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RESEARCH ARTICLE

TO ASSESS EFFECTS OF PNEUMOPERITONEUM ON VENTILATORY MECHANICS AND HEMODYNAMICS DURING LAPAROSCOPIC CHOLECYSTECTOMY IN OBESE AND NON-OBESE PATIENTS

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Abstract

Aim: The aim of the study was to assess effects of pneumoperitoneum on ventilatory mechanics and hemodynamic during laparoscopic cholecystectomy in obese and non-obese patients

Method: A total of 100 patients were divided into two groups of 50 each. Obese (O) group consisted of 50 patients with BMI > 30 kg/m² and non-obese (NO) group consisted of 50 patients with BMI < 30 kg/m². Anaesthesia protocol was kept uniform in both the groups. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), peripheral oxygen saturation (SpO₂), end-tidal carbon dioxide (EtCO₂), perfusion index (PI), peak (PPEAK), plateau (PPLAT) and driving (PDRIVING) pressures, static (CST) and dynamic (CDYN) lung compliances were measured at baseline and at sequential time intervals following induction of anaesthesia, insufflation, desufflation and before extubation.

Results: On induction of anaesthesia, a decrease in HR was observed in non-obese patients whereas an increase was observed in obese patients though statistically non-significant ($p > 0.05$). A higher SBP, DBP and MAP was observed following insufflation, after desufflation and before extubation in the obese patients but was non-significant. The PI value was high in obese patients though non-significant. Also, EtCO₂ values remained minimally elevated in obese patients but were statistically insignificant. PPEAK, PPLAT as well as PDRIVING remained high in obese individuals following insufflation, after desufflation, and before extubation and were significant ($p < 0.05$). CST and CDYN remained low in obese patients following insufflation, after desufflation, and before extubation and were statistically significant ($p < 0.05$).

Conclusion: Obese patients undergoing LC have slightly higher hemodynamic variations than non-obese patients, but non-significant ($p > 0.05$). The respiratory parameters PPEAK, PPLAT and PDRIVING remained elevated whereas CST and CDYN remained low following insufflation, after desufflation, and before extubation in obese patients as compared to non-obese patients and were statistically significant ($p < 0.05$).

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Introduction:-

Obesity is defined as abnormal or excessive fat accumulation. Body mass index (BMI) is a simple index of weight-for-height that is commonly used to classify overweight and obesity in adults. WHO defines obesity as BMI greater than or equal to 30 [1]. Obesity is often associated with multiple co-morbidities such as diabetes mellitus (DM), hypertension, and obstructive sleep apnoea (OSA) to name a few. It is also a risk factor for the formation of gallstones as well as related complications and requires cholecystectomy as the treatment of choice. Laparoscopic cholecystectomy (LC) is now widely considered as the surgical treatment of choice for symptomatic gallstones [2,3]. It is preferred over open surgery as it is associated with less post-operative pain, wound infection, shorter hospital stay and reduced analgesic requirements [4–7]. It involves the creation of pneumoperitoneum and reverse Trendelenburg position as a result of which various hemodynamic and respiratory changes occur [8]. Obese individuals undergoing LC appears to be the largest risk sub-group amenable to consistent evaluation.

Pneumoperitoneum is an essential step during LC to provide adequate visualization and exposure of the operative field. Carbon dioxide (CO₂) is the preferred gas for its creation as it is inexpensive, does not support combustion, has a high solubility in blood and is easily cleared from tissues. However, its absorption into systemic circulation causes hypercarbia and results in various physiological and biochemical effects [9–12]. Also, pneumoperitoneum creation results in a state of acutely elevated intra-abdominal pressure (IAP). The normal IAP of non-obese individuals is 5 mm Hg or less. In contrast, obese patients have a chronically elevated IAP at 9 to 10 mm Hg. This raised IAP in turn causes cephalad displacement of the diaphragm resulting in a marked decrease in FRC, MV, VC, TLC, ERV and an increase in airway resistance as well as peak and plateau pressures [13–15]. Compliance and airway resistance are further worsened by laparoscopy in obese as compared to non-obese individuals [16–20]. Increased soft tissue mass and fat deposits around neck and chest wall render obese individuals difficult to position, mask ventilate and intubate. An increase in adipose tissue, high basal metabolic rate and increased oxygen consumption is also observed in them. Hence, an overall synergistic effect of the above-mentioned factors is responsible for an instantaneous desaturation following induction of anaesthesia and during laparoscopic procedure.

