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The haemodynamics and the consequent arterial blood gas changes during carbon dioxide insufflation in laparoscopic surgeries in hypertensive patients.

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Abstract:
Objective: *The pneumoperitoneum caused during laparoscopic surgeries by the insufflation of carbon dioxide is known to cause significant changes in arterial blood gas values and in the haemodynamics. The aim of our study was to compare the effects of pneumoperitoneum on haemodynamics and arterial blood gas parameters in hypertensive and normotensive groups.*
Methods:*After getting approval from the Institutional Ethics Committee (Approval number MGIMS/IEC/ANAE/64/2014 dated 14th November 2014), 30 hypertensive patients undergoing laparoscopic surgery were considered as cases with 30 normotensive patients undergoing laparoscopic surgery considered as control population. The ABGs were sent before insufflation and immediately after desufflation. The patients were monitored with SBP, DBP, MBP HR, and* $ETCO_2$ *throughout the procedure. All patients in our study did not suffer blood loss and no transfusion were required intraoperatively. All patients had same anaesthetic parameters and measurements performed under same standardized procedure.*
Results: *Demographic parameters were comparable between both the groups. Mean arterial pressure was elevated more in hypertensive patients as compared to normotensive group (statistically significant). Acidosis though mild but statistically significant (both metabolic and respiratory) was seen more in hypertensive patient group as compared to normotensive group.*
Conclusion: *Hypertensive patients undergoing laparoscopic surgeries appears to be more prone to increase in SBP, DBP, and MAP. There also appears a trend for increased acidosis (metabolic and respiratory) in hypertensive patients. This factors needs to be monitored and considered when maintaining anaesthesia in hypertensive patients for laparoscopic surgeries. More studies needs to be done with larger sample size.*
Keyword*: haemodynamic changes, laparoscopic surgery, arterial blood gas (ABG), pneumoperitoneum.*

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I. Introduction:

Laparoscopic surgeries are nowadays being considered as day-care procedures, thus decreasing the cost, hospital stay, and early mobility with early back to daily routine activities. Constant work was done in developing skills and science behind laparoscopic surgeries and it has become a common, daily-performed procedure worldwide, replacing many types of open surgeries. Laparoscopic surgeries hold several advantages over open procedures because of its smaller incisions, less postoperative pain, better wound healing, cosmetically better scar. The patients recover early and hence lesser is the hospital stay. The total cost in-admission to discharge is lesser as compared to the open procedures and so is the morbidity.

A lot of research was done to conclude on ideal gas for creating pneumoperitoneum. Carbon dioxide (CO2) and helium were found to meet the desired requirement. Air, Helium, Nitrous Oxide, Oxygen, Nitrogen, and Argon were the other gases studied for pneumoperitoneum. CO2 is superior because of its colourless, odourless, extremely soluble, non- flammable (when used with electro-cautery), not supporting combustion, non-toxic, non-irritating and easy availability which had almost the qualities of an ideal gas for insufflation, and is preferred gas for Laparoscopic surgeries. Though CO2 insufflation to create pneumoperitoneum has a lot of benefits over other gases, it comes with its own side effect profile too, mainly due to its high blood solubility status. Due to the absorption of CO2 after pneumoperitoneum, there is an increase in the cardiac output (CO), mean arterial pressure (MAP), heart rate (HR), and plasma catecholamine concentrations. The direct effect of CO2 is myocardial depression, vasodilatation, and increased systemic vascular resistance (SVR) [1]. This limits the use of CO_2 pneumoperitoneum in laparoscopic surgeries in higher ASA grades and critically ill patients. An attempt was made to observe the haemodynamics and Arterial Blood Gas changes occurring in the hypertensive patients posted for elective laparoscopic surgeries under general anaesthesia.

II. Materials and Methods:

This was an observational cross-section study carried out at a tertiary care hospital in rural India. The Institutional Ethics Committee approved the study (approval number MGIMS/IEC/ANAE/64/2014 dated 14th November 2014). All procedures performed in the study followed the ethical guidelines of the Declaration of Helsinki. Voluntary written informed consent was obtained from all participants. The study was undertaken for 18 months period starting from January 2015.

