Evaluation of Respiratory Functions in Morbid Obese during Laparoscopic and Open Surgery: A Comparative Study

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ABSTRACT
30 morbid obese patients, with BMI>40 Kg/m², ASA physical status II and III, were randomly divided into two equal groups, 15 patients each: GI (n=15): scheduled for laparoscopic surgery, and GII (n=15): scheduled for open upper abdominal surgery. Their preoperative pulmonary function tests, were the following average: FVC 85.10%±3.21%, FEV1s was 83.12%±6.08%, and FEF 25-75 was 88.76%±7.22% of the normal predicted values (S0). Spirometric data were repeated after 24h (S24), and 72h (S72). Blood gas measurements during spontaneous respiration at room air before surgical intervention included an arterial oxygen tension (PaO2) of 76.49±7.64 mmHg, arterial Carbon dioxide tension (PaCO2) of 37.73±2.04, and pH of 7.40±0.02. All measurements were performed with the patient in supine position at 5 time points; A1: 15 min after tracheal intubation. A2: GI: 15 min after CO2 insufflation. GII: 15 min after opening the abdomen. A3: 30 min after A2. A4: GI: 15 min after deflation of the abdomen. GII: 15 min after closure of peritoneum. A5: directly before extubation. The measurements were Arterial Blood Gases (ABG), alveolar-arterial oxygen difference was calculated as an index of pulmonary shunt, arterial to end-tidal CO2 tension difference (Δ P) was calculated as an index of physiological dead space, The lung/chest wall compliance, and the maximal and minimal resistance of the total respiratory system. In GI (laparoscopy), PaCO2, ETCO2, P (A-a)O2 alveolar-arterial oxygen difference and arterial to end-tidal CO2 tension difference and resistance showed significant rise after CO2 peritoneal insufflation. All measurements returned to normal by the end of surgery. In GII (open surgery), those measurements did not show any considerable change. Postoperative pulmonary functions showed marked reduction in both groups, but to a lesser degree in GI. However, GI returned to normal by the 3rd day postoperative.

INTRODUCTION
Obesity is increasing in epidemic proportions worldwide. Even mild degrees of obesity have adverse health effects and are associated with diminished longevity. Body mass index (BMI; weight per unit surface area) is the scientific yardstick by which overweight is gauged relative to the population norm. Morbid obesity is considered when BMI≥35 Kg/m². Morbid obesity is associated with a 2-fold increase in overall mortality, a 7-fold increase in cardiopulmonary mortality, and a 40 times more frequent incidence of sudden death. [1]

The interference of anesthesia with cardiopulmonary compensatory mechanisms in already compromised obese increases the propensity for peri-operative complications in this population. Those patients are characterized by reduced lung volume, compared with non-obese subjects. [2] It has been suggested that such reductions in lung volume are associated with an increasing incidence of atelectasis or airway closure. This results in arterial hypoxemia and marked alterations in respiratory mechanics. There is also increase in total respiratory system, and lung resistance. [3]

Excess body fat has several effects on the respiratory system most notably, a decline in the expiratory reserve volume and an increase in the FEV1/ FVC ratio. The vital capacity (VC), total lung capacity (TLC), and functional residual volume (FRV) are generally maintained in otherwise normal individuals with mild-to-moderate obesity but are reduced by up to 30% in morbidly obese patients. [4] The work of breathing (WOB) is increased by abnormal chest elasticity, increased chest wall resistance, increased airway resistance (Raw), abnormal diaphragmatic position, and upper airway resistance as well as by the need to eliminate a higher daily production of carbon dioxide. [5][6] Severely obese patients are often hypoxic, with a widened alveolar-arterial oxygen gradient, caused primarily by ventilation-perfusion (V/Q) mismatching. [7][8] In addition, obesity is a major risk factor for the development of pulmonary thromboembolism. [9][10]

General anesthesia, upper abdominal surgery, and supine position may accentuate respiratory mechanical abnormalities, worsening gas exchange, which can be a challenge for the anesthesiologist. The evaluation of patients for impairment cause by respiratory diseases requires a comprehensive history and physical examination, followed by appropriate standardized testing such as spirometry, diffusing capacity, and often chest radiography to establish a diagnosis and determine the severity of the disease. In the minority of cases, more comprehensive cardiopulmonary exercise testing may be indicated to define the extent of impairment. [11]

Key words
Morbid, obese, FEV1, FVC, FEF 25-75, BMI, Compliance, laparoscopy, ABG.
Aim of work
The aim of this work was to evaluate the respiratory function during and post anesthesia in laparoscopic surgery, in comparison with the open upper abdominal surgery, in morbid obese patients.