During LC, a reverse Trendelenburg position is employed to produce gravitational displacement of viscera away from the surgical view for a better operating field. This results in improved respiratory mechanics as lesser pressure is exerted on the diaphragm by abdominal contents and pneumoperitoneum [21]. However, there is peripheral pooling of blood in the lower extremities causing a decrease in preload and mean arterial pressure (MAP).

Thus, the cumulative effects of pneumoperitoneum and positioning maneuvers result in alterations in hemodynamic parameters, pulmonary functions, ventilation and perfusion (V/Q) mismatch [22,23]. All these pathophysiological changes are more exaggerated in obese individuals complicating their anesthetic management [24,25]. They pose a challenge to anesthesiologists as well as surgeons. So, a thorough understanding of these changes is a must for anesthesiologists so that they are always prepared to prevent, detect and treat the possible complications that can occur during and after laparoscopic procedures.

Considering the aforementioned changes occurring due to pneumoperitoneum, positioning maneuvers and obesity-related complications, this study is performed to compare and evaluate the effects of pneumoperitoneum on various ventilatory and hemodynamic parameters in obese and non-obese individuals undergoing LC.

Materials and Methodology:-

The study was conducted in the Anaesthesiology and Intensive Care Department of the Max Super Speciality Hospital in Mohali. It received formal approval from the review board and institutional ethics committee before it started. In addition, the patients who took part in this study gave their express written consent. A cohort of one hundred patients was recruited for the study, randomly assigned, and classified according to predetermined criteria. The requirements for inclusion were anyone over the age of eighteen, male or female, in ASA grades I, II, or III, and able to provide written agreement at the time of entrance. On the other hand, patients who refused to take part, those who required conversion to open surgery, and those who had lung conditions prior to the study were excluded. Patients were assigned to groups based on their Body Mass Index (BMI), which divided them into two categories: group O, which included obese persons with a BMI greater than 30 kg/m², and group NO, which included non-obese individuals with a BMI less than 30 kg/m².

Study Design

The study conducted over a span of two years was a hospital-based prospective and randomized comparative analysis. It aimed to investigate and compare specific parameters between two distinct groups. The sample size determination involved meticulous calculations utilizing established formulas tailored to this study's requirements. With a meticulous consideration of various factors such as the desired power of 80%, a confidence interval set at 95%, a predefined difference in group means of 7.48, and a fixed ratio of sample sizes (1:1 for group 1 to group 2), the study arrived at a derived sample size of 102, translating to 51 individuals allocated to each group. This comprehensive approach ensured that the study possessed the statistical robustness necessary to yield meaningful and reliable insights within the given parameters and objectives.

The estimation of the sample size was based on specific assumptions: an alpha error of 50% and a beta error of 20%. The readings for group 1 and group 2 were 94.3 and 86.82, respectively, with a shared standard deviation of 12.37. Using a calculation tool (<http://powerandsamplesize.com/Calculators/Compare-2-Means/2-Sample-Equality>), the minimum required sample size was determined to be 43. Consequently, this figure established the minimum sample size per group, aligning the study's parameters with this designated value of 43.

Anaesthesia Technique

Before surgery, a pre-anaesthesia check-up was conducted, which involved documenting demographic details, age, gender, comorbidities, medication history, ASA status, and laboratory parameters. Patients were instructed to fast for 8 hours prior to surgery and were administered Oral Pantoprazole 40 mg the night before the procedure. Additionally, patients were informed about the study and provided consent to participate. During the pre-induction phase, the anaesthesia machine was verified preoperatively, and all patients had a wide bore cannula inserted. Intravenous fluids (crystalloids) were administered at a minimum volume of 10ml/kg, with an additional 10-15 ml/kg during laparoscopic cholecystectomy. Standard monitors including non-invasive blood pressure, 5-lead electrocardiogram, and pulse oximetry were applied after the patient was transferred to the operation theatre.

Patients preoxygenated with 100% oxygen for 3 mins. Analgesia: fentanyl (2 mcg/kg). Induction: propofol (1.5-2.5mg/kg). Muscle relaxation: atracurium (0.5mg/kg). Tracheal intubation: cuffed endotracheal tube (females: 7.5mm, males: 8.5mm) using direct laryngoscopy. Controlled ventilation: 14 breaths/min, tidal volume (6-8ml/kg ideal body weight), 1:2 inspiratory/expiratory ratio, EtCO₂ (30-40 mmHg). Maintenance: sevoflurane, low-flow oxygen and nitrous oxide (1:1). CO₂ insufflation during pneumoperitoneum, patients in horizontal position. Post-pneumoperitoneum: reverse trendelenberg, maintained throughout surgery. Neutral positions post-gall bladder removal and peritoneal wash.

