

Effect of protective lung ventilation on oxygenation & hemodynamics in obese patients

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ABSTRACT

Background: The number of obese patients undergoing surgery, either bariatric or non-bariatric, is steadily increasing. These patients are more labile to the perioperative complications, such as hypoxemia, hypercapnia, and atelectasis. Intraoperative protective ventilation consisting of low tidal volume, high PEEP and recruitment maneuvers resulted in alveolar recruitment and optimization of intraoperative respiratory mechanics.

Objective: This study tested two strategies of mechanical ventilation in obese patients during pneumoperitoneum to conclude which is better as regard gas exchange optimization and hemodynamic stability.

Methods: Study was a randomized prospective comparative control study which was carried out on 50 obese patients with BMI 30-50 kg/m². Patients were prepared for laparoscopic cholecystectomy. Patient's selection according to attendees at time of operation as a single numbers were protective ventilation (group A) and a double numbers were conventional ventilation (group B).

Results: Study showed significance between oxygenation in both groups. Post-operative oxygenation in protective ventilation (group A). Mean Post P (A-a) O₂ in group A was 27.93 (±7.76) mmHg, while in group B was 35.82 (±11.98) mmHg, p value (0.022). Hemodynamic instability observed in 24% in group A, but only occurred in 8% in group B.

Conclusion: Study found that protective ventilation was superior to conventional ventilation as it was associated with better oxygenation in the post-operative in obese laparoscopic cholecystectomy. In spite of it was very effective in optimizing gas exchange, but associated with more hemodynamic affection.

Keywords: Obese; Laparoscopy; Protective; Lung ventilation; Conventional; Recruitment; Pulmonary function tests

INTRODUCTION

Obesity is defined as having a body mass index (BMI [calculated as weight in kilograms divided by height in meters squared]) of 30 or greater, and it can be further subdivided: class I obesity is defined as a BMI of 30 to less than 35; class II obesity is defined as a BMI of 35 to less than 40; and class III obesity is defined as a BMI of 40 or greater ^{1}.

Obesity is characterized by several alterations in the mechanics of the respiratory system that tend to further exaggerate impairment of gas exchange. It has been demonstrated that in anaesthetized patients, arterial partial pressure of oxygen (PaO₂) is inversely related to BMI ^{2}.

Postoperative lung atelectasis develops with both IV and inhaled anesthesia and whether the patient is breathing spontaneously or is paralyzed and ventilated mechanically. The adverse effects of atelectasis persist into the postoperative period and can affect patient recovery ^{3}. Up to 15% of the entire lung may be atelectatic during anesthesia, particularly in the basal region, resulting in a true pulmonary shunt of approximately 5%–10% of cardiac output ^{4}. During general anesthesia and the immediate postoperative period, obese patients are more likely to develop postoperative pulmonary complications such as atelectasis and exhibit impaired pulmonary function compared to non-obese individuals.

Therefore, the prevention of atelectasis is of utmost importance in this population because atelectasis affects respiratory mechanics, the volume of airway closure, and the oxygenation index (PaO₂/FiO₂) ^{5}.

Laparoscopy is a well-established procedure for laparoscopic cholecystectomy surgery often performed in reversed trendelenburg position. To facilitate laparoscopic surgical manipulation, a pneumoperitoneum is usually induced through carbon dioxide inflation. The increase in abdominal pressure as a result of carbon dioxide inflation has been shown to impair the respiratory function during the procedure, mainly inducing atelectasis formation in the dependent lung regions. The resulting decrease in functional residual capacity poses patients at risk of perioperative complications, particularly if they are obese and/or submitted to intricate surgical procedures ^{6}. For decades, it has been known that general anesthesia can impair oxygenation, even in patients with healthy lungs and it is possible that the application of mechanical ventilation is a contributing factor.

A strategy of protective ventilation, consisting of low tidal volumes and plateau pressures and application of positive end expiratory pressure (PEEP) has gained widespread acceptance in intensive care units after large studies showed an associated reduction in morbidity and mortality in patients with acute lung injury. Information about

the respiratory effects of protective mechanical ventilation in the operating room where patients with normal lung function receive mechanical ventilation for a short period is limited ^{7}.