60 patients were selected who were scheduled to undergo abdominal laparoscopic surgery, and divided into groups of 30 each, of hypertensive and normotensive. All the patients, aged between 35 years and 60 years, ASA 2 and 3, who were hypertensive at least from one year, irrespective of their drug regimen and their baseline blood pressure readings were included in the hypertensive group (group H). No changes were made in the dosing regimens of their antihypertensive drugs. All ASA 1 patients were included in the normotensive group (group N). Patients not willing for consent, out of the age criteria, with ASA status 4 or above were excluded.

Inclusion criteria:

1) Normotensive subjects between 35 and 60 years of age with ASA physical status I.
2) Hypertensive subjects with ASA I/II/III with on/off treatment since at least 1 year.

Exclusion criteria:

1) Patients not willing for the study
2) Patients with ASA physical status IV or more,
3) Patients less than 35 years and more than 60 years of age,
4) Patients with BMI more than 30.
5) Pregnant ladies.

Procedure Methodology:

After selecting the patients for the study, they were explained about the nature of the study, and the necessary consents were obtained. These patients received oral sedation with Tab Diazepam 5 mg and antacid prophylaxis with Tab Ranitidine 150 mg night before the surgery. The antihypertensive medicines of the patients were continued as per the schedule and no changes were suggested or made regarding their dosing. Standard fasting guidelines were strictly followed.

Baseline parameters were recorded in the Operation Theatre. After performing modified Allen's test on each patient the radial artery was cannulated with 20 G cannula, for invasive pressure monitoring and arterial blood samples for ABG analysis. A preoperative arterial blood sample was sent for the blood gas analysis in a pre-heparinized syringe by standard technique and sample tested for arterial blood gas analysis before induction of anaesthesia. The same line was then used for invasive monitoring of systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP), and heart rate (HR).

Patients received the following drugs: Inj. Ondansetron 0.08 mg/kg, Inj Ranitidine 1 mg/kg, Inj. Glycopyrrolate 5 μgm/kg, Inj. Midazolam 0.03 mg/kg, Inj. Fentanyl 2 μg/kg immediately before induction of anaesthesia. Patients were pre-oxygenated with 100 % Oxygen for 3 minutes before inducing with Inj. Propofol 2 mg/kg. The patients' lungs were manually ventilated with 100% oxygen before intubation. To facilitate tracheal intubation Inj. Succinyl Choline 1.5 mg/kg was given. Haemodynamic parameters were recorded every 5 minutes from the start of the induction of anaesthesia. Tracheal intubation was done with an appropriately sized cuffed oral endotracheal tube and a nasogastric tube was inserted. Anaesthesia was maintained by 50% Oxygen with medical-grade Air, Isoflurane, and intermittent boluses with Inj. Atracurium 0.1 mg/kg. The tidal volume and ventilator frequency was adjusted and intermittent positive pressure ventilation was continued by a mechanical ventilator.

Pneumoperitoneum was created with CO_2 and Intra-abdominal pressure was maintained between 12 and 15 mmHg throughout the procedure. After pneumoperitoneum was established ventilator settings (Tidal Volume, Respiratory Rate) were changed accordingly to maintain $EtCO_2$ levels below 45 mm hg and SBP, DBP, MAP, HR, SPO_2, and $EtCO_2$ were documented every 2 minutes till desufflation. Immediately after desufflation, the second sample of arterial blood was analyzed. The mean of the values until 30 minutes of each of the parameters were measured and were considered as the post-insufflation values. At the end of surgery, the residual neuromuscular blockade was reversed by Inj. Neostigmine (0.05 mg/kg) with Inj. Glycopyrrolate (0.01 mg/kg) intravenously. The trachea was extubated and the patients were shifted to the post-operative recovery room.

Study variables:

Haemodynamic variables studied were heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MBP), and end-tidal carbon dioxide concentration (EtCO2). In the arterial blood gases we studied pH, PaCO2, and HCO3. All these data were documented and analyzed for the possible changes that occur due to carbon dioxide pneumoperitoneum in hypertensive patients.

