Research Article

Efficacy of PCV as compared to VCV in obese patients undergoing laproscopic cholecystectomy

Jyoti Khanna[1*], Priya[2], Ashwani Kumar[3], Smriti Gulati[4]

[1]Associate Professor, [2]PG, [3]Lecturer, [4]HOD, Department of Anaesthesiology, Government Medical College, Jammu, Jammu & Kashmir, INDIA.
Email: khannajyoti1960@gmail.com

Abstract

Introduction: Laparoscopic surgery requires the creation of pneumoperitoneum. Pneumoperitoneum decreases thoracopulmonary compliance by 30% to 50% in healthy and obese patients. An elevated intra-abdominal pressure and abdominal expansion shifts the diaphragm upwards. Thus, as the intra-thoracic pressure increases, the abdominal part of the chest wall becomes stiff and expansion of the lung is restricted. This is followed by a significant decrease in pulmonary dynamic compliance and changes in the ventilation and perfusion from increased airway reassure. However, high airway pressures and decreased compliance can be associated with pulmonary barotrauma. Obesity is a well-established risk factor for cholelithiasis for which laparoscopic cholecystectomy is routinely performed. **Aims and objectives:** To study the efficacy of PCV as compared to VCV in obese patients undergoing laproscopic cholecystectomy. **Materials and method:** Present study was undertaken to compare the volume controlled mode of ventilation with the pressure controlled mode for providing better oxygenation in obese patients undergoing laparoscopic cholecystectomy. 60 patients as ASA Grade I/II, between the ages of 18-55 years belonging to either sex and posted for laparoscopic cholecystectomy under general anesthesia were divided into two groups of 30 each. Patients in Group V were ventilated with volume controlled mode of ventilation whereas patients in Group P were ventilated with pressure controlled mode of ventilation. In both the groups, patients were intubated with a PVC cuffed endotracheal tube of appropriate size after achieving adequate relaxation with inj. suxamethonium 1.5 mg/kg. They were ventilated with 60% nitrous oxide, 40% oxygen and isoflurane 09.-1%. Muscle relaxation was maintained with Inj. Vecronium Bromide 0.08 mg/kg followed by additional top up doses of 0.02 mg/kg. at the end of surgery, neuromuscular blockade was reversed with Inj. Neostigmine 0.05 mg/kg and Inj. Glycoprrolate 0.01 mg/kl. The patients were extubated after fulfilling the criteria of adequate reversal. **Results:** Both the groups were comparable in respect to age (yrs), weight (Kg), Height (m), BMI (Kg/m2), sex (F:M) and ASA I/II grading. Both the groups were comparable in respect to duration of surgery (min), duration of anaeshtesia (min), duration of CO2 insufflation (min) and intra-abdominal pressure (mmHg).The difference between ventilation parameters i.e. Respiratory rate (per Min), Tidal Volume (mL), Minute Ventilation (L) and Peak airway pressure (cm H2O) was statistically insignificant 10 and 20 minutes after insufflations of Pneumoperitoneum but it was statistically significant at 30 minutes after Pneumoperitoneum with respiratory rate (per min), Tidal volume (mL), minute ventilation (L), peak airway pressure (mmHg) being significantly less in PCV group (Group P)as compared to VCV group (Group V). The oxygen saturation (SpO2)was comparable in both the groups, preoperatively as well as at 10 minutes after Pneumoperitoneum but it was higher in PCV group (Group P) as compared to VCV group (Group V) at 30 minutes after Pneumoperitoneum i.e. at 30 munities after Pneumoperitoneum the difference in both the groups was statistically significant.Both the groups were statistically comparable to each other in respect to pH, PaO2, (mmHg), PaCO2 (mmHg),)PAO2-PaO2 (mmHg), PaCO2-EtCO2 (mmHg) preoperatively and 10 minutes after the establishment of Pneumoperitoneum but there was statistically significant difference in both the groups in respect to PaO2, (mmHg), PaCO2 (mmHg),)PAO2-PaO2 (mmHg), PaCO2-EtCO2 (mmHg) at 30 minutes after the establishment of Pneumoperitoneum. **Conclusion:** Thus in the end we conclude that pressure controlled ventilation as a batter option for ventilating obese patients undergoing laparoscopic cholecystectomy.
Key words: PCV, VCV, obese patients, laproscopic cholecystectomy