The hypothesis of the current study is that during laparoscopic surgery, both the positioning of the patient and pneumoperitoneum worsen chest wall elastance, concomitantly decrease trans-pulmonary pressure, and that protective lung strategy consisting of low tidal volume, high PEEP and recruitment maneuvers by increasing the trans pulmonary pressure, would result in alveolar recruitment and improvement in respiratory mechanics and gas exchange.

AIM OF THE WORK

To evaluate the efficacy of intraoperative ventilation strategy either protective or conventional in obese patients undergoing abdominal laparoscopy as regard efficiency of oxygenation and hemodynamic stability.

PATIENT AND METHODS

A- Patients

The study was a prospective comparative study which was carried out in Aswan university hospital starting from November 1st 2015 until reaching the target sample size. It was carried out on 50 obese patients recruited from General Surgery clinics that were prepared for laparoscopic cholecystectomy. **The study was approved by the Ethics Board of Aswan University.**

Inclusion criteria: ASA class I & II, Age \geq 18 years, BMI \geq 30 kg/m² and $<$ 50 kg/m². Free of cardiac, pulmonary, renal or neuromuscular disease, & nonsmoker.

Exclusion criteria: ASA III & IV, BMI $<$ 30kg/m², previous abdominal surgery, previous lung surgery & hemodynamic instability or intractable shock.

Prior to initiation of the study; every subject was informed about the aim of the study and gave a written consent, and approval of the university hospital ethical committee was taken.

B- Methods

All participants in this study were subjected to the following: History taking: Full detailed history was taken from each participant. Questions such as name, age, residence, occupation, history of any other medical problems as diabetes, hypertension, history of cardiac

disease, renal or pulmonary problem and history of previous abdominal or lung surgery.

On operating theatre: On their arrival to the operating theatre, patients were premeditated with IV metoclopramide 10 mg, ranitidine 50mg and midazolam 0.1 mg/kg in the pre-anesthesia room.

After applying intraoperative monitors (**Drager Vista 120 monitor, Germany**) using 5 leads ECG, pulse oxymetry, capnography and non-invasive blood pressure, patients were pre-oxygenated with 100% O₂ for 5 minutes and general anesthesia was induced with propofol 2 mg/kg, fentanyl 2 μ g/kg and succinylcholine 1.5 mg/kg. After oral endotracheal intubation with appropriate size cuffed endotracheal tube, anesthesia was maintained using 1-2 MAC Isoflurane over the period of the operation with fentanyl shots when needed. Neuromuscular blockade obtained by using Atracurium besaylate 0.25 μ g/kg as a bolus dose and 5 μ g/kg/min as a maintenance dose.

All patients were mechanically ventilated with volume control mode (VCV) (**Drager Fabius plus XL anesthesia machine, Germany**) with a respiratory rate 12/min, I: E ratio = 1:2 and FiO₂ 60%.

After induction of anesthesia and positioning of the patient, carbon dioxide was insufflated into the peritoneal cavity until the intra-abdominal pressure reached 10–15 mmHg, which was maintained throughout the procedure.

Patients were given 12-15 ml/kg of normal saline intravenously before the induction of anesthesia and were then maintained with 5 ml/kg/h of normal saline solution until the end of the surgery. Intraoperative hypotension (decrease in mean arterial blood pressure [MAP] $>$ 25% of baseline) was treated with a bolus of normal saline 0.9% 250 mL and/or incremental doses of IV vasoactive drugs (ephedrine 5 mg).

Intraoperative analgesia was achieved by peralfgan 15 mg/kg and nalbuphine 0.25 mg/kg once.

At the end of the surgery, Isoflurane was discontinued and FiO₂ was increased to 100%. The muscle relaxant was reversed by neostigmine 50 μ g/kg and 0.015 mg/kg atropine sulfate. Tracheal extubation was performed after reaching satisfactory criteria for extubation. Patients were transferred to the recovery room (PACU) and duration of operation was recorded.

In the recovery room: Patients were put in semi-sitting position under basic monitoring and observed for 30 minutes for occurrence of any postoperative complications. Oxygen was applied

if oxygen saturation decreased < 94%. Intraoperative and immediate postoperative complications such as hypoxia, hemodynamic instability either alone or during recruitment maneuver (we marked it as having MAP <60 mmHg or HR < 50 b/min), increased ET_{CO₂}> 45 for more than 1 min, need for reintubation or need for ICU admission were recorded. Postoperative analgesia during the first 24 hours postoperative was maintained by IV ketorolac amp 30 mg every 6 hour. All medications and parameters were calculated on an ideal body weight basis.