Hemodynamic (HR, SBP, DBP, MAP, perfusion index, SpO₂, MAC, EtCO₂) and Ventilatory (RR, TV, PEEP, Ppeak, Pplat, driving pressures, static and dynamic compliance) parameters were logged. Readings taken: baseline, post-anaesthesia induction, post-intubation, at CO₂ insufflation (0, 1, 3, 5 mins), 1 and 3 mins post-desufflation, pre-extubation. All data documented for further assessment. Patients observed in recovery for 30-45 mins pre-ward transfer. Intra-op measurements derived:

- TV, PEEP, Ppeak, Pplat from the ventilator.
- Driving pressure (PDRIVING) = Pplat – PEEP [26]
- Static compliance (CSTAT) = TV / (Pplat - PEEP)
- Dynamic compliance (CDYNAMIC) = TV / (Ppeak - PEEP) [27-29]

Statistical Analysis

The statistical analysis was carried out using IBM SPSS (Statistical Package for Social Sciences) statistical version 20. The analysis includes frequency table, bar, pie chart, association of variables based on Chi-square test. All quantitative variables were estimated using measures of central location (mean and median) and measures of dispersion (standard deviation). For normality distributed data, Mean was compared with respect to t-test (for two groups) and Paired t-test (for Compare Baseline to follow-up). All statistical tests were seen at two-tailed level of significance ($p \leq 0.01$ and $p \leq 0.05$).

Result:-

Table 1 represents the demographic distribution of both the groups. The mean age in group NO was 51.46 ± 15.07 years and in group O was 48.74 ± 10.77 years.

Table 1:- Demographic distribution of the studied population.

		Group-A	Group-B	Chi-Square	p-value
Age		51.46±15.07	48.74±10.77	7.207	0.302
Sex	Male	22 (44%)	14 (28%)	2.778	0.096
	Female	28 (56%)	36 (72%)		

In both group A and group B, majority of them were females (56% and 64% respectively). However, no statistically significant difference in age and gender distribution was noted between the two groups ($P>0.05$).

Table 2:- Distribution of the studied population based on their weight, height and BMI.

Parameters	Group-A	Group-B	t	p
Weight (Kg)	66.66±8.65	87.06±10.53	10.583	.0001**
Height (Cm)	160.82±10.58	156.98±8.85	1.968	0.052
BMI	25.08±2.41	35.02±2.87	18.749	.0001**

Table 2 represents the distribution of the studied population based on their average Weight, Height and BMI in group A(NO) and group B(O). In the **NO** group, the mean weight was 66.66 ± 8.65 kg whereas in the **O** group it was 87.06 ± 10.53 kg and was statistically significant ($p<0.05$). The mean height in the **NO** group was 160.82 ± 10.58 cm whereas in the **O** group it was 156.98 ± 8.85 cm and was statistically non-significant ($p>0.05$). The mean BMI observed in the **NO** group was 25.08 ± 2.41 kg/m² whereas in the **O** group it was 35.02 ± 2.87 kg/m² and was statistically significant ($p<0.05$).

Table 3 presents a comprehensive comparison of two study groups (Non-Obese and Obese) concerning changes in various physiological parameters at different time points (T1 to T10). The parameters include heart rate, systolic and diastolic blood pressure, mean arterial pressure (MAP), oxygen saturation (SpO₂), perfusion index (PI), and end-tidal carbon dioxide (ETCO₂). The statistical significance (Sig.) of differences between the two groups at each time point is assessed using the p-value.

Table 3:- Comparison of study groups as per changes in heart rate, systolic, diastolic pressure, atrial pressure, oxygen saturation (SpO₂) and Perfusion index (PI).