***Address for Correspondence:**
Dr. Jyoti Khanna, Associate Professor, Department of Anaesthesiology, Government Medical College, Jammu, Jammu & Kashmir, INDIA.
Email: khannajyoti1960@gmail.com
Received Date: 30/05/2015 Revised Date: 07/06/2015 Accepted Date: 10/06/2015

How to site this article: Jyoti Khanna, Priya, Ashwani Kumar, Smriti Gulati. Efficacy of PCV as compared to VCV in obese patients undergoing laproscopic cholecystectomy. *International Journal of Recent Trends in Science and Technology* June 2015; 15(2): 298-304 http://www.statperson.com (accessed 12 June 2015).

Access this article online

Quick Response Code:	Website: www.statperson.com
	DOI: 11 June 2015

INTRODUCTION

Laparoscopic surgery requires the creation of pneumoperitoneum. Pneumoperitoneum decreases thoracopulmonary compliance by 30% to 50% in healthy[1,2,3] and obese patients. An elevated intra-abdominal pressure and abdominal expansion shifts the diaphragm upwards. Thus, as the intra-thoracic pressure increases, the abdominal part of the chest wall becomes stiff and expansion of the lung is restricted. This is followed by a significant decrease in pulmonary dynamic compliance and changes in the ventilation and perfusion from increased airway reassure [4,5]. However, high airway pressures and decreased compliance can be associated with pulmonary barotrauma. Obesity is a well-established risk factor for cholelithiasis for which laparoscopic cholecystectomy is routinely performed.[6] In healthy subjects undergoing laparoscopic cholecystectomy; Dexter *et al.*[7], using doppler found that cardiac output was depressed to a maximum of 28% at an insufflations pressure of 15 mm Hg but was maintained at an insufflations pressure of 7 mm Hg (Dexter SP *et al.* 1999). In an animal model, Ishizaki *et al.* reported the threshold IAP that had minimal effects on hemodynamic function was <12 mm Hg and recommend this pressure limit to avoid cardiovascular compromise during CO2 insufflations.[8] Respiratory changes during the laparoscopic procedure may contribute to increasing CO2 tension. Mismatched ventilation and pulmonary perfusion can result from the position of the patient and from the increased airway pressures associated with abdominal distension. [9,10]

AIMS AND OBJECTIVES

To study the efficacy of PCV as compared to VCV in obese patients undergoing laproscopic cholecystectomy.

MATERIALS AND METHOD

The present study was undertaken in the Department of Anaesthesiology and Intensive care, Government medical college, Jammu after obtaining approval from the hospital ethics committee.

For the purpose of study total 60 patients were selected by using following inclusion and exclusion criteria.

Inclusion Criteria

- Age between 18-55 years
- ASA physical status I and II
- BMI between 30 -40 kg/m2

Exclusion Criteria

- Anticipated difficult intubation.
- Major obstructive or restrictive type of lung disease.
- Pulmonary functions tests < 70% of predicted values for volume and flow.
- Known hypersentitivity to anaesthetic drugs used in this study.
- ASA physical status III & IV
- Anticipated inability to perform early post-operative extubation.

Informed written consent was obtained from each patient. A preanesthetic checkup was done a day before the surgery.All patients were subjected to detailed history taking to rule out any medical illness. They were subjected to complete general physical as well as systemic examination. Basic demographic characters like age, sex, height, weight and Body Mass Index were recorded. Routine investigations were performed in all the patients. All the patients were randomly allocated to two groups.

- **Group V (n = 30):** patients were put on volume controlled ventilation and
- **Group P (n = 30):** patients were put on pressure controlled ventilation.