Participants in the study were allocated into two equal groups: (1) Protective lung ventilation group (group A): Ventilation strategy consists of: Tidal volume (VT) 6 ml/kg IBW, PEEP 10 cmH₂O & recruitment maneuvers performed after induction of anesthesia, after pneumoperitoneum induction and before extubation. To conduct the alveolar recruitment maneuver we changed the ventilation settings from VCV into pressure control mode with driving pressure 15 cmH₂O with PEEP 20 cmH₂O, RR 10/min, I:E 1:1, FiO₂ 1.0 for 2 minutes then return to the previous parameters with close monitoring for the hemodynamics of the patient. **(2) Conventional ventilation group (group B):** Ventilation strategy consists of: Tidal volume (VT) 10 ml/kg IBW, PEEP 0 cmH₂O (ZEEP) & No recruitment maneuvers.

C- Measurements

1. After anesthesia induction in supine position (T0).
2. After CO₂ pneumoperitoneum (T1).
3. After positioning of patient by 10 min (T2)
4. After positioning of patient by 30 min (T3).
5. At the end of surgery, after abdominal deflation in supine position (T4).

Statistical analysis

The statistical analysis was performed using SPSS 20 (statistical Package for the Social Sciences). Pearson chi-square test was used in analysis of the qualitative variables and the student-t test was used for the continuous variables. P value < 0.05 was considered statistically significant.

RESULTS

The current study is a randomized prospective comparative control study which conducted on 50 obese patients with a body mass index (BMI) between 30 and

50 kg/m², aged ≥ 18 years, scheduled to undergo elective abdominal laparoscopic cholecystectomy surgery and categorized into two equal groups to evaluate the efficiency of protective lung ventilation strategy and conventional ventilation in this type of patients and surgery.

Table 1 and Figure 1 represent sex & age distribution of the studied cases, body mass index (BMI) & time of operation. They reveal that the mean age in group A is 39.28 (± 11.56) years while in group B is 37.65 (± 9.71) years. Mean BMI in group A is 35.21 (± 5.07) kg/m², while in group B is 34.57 (± 3.43) kg/m². The mean time of operation in group A is 63.6 min, while in group B is 61.6 min.

Table 2 and Figure 2 reveal a better post-operative alveolar-arterial O₂ pressure gradient (Post P (A-a) O₂) in group A where the mean Post P (A-a) O₂ in group A is 27.93 (± 7.76) mmHg, while in group B is 35.82 (± 11.98) mmHg with statistically significant p value (0.022).

Table 3 and Figures 3, 4 show the changes in the intraoperative hemodynamics (MAP & HR) during the surgery time. MAP reduced significantly in group A at T₂, T₃ & T₄ (mmHg; Mean ± SD: 85.76±13.87, 82.84±13.53, 79.2±11.77), p value (0.011, 0.0010.001).

Table 4 and Figure 5 show that the incidence of haemodynamic instability was higher in group A (24%), but only occurred in 8% of the cases in group B.

Table (1): Patient characteristics of the two groups and time of operation.

	A	B	P. Value
	Mean ± SD	Mean ± SD	
Sex			
Male	1(4%)	1(4%)	1.000
Female	24(96%)	24(96%)	
Age	39.28±11.56	37.65±9.71	0.592
Body mass index (BMI)	35.21±5.07	34.57±3.43	0.602
Time of operation (min)	63.6±10.85	61.6±8.26	0.467

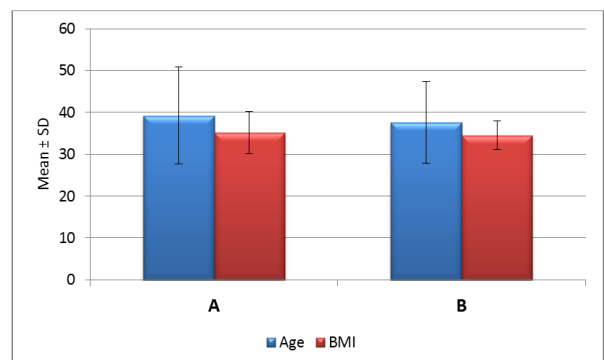


Fig. (1): Age and BMI of the participants in the study.