Material and method
This study was performed in Kasr Al Aini teaching hospital on 30 morbid obese patients, with BMI>40 Kg/ m², ASA physical status II. Their preoperative pulmonary function tests, performed on a standard spirometer, were the following average: forced vital capacity of 85.10% ±3.21% of that predicted by Crepo et al [11], the forced expiratory volume in 1s was 83.12%±6.08%, and the average forced expiratory flow rate measured over the middle portion of the forced vital capacity (FEF 25-75), was 88.76%± 7.22% of the predicted values. Spirometric studies were performed again 24 and 72h, postoperative. The preoperative data were considered the reference value (S0), 24h readings were (S24), and 72h readings were (S72). Blood gas measurements during spontaneous respiration at room air before surgical intervention included an arterial oxygen tension (PaO₂) of 76.49±7.64 mmHg, arterial Carbon dioxide tension (PaCO₂) of 37.73±2.04, and pH of 7.40±0.02.

All patients were scheduled for performing either open upper abdominal surgery, or laparoscopic abdominal or surgery. Patients were randomly divided into two equal groups, 15 patients each.
GI (n=15): scheduled for laparoscopic surgery.
GII (n=15): scheduled for open upper abdominal surgery.

BMI was calculated by dividing the body weight by the height of the patients in square meters. Subtracting 100 from the height of males in centimeters and 105 from females calculated the effective body weight roughly.
Exclusion criteria included clinical evidence of cardiac and/or respiratory diseases. Patients affected by obesity hypoventilation syndrome or chronic obstructive lung disease, and any patient with a previous history of myocardial infarction or congestive heart failure were excluded from the study. In all patients, a complete medical history and physical examination were obtained at the time of the preoperative evaluation. All drug doses were based on the effective body weight.
The laparoscopic procedure was performed using a carbon dioxide pneumoperitoneum, through a paraumbilical trocar. Abdominal pressure was maintained at 15-20 mmHg.
Intravenous midazolam premedication (2-3 mg) was given 15 minutes before induction; then general anesthesia was induced with fentanyl (1 µg/kg) intravenously and propofol (sleep dose). Orotracheal intubation with cuffed tube was facilitated with succinyl choline (1.5 mg/ kg IV). Anesthesia was maintained with N₂O and O₂ (FIO₂ = 0.5), and isoflurane 1.5%. Additional muscle relaxation was provided with atracurium (0.5 mg/ kg, 10-15 mg were added with the appearance of the second twitch of train of four. For the first 15 minutes after general anesthesia induction and intubation, a 6 L/min fresh gas flow was used, and then the flow is adjusted to 2 L/min. intermittent positive pressure ventilation was used to keep CO₂ tension (ETCO₂) ranging between 35 and 40 mmHg throughout the procedure. Standard monitoring was used throughout the operations included continuous electrocardiography (ECG) (lead II), heart rate (HR), non-invasive blood pressure (BP), pulse oximetry, inspired oxygen fraction (FIO₂), ETCO₂, end-tidal concentration of the inhalation drug, and minute ventilation. Muscle relaxation was assessed by a peripheral neuromuscular monitor, (TOF GUARD, Organon). The attending anesthetist’s aim was to maintain HR, and BP values within ±20% of baseline values. Occurrence of hypotension (reduction in systolic arterial blood pressure SBP> 30% of baseline), hypertension (increase in SBP> 30% from baseline), or bradycardia (HR decrease to less than 50 bpm) provoked stepwise changes in the inspired concentration on isoflurane to restore the hemodynamic variables. Management with other drugs (atropine, ephedrine, etc) may be needed accordingly.
To avoid the effect of the time of anesthesia on the lung, preceding the airway occlusion, recruitment maneuver (inspiratory inflation to a maximal capacity repeated three times. All measurements were performed with the patient in supine position at 5 time points
A1: 15 min after tracheal intubation.
A2:
GI: 15 min after CO₂ insufflation.
GI: 15 min after opening the peritoneum.
A3: 30 min after A2.
A4:
GI: 15 min after deflation of the abdomen.
GII: 15 min after closure of peritoneum.
A5: directly before extubation.
During the measurements, surgery was stopped, and in GII, all retractors were removed.