The patient were kept fasting overnight and premedicated with Tab. Alprazolam 0.5 mg at bedtime.A peripheral venous access and an arterial access, under local anesthetic were secured and a baseline arterial sample was drawn for Arterial Blood Gas analysis pre-operatively. Inj. Pantoparxole 40 mg i.v., inj. Glycopyrrolate 0.2 mg i.m., and inj. Diclofenac sodium 1 mg/kg i.m. were administered 45 minutes before surgery. Standard technique was used to induce anesthesia in both the groups. In Group V, ventilation was continued with a tidal volume of 8 mL/kg and a respiratory rate of 12/min. the initial tidal volume was increased by 1 mL/kg every 5 min until 12 mL/kg, and the respiratory rate was increased by 2/min every 5 min till 20/min ot maintain EtCO2 < 35 mmHg, the respiratory rate was decreased by 2/min every 5 min till 8/min, with a decrease in tidal volume of 1 mL/kg every 5 min until 6 mL/kg.In Group P, an airway pressure not exceeding 30 cmH2O was set to provide a tidal volume of 8 mL/kg. respiratory rate of 12/min to begin with, was adjusted to keep an EtCO2 of

35-40 mmHg. An inspiratory/expiratory ratio of 1:2 was maintained. Following an increase in EtCO2 the respiratory rate was increased by 2/min every 5 min till 20/min, achieving the target EtCO2. Following a fall in EtCO2 < 35 mmHg, the respiratory rate was decreased by 2/min every 5 min till 8/min, with a decrease in airway pressure by 2 mmHg every 5 min until 10 cmH2O. The patients who were unable to maintain SpO2 > 95% and EtCO2 < 40 mmHg were supplemented by increasing Positive and expiratory pressure (PEEP) to 8 cm H2O, followed by increasing FiO2. Hypotension, if developed after increasing PEEP, was managed with intravascular fluid rush and short acting vasoconstrictors. Heart rate, blood pressure, respiratory rate, oxygen saturation, tidal volume, peak airway pressure and EtCO2 were monitored and recorded at 10 min interval intra-operatively. Time of start of anesthesia, surgery and CO2 insufflations were noted along with duration of anaesthesia, surgery and CO2 insufflations. Arterial blood sample were drawn 10 min after establishment of pneumopertoneum and at 20 min intervals thereafter till the end of the surgery. pH, PoO2, PaCO2,PAO2-PaO2, PaCO2-EtCO2 were noted at each interval. Neuromuscular blockade was reversed with Inj. Neostigmine 0.05 mg/kg and Inj. Glycopyrrolate 0.01 mg/kg. the patient was extubated after fulfilling the criteria of adequate reversal. The analysis was conducted with the help of computer software MS excel and SPSS version 17 of windows.Baseline comparability between the two groups was assessed and the data presented as Mean and Standard variations deemed appropriate for quantities variables.

RESULTS

Table 1: Demographic profile of patients

Characteristics	Group V	Group P	P value
Age (years)	43.87±709	41.83±8.32	0.31
Weight (Kg)	79.83±9.19	79.73±8.55	0.97
Height (M)	1.54±0.051	1.56±0.058	0.18
BMI (Kg/m2)	33.54±3.46	32.63±2.38	0.24
Sex (F:M)	28/2	26/4	0.67
ASA I/II	21/9	22/8	1.00s

The above table showed the mean age (years), weight (kg), height (m), BMI (Kg/m2), Sex (F:M), and ASA I/II in the two groups. It was observed that the difference among the two groups with respect to age, weight, height and BMI was statistically insignificant (p value > 0.05).

Table 2: Distribution according to Duration of surgery, anesthesia, CO2 insufflation time and intra-abdominal pressure (mmHg)

Characteristics	Group V	Group P	P value
Duration of surgery (min)	35.63±2.93	34.57±2.37	0.127
Duration of anaesthesia (min)	45.87±3.35	45.47±3.07	0.632
Duration of CO2 insufflation (min)	32.57±2.01	31.60±1.98	0.065
Intra-abdominal pressure (mmHg)	12	12	0

It was observed that the duration of surgery (min), duration of anesthesia (min), duration of CO2 insufflations (min) and intra-abdominal pressure (mmHg) was nearly similar in both the groups and the difference observed was statistically insignificant.