Group	Time									
	T1	T2	T3	T4	T5	T6	T7	T8	T9	T10
Heart Rate										
Non-Obese	87.46±17.43	84.64±16.47	92.04±16.01	83.20±14.24	86.80±17.84	85.32±13.99	82.30±13.56	85.68±14.27	83.04±14.27	89.74±18.60
Obese	82.32±9.66	85.46±5.72	89.54±11.82	79.02±14.04	82.82±14.29	82.36±14.26	81.90±12.68	81.14±10.77	81.08±9.57	83.66±11.34
Sig.	0.071	0.74	0.376	0.143	0.221	0.297	0.879	0.076	0.422	0.051
Systolic blood pressure										
Non-Obese	128.62±19.74	116.06±18.44	116.72±21.19	105±14.23	115.46±16.93	123.38±18.30	128.16±17.97	129.26±15.27	125.24±13.22	125.86±14.94
Obese	134.90±13.02	119.22±13.04	122.72±18.95	108.02±15.40	118.02±14.06	125.28±13.01	127.20±15.61	125.36±22.16	128.56±31.54	131.30±14.80
Sig.	0.063	0.325	0.139	0.201	0.413	0.551	0.776	0.308	0.494	0.07
Diastolic blood pressure										
Non-Obese	79.28±12.94	76.86±12.69	77.54±13.82	71.12±11.71	80.90±12.48	85.24±12.86	86.96±13.22	83.62±11.31	81.42±10.39	82.68±15.05
Obese	84.38±14.35	79.62±12.24	83.38±15.98	76.98±12.32	83.52±12.52	88.22±11.54	85.40±12.58	82.50±12.47	81.50±13.50	14.84±14.84

Sig.	0.065	0.271	0.053	0.017*	0.297	0.225	0.547	0.639	0.974	0.075
MEAN ARTERIAL PRESSURE (MAP)										
Non-Obese	94.44±15.34	89.28±13.10	90.02±14.56	82.70±11.26	92.80±13.22	96.66±13.17	99.56±13.92	97.08±10.69	94.58±10.01	96.04±11.49
Obese	99.62±12.49	92.76±12.39	94.50±14.59	86.98±1.57	94.48±11.53	99.56±10.86	98.74±13.43	96.62±11.85	94.70±13.25	99.60±15
Sig.	0.067	0.175	0.128	0.064	0.5	0.232	0.765	0.839	0.959	0.186
Oxygen saturation (SpO2)										
Non-Obese	98.28±0.73	99.98±0.14	100±0.0	100±0.0	100±0.0	100±0.0	100±0.0	100±0.0	100±0.0	100±0.0
Obese	98.12±0.56	99.88±0.33	100±0.0	100±0.0	100±0.0	100±0.0	100±0.0	100±0.0	100±0.0	100±0.0
Sig.	0.221	0.051	1	1	1	1	1	1	1	1
Perfusion index (PI)										
Non-Obese	1.50±1	1.85±1.37	2.89±1.96	2.41±1.38	2.47±2.02	2.72±2.86	2.86±2.54	3.04±2.56	4.08±1.436	2.01±1.43
Obese	1.88±1.35	2.38±1.79	3.72±2.40	2.71±1.58	3.03±1.86	3.24±2.02	3.31±2.29	3.20±2.42	3.56±1.91	2.66±2.52
Sig.	0.115	0.098	0.062	0.314	0.158	0.304	0.348	0.751	0.8	0.115
ETCO2										
Non-Obese	24.7±2.43	25.56±2.33	27.54±3.21	26.86±3.30	27.48±3.27	29.72±3.48	31.50±3.43	32.10±3.49	33.0±2.78	29.08±5.19
Obese	25.20±2.13	26.38±28.50±2.26	28.50±2.26	27.32±2.43	28.04±2.78	29.18±2.62	31.10±3.37	32.22±3.69	34.0±2.04	31.20±3.02
Sig.	0.276	0.064	0.087	0.429	0.358	0.383	0.558	0.868	.043*	.014*

Non-Obese individuals show a slightly higher mean heart rate throughout the study compared to Obese individuals, but the differences are not statistically significant at any time point ($p > 0.05$). Non-Obese individuals generally exhibit lower systolic blood pressure compared to Obese individuals, with borderline significance observed at T10 ($p = 0.07$). Non-Obese individuals consistently show lower diastolic blood pressure compared to Obese individuals. The difference becomes statistically significant at T4 ($p = 0.017$). No statistically significant differences are observed in mean arterial pressure between Non-Obese and Obese groups at any time point ($p > 0.05$). Both groups maintain high and comparable levels of oxygen saturation throughout the study, with no statistically significant differences at any time point ($p > 0.05$). Non-Obese individuals generally exhibit lower perfusion index values compared to Obese individuals, with a borderline significance observed at T3 ($p = 0.062$). Non-Obese individuals show lower ETCO2 levels compared to Obese individuals, with a statistically significant difference at T9 ($p = 0.043$) and T10 ($p = 0.014$).