Statistical analysis:

All statistical analysis was done by using descriptive and inferential statistics using Chi-Square Test, and Student's Paired t-Test. Software used for analysis was SPSS version 17.0, GraphPad Prism version 6.0, and EPI INFO version 6.0. 'p' value < 0.05 was considered as the level of significance.

III. Results:

Out of the 60 patients, 30 were hypertensive and 30 were normotensive. The mean age was 46.63 ± 8.5 years in the hypertensive group while it was 41.26 ± 7.0 years in the normotensive group. 25 females and 35 males participated in the study. 28 patients were in the American Society of Anaesthesiologists physical classification of II and 2 patients were in class 3. All the patients in the group N were ASA physical status class I.

In both the groups, the systolic, the diastolic, and the mean arterial pressures increased after the insufflation and then had a slow decline till desufflation. The values were statistically higher from the initial values at the start of insufflation. There were statistically significant differences in the values in both groups. At post desufflation, the values were significantly higher than the baseline pre insufflation values. Whereas, the heart rate increased significantly from the initial values after insufflation in both the groups, the rise was statistically insignificant amongst the groups. The heart rate increased after the creation of the pneumoperitoneum, and the difference at the desufflation time was significant. (Tables 1,2,3,4. Graph 1, 2, 3, 4).

There was a statistically significant increase in the EtCO2 after the CO2 insufflation in both the groups. The CO2 increased progressively after pneumoperitoneum was created and decreased after desufflation, though the final value was still higher than the initial value. In between the groups, there was no clear statistical significance as there were non-significant values at various time intervals. EtCO2 showed a gradual increase in both the groups, though the increase was significant, it remained clinically non-significant and within acceptable physiological limits.The values were statistically insignificant amongst the groups. (Table 5, graph 5).

The pneumoperitoneum caused in both the groups, which was significant as compared to the pre-pneumoperitoneum values. However, the change was insignificant after pneumoperitoneum amongst the groups. In both the groups, the PaCO2 values increased after the pneumoperitoneum. During our study, the EtCO2 was not allowed to exceed 45 mm Hg, which might have kept the rise in PaCO2 to an acceptable range which was well within normal values.Since Carbon dioxide is highly diffusible gas that gets absorbed through the peritoneal surface, it causes marked hypercarbia, more so in absence of compensatory hyperventilation. In both the groups, the bicarbonates dropped a bit, though they were maintained well within normal ranges.

IV. Discussion:

Laparoscopic surgery compared to conventional surgery has many advantages for patients including small incision less post-operative pain, earlier oral intake, quicker mobilization, faster discharge, and a better cosmetic effect. During laparoscopy, pneumoperitoneum is essential to provide a good surgical field, allowing visibility and performing surgical procedures. Carbon dioxide (CO2) is the most common gas used for pneumoperitoneum. Because of its property of high solubility after CO2 insufflation, it diffuses rapidly through the tissues and peritoneal membrane and enters the systemic circulation. This is then removed from the body by the lungs. Hence the process of creating pneumoperitoneum causes haemodynamic and respiratory alterations.

The systemic vascular resistance increases after CO2 pneumoperitoneum. This increase in systemic vascular resistance (SVR) is because of the mechanical and neurohumoral factors[1]. Catecholamines, the renin-angiotensin system, prostaglandins, and especially vasopressin are released during pneumoperitoneum which contributes to the increase in the afterload[2, 3]. Increased IAP due to CO2 pneumoperitoneum affects the venous return and systemic vascular resistance, and myocardial function. The changes occur due to the autotransfusion of pooled blood from the splanchnic circulation, which increases the venous return and cardiac output that leads to increased blood pressures and heart rate[4]. The increased plasma vasopressin concentrations along with the CO2 absorption from the peritoneal surfaces have been correlated with the intra-thoracic pressures and the transmural right atrial pressures. Mechanical stimulation of peritoneal receptors results in increased vasopressin release, SVR, and arterial pressure. The increase in the systemic vascular resistance also explains why arterial pressure increases, with cardiac output decreasing. A similar finding was noted by Gupta S et al. when they studied the haemodynamic parameters in patients undergoing laparoscopic surgeries and found that the systolic blood pressure increased non-significantly after pneumoperitoneum, but was within normal limits[5].