MEASUREMENT
The following parameters were measured:
- Arterial Blood Gases (ABG).
- Alveolar-arterial oxygen difference was calculated as an index of pulmonary shunt from the following equation [13]
  \[ P (A-a)O_2 = FIO_2 \times (Pb - PH_2O - PaCO_2/RQ - PaO_2) \]
Pb = actual barometric pressure.
PH_2O = water vapor tension at 37°C.
RQ = respiratory quotient measured to be 0.8.
PaO_2 = arterial oxygen tension.
PaCO_2 = arterial CO_2 tension.
- Arterial to end-tidal CO_2 tension difference (\( \Delta P \)) was calculated as an index of physiological dead space. [13]
- The lung/chest wall compliance (C_{tot}) was obtained by dividing the expired tidal volume by the plateau pressure measured after 1-second inspiratory pause. [14]
- The maximal and minimal resistance of the total respiratory system (cm H_2O/L/s) (R ‘max, R’ min respectively), were calculated by dividing peak airway pressure minus plateau pressure (P’ max-P2), and peak pressure minus initial pressure (P’ max-P1), by the flow immediately preceding the occlusion. [15] A rapid airway occlusion was performed during 5s of inspiratory pause by holding the inspiration and decrease fresh gas flow to zero. This maneuver was applied 3 times with a brief interval of normal ventilation between each one. An average of the three measurements was used to estimate the elastic and resistive values of the respiratory system.

Data were presented as mean ± standard deviation (SD), P value <0.05 was considered statistically significant. Analysis of variance (ANOVA) for repeated measures was used to evaluate changes over time followed by Dunnet test to compare each measurement with baseline.

RESULTS
Table (1) Patient’s Demographic Data

<table>
<thead>
<tr>
<th></th>
<th>GI (n=15)</th>
<th>GII (n=15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SEX (male: female)</td>
<td>11:4</td>
<td>9:6</td>
</tr>
<tr>
<td>AGE (years)</td>
<td>34.7±9.14</td>
<td>41±5.21</td>
</tr>
<tr>
<td>WEIGHT (kg)</td>
<td>135.7±12.6</td>
<td>127.9±11.8</td>
</tr>
<tr>
<td>HEIGHT (cm)</td>
<td>164.2±9.4</td>
<td>158.9±11.3</td>
</tr>
<tr>
<td>BMI (kg/m^2)</td>
<td>41.7±3.5</td>
<td>39.3±6.4</td>
</tr>
<tr>
<td>DURATION OF SURGERY (min)</td>
<td>112.3±17.3</td>
<td>121.7±11.4</td>
</tr>
</tbody>
</table>
Data are presented as mean ±SD.

*p<0.05 is significant.

GI: laparoscopic surgery.

GII: open upper abdominal surgery.

BMI: Body Mass Index.

There was no statistical significant difference between the two groups regarding the age, weight, height, BMI, and duration of surgery.

Table (2) PaO$_2$, PaCO$_2$, PETCO$_2$ and Respiratory Rate

<table>
<thead>
<tr>
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<th>A1</th>
<th>A2</th>
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<tbody>
<tr>
<td>PaO$_2$ (mmHg)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>GI</td>
<td>184.3±16.8</td>
<td>174.6±22.2</td>
<td>142.7±18.1*</td>
<td>137.0±25.7*</td>
<td>177.9±19.2</td>
</tr>
<tr>
<td>GII</td>
<td>178.7±23.1</td>
<td>171.5±14.1</td>
<td>183.1±21.6</td>
<td>187.2±17.3</td>
<td>189.7±23.4</td>
</tr>
<tr>
<td>PaCO$_2$ (mmHg)</td>
<td></td>
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</tr>
<tr>
<td>GI</td>
<td>32.4±3.7</td>
<td>35.7±3.1</td>
<td>41.2±3.1*</td>
<td>46.6±3.7*</td>
<td>34.1±5.3</td>
</tr>
<tr>
<td>GII</td>
<td>34.1±4.1</td>
<td>31.3±6.1</td>
<td>32.7±4.6</td>
<td>31.9±4.5</td>
<td>29.8±5.3</td>
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<tr>
<td>PETCO$_2$ (mmHg)</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>GI</td>
<td>29.7±2.4</td>
<td>32.1±3.6</td>
<td>34.8±2.1</td>
<td>39.6±2.7*</td>
<td>31.2±3.1</td>
</tr>
<tr>
<td>GII</td>
<td>30.7±2.3</td>
<td>29.1±1.5</td>
<td>27.8±4.1</td>
<td>28.4±1.7</td>
<td>26.3±2.4</td>
</tr>
<tr>
<td>RR (Br/min)</td>
<td></td>
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<tr>
<td>GI</td>
<td>12.3±2.4</td>
<td>12.7±3.4</td>
<td>14.7±1.2*</td>
<td>15.1±4.1*</td>
<td>13.6±1.7</td>
</tr>
<tr>
<td>GII</td>
<td>10.7±1.3</td>
<td>9.8±2.8</td>
<td>11.2±3.1</td>
<td>12.3±2.4</td>
<td>11.5±3.7</td>
</tr>
</tbody>
</table>