The study indicates that Non-Obese and Obese individuals exhibit differences in cardiovascular and respiratory parameters, with some parameters showing statistically significant variations at specific time points. The findings suggest potential physiological distinctions between the two groups, emphasizing the importance of considering obesity in the context of cardiovascular and respiratory health.

Table 4 presents the Minimum Alveolar Concentration (MAC) distribution for two groups, Non-Obese and Obese, at different time points (T2 to T10). MAC represents the concentration of an inhaled anesthetic required to prevent movement in response to a surgical stimulus in 50% of patients. The table includes mean values with standard deviations, and the statistical significance (Sig.) of differences between the two groups at each time point is assessed using the p-value.

Table 4:- Mac Distribution.

Groups	Time								
	T2	T3	T4	T5	T6	T7	T8	T9	T10

Non-Obese	0.34±0.05	0.62±0.12	1±0.0	1±0.0	1±0.0	1±0.0	1±0.0	1±0.0	0.10±0.0
Obese	0.32±0.06	0.65±0.14	1±0.0	1±0.0	1±0.0	1±0.0	1±0.0	1±0.0	0.10±0.0
Sig.	0.053	0.254	1	1	1	1	1	1	1

Both Non-Obese and Obese groups show similar MAC distribution across all time points, with no statistically significant differences observed ($p > 0.05$). At T2, the mean MAC for Non-Obese is 0.34 ± 0.05 , while Obese is 0.32 ± 0.06 . The difference is not statistically significant ($p = 0.053$). From T3 to T10, the MAC values are consistently 1 ± 0.0 for both groups, indicating that the concentration of the inhaled anesthetic required for the intended effect is uniform and not significantly different between Non-Obese and Obese individuals ($p = 1$).

The findings suggest that, the Minimum Alveolar Concentration remains stable and comparable between Non-Obese and Obese individuals at different time points. The lack of significant differences in MAC distribution implies that the anesthetic requirements for achieving a specified level of anesthesia do not vary between the two groups. It is essential to note that a p-value close to the significance threshold (e.g., $p = 0.053$) may warrant further investigation in larger studies to validate the trends observed. The study provides insights into MAC distribution in relation to obesity, suggesting that anesthetic management may not need significant adjustments based on obesity status in the studied population.

Table 5 presents a detailed comparison of two study groups, Non-Obese and Obese, concerning changes in various respiratory parameters at different time points (T3 to T10). The parameters include respiratory rate (RR), tidal volume (TV), positive end-expiratory pressure (PEEP), peak inspiratory pressure, plateau pressure, driving pressure, static compliance, and dynamic compliance. The table includes mean values with standard deviations, and the statistical significance (Sig.) of differences between the two groups at each time point is assessed using the p-value.

Table 5:- Comparison of study groups as per changes in tidal volume, peak inspiratory, plateau pressure, End-tidal carbon dioxide level, Driving pressure, Static and Dynamic compliance.

Groups	Time							
	T3	T4	T5	T6	T7	T8	T9	T10
RESPIRATORY RATE (RR)								
Non-Obese	14.0±0.0	14.0±0.0	14.0±0.0	14.0±0.0	14.0±0.0	14.0±0.0	14.0±0.0	14.0±0.0
Obese	14.0±0.0	14.0±0.0	14.0±0.0	14.0±0.0	14.0±0.0	14.0±0.0	14.0±0.0	14.0±0.0
Sig.	1	1	1	1	1	1	1	1
TIDAL VOLUME (TV)								
Non-Obese	436.60±23.44	436.60±23.44	436.60±23.44	436.60±23.44	436.60±23.44	436.60±23.44	436.60±23.44	436.60±23.44
Obese	489.0±17.17	489.0±17.17	489.0±17.17	489.0±17.17	489.0±17.17	489.0±17.17	489.0±17.17	489.0±17.17
Sig.	.0001**	.0001**	.0001**	.0001**	.0001**	.0001**	.0001**	.0001**
PEEP								
Non-Obese	5±0.0	5±0.0	5±0.0	5±0.0	5±0.0	5±0.0	5±0.0	5±0.0
Obese	5±0.0	5±0.0	5±0.0	5±0.0	5±0.0	5±0.0	5±0.0	5±0.0
Sig.	1	1	1	1	1	1	1	1
PEAK INSPIRATORY PRESSURE								
Non-Obese	16.54±2.99	21.88±4.19	22.42±3.29	21.80±2.81	21.74±2.85	18.78±2.71	18.22±2.46	16.28±2.15
Obese	17.44±2.54	22.58±3.31	27.36±4.27	28.18±3.67	28.20±3.65	23.10±3.17	21.02±2.45	21.16±2.82
Sig.	0.108	0.357	.0001**	.0001**	.0001**	.0001**	.0001**	.0001**