The direct and indirect effects of hypercarbia due to CO2 insufflation are dual and opposite. Directly hypercarbia causes vasodilation and depression of myocardium. Indirect effects of hypercarbia causes increased sympathoadrenal activity by the release of catecholamines. This causes abnormal inotropic and chronotropic effects which increases myocardial oxygen consumption. The indirect vascular effects of enhanced sympathoadrenal activity depends on the predominance of the α-adrenergic (norepinephrine) or β-adrenergic (epinephrine) activity. In patients with heart disease, β-adrenergic cardiac activity and vascular effects prevailed due to the β-adrenergic cardiac activity and vascular effects, results in hypertension, tachycardia, increased cardiac output, and stroke volume and decreased SVR. These were associated with a 2-3 fold increase in the epinephrine and norepinephrine levels[6].

Odberg et al. also observed 11 patients with ASA I status and found that MAP increased by almost 39% after pneumoperitoneum [7]. O'Leary et al. studied the haemodynamic changes occurring in laparoscopic surgery and found that mean arterial pressure increased significantly (p=0.01) after pneumoperitoneum[8]. Joris et al. studied the haemodynamic changes occurring in the laparoscopic cholecystectomies in healthy individuals where they found similar results. In their study they found that the heart rate did not change or changed only slightly[3].

Since Carbon dioxide is a highly diffusible gas that gets absorbed through the peritoneal surface, it causes marked hypercarbia, more so in the absence of compensatory hyperventilation. The values remained in the normal physiological range as the EtCO2 was not allowed to exceed 45 mm Hg (as can be seen from Table 5) by adjusting the tidal volume and respiratory rate as needed during the procedure.

PaCO2 values noted at pre insufflation and post desufflation corresponded well with EtCO2 values. A significant increase was noted at post desufflation, which might have occurred due to the systemic absorption of CO2 through peritoneal vessels.

It was seen in our study that the pH changed from normal to mild acidosis. The PaCO2 increased from baseline to higher normal values while the bicarbonates did not change significantly which suggests that the acidosis caused was due to respiratory cause. It is possible that the acidosis developed from CO2 pneumoperitoneum might be related to CO2 absorption from the peritoneal cavity rather than the secondary ventilator effects because of the increased intra-abdominal pressure [4, 9]. The CO2 that is absorbed from the peritoneal surfaces gets partly excreted and partly stored which results in increases in PaCO2. The CO2 insufflation further decreases the FRC due to the elevation of the diaphragm[6]. This also causes an increase in the V/Q mismatch and alveolar dead space[10].

Makwana et al. studied the changes in laparoscopic surgery and observed that the pH decreased from 7.43 ± 0.01 to 7.34 ± 0.01 that was statistically significant[11]. As studied by Gupta S et al. they found that after creating pneumoperitoneum, the pH decreased from 7.4156 to 7.400 ($p>0.05$), which was clinically as well as statistically non-significant[5]. In both the groups, the PaCO2 values increased after the pneumoperitoneum. During our study, the EtCO2 was not allowed to exceed 45 mm Hg, which might have kept the rise in PaCO2 to an acceptable range which was well within normal values. Hirvonen et al. studied the arterial blood gas changes during laparoscopic hysterectomy in 20 patients. They also noted that partial pressure of arterial CO2 (PaCO2) increased after CO2 insufflation. They found that there was mild metabolic acidosis during laparoscopic surgery but after the laparoscopic procedure, there was predominantly respiratory acidosis[12].

Another comparative study conducted by Gupta Set al. on pre-insufflation versus post insufflation arterial blood gas analysis in laparoscopic surgeries concluded that there occurred non-significant hemodynamic changes, except the rise in diastolic blood pressure which changed significantly. There were non-significant changes in PaCO2 and HCO3, but significant change in pH, which was within the normal physiological range[5]. Our findings are similar to a study conducted by Kwak et al. on acid-base alterations during laparoscopic abdominal surgery where they compared laparoscopy with laparotomy that showed carbon dioxide insufflation during laparoscopic surgery resulted in acid-base imbalance. The decrease in pH during the pneumoperitoneum is caused by the increase in PaCO2, which promptly returned to normal value after desufflation. On the other hand, the decrease in pH after laparotomy was affected by the metabolic factors which persisted an hour after surgery [10].