Data are presented as mean ±SD.

*p<0.05 is significant.

GI: laparoscopic surgery.

GII: open upper abdominal surgery.

PaO$_2$ = arterial oxygen tension.

PaCO$_2$ = arterial CO$_2$ tension.

PETCO$_2$ = end tidal CO$_2$.

RR = respiratory rate.

Br/min = breath per minute.

A1: 15 min after tracheal intubation.

A2:

GI: 15 min after CO$_2$ insufflation.

GII: 15 min after opening the peritoneum.

A3: 30 min after A2.

A4:

GI: 10 min after deflation of the abdomen.

GII: 10 min after closure of peritoneum.

A5: directly before extubation.

There was no statistical significant difference between the two groups regarding the baseline readings of blood gases, with comparable respiratory rate. However, in GI (laparoscopic group), with the beginning of CO$_2$ insufflation, both PaCO$_2$ and PETCO$_2$ showed significant rise. The respiratory rate was increased significantly in an attempt to keep the carbon dioxide within the clinically accepted limits. Oxygen tension showed significant decline with the abdominal insufflation, although it was only of statistical value. In GII, (upper abdominal open surgery), both PaO$_2$ and PaCO$_2$ showed no significant change, and also the respiratory rate.

Table (3) Indices of Dead Space and Shunt

<table>
<thead>
<tr>
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<th>A1</th>
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<tbody>
<tr>
<td>Pa-PETCO$_2$</td>
<td></td>
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</tr>
<tr>
<td>GI</td>
<td>2.64±3.1</td>
<td>3.71±2.3*</td>
<td>5.13±3.5*</td>
<td>4.71±1.4*</td>
<td>3.53±2.1</td>
</tr>
<tr>
<td>GII</td>
<td>2.57±1.23</td>
<td>3.08±3.7</td>
<td>3.17±2.7</td>
<td>2.83±2.1</td>
<td>2.91±1.17</td>
</tr>
<tr>
<td>P (A-a) O$_2$</td>
<td></td>
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<tr>
<td>GI</td>
<td>126.7±9.4</td>
<td>131.4±5.7</td>
<td>213.2±4.1*</td>
<td>209.3±5.8*</td>
<td>137.2±4.2</td>
</tr>
<tr>
<td>GII</td>
<td>134.2±5.1</td>
<td>136.7±2.3</td>
<td>141.2±6.3</td>
<td>138.9±7.1</td>
<td>147.7±8.1</td>
</tr>
</tbody>
</table>

Data are presented as mean ±SD.
*p<0.05 is significant.

GI: laparoscopic surgery.
GII: open upper abdominal surgery.
PaO$_2$ = arterial oxygen tension.
Pa-PEtCO$_2$ = (arterial - end tidal CO$_2$) in mmHg.
P(A-a)O$_2$ = alveolar-arterial oxygen difference, in mmHg.
A 1: 15 min after tracheal intubation.
A2:
  GI: 15 min after CO$_2$ insufflation.
  GII: 15 min after opening the peritoneum.
A3: 30 min after A2.
A4:
  GI: 10 min after deflation of the abdomen.
  GII: 10 min after closure of peritoneum.
A5: directly before extubation.