Table (2): Preoperative and postoperative alveolar-arterial O₂ pressure gradient in group A & B.

	A	B	P. Value
	Mean ± SD	Mean ± SD	
Pre P(A-a)o ₂	14.24± 10.75	16.91± 10.05	0.370
Post P(A-a)o ₂	27.93± 7.76	35.82± 11.98	0.022*

* Statistically significant difference (p<0.05)

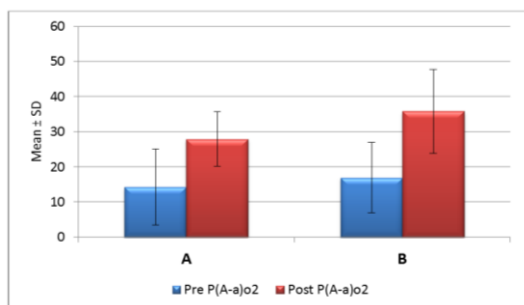


Fig. (2): preoperative and postoperative alveolar-arterial O₂ pressure gradient in group A & B.

Table (3): Intraoperative hemodynamics during the surgery time.

	Mean arterial blood pressure			Heart rate (HR)		
	A	B	P. Value	A	B	P. Value
	Mean±SD	Mean±SD		Mean±SD	Mean±SD	
Baseline	99.4±7.17	101.96±9.70	0.212	93.16±10.65	93.6±11.29	0.562
T0	86.24±11.59	92.24±9.8	0.054	90.44±11.11	93.92±12.08	0.294
T1	88.68±13.24	91.64±9.14	0.362	86±9.9	90±10.9	0.181
T2	85.76±13.87	94.88±10.39	0.011*	83.96±11	89.08±10.34	0.096
T3	82.84±13.53	94.4±9.52	0.001**	81.4±10.63	86.2±10.78	0.119
T4	79.2±11.77	94.96±11.41	0.001**	79.16±13.89	89.72±13.83	0.010*

** Highly statistically significant difference (p<0.01)

* Statistically significant difference (p<0.05)

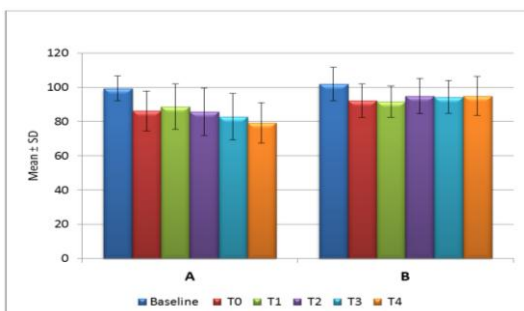


Fig. (3): MAP during the surgery time.

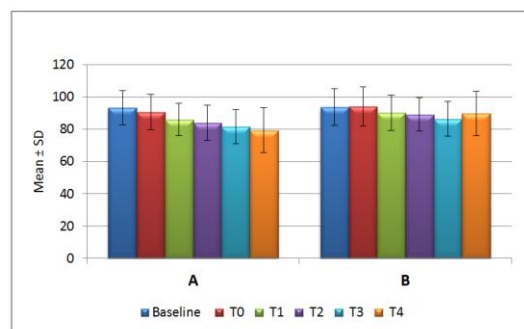


Fig. (4): Heart rate during the surgery time.

Table (4): Incidence of hemodynamic instability.

	Mean hemodynamic instability				P. Value
	Group A		Group B		
	NO.	%	NO.	%	
-ve	19	76	23	92.0	0.247
±ve	6	24	2	8.0	

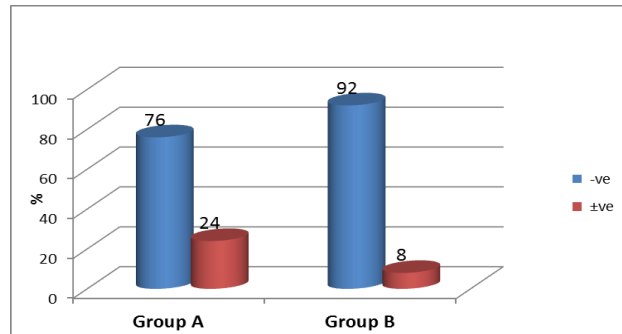


Fig. (5): Incidence of the hemodynamic instability in the two groups during the surgery time.