Table 3: Distribution according to Ventilation parameters

	Parameters	Group V	Group P	P value
Respiratory rate(per Min)	10 min after pneumoperitoneum	12.40±0.81	12.33±0.76	0.744
	20 min after pneumoperitoneum	13.33±1.09	12.87±1.14	0.11
	30 min after pneumoperitoneum	13.53±1.01	12.87±1.14	0.019
Tidal Volume (mL)	10 min after pneumoperitoneum	635.83±70.31	636.67±68.14	0.963
	20 min after pneumoperitoneum	660.00±61.45	639.17±68.13	0.218
	30 min after pneumoperitoneum	680.00±49.31	635.83±57.87	0.002
Minute Ventilation (L)	10 min after pneumoperitoneum	7.88±0.97	7.88±1.21	0.981
	20 min after pneumoperitoneum	8.78±0.95	8.25±1.36	0.082
	30 min after pneumoperitoneum	9.18±0.68	8.19±1.13	0.001
Peak airway pressure (cm H2O)	10 min after pneumoperitoneum	22.43±1.87	21.60±2.57	0.156
	20 min after pneumoperitoneum	23.76±1.74	22.83±2.17	0.071
	30 min after pneumoperitoneum	24.37±1.65	22.80±2.01	0.001

It was observed that there was no significant change in respiratory rate, tidal volume, minute ventilation and peak airway pressure after 10 and 20 min of pneumoperitoneum. But the difference in respiratory rate, tidal volume, minute ventilation and peak airway pressure in group V and P,after 30 min of pneumoperitoneum was statistically significant.

Table 4: Distribution according to SpO2 and EtCO2

Parameters		Group V	Group P	P value
SpO2 (%)	Pre-operative	98.67±0.84	98.77±0.68	0.62
	10 min after pneumoperitoneum	98.37±0.96	98.33±1.00	0.88
	30 min after pneumoperitoneum	38.33±0.96	38.77±0.63	0.04
EtCO2 (mmHg)	Pre-operative	35.33±1.03	35.57±0.73	0.32
	10 min after pneumoperitoneum	37.13±1.33	37.07±1.39	0.85
	30 min after pneumoperitoneum	37.07±1.28	37.03±1.35	0.92

SpO2 (%) and EtCO2 (mmHg) preoperatively as well as at 10 and 30 min after pneumoperitoneum in both the groups was measured. The oxygen saturation in Group V and group P was decreasing with time. The fall in saturation after 30 min of pneumoperitoneum was statistically significant in group V as compared to group P. The end tidal CO2 was increasing in both the groups and the difference observed was not statistically significant.

Table 5: Distribution according to Arterial blood gas analysis

Parameters		Group V	Group P	P value
pH	Pre-operative	7.41±0.04	7.39±0.04	0.114
	10 min after pneumoperitoneum	7.37±0.05	7.38±0.05	0.391
	30 min after pneumoperitoneum	7.37±0.05	7.38±0.05	0.391
PaO2 (mmHg)	Pre-operative	103.77±11.16	102.12±8.87	0.529
	10 min after pneumoperitoneum	107.20±17.56	103.94±17.38	0.473
	30 min after pneumoperitoneum	120.81±16.67	139.80±9.96	0.0001
PaCO2 (mmHg)	Pre-operative	38.17±3.06	38.62±1.55	0.475
	10 min after pneumoperitoneum	39.37±1.78	40.20±2.36	0.129
	30 min after pneumoperitoneum	39.92±1.25	39.05±1.21	0.009
PAO2-Pao2 (mmHg)	Pre-operative	5.09±6.44	6.39±1.93	0.455
	10 min after pneumoperitoneum	7.16±6.69	9.87±9.51	0.206
	30 min after pneumoperitoneum	12.20±8.46	5.53±6.28	0.001
PaCO2-EtCO2 (mmHg)	Pre-operative	3.54±2.44	3.06±1.68	0.374
	10 min after pneumoperitoneum	2.23±1.40	3.13±2.21	0.065
	30 min after pneumoperitoneum	2.85±1.57	2.02±1.35	0.043

The Arterial blood gas analyses in both groups preoperatively and at 10 and 30 min after pneumoperitoneum was also compared. It was observed that there was no significant change in preoperative pH and at 10 and 30 min after pneumoperitoneum ingroup V and P. PaO2 (mmHg) and PaCO2 (mmHg) were increasing in both the group. The difference observed in preoperative PaO2 and PaCO2 (mmHg) and 10 min after pneumoperitoneum was statistically insignificant whereas difference observed 30 min after pneumoperitoneum was statistically significant. It was seen that PAO2-Pao2 (mmHg) was increasing in both the groups and the difference observed 30 min after pneumoperitoneum was statistically significant in group V and P. PaCO2-EtCO2 (mmHg) was decreasing with the time in both the group. And the difference observed in group V and P of PaCO2-EtCO2 (mmHg) 30 min after pneumoperitoneum was statistically significant.