The corresponding changes were seen in the HCO3 values in both groups. The mean HCO3 noted at Post desufflation in Group N was 21.87, slightly below normal value showing a metabolic component in an attempt to compensate for the respiratory acidosis during pneumoperitoneum. This metabolic component might be due to the splanchnic hypoperfusion due to the increased intra-abdominal pressure [4]. With the above observations we can see that there is respiratory acidosis and a metabolic component as well. This suggests that there are chances of respiratory as well as mixed acidosis which can be even more in the hypertensive patients[13]. This can be probably since the patient cannot generate the respiratory activity efficaciously after general anaesthesia because of the residual effects of anaesthetics and analgesics which makes it difficult to compensate for these changes[4].

The PaO2 and SaO2 values corresponded well with the FiO2 being used which might be because of the mechanical ventilation and were comparable.

V. Conclusion:

An attempt has been made to study the effects of CO2 insufflation on haemodyanamics and arterial blood gas changes in hypertensive patients undergoing laparoscopic surgeries. The conclusions drawn from our study were: Pneumoperitoneum used during laparoscopic surgeries cause sympathetic activation leading to alteration in haemodynamics. Though the changes in the haemodynamics are more pronounced in hypertensive patients than in normotensive patients, laparoscopic surgeries remain the preferred modality of the procedure. With more haemodynamic changes in hypertensive patients, care has to be taken in them considering the haemodynamics in them and also in patients with higher grades of ASA physical status. Laparoscopic procedures with Carbon Dioxide (CO2) pneumoperitoneum are associated with increased risks of hypercapnia and acidosis due mainly to the increase in the intra-abdominal pressure and CO2 absorption mostly through the peritoneum. It may be possible that acidosis which develops from the CO2 pneumoperitoneum may be related to its absorption from the peritoneal cavity rather than to the secondary ventilatory effects due to increased intra-abdominal pressures. This should be considered in all the major laparoscopic cases but needs further evaluation on a larger sample size.

Tables:

1) Comparison of Mean post-insufflation Systolic Blood Pressure (SBP) in two groups at various time interval.

Time Interval	Group H			Group N			p-value
	Mean	SD	p-value	Mean	SD	p-value	
SBPCO0	112.37	11.24	-	108.23	5.95	-	0.080,**NS**
SBPCO2	128.30	9.96	0.0001,**S**	115.03	8.84	0.0001,**S**	0.0001,**S**
SBPCO4	130.43	9.74	0.0001,**S**	119.47	10.12	0.0001,**S**	0.0001,**S**
SBPCO6	134.57	10.31	0.0001,**S**	122.93	8.20	0.0001,**S**	0.0001,**S**
SBPCO8	137.73	13.96	0.0001,**S**	124.90	7.50	0.0001,**S**	0.0001,**S**
SBPCO10	138.07	15.63	0.0001,**S**	126.80	7.49	0.0001,**S**	0.001,**S**
SBPCO12	136.83	15.27	0.0001,**S**	126.60	7.88	0.0001,**S**	0.002,**S**
SBPCO14	135.70	15.38	0.0001,**S**	126.27	8.84	0.0001,**S**	0.005,**S**
SBPCO16	133.40	14.85	0.0001,**S**	125.00	8.85	0.0001,**S**	0.010,**S**
SBPCO18	129.90	14.09	0.0001,**S**	121.97	10.01	0.0001,**S**	0.015,**S**
SBPCO20	129.93	13.57	0.0001,**S**	121.07	21.79	0.002,**S**	0.064,**NS**
SBPCO22	128.53	13.14	0.0001,**S**	118.57	10.15	0.0001,**S**	0.002,**S**
SBPCO24	127.67	12.36	0.0001,**S**	118.97	11.45	0.0001,**S**	0.006,**S**
SBPCO26	125.37	12.04	0.0001,**S**	117.77	10.19	0.0001,**S**	0.011,**S**
SBPCO28	125.27	11.75	0.0001,**S**	116.90	9.65	0.0001,**S**	0.004,**S**
SBPCO30	123.80	11.66	0.0001,**S**	116.93	10.39	0.0001,**S**	0.019,**S**