In GI (laparoscopic surgery), there was a statistically significant increase in dead space as presented by the increase in Pa-PEtCO$_2$, and significant increase in shunt as shown in the P (A-a)O$_2$, after abdominal insufflation by CO$_2$. However, those parameters returned to normal by the end of the surgical procedure, after deflation of the peritoneum. There was no change in dead space and shunt ratio, as compared to baseline readings in GII (the open abdominal surgery).

Table (4) Lung and Chest Wall Compliance

<table>
<thead>
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<tbody>
<tr>
<td>P$_{aw}$ (Peak)</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Gi</td>
<td>27.8±7.3</td>
<td>38.1±8.2</td>
<td>42.7±7.1</td>
<td>33.6±3.7</td>
<td>29.3±4.2</td>
</tr>
<tr>
<td>GII</td>
<td>28.3±6.1</td>
<td>24.0±5.1</td>
<td>21.7±4.1</td>
<td>22.7±2.2</td>
<td>27.9±4.8</td>
</tr>
<tr>
<td>P$_{aw}$ (Plateau)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gi</td>
<td>24.3±6.1</td>
<td>24.7±5.0</td>
<td>38.1±3.7</td>
<td>30.4±2.3</td>
<td>26.1±6.3</td>
</tr>
<tr>
<td>GII</td>
<td>24.7±5.0</td>
<td>23.1±3.8</td>
<td>19.6±7.9</td>
<td>20.2±4.1</td>
<td>23.9±3.7</td>
</tr>
<tr>
<td>C$_{tot}$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gi</td>
<td>46.3±3.7</td>
<td>31.7±7.3</td>
<td>30.2±5.9</td>
<td>42.4±5.1</td>
<td>43.8±3.7</td>
</tr>
<tr>
<td>GII</td>
<td>44.7±4.7</td>
<td>48.3±6.1</td>
<td>47.2±4.7</td>
<td>45.6±1.2</td>
<td>44.4±2.5</td>
</tr>
</tbody>
</table>

Data are presented as mean ±SD.

* *p<0.05 is significant.

GI: laparoscopic surgery.
GII: open upper abdominal surgery.
P$_{aw}$ (Peak)= Peak airway pressure, cm H$_2$O.
P$_{aw}$ (Plateau) = Plateau airway pressure, cm H$_2$O.
C$_{tot}$= lung/ chest wall compliance, ml/ cm H$_2$O.
A 1: 15 min after tracheal intubation.
A2:
  GI: 15 min after CO$_2$ insufflation.
  GII: 15 min after opening the peritoneum.
A3: 30 min after A2.
A4:
  GI: 10 min after deflation of the abdomen.
  GII: 10 min after closure of peritoneum.
A5: directly before extubation.

In GI (laparoscopic group), there was a statistically significant decline in total lung and chest wall compliance with abdominal inflation. There was also increase in both peak and plateau pressure. However, the compliance and pressure returned to almost normal levels with the end of surgery before extubation.

In contrary, GII (the open surgery), showed significant increase in compliance, with a decrease in both plateau and peak pressure. Parameters returned to near normal after peritoneum was closed, before extubation.
Table (5) Maximal and Minimal Resistance of the Total Respiratory System

<table>
<thead>
<tr>
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<th>A1</th>
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<th>A4</th>
<th>A5</th>
</tr>
</thead>
<tbody>
<tr>
<td>R max</td>
<td>GI</td>
<td>8.32±2.7</td>
<td>10.17±0.75*</td>
<td>11.34±0.87*</td>
<td>10.61±1.43*</td>
</tr>
<tr>
<td></td>
<td>GII</td>
<td>9.12±1.68</td>
<td>7.28±0.81*</td>
<td>6.21±0.73*</td>
<td>6.33±1.20*</td>
</tr>
<tr>
<td>R min</td>
<td>GI</td>
<td>5.25±3.51</td>
<td>6.81±0.61*</td>
<td>7.73±0.99*</td>
<td>7.12±1.03*</td>
</tr>
<tr>
<td></td>
<td>GII</td>
<td>5.17±3.60</td>
<td>3.18±0.27*</td>
<td>2.88±0.93*</td>
<td>2.92±0.81*</td>
</tr>
</tbody>
</table>

Data are presented as mean ±SD.

*p<0.05 is significant.