DISCUSSION

The age (mean±SD) (years) in Group V was 43.87±7.09 years whereas in Group P it was 40.38±8.32 years. The weight (mean±SD) (kg) in Group V was 79.83±9.19 kg whereas in Group P it was 79.73±8.55 kg. The height (mean±SD) (m) in Group V was 1.54±0.051 m whereas in Group P it was 1.56±0.058 m.The BMI (mean±SD) (kg/m2) in Group V was 33.54±3.46 kg/m2 whereas in Group P it was 32.63±2.38 kg/m2.The Female: Male ratio in Group V was 28:2 were as in Group P it was 26:4.In Group V there were 21 patients belonging to ASA grade I and 9 patients belonging to ASA grade II where as in Group p there were 22 patients belonging to ASA grade I and 8 patients belonging to ASA grade II.Both the groups were statistically comparable in respect to age (yrs), weight (kg), height (m), BMI (kg/m2), sex (F: M) and ASA I/II grading. The duration of surgery (mean±SD) in Group V was 35.63±2.93 min whereas in Group P it was 34.57±2.37 min. The duration of anesthesia (mean±SD) in Group V was 45.87±3.35 min whereas in Group P it was 45.47±3.07 min. The duration of carbon dioxide (CO2) insufflations (mean±SD) in Group V was

32.57±2.01min whereas in Group P it was 31.60±1.98 min. The intra-abdominal pressure (mmHg) in both the groups was kept at 12 mmHg. Both the groups were statistically comparable in respect to duration of surgery (min), duration of anesthesia (min), duration of carbon dioxide (CO2) insufflations (min) and intra-abdominal pressure (mmHg). It was observed that in Group V the respiratory rate (mean±SD) increased form 12.40±0.81per min at 10 min to 13.33±1.09 and 13.53±1.01 per min at 20 min and 30 minutes respectively whereas in Group P the respiratory rate (mean±SD) increased form 12.33±0.76 per min at 10 min to 12.87±1.14 per min at 20 min and 30 minutes. The difference observed in both the groups at 10 and 20 minutes after pneumoperitoneum was statistically non-significant (P> 0.05) but the difference observed in both the groups at 30 minutes after pneumoperitoneum was statistically significant (P> 0.05). This implies that significantly lower respiratory rate was required to maintain normocarbia in Group P compared with Group V. although the difference in repiratory rate was statistically significant, it was not clinically relevant (13.53±1.01 in Group V vs 12.87±1.14 in Group P).

