopy was used to achieve endotracheal intubation with a tube having an inner diameter of 7.0-8.5 mm.

Patients were divided into two randomised groups as VCV group and PCV group according to the envelope method. Ventilation in the VC group began with a Vt of 8 ml/kg. The peak inspiratory pressure (P_{peak}) in the PCV group was adjusted to give an 8ml/kg Vt while not exceeding the maximum limit of 35 cm H_2O. During the continuance of anaesthesia, the respiratory rate (f) in both groups, which started at 12 /min, was adjusted to keep the $PetCO_2$ in the range of 32-38 mm Hg.

In both groups, anaesthesia was given with sevoflurane in an O_2/air mixture with FiO_2: 50%. The flow was kept constant at 2 l/min, I/E ratio 1:2, PEEP: 5 cm H_2O. IAP was set at 14±2 mm Hg.

The following parameters were recorded at 3 different time points: T1: in supine position 10 minutes after induction of anaesthesia, T2: 15 minutes after CO_2 insufflation in inverted Trendelenburg position (head 30 degrees up), T3: 10 minutes after CO_2 desufflation.

At these times, the following parameters were recorded: HR, SpO_2, SAP, DAP, MAP, $PetCO_2$, P_{peak}, $P_{plateau}$, P_{mean}, Vt, and compliance (C). With the available data, the cases in both groups' dead space (Vd), Vd/Vt ratios and $P(A-a)O_2$ were calculated. Arterial blood gas samples were also taken at the same time. It was evaluated with a blood gas machine. Arterial blood gas parameters (pH, PaO_2, $PaCO_2$, SaO_2 and $P(a-et)CO_2$) values were recorded.

Inhalation agents were turned off and 100% oxygen was supplied once the operation was completed and the patient was returned to the supine position. After the onset of spontaneous respiratory effort, neuromuscular blockade was reversed using atropine 0.01 mg/kg and neostigmine 0.04 mg/kg. When the patient's spontaneous breathing was adequate, the patient was extubated. Operation time and pneumoperitoneum times were recorded.

For postoperative analgesia, the nonsteroidal anti-inflammatory drug tenoxicam 20 mg IV was administered 30 minutes before the end of surgery. At the end of the second hour of the postoperative period, the patients hemodynamic parameters were recorded and ABP analyses were conducted.

The statistical package program SPSS for Windows 10.0 was used to analyse the statistical data. Comparisons were made using the student's t test, Mann-Whitney U test, Chi-square and paired t test. The significance level was set at $p<0.05$.

RESULTS

No significant difference in demographic data (age, height, weight, BMI, operation time, duration of pneumoperitoneum, gender) was found when comparing the groups ($p>0.05$) (Table 1).

In all measurement periods, there was no statistically significant difference between the groups in terms of DAP and MAP values ($p>0.05$) (Table 2). In the T1 period, however, SAP values were more significant in the VC group. In the postoperative period, HR values were determined to be higher in the VC group ($p<0.05$) (Table 2).

It was found that P_{peak} and $P_{plateau}$ values were significantly higher in the VC group ($p<0.05$) (Table 3). It was found that compliance was significantly higher in the PC group ($p<0.05$) ($p<0.01$) (Table 3).

There was no significant difference between the groups in the values of P_{mean}, $PetCO_2$, Vt, Vd, Vd/Vt ($p>0.05$) (Table 3).

In terms of pH, $PaCO_2$, SaO2, and P(a-et)CO2 values, there was no difference between the groups ($p>0.05$) (Table 4).

In the postoperative period, it was found that PaO_2 values were significantly higher in the PC group compared to the VC group ($p<0.05$) (Table 4).

It was found that the $P(A\text{-}a)O_2$ values of the PC group were significantly higher than those of the VC group during the desufflation phase ($p<0.05$) (Table 4).

Table 1: Demographic characteristics of the groups.

Demographic characteristics	VC$^{\alpha}$ group (N=25)		PC$^{\beta}$ group (N=25)		P value
	Mean	SD	Mean	SD	
Age	51.64	12.52	47.32	12.95	0.236
Height	159.64	7.38	160.92	9.62	0.600
Weight	76.88	15.47	77.96	14.51	0.800
BMI	29.60	4.95	29.68	5.59	0.957
Operation time	72.80	17.80	79.00	27.31	0.346
Duration of pneumoperitoneum	44.60	16.52	46.20	22.88	0.778
Male	5.00	20.00	7.00	28.00	0.742
Female	20.00	80.00	18.00	72.00	0.742

$^{\alpha}$: volume controlled; $^{\beta}$: pressure controlled.

Table 2: Comparison of the hemodynamic parameters of the groups.