PLATEAU PRESSURE								
Non-Obese	14.04±2.72	18.42±3.41	19.52±2.98	18.90±2.35	19.18±2.49	16.12±2.43	15.66±2.32	13.82±1.78
Obese	14.96±2.04	19.52±3.26	24.22±3.81	25.10±3.47	25.12±3.17	20.62±2.77	18.62±2.12	18.70±2.35
Sig.	0.058	0.102	.0001**	.0001**	.0001**	.0001**	.0001**	.0001**
DRIVING PRESSURE								
Non-Obese	8.92±2.66	13.42±3.41	14.52±2.98	13.90±2.35	14.18±2.49	11.12±2.43	10.66±2.32	8.82±1.78
Obese	9.84±2.23	14.34±3.10	18.78±3.89	19.66±3.50	19.70±3.25	15.20±2.89	13.20±2.38	13.28±2.43
Sig.	0.064	0.161	.0001**	.0001**	.0001**	.0001**	.0001**	.0001**
STATIC COMPLIANCE								
Non-Obese	53.67±16.85	35.02±10.36	31.29±6.55	32.36±6.16	31.85±6.46	41.53±11.10	43.33±12.30	51.52±10.72
Obese	48.12±11.70	32.23±7.91	27.12±5.78	25.64±4.73	25.55±4.68	33.43±7.12	38.28±7.85	38.05±7.08
Sig.	0.058	0.134	.001**	.001**	.001**	.001**	.018*	.001**
DYNAMIC COMPLIANCE								
Non-Obese	42.08±10.99	27.68±7.98	26.05±6.68	26.76±5.05	26.94±5.45	33.18±8.10	34.38±7.89	40.14±8.18
Obese	38.29±9.16	27.11±6.22	23.11±4.47	22.03±3.58	22.05±3.83	28.73±6.0	32.33±6.11	32.14±6.32
Sig.	0.079	0.692	.005**	.0001**	.0001**	.002**	0.149	.0001**

Both Non-Obese and Obese groups maintain a constant respiratory rate of 14.0 breaths per minute throughout the study, and the difference is not statistically significant ($p = 1$). Non-Obese individuals consistently exhibit lower tidal volumes (436.60 ± 23.44 ml) compared to Obese individuals (489.0 ± 17.17 ml), and the differences are highly statistically significant at all time points ($p < 0.0001$). PEEP values are constant at 5 cmH₂O for both Non-Obese and Obese groups at all time points, with no statistically significant differences ($p = 1$). Non-Obese individuals generally exhibit lower peak inspiratory pressures compared to Obese individuals, but the differences are not statistically significant ($p > 0.05$). Non-Obese individuals tend to have lower plateau pressures compared to Obese individuals, with borderline significance observed at T1 ($p = 0.058$) and T2 ($p = 0.102$). Similar to plateau pressure, Non-Obese individuals generally exhibit lower driving pressures compared to Obese individuals, with borderline significance at T1 ($p = 0.064$) and T2 ($p = 0.161$). Non-Obese individuals consistently show higher static compliance compared to Obese individuals, with statistically significant differences observed at all time points ($p < 0.001$). Similar to static compliance, Non-Obese individuals consistently show higher dynamic compliance compared to Obese individuals, with statistically significant differences observed at all time points ($p < 0.005$).

Overall, the study indicates significant differences between Non-Obese and Obese individuals in terms of tidal volume, static compliance, and dynamic compliance. These findings suggest that respiratory parameters are influenced by obesity status, highlighting the importance of considering weight-related factors in respiratory management and ventilator settings. The study provides valuable insights into the respiratory characteristics of Non-Obese and Obese individuals in a controlled clinical setting.

Discussion:-

Laparoscopic surgery, known for less pain and quicker recovery, like Laparoscopic cholecystectomy (LC), now preferred over open surgeries for gallstones. In LC, carbon dioxide (CO₂) pneumoperitoneum and reverse Trendelenburg positioning are vital steps but can affect cardio-pulmonary functions, especially in obese patients [20]. In obesity, laparoscopy may heighten CO₂ absorption, causing hypercarbia, needing higher ventilation. Increased intra-abdominal pressure during pneumoperitoneum impacts blood flow, urinary output, respiratory compliance, airway pressures, and cardiac function [26].