In Group V the tidal volume (mean±SD) at 10 minute after pneumoperitoneum was 635.83±70.31ml which increased to 660.00±61.45ml at 20 minutes and 680.00±49.31ml at 30 minutes. In Group P the tidal volume (mean±SD) at 10 minute after pneumoperitoneum was 636.67±68.14 ml which increased to 639.17±68.13 ml at 20 minutes followed by decrease and 635.83±57.87 ml at 30 minutes. The difference observed in both the groups at 10 and 20 minutes after pneumoperitoneum was statistically non-significant (P> 0.05) but difference observed in both the Group at 30 minutes after pneumoperitoneum was statistically significant (P>0.05). Thus significantly less tidal volume was required to ventilate the patients in Group P as compared to that of Group V at 30 minutes after pneumoperitoneum. In Group V, the minute ventilation volume (mean±SD) at 10 minute after pneumoperitoneum was 7.88±0.97 l which increased to 8.87±0.95 l at 20 minutes and 9.18±0.68 l at 30 minutes. In Group P, the minute ventilation volume (mean±SD) at 10 minute after pneumoperitoneum was 7.88±1.21 l which increased to 8.25±1.36 l at 20 minutes and 8.19±1.13 l at 30 minutes. The difference observed in both the groups at 10 and 20 minutes after pneumoperitoneum was statistically non-significant (P> 0.05) but difference observed in both the Group at 30 minutes after pneumoperitoneum was statistically significant (P>0.05). Thus significantly less tidal volume was required to ventilate the patients in Group P as compared to that of Group V at 30 minutes after pneumoperitoneum. Gupta SD *et al.*[11], also found in their study the requirement of higher tidal volume and minute ventilation in VCV group in comparison with PCV for patients undergoing laparoscopic cholecystectomy. It can be explained by the fact that large tidal volume in VCV mainly ventilates the non-dependent portion of the lung, leading to excessive stretching of those regions without improving the overall ventilation.[12]On the contrary, in PCV, recruitment of collapsed alveoli due to high flow rate in the early respiratory phase leads to improved lung ventilation. Although the delivery of tidal volume and minute ventilation were lower in PCV, adequate CO2 elimination was achieved due to overall improvement in lung ventilation. Thus, the adverse consequences of large tidal volume delivery like rise in peak pressure, plateau pressure, volutrauma and inflammatory lung injury could be avoided in PCV. In Group V, the Peak airway pressure (mean±SD) at 10 minute after pneumoperitoneum was 22.43±1.87 cmH2O which increased to 23.76±1.74 cmH2O at 20 minutes and reached up to 34.37±1.65 cmH2O at 30 minutes. In Group P, the Peak airway pressure (mean±SD) at 10 minute after pneumoperitoneum was 21.60±2.57cmH2O which increased to 22.83±2.17cmH2O at 20 minutes with a subsequent fall to 22.80±2.01cmH2O at 30 minutes. The difference observed in both the groups at 10 and 20 minutes after pneumoperitoneum was statistically non-significant (P> 0.05) but difference observed in both the Group at 30 minutes after pneumoperitoneum was statistically significant (P>0.05). Thus significantly less peak airway pressure was required to ventilate the patients in Group P as compared to that of Group V at 30 minutes after pneumoperitoneum. Our study was in consonance with that reported by Balick-weber *et al.*[13]who found lower peak airway pressures during PCV than during VCV in patients undergoing laparoscopic urological procedures. In can be explained by the decelerating flow pattern and the earlier dissipation of flow resistance in PCV. Thus, in PCV, the peak pressure is limited, reducing the chance of barotraumas. Ayman S A EI-Aziz[14] also found more improvement in lung mechanics with pressure controlled ventilation mode than with volume controlled ventilation mode in form of reduction of peak airway pressure, plateau pressure, mean airway pressure, improvement of lung compliance and airway resistance. M Tugrul*et al.*[15]found consistently lower peak airway pressures as well as plateau pressures during PCV as compared to VCV.