Comparison	VC$^{\alpha}$ group		PC$^{\beta}$ group		P value
	Mean	SD	Mean	SD	
SAP$^{¥}$ (mmHg)					
Preop	145.84	22.58	139.60	20.55	0.312
T1	143.44	38.65	125.36*	18.27	0.040*
T2	138.84	21.30	129.08	20.11	0.102
T3	136.00	19.00	129.16	16.81	0.184
Postop	123.20	15.47	117.68	12.76	0.175
DAP$^{¥}$ (mmHg)					
Preop	83.88	11.49	82.04	10.31	0.554
T1	88.28	16.83	82.92	11.68	0.197
T2	87.56	14.47	88.48	14.45	0.823
T3	79.56	11.64	80.80	11.98	0.712
Postop	77.60	10.91	73.40	9.21	0.148
MAP$^{¥}$ (mmHg)					
Preop	110.80	16.55	105.20	15.50	0.223
T1	109.24	23.05	99.32	13.80	0.071
T2	108.80	16.96	104.68	18.27	0.413
T3	104.12	13.39	100.60	13.71	0.363
Postop	93.08	11.38	88.96	10.84	0.196

Continued.

Comparison	VC[α] group		PC[β] group		P value
	Mean	SD	Mean	SD	
HR[¥] (beats/min)					
Preop	77.80	12.29	81.76	16.85	0.347
T1	81.92	17.62	83.80	21.49	0.737
T2	81.20	16.74	86.08	13.83	0.267
T3	77.36	15.21	80.80	12.60	0.388
Postop	76.56	6.12	80.04*	5.37	0.038*

[α]: Volume controlled; [β]: Pressure controlled; [¥]: SAP, systolic arterial pressure; DAP, diastolic arterial pressure; MAP, mean arterial pressure; HR, heart rate; *: p<0.05.

Table 3: Comparison of respiratory mechanics parameters of the groups.

Comparison	VC[α] group		PC[β] group		P value
	Mean	SD	Mean	SD	
Ppeak[£] (cmH_20)					
T1	17.84	2.48	15.48*	2.62	0.002*
T2	24.00	2.89	20.12*	2.73	0.000*
T3	18.68	2.32	16.28*	2.69	0.001*
Pplateau[£] (cmH_2O)					
T1	17.08	2.58	15.32*	2.46	0.017*
T2	22.40	3.18	19.88**	2.57	0.003**
T3	17.60	2.55	15.96*	2.64	0.030*
Pmean[£] (cmH_2O)					
T1	8.68	0.69	8.92	1.08	0.353
T2	10.08	0.86	10.36	0.91	0.269
T3	9.32	1.14	9.00	0.91	0.280
$PetCO_2$[£] (mmHg)					
T1	30.20	1.44	30.08	1.68	0.788
T2	33.00	1.71	32.48	2.10	0.342
T3	31.84	2.27	31.68	1.60	0.774
Vt[£] (ml)					
T1	521.56	69.89	531.60	80.40	0.640
T2	535.80	68.98	551.68	67.18	0.414
T3	526.16	63.13	545.60	83.69	0.358
C[£] (ml/cmH_2O)					
T1	45.72	13.81	55.08 *	11.81	0.013*
T2	30.84	6.03	36.68 **	7.15	0.003**
T3	43.48	10.40	50.28 *	8.01	0.013*
Vd[£] (ml)					
T1	55.38	33.34	50.49	28.83	0.582
T2	57.47	33.12	56.25	26.86	0.887
T3	55.37	29.96	51.92	26.80	0.669
Vd/Vt[£]					
T1	0.11	0.05	0.09	0.04	0.443
T2	0.12	0.07	0.10	0.04	0.328
T3	0.11	0.05	0.09	0.05	0.433

[α]: Volume controlled; [β]: Pressure controlled; [£]: Ppeak, peak pressure; Pplateau, plateau pressure; Pmean, mean pressure; $PetCO_2$, end-tidal carbon dioxide pressure; Vt, tidal volume; C, compliance; Vd, dead space; Vd/Vt, dead space/tidal volume ratios; *: p<0.05; **: p<0.01.

Table 4: Comparison of arterial blood gas parameters of the groups.

Comparison	VC[α] group		PC[β] group		P value
	Mean	SD	Mean	SD	
pH					
Preop	7.42	0.04	7.42	0.01	0.850
T1	7.44	0.04	7.44	0.04	0.798

Continued.