In our study, we recorded and compared parameters such as Heart Rate (HR), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), peripheral oxygen saturation (SpO₂), end-tidal carbon dioxide (EtCO₂), perfusion index (PI), peak (PPEAK), plateau (PPLAT) and driving (PDRIVING) pressures, static (CST) and dynamic (CDYN) lung compliance at varying time intervals in both obese and non-obese patients undergoing LC.

This prospective study was carried out on 100 patients divided randomly into two groups of 50 each viz. non-obese (NO) and obese (O). The mean age in the NO group was 51.46 ± 15.07 years and that of the O group was 48.74 ± 10.77 years. NO group consisted of 22 males and 28 females whereas the O group had 14 males and 36 females.

The mean weight in the NO group was 66.66 ± 8.65 kg and 87.06 ± 10.53 kg in the O group and the difference was statistically significant ($p < 0.05$). The mean height in the NO group was 160.82 ± 10.58 cm whereas in the O group it was 156.98 ± 8.85 cm and was statistically non-significant ($p > 0.05$). The mean BMI observed in the NO group was 25.08 ± 2.41 kg/m² while in the O group it was 35.02 ± 2.87 kg/m² and was statistically significant ($p < 0.05$).

The baseline means heart rate of the NO group and O group was 87.46 ± 17.43 bpm and 82.32 ± 9.66 bpm respectively, which was comparable. On induction of anaesthesia, a decrease in heart rate was observed in the NO group whereas an increase was observed in the O group however, it was statistically non-significant. Rossi R et al. and Wu J et al. noted higher heart rates in obese individuals, suggesting an autonomic imbalance with reduced parasympathetic activity and increased sympathetic dominance [30,31]. post-intubation, both groups showed increased heart rates, which was not statistically significant. One minute after insufflation, a rise in heart rate was seen in both groups, more pronounced in obese patients, yet statistically insignificant. This rise is linked to reduced venous return due to increased intra-abdominal pressure, decreasing cardiac output, and prompting a compensatory heart rate increase. Fried et al. also observed similar trends in obese vs. non-obese individuals during laparoscopic surgery [32].

Baseline SBP, DBP, and MAP were similar between both groups, lacking statistical significance. Although not statistically significant, the O group displayed higher resting SBP, DBP, and MAP. Weil et al observed obese individuals are more prone to hypertension, likely due to increased vasoconstrictor tone and reduced vasodilation in obese patients [33]. During induction, both groups saw a non-significant decrease in SBP, DBP, and MAP. Post-intubation, the O group exhibited higher mean SBP, DBP, and MAP than the NO group, statistically insignificant ($p\text{-value} > 0.05$). Obese individuals often develop LV hypertrophy due to increased blood volume to meet high metabolic demands from excess fat, explaining the higher SBP, DBP, and MAP in the O group.

Chopra G et al compared various hemodynamic parameters like SBP, DBP, and MAP in 2 groups of patients posted for LC and open cholecystectomy and found a significant increase in these values in patients undergoing laparoscopies [12]. The authors proposed an increase in SVR and catecholamine release due to abdominal distension and hypercarbia as possible mechanisms for these alterations [34].

PI reflects peripheral perfusion, measured non-invasively from an oximeter. Baseline mean PI for NO and O groups: 1.48 ± 0.73 and 1.982 ± 0.56 , comparable. PI increased post-induction, intubation, insufflation, and desufflation. Similar changes during induction and intubation seen in Choudhary et al's study, linked to catecholamine surge from laryngoscopy [35]. Increased IAP decreases venous return, elevates SVR, affecting blood pressure, and impacting PI. Throughout laparoscopy, PI rose in our study. However, Liu F et al observed a contrasting decrease in PI with rising IAP during LC [36].

The mean EtCO₂ of the NO group and O group was 27.24 ± 3.22 and 28.78 ± 2.29 respectively which was comparable. It increased following induction, after insufflation and post-desufflation in both groups though non-significant. Bhadauria A S et al observed a similar increase in EtCO₂ values throughout the pneumoperitoneum and post-desufflation in obese individuals undergoing LC [37]. The authors attributed this rise of EtCO₂ values to the increase in absorption of CO₂ into the systemic circulation through the peritoneal surface and hypoventilation due to elevated diaphragm during pneumoperitoneum.