The oxygen saturation (SpO2) was comparable in both the groups, preoperatively as well as at 10 minutes after pneumoperitoneum but it was higher in PCV group (Group P) as compared to VCV group (Group V) at 30 minutes after pneumoperitoneum. Monitoring EtCO2 is an adequate guide for determining the minute ventilation required to maintain normocarbia, and it provides a reasonable approximation of PaCO2 in healthy patients undergoing laparoscopic cholecystectomy. Both the groups were statistically comparable to each other in respect to preoperative values as well as those at 10, 20 and 30 minutes after pneumoperitoneum. It was observed that there was no significant change in preoperative pH and at 10 and 30 min after pneumoperitoneum in group V and P. In the present study, 30 min after establishment of pneumoperitoneum, a significantly higher PaO2 value was found in Group P than that in Group V. Gupta SD *et al.*[11], and Cadi *et al.*[16], observed a similar finding in their study. Balick-Weber *et al.*[13], noted no significant difference to arterial oxygenation in patients receiving PCV compared with the patients receiving VCV. The improved oxygenation in PCV may be explained by the delivery of "square wave" of pressure to the patient's airway. Pressure develops rapidly due to the very high flow at the initiation of inspiration followed by rapid flow deceleration. By delivering a larger proportion of tidal volume early in the inspiratory phase, the lung is maintained at a higher volume recruiting more alveoli to participate in gas exchange. In Group V the pre-operative alveola-arterial oxygen gradient (mean±SD) was 5.09±6.44 mmHg which increased to a value of 7.16±6.69 mmHg at 10 min after pneumoperitoneum followed by a subsequent increase to 12.20±8.46 mmHg at 30 min after pneumoperitoneum whereas in Group P, the pre-operative alveola-arterial oxygen gradient (mean±SD) was 6.39±6.93 mmHg which increased to a value of 9.87±9.51 mmHg at 10 min after pneumoperitoneum followed by a subsequent decrease to 5.53±6.28 mmHg at 30 min after pneumoperitoneum . The difference in respect to alveolar-arterial oxygen gradient was statistically significant at 30 min after pneumoperitoneum in the two groups. The patients receiving PCV (Group P) showed a significantly lower value of alveolar-arterial oxygen gradient (PAO2-PaO2) compared with the result of the study conducted by Cadi *et al.*[16] and Gupta SD *et al.*[11]. This can be explained by the decelerating inspiratory flow profile in PCV that enhances the distribution of ventilation among alveolar lung units improving gas exchange. Moreover, in VCV, higher intra-thoracic pressure decreases the venous inflow into the thorax, leading to a fall in preload and the compression of pulmonary blood vessels raises pulmonary vascular resistance, which can impede right ventricular stroke output. Fall in right ventricular stroke output causes ventilation-prefusion mismatch in the lung, resulting in hypoxaemia. It can be supported by the study conducted by Auler JO *et al.*[17], who found lower pulmonary vascular resistance and higher cardiac index in patients receiving PCV in comparison with the patients receiving VCV. With a pre-operative value (mean±SD) was 38.17±3.06 mmHg in Group V the arterial carbon dioxide increased to a value of 39.37±1.78 mmHg at 10 min after pneumoperitoneum followed by a subsequent increase to 39.92±1.25 mmHg at 30 min after pneumoperitoneum whereas from a pre-operative value (mean±SD) was 38.62±1.55 mmHg in Group P the arterial carbon dioxide increased to a value of 40.20±2.36 mmHg at 10 min after pneumoperitoneum followed by a subsequent increase to 39.05±1.21 mmHg at 30 min after pneumoperitoneum. In our study, though the difference in arterial carbon dioxide between the two groups was statistically significant at 30 min after pneumoperitoneum, this result may be because of the characteristics of PCV. For a given tidal volume, the inspiratory flow reaches much higher values with PCV than with the VCV mode. The alveoli with a short time constant may be initially over-inflated, but then a more homogenous distribution of the tidal volume in all the ventilated alveoli could follow. Therefore, the differences of the PaCO2 may be due to a better ventilation/perfusion ratio in the PCV group. With a pre-operative value (mean±SD) was 3.54±2.44 mmHg in Group V the arterial end tidal carbon dioxide decreased to 2.23±1.40 mmHg at 10 min after pneumoperitoneum followed by a subsequent increase to 2.85±1.57 mmHg at 30 min after pneumoperitoneum, whereas from a pre-operative value (mean±SD) was 3.06±1.68 mmHg in Group P the arterial and end tidal carbon dioxide increased to 3.13±2.21 mmHg at 10 min after pneumoperitoneum followed by a subsequent decrease to 39.05±1.21 mmHg at 30 min after pneumoperitoneum.Both the groups were comparable at preoperative level as well as at 10 minutes after pneumoperitoneum. At 30 min after pneumoperitoneum the difference between the two groups was statistically significant with the aetPCO2 gradient being 2.14±1.57 mmHg Group V and 2.02±1.53 mmHg in Group P. our study is in concordance with Gupta SD *et al.*[11].Generally, the a-etPCO2 gradient is considered to be an index of alveolar dead space. Many factors influence the magnitude of the a-etPCO2 gradient. It increases with age, pulmonary disorders, pulmonary embolism, decreased cardiac output, hypovolemia, and anesthesia. It decreases with increased cardiac output and with large tidal volume and low frequency ventilation. Thus in the end we summarize that significantly less minute ventilation and peak airway pressures were required by the patients in Group P as compared to Group V at 30 min after pneumoperitoneum. Significantly higher values of arterial oxygenation i.e. PaO2 (mmHg) were achieved in Group P as compared to Group V at 30 min after pneumoperitoneum. Significantly lower values of arterial carbon dioxide i.e. PaCO2 (mmHg), alveolar-arterial oxygen gradient i.e. PAO2-PaO2 (mmHg) and arterial-end tidal carbon dioxide gradient i.e. PaCO2-etCO2 (mmHg) were achieved in Group P as compared to Group V at 30 min after pneumoperitoneum.