2) Comparison of Mean post-insufflation Diastolic Blood Pressure (DBP) in two groups at various time interval

Time Interval	Group H			Group N			p-value
	Mean	SD	p-value	Mean	SD	p-value	
DBPCO0	71.90	7.80	-	67.60	5.77	-	0.018,**S**
DBPCO2	80.50	7.76	0.0001,**S**	73.80	8.94	0.0001,**S**	0.003,**S**
DBPCO4	87.20	7.98	0.0001,**S**	77.13	8.54	0.0001,**S**	0.0001,**S**
DBPCO6	87.80	8.73	0.0001,**S**	79.87	8.27	0.0001,**S**	0.001,**S**

DBPCO8	89.87	9.88	0.0001,**S**	80.63	6.83	0.0001,**S**	0.0001,**S**
DBPCO10	86.97	10.72	0.0001,**S**	81.20	6.88	0.0001,**S**	0.016,**S**
DBPCO12	86.63	10.98	0.0001,**S**	80.83	7.08	0.0001,**S**	0.018,**S**
DBPCO14	86.00	11.27	0.0001,**S**	80.80	7.49	0.0001,**S**	0.040,**S**
DBPCO16	85.10	10.65	0.0001,**S**	79.97	7.01	0.0001,**S**	0.031,**S**
DBPCO18	85.10	9.91	0.0001,**S**	79.53	7.60	0.0001,**S**	0.018,**S**
DBPCO20	84.10	10.23	0.0001,**S**	79.93	8.25	0.0001,**S**	0.088,**NS**
DBPCO22	82.97	9.58	0.0001,**S**	79.23	8.10	0.0001,**S**	0.109,**NS**
DBPCO24	82.43	9.36	0.0001,**S**	78.97	8.67	0.0001,**S**	0.142,**NS**
DBPCO26	81.80	9.20	0.0001,**S**	78.57	8.94	0.0001,**S**	0.173,**NS**
DBPCO28	80.67	8.90	0.0001,**S**	78.47	8.49	0.0001,**S**	0.331,**NS**
DBPCO30	81.27	8.49	0.0001,**S**	77.83	8.40	0.0001,**S**	0.121,**NS**

3) Comparison of Mean post-insufflation Mean Arterial Blood Pressure (MBP) in two groups at various time interval.

Time Interval	Group H			Group N			p-value
	Mean	SD	p-value	Mean	SD	p-value	
MBPCO0	84.53	8.37	-	80.37	5.26		0.025,**S**
MBPCO2	95.57	7.77	0.0001,**S**	86.63	8.41	0.0001,**S**	0.0001,**S**
MBPCO4	100.53	7.86	0.0001,**S**	90.30	8.63	0.0001,**S**	0.0001,**S**
MBPCO6	102.33	8.67	0.0001,**S**	93.23	7.75	0.0001,**S**	0.0001,**S**
MBPCO8	104.73	10.46	0.0001,**S**	94.37	6.53	0.0001,**S**	0.0001,**S**
MBPCO10	102.93	11.67	0.0001,**S**	95.43	6.62	0.0001,**S**	0.003,**S**
MBPCO12	102.43	11.79	0.0001,**S**	95.00	6.94	0.0001,**S**	0.004,**S**
MBPCO14	101.57	11.85	0.0001,**S**	94.97	7.34	0.0001,**S**	0.012,**S**
MBPCO16	100.30	11.33	0.0001,**S**	93.93	7.16	0.0001,**S**	0.012,**S**
MBPCO18	98.93	10.76	0.0001,**S**	92.77	7.81	0.0001,**S**	0.014,**S**
MBPCO20	98.47	10.79	0.0001,**S**	92.67	11.07	0.0001,**S**	0.044,**S**
MBPCO22	97.27	10.33	0.0001,**S**	91.43	8.27	0.0001,**S**	0.019,**S**
MBPCO24	96.63	9.83	0.0001,**S**	91.37	9.00	0.0001,**S**	0.035,**S**
MBPCO26	95.43	9.64	0.0001,**S**	90.67	8.75	0.0001,**S**	0.050,**NS**
MBPCO28	94.57	9.27	0.0001,**S**	90.27	8.33	0.0001,**S**	0.064,**NS**
MBPCO30	94.63	8.90	0.0001,**S**	89.93	8.29	0.0001,**S**	0.039,**S**