DISCUSSION

Ventilating obese patients is becoming a frequent challenge since the prevalence of obesity is steadily increasing and reaching epidemic proportions worldwide.

Morbid obesity can promote a restrictive syndrome due to the accumulation of thoracic and abdominal fat, decreasing pulmonary volumes, expiratory reserve volume, and FRC due to reduced thorax wall movement, decreased pulmonary compliance, and increased airway resistance. Thus, anomalies in ventilation/perfusion appear, causing hypoxemia at rest and in the supine position, most likely due to the closure of small airways observed in this type of patient^{8}. The development of atelectasis (i.e., collapse of part of a lung) after induction of anesthesia is exaggerated in patients with morbid obesity, and it is the main contributor to respiratory dysfunction^{9}.

Because the diaphragm is mechanically coupled to the abdominal wall, any increase in abdominal pressure may decrease functional residual capacity. During laparoscopy, the raised abdominal pressure distends the abdominal wall, increases its elastance, shifts cranially the diaphragm, and moves a large part of the ventilation-related volume changes through the rib cage^{10}.

Intra-operative oxygenation in patients with morbid obesity is largely determined by the magnitude of post-induction atelectasis. Thus, increasing intra-operative PaO₂ (i.e., partial pressure of oxygen in arterial blood) depends on recruiting the atelectatic lung and maintaining the expansion. This should ideally be done while avoiding mechanical lung injury.

Mechanical ventilation should not be seen as a simple and safe intervention, either in patients under general anesthesia for surgery or in critically ill patients. Indeed, ventilation is increasingly recognized as a harmful intervention as it may cause lung injury, frequently referred to as ventilator-induced lung injury (VILI), and may harm the respiratory muscles, also named ventilator-induced diaphragm dysfunction (VIDD)^{11}.

Several studies have been conducted about determining the best ventilatory strategies for obese patients under general anesthesia. Pressure-controlled ventilation (PCV) is often the preferred ventilatory mode in obese patients, because of the more homogeneous distribution of delivered gas mixture and the increased possibility of avoiding alveolar distension and improving ventilation perfusion mismatch when compared with volume-controlled ventilation (VCV)^{12}. Some studies also demonstrated improved oxygenation with intraoperative PCV, compared to VCV in obese patients. However, no ventilatory mode significantly improved optimum delivered VT or mean airway pressures. There is also no information suggesting superior clinical outcomes with intraoperative PCV or VCV use in obese patients and they should be therefore, selected under adequate understanding of their different operation and characteristics to achieve the goals of lung protective ventilation and avoid both volu/barotrauma and hypoventilation^{13}.

Tidal volume settings require special attention in obese patients. Obese patients are more often exposed to greater VT, an observation that likely reflects the practice of basing VT computations on actual instead of predicted body weight. It is important to highlight, particularly in obese patients, that the desired VT should be calculated based on the predicted body weight and not on the actual body weight because the increased thoracic appearance is due to excessive adipose tissue but not a greater intrathoracic (lung) volume^{14}. The use of higher tidal volumes was the standard of care in the operating room for many years since the use of higher tidal volumes per se constantly opens those lung parts that collapse at the end of expiration and as such prevents the need for high oxygen fractions. Furthermore, relatively short use (that is, hours) of higher tidal volumes was considered relatively safe, although animal as well as clinical studies show that VILI can develop shortly after initiation of ventilation. Finally, it was hypothesized that the use of lower tidal volumes could actually increase the risk of repetitive opening and closing of partly atelectatic tissue^{15}.

Lung protective ventilation with low tidal volumes (VT) and the use of positive end-expiratory pressure (PEEP) are now considered routine for ICU patients, but the implementation of protective ventilation strategies in the operating room is not widespread^{16}. In obese patients, the efficiency of recruitment maneuvers and PEEP on postoperative outcomes such as oxygenation and pulmonary function remains controversial. Thus, the postoperative effect and impact on clinical outcomes of these intraoperative lung recruitment efforts needs to be further studied. The main aim of mechanical ventilation in obese patients is to 'keep the lung open' during the entire breathing cycle despite all the pathophysiological alterations. Adequate opening pressure can be obtained through periodic large, manual lung inflations (recruitment maneuvers)^{17}.