CONCLUSION

Thus in the end we conclude that pressure controlled ventilation as a better option for ventilating obese patients undergoing laparoscopic cholecystectomy.

REFERENCES

1. Bradoczky GI, Engelaman E, Lavarlet M, *et al.* . Ventilation effects of pneumoperitoneum monitored with continuous spiraometryAnaesthesia 1993; 48:309.
2. Fahy BG, Barnas GM, flowers JL *et al.*. the effects of increased abdominal pressure on lung and chest wall mechanics during laparoscopic surgery. AnesthAnalg 1995; 81: 744.
3. Fahy BG, Barnas GM, ngle SE *et al.*. changes inlung and chest wall properties with abdominal insufflations of carbon dioxide art immediately reversible. AnesthAnalg 1996; 82: 511.
4. Koivusalo AM, Lindgren L. Effects of carbon dioxide pneumoperitoneum for laparoscopic cholecystectomy. ActaAnaesthesiolScand 2000; 44; 834-41.
5. Hirvone EA, nuutinen LS, kauko M. ventilatory effects, blood gas changes and oxygen cnsumpation during laparoscopic hysterectomy. AnesthAnalg 1995: 80-961.
6. Neugebauer E, Troidl H, Kum CK *et al.*. The E.A.E.S. consensus development conferences on laparoscopic cholecystectomy, apperndicectomy and hernia repair. Consensus statements. SurgEndose 1995; 9: 550-63.
7. Dexter SP, Vucevic M, Gibson J *et al.*. Hemodynamic consequernces of high and low pressure capnoeritoneumduting laparoscopic cholecystectomy.
8. Ishizaki Y, Bandai y, Shimomura k *et al.*. Safe intraabdomianl pressure of pneumoperitoneum during laparoscopic surgery. Surgery 1993; 114-549.
9. CiofoloMj, Clergue F, Seebacher J *et al.*. Ventilation effects of laparoscopy undr epidural anesthesia. AnesthAnalg 1990; 70: 357.
10. Wilcox S, Vandam LD. Alas, Poor Trendelenburg and his position! A critique of its uses and effectiveness. AneshtAnalg 1988; 67: 574.
11. Gupta SD, Kundu SB, Ghose T *et al.*. A comparison between volume-controlled ventilation and pressure-controlled ventilation in proveidng better oxygenation in obese patients undergoing laparoscopic cholecystectomy. Indian J Anaesth 2012; 56: 276-82.
12. Marion PL, Sutin KM, Principles of Mechanical Ventilation, the ICU Book 3rd ed. 2007.
13. Balick-Weber CC, Nicolas P, Hedreville-Montout M *et al.*. Respiratory the hemodynamic effects of volume-controlled vs. pressure-controlled ventilation during laparoscopuy: A cross-over study with echocardiographic assessment. Br. J. Anaesh 2007; 99: 429-35.
14. Ayman S A EI-Aziz. New supraglottic airway decive; I-Gel in elective laparoscopic cholecystectomy using volume controlled ventilation versus pressure controlled ventilation. Journal of American Science 2011; 7(11): 447-53.
15. Tugrul M, Camci E, karadeniz H *et al.*. Comparson of volume controlled with pressure controlled ventilation during one-lung anaesthesia. Br J Anaesth 1997; 79: 306-10.
16. Cadi P, Guenoun T, journois D *et al.*. Pressure-controlled ventilation improves oxygenation during laparoscopic obesity surgery compared with volume-controlled ventilation. Br. J. Anaesth 2008; 100: 709-16.
17. Auler JO Junior, Carmona MJ, Silva MH *et al.*. Hemodynamic effects of pressure-controlled ventilation versus volume-controlled ventilation in patients submitted to cardiac surgery. Clin Intensive Caer 1995; 6:100-06.

Source of Support: None Declared
Conflict of Interest: None Declared