GI: laparoscopic surgery.
GII: open upper abdominal surgery.
R max = maximal resistance of the total respiratory system, cm H₂O/L/s.
R min = minimal resistance of the total respiratory system, cm H₂O/L/s.
A 1: 15 min after tracheal intubation.
A2: GI: 15 min after CO₂ insufflation.
GII: 15 min after opening the peritoneum.
A3: 30 min after A2.
A4: GI: 10 min after deflation of the abdomen.
GII: 10 min after closure of peritoneum.
A5: directly before extubation.

There was a significant rise in the total respiratory system resistance after abdominal inflation in GI, (laparoscopic group). This rise tends to lower down towards the end of the procedure although it was not back to the starting measures. Meanwhile, patients in GII, (open surgery), showed significant decrease in resistance after opening the abdomen, while the resistance tends to rise once again after closure of the peritoneum.

Table (6) Post-operative Respiratory Functions

<table>
<thead>
<tr>
<th></th>
<th>S0</th>
<th>S24</th>
<th>S72</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC (L)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>3.71 ± 1.12</td>
<td>2.14±0.21*</td>
<td>3.24±0.97</td>
</tr>
<tr>
<td>GII</td>
<td>3.91±1.13</td>
<td>2.12±0.78*</td>
<td>3.02±0.04*</td>
</tr>
<tr>
<td>FEV1 (L)</td>
<td></td>
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</tr>
<tr>
<td>GI</td>
<td>2.87 ± 0.91</td>
<td>1.78±0.39*</td>
<td>2.26±0.53</td>
</tr>
<tr>
<td>GII</td>
<td>2.95±0.87</td>
<td>1.64±0.26*</td>
<td>2.13±0.62*</td>
</tr>
<tr>
<td>FEF25-75 (L/s)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>2.29±0.77</td>
<td>1.97±0.13*</td>
<td>2.14±0.16</td>
</tr>
<tr>
<td>GII</td>
<td>2.31±1.03</td>
<td>1.19±0.06*</td>
<td>1.87±0.22*</td>
</tr>
</tbody>
</table>

Data are presented as mean ±SD.

*p<0.05 is significant.

GI: laparoscopic surgery.
GII: open upper abdominal surgery.
S0: pre-operative values.
S24: 24h post-operative values.
S72: 72h post-operative values.
FVC: forced vital capacity.
FEV 1: forced expiratory volume in 1s.
FEF 25-75: forced expiratory flow rate

Post-operative FVC, FEV1 and FEF 25-75 readings, compared to pre-operative values, showed significant decline in both groups after 24h. However, GI (laparoscopic group), returned to the preoperative levels in the 72h readings. GII (open surgery) remained lower than preoperative values after 72h.

**DISCUSSION**
The short-term benefits of minimal access techniques include less pain, early mobilization, and shorter hospital stay. Nonetheless, significant data have accumulated regarding the complications associated with laparoscopic techniques, including those that are unique to laparoscopic surgery such as bile duct injury and disruption of major blood vessels. Other problems such as myocardial ischemia and respiratory acidosis are associated with the cardiopulmonary effects of pneumoperitoneum and systemic CO₂ absorption. Following peritoneal insufflation, CO₂ is absorbed trans-peritoneally, and the rate at which this occurs depends on the gas solubility, the perfusion of the peritoneal cavity, and the duration of pneumoperitoneum. However, Mullet and colleagues found that end-tidal CO₂ (ETCO₂) and pulmonary CO₂ elimination (VCO₂) increased between the eighth and tenth minutes, regardless of site and duration of insufflation. [16]

These physiologic changes, although tolerated by healthy patients, could have particular adverse consequences for infirm and critically ill patients. [5] A CO₂ intra-abdominal insufflation, induced important changes in pulmonary mechanics leading to moderate variation in gas exchange as well as an increased cardiac index, heart rate, mean arterial pressure and mean pulmonary arterial pressure - and that without changes in the oxygen transport, delivery and consumption. [17][18]