This study compared a lung-protective mechanical ventilation strategy combining the use of lower tidal volume (TV), higher PEEP levels, and intraoperative RMs, with a conventional standard mechanical ventilation (higher tidal volume, ZEEP without intraoperative RMs) during abdominal laparoscopic cholecystectomy surgery. All patients were submitted to careful preoperative assessment especially cardiovascular and respiratory history and examination. The airway was carefully evaluated to avoid the risk of difficult intubation dilemma. Extensive patient counseling was done to all patients to understand the practical and technical difficulties that may be encountered by the staff and surgeons during laparoscopy (such as intravenous access and need for central lines, panniculus repositioning, and conversion to laparotomy). All patients performed a preoperative chest x-ray and arterial blood gases to be compared with the postoperative results.

Demographic characteristics showed non-significant difference between the two groups regarding age, sex, BMI and operative time.

The study showed improvement in the oxygenation of the cases subjected to protective ventilation. This agreed with *Pang et al.*^{18} who evaluated the effect of PEEP and RM in laparoscopic cholecystectomy arterial oxygenation, a group of patients were ventilated with zero PEEP and 10 mL/kg of VT, and the second group with 10 cmH₂O of PEEP and RM (airway pressure set at 40 cmH₂O for 1 minute). The group with PEEP and RM showed an improvement in intraoperative oxygenation. Also agreed with *Futier et al.*^{19} and *Aldenkortt et al.*^{13} as they concluded that adding recruitment maneuvers to PEEP in these obese patients improved oxygenation. This disagreed with *Whalen et al.*^{20} who investigated the

effect of different PEEP levels (4 and 8 cmH₂O) and recruitment maneuvers at a fixed level of VT set at 8 mL/kg on intraoperative arterial oxygenation. Recruitment maneuvers (RM) were obtained with a progressive PEEP increase from 10 to 20 cmH₂O. As a result, the PaO₂/FiO₂ ratio significantly increased during pneumoperitoneum in the PEEP+RM-group, but it was promptly dissipated at extubation.

The use of high PEEP levels is potentially associated with an increase in mean airway pressure within the respiratory system, likely promoting higher incidence of hemodynamic complications and higher fluids' requirement. The current study tried to overcome the deleterious effects of PEEP, recruitment maneuvers and large tidal volumes in both groups by giving sufficient preoperative preload with crystalloid solution (12-15 ml/kg of normal saline intravenously before the induction of anesthesia and were then maintained with 5 ml/kg/h of normal saline solution until the end of the surgery). However, there was a greater affection of the hemodynamics in patients received protective ventilation and RMs. Indeed hemodynamic instability necessitating the use of further fluid boluses and vasopressors, was much higher in group A (observed in 24% of the cases), but only occurred in 8% of the cases in group B. This agreed with *Grasso et al.*^{21} who reported reduction in cardiac output and MAP after application of the RM in ARDS patients and *Nielsen et al.*^{22} who reported that RM lead to a significant reduction in cardiac output in critical care patients. Also agreed with *Almarakbi et al.*^{23} who reported that patients during RM required more vasopressor agents, and with *Jo et al.*^{24} who reported a decrease in MAP and HR when PEEP was added to pneumoperitoneum. This disagreed with *Talab et al.*^{5} who stated that application of PEEP and VCM was not accompanied by a significant reduction in MAP, even after pneumoperitoneum and positioning, perhaps because they administrated more fluids to the patients before positioning (20 ml/kg/h) and also the vital capacity maneuver (VCM) was applied only once immediately after intubation and maintained for 7–8 s. Also the study disagreed with *Severgnini et al.*^{25} who found that the use of higher PEEP levels was associated neither with major hemodynamic impairment nor with higher intraoperative requirement of fluids or blood losses, perhaps because they used a modified RMs by allowing a progressive increase in tidal volumes, which may have promoted less negative hemodynamic impairment instead of using sustained inflation as commonly suggested.

CONCLUSION

The study found that protective ventilation was superior to the conventional ventilation in optimization of oxygenation. However, it was associated with more hemodynamic compromise which was managed with either fluids or vasopressors.

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