Baseline PPEAK and PPLAT were higher in O vs. NO groups post-intubation but not statistically significant ($p > 0.05$). Insufflation raised PPEAK/PPLAT in both, more in O. Post-insufflation, O group consistently showed significantly higher PPEAK/PPLAT ($p < 0.05$). On desufflation, both groups decreased, yet O remained notably

higher and significant ($p < 0.05$). Before extubation, values neared baseline but were significantly higher in O ($p < 0.05$). Sprung J et al found similar trends in obese patients, attributing it to reduced FRC [38]. Dumont L et al and Bhadauria A et al also noted increased airway pressures post-insufflation, suggesting mechanical effects from increased IAP causing abdominal distension and altering respiratory mechanics [19,37].

Driving pressure (PDRIVING) measures alveolar opening pressure (PPLAT-PEEP), inversely linked to lung compliance. Post-intubation, the O group had higher PDRIVING but was statistically insignificant ($p > 0.05$). Insufflation increased PDRIVING in both, significantly more in O ($p < 0.05$). On desufflation, PDRIVING decreased in both, more in NO vs. O ($p < 0.05$). Pre-extubation, values neared baseline, significantly lower in NO vs. O ($p < 0.05$). Casati A et al. found similar trends in obese patients, attributing them to increased IAP elevating central venous pressure and affecting lung elastance, raising driving pressures [20]. Mazzinari G et al. linked increased IAP during pneumoperitoneum to stiffer chest walls, escalating driving pressures during laparoscopy [39].

Static compliance (CST) gauges lung elasticity without airflow and correlates inversely with plateau pressures, always exceeding dynamic compliance. Post-intubation, NO had higher but statistically insignificant CST ($p > 0.05$). Insufflation reduced CST in both, significantly more in O ($p < 0.05$). After desufflation, CST improved, notably more in NO vs. O ($p < 0.05$). Pre-extubation, CST approached baseline, better in NO vs. O ($p < 0.05$). In awake obese patients, chest wall compliance diminishes up to 35% due to fat accumulation around ribs and abdomen, increasing physiological dead space. Casati A et al and Dumont L et al found decreased compliance in obese patients during laparoscopy, attributing it to fat accumulation, limited diaphragm movement, and altered chest wall excursion [19,20]. Oikkonen M et al and Antini D et al observed decreased compliance post-pneumoperitoneum, linking it to increased IAP, which stiffens the chest wall, reducing lung compliance [40,41].

Dynamic compliance (CDYN) measures lung compliance during normal breathing, representing airway resistance and lung/chest wall elasticity. Post-intubation, NO had a higher but statistically insignificant CDYN ($p > 0.05$). Insufflation decreased CDYN in both, significantly lower in O ($p < 0.05$). After desufflation, CDYN improved in both, more in NO ($p < 0.05$). Pre-extubation, CDYN approached baseline, better than NO ($p < 0.05$). Obesity increases respiratory resistance, limits chest wall mobility, and reduces pulmonary volumes, decreasing compliance. Araujo O. C. et al. found similar findings in obese vs. non-obese patients during LC, attributing them to anesthesia's effects on respiratory drive and reduced FRC [42]. Obeid F. et al. observed decreased pulmonary compliance post-insufflation during laparoscopic cholecystectomy due to reduced diaphragmatic compliance [43]. Hemodynamic and ventilatory variations were more pronounced in obese patients but statistically significant only in ventilatory parameters. No post-op adverse events like delayed recovery or CO₂ retention were observed in either group.

Our study had few limitations. Firstly, baseline measurements of hemodynamic parameters were taken in the supine position, whereas, patient's position was changed after creation of pneumoperitoneum. Secondly, patient population above BMI $> 30 \text{ kg/m}^2$ were considered obese. No further classification of obesity was done.

Conclusion:-

This study concludes that obese patients exhibit more variations in hemodynamic and respiratory parameters as compared to non-obese patients. HR, SBP, DBP, MAP, PPEAK, PPLAT and PDRIVING were higher whereas, CST and CDYN were lower in obese patients. Variations in hemodynamic parameters were more pronounced in obese patients, but statistically non-significant. Whereas the higher variations in respiratory parameters in the obese group was statistically significant. No adverse events like delayed recovery, post-operative ventilatory support, signs of CO₂ retention were observed in our study population.

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