Comparison	VC$^{\alpha}$ group		PC$^{\beta}$ group		P value
	Mean	SD	Mean	SD	
T2	7.40	0.03	7.40	0.03	0.938
T3	7.38	0.04	7.39	0.03	0.557
Postop	7.41	0.03	7.41	0.02	0.857
$PaO_2$$^{\mu}$ (mmHg)					
Preop	93.60	17.54	90.80	14.28	0.539
T1	175.44	44.45	186.44	42.47	0.375
T2	142.40	40.53	157.76	41.19	0.190
T3	156.36	61.39	185.52	51.57	0.075
Postop	78.60	11.11	85.88*	13.45	0.042*
$PaCO_2$$^{\mu}$ (mmHg)					
Preop	34.67	5.38	35.34	3.15	0.593
T1	33.94	2.31	32.02	6.63	0.178
T2	37.10	3.33	36.28	3.10	0.372
T3	35.78	3.67	35.20	2.71	0.525
Postop	35.22	3.83	34.88	3.63	0.751
$SaO_2$$^{\mu}$					
Preop	97.00	1.73	97.02	1.09	0.961
T1	98.73	0.52	98.84	0.56	0.500
T2	98.15	1.50	98.55	0.69	0.227
T3	98.22	0.89	98.46	1.06	0.382
Postop	95.62	2.24	96.32	1.39	0.196
$P(A\text{-}a)O_2$$^{\mu}$					
T1	137.76	43.35	123.52	40.20	0.234
T2	166.68	39.57	152.44	39.82	0.211
T3	158.68	40.56	126.12*	51.00	0.016*
$P(a\text{-}et)CO_2$$^{\mu}$					
T1	3.70	2.27	3.22	1.93	0.424
T2	4.10	2.57	3.80	1.97	0.645
T3	3.91	2.38	3.46	2.05	0.475

$^{\alpha}$: Volume controlled; $^{\beta}$: Pressure controlled; $^{\mu}$: PaO_2, partial pressure of oxygen; $PaCO_2$, partial pressure of carbon dioxide; SaO_2, oxygen saturation; $P(a\text{-}et)CO_2$, arterial to end-tidal partial pressure gradient of carbon dioxide; $P(A\text{-}a\ O_2$, alveoli-arterial oxygen pressure gradient; *: $p<0.05$.

DISCUSSION

Pneumoperitoneum causes an increase in IAP, a decrease in lung volumes and FRC, a decrease in pulmonary compliance, but an increase in airway resistance, diaphragm push, development of atelectasis in the basal parts of the lung, redistribution of hydrostatic forces and ventilation-perfusion disorder during laparoscopic cholecystectomy surgeries.[9,10] In studies comparing different mechanical ventilation modes used to prevent and correct these physiopathological changes, different results were observed.[11-21]

In our study, we compared the changes induced by VCV and PCV modes in hemodynamics (HR, SAP, DAP, MAP), respiration (airway pressures, gas exchange parameters) and metabolism (acid-base balance) in patients undergoing laparoscopic cholecystectomy.

Similar to other studies, we determined no difference between the groups in terms of SAP, DAP and MAP values when evaluating hemodynamic parameters.[11-18] Although the SAP values of only the VC group were significantly higher than those of the PC group in the T1 period, they were within physiological limits in terms of clinical values. When the respiratory mechanical parameters were evaluated, it was found that the pressures P_{peak} and $P_{plateau}$ were higher in the VC group compared to the PC group in all periods. Between the groups, there was no difference in P_{mean} values. We think that the reason for the lower airway pressures in the PC group was due to the decreasing flow pattern of the PCV mode. The decreasing flow pattern of PCV mode and thus the earlier dissipation of flow resistance provides a low pressure. Lower airway pressures were observed in similar research of patients undergoing laparoscopic surgery in the PCV mode.[11,16,19,20] It has also been highlighted that the airway pressure was higher, especially in patients with high BMI.[16,17]

The rise in mean airway pressure was caused by the initial rapid flow leading to early alveolar inflation, according to studies that found the P_{peak} value to be lower in PCV mode and the P_{mean} value to be higher.[15,18]

In our study, no significant difference was found between groups in pH, $PaCO_2$, $PetCO_2$, $P(a\text{-}et)CO_2$ and SpO_2. When we look at the PaO_2 values between the groups, it was found that the postop PaO_2 values were significantly higher in the PC group than in the VC group. The high postoperative PaO_2 values in the PCV group may be due to the higher inspiratory flow rate and the decreasing flow pattern. With a short-time constant, this flow pattern causes excessive swelling of the alveoli. This later-created tidal volume provided a more homogeneous distribution in the ventilated alveoli, created recruitment in the alveoli, and prevented the development of atelectasis.

The higher compliance values in the PCV mode were due to the decreasing flow pattern, which reduced pulmonary tension dependent on the gas distribution in this mode.

The $P(A\text{-}a)O_2$ value is one of the findings indicating that the ventilation/perfusion ratio was better preserved in PC mode. $P(A\text{-}a)O_2$ values in the T3 period were higher in VC ventilation than in PC ventilation in our study. This showed us that the ventilation/perfusion disorder rate was higher with VC ventilation.

Studies that find no difference in oxygenation between PCV and VCV in laparoscopic surgery may be due to differences in patient groups, surgical methods, surgical position, or type of surgery.[13,19,21,22]

In patients who had undergone laparoscopic cholecystectomy, peak and plateau airway pressures were lower and compliance was higher in PCV mode than in VCV mode, according to the findings of this study. An increase in postoperative oxygenation was observed in the PCV mode. It was found that the ventilation/perfusion ratio was better maintained at lower $P(A\text{-}a)O_2$ values in the PCV mode.

CONCLUSION

In conclusion, we think that PCV mode can be a good alternative for the prevention and correction of physiopathological changes due to laparoscopic surgery.

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