As was obvious in this study, soon after CO₂ insufflation, ETCO₂ and PaCO₂ values rose up. Increasing minute ventilation in most cases maintains PaCO₂ within normal limits clinically, despite the statistical rise, but inevitably leads to some increase in airway pressure. Furthermore, arterial CO₂ might not be accurately reflected by ETCO₂, perhaps owing to increased in dead-space ventilation during pneumoperitoneum. This was also approved by Perilli et al. [3] All these changes were of minor clinical relevance and were well tolerated by the ASA II morbidly obese patient. The increase in minute ventilation, required to maintain PaCO₂ values constant in ASA I and II patients, has been variously reported as; 12% to 16%, 30%, and 55%. [19][20][21] In ASA III or IV patients, however, PaCO₂ can remain elevated despite adjusting minute ventilation to normalize ETCO₂ and refractory hypercapnia and acidosis can supervene. [14] Preoperative evaluation and pulmonary function testing therefore might be prudent. Forced expiratory volumes (FEV₁) less than 70% of predicted values and diffusion defects less than 80% of predicted values could identify patients at risk. [22]

FRC and lung compliance decrease with supine positioning, and a cephalad shift of the diaphragm follows the induction of general anesthesia. CO₂ insufflation reduces compliance but reverse Trendelenburg alleviates this to a degree. Conversely, the head-down tilt is accompanied by a decrease in vital capacity and diaphragmatic excursion, and further decreases the reduction in compliance associated with pneumoperitoneum. Increased airway pressures are also seen. [23] Patients, who were subjected to laparoscopic surgery, suffered an increase in resistance with a decline in compliance. These findings were in accordance with those of Dumont et al [6][17], and Carol et al [8]. Fifty percent and 81% increases in peak airway and plateau pressures, respectively, were observed, in association with 47%-decreased compliance during laparoscopy in seven healthy women. [5] Significant reversible increases in lung and chest wall elastance and resistance with rising intra-abdominal pressure have been reported during CO₂ pneumoperitoneum. These perioperative changes in the passive mechanical properties of the lung and chest wall can be critical in obese patients or in patients with underlying pulmonary disease. [24] A reduction in compliance leading to a diminished functional residual capacity (FRC) relative to closing volume and ventilation–perfusion mismatch could result in perioperative hypoxemia. Hypoxemia is, however, uncommon during laparoscopic surgery in otherwise healthy patients. When a deoxygenation is observed, there can be many potential causes. [5]

In the mean time all measurements in GII (open surgery), including PaCO₂ and ETCO₂ showed no significant change compared to the baseline values (A1), indicating no major change in dead space and shunt relative to the original readings. Moreover, resistance decreased and compliance increased after opening of the peritoneum. Now it seems that open surgery is the choice in morbid obese patients, compared to laparoscopic surgery. However, laparoscopic surgery can reduce postoperative pulmonary complications by avoiding the restrictive pattern of breathing that usually follows upper abdominal surgery. In fact, the defects in respiratory function after laparoscopic surgery were qualitatively similar, although less severe, than those following open surgery. Respiratory functions returned to normal pre-operative level in the 3rd day postoperative. Karayiannakis and colleagues compared 42 patients undergoing laparoscopic
cholecystectomy with 40 undergoing open cholecystectomy in a prospective, randomized study to determine differences in postoperative respiratory function and complications. They found significant reductions in total lung capacity, FRC, FEV₁, forced vital capacity (FVC), and mid-expiratory flow (FEF, 25%–75%) after both procedures. The reductions in FRC, FEV₁, FVC, and mid-expiratory flow were significantly smaller following the laparoscopic approach. [25] It was suggested that the site of surgery could be significant because greater reductions in pulmonary function were observed following laparoscopic cholecystectomy than after gynecologic laparoscopy. In contrast, other investigators found no significant difference in postoperative pulmonary changes following upper abdominal, lower abdominal, or pelvic laparoscopy. [26]

In conclusion all the harmful effects of laparoscopy on morbid obese, seem to be of moderate clinical importance, and well tolerated by ASA II morbid obese patients. Compared to the benefits the patient gain post-operative: laparoscopy seems to be of choice in morbid obese. However, laparoscopy in the critically ill patient is questionable because the role is not established. An ICU patient has little to gain from the benefits of early mobilization. Conversely, in the presence of raised intra-cranial pressure (ICP) or borderline organ function, the physiologic changes associated with pneumoperitoneum and laparoscopy could have profound detrimental effects. Judicial control of minute ventilation together with minimizing intra abdominal pressure (IAP) during insufflation, and post-operative pain control, will lead to decrease of the risk of potentially marked cardiovascular and respiratory changes. [4] In turn, this will decrease the risk of perioperative myocardial events, or organ dysfunction or failure.

REFERENCES