4) Comparison of heart rate

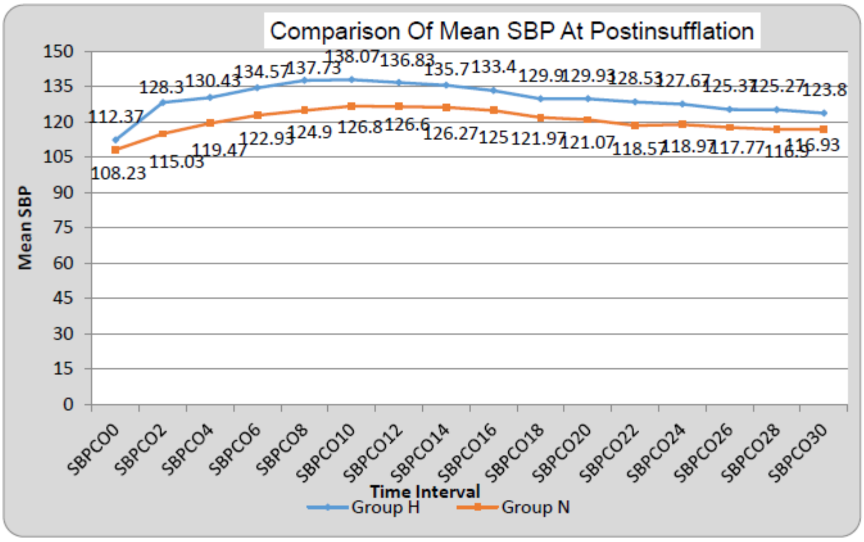
Time Interval	Group H			Group N			p-value
	Mean	SD	p-value	Mean	SD	p-value	
HRCO0	81.27	14.45	-	86.27	14.20	-	0.182,**NS**
HRCO2	87.17	14.47	0.0001,**S**	91.63	15.42	0.0001,**S**	0.252,**NS**
HRCO4	92.23	13.71	0.0001,**S**	94.73	15.12	0.0001,**S**	0.505,**NS**
HRCO6	96.07	14.08	0.0001,**S**	96.87	14.51	0.0001,**S**	0.829,**NS**
HRCO8	99.50	15.00	0.0001,**S**	98.23	12.81	0.0001,**S**	0.726,**NS**
HRCO10	99.60	15.09	0.0001,**S**	98.60	12.73	0.0001,**S**	0.782,**NS**
HRCO12	99.00	14.85	0.0001,**S**	98.93	14.28	0.0001,**S**	0.986,**NS**
HRCO14	98.00	14.27	0.0001,**S**	99.20	14.21	0.0001,**S**	0.745,**NS**
HRCO16	97.57	13.70	0.0001,**S**	99.37	15.05	0.0001,**S**	0.630,**NS**
HRCO18	96.43	13.53	0.0001,**S**	98.60	14.86	0.0001,**S**	0.557,**NS**
HRCO20	95.83	13.65	0.0001,**S**	97.90	14.32	0.0001,**S**	0.569,**NS**
HRCO22	94.90	12.35	0.0001,**S**	97.13	14.26	0.0001,**S**	0.519,**NS**
HRCO24	94.30	12.27	0.0001,**S**	97.30	14.84	0.0001,**S**	0.397,**NS**
HRCO26	93.17	11.87	0.0001,**S**	96.80	14.39	0.0001,**S**	0.291,**NS**
HRCO28	92.83	11.19	0.0001,**S**	96.47	14.16	0.0001,**S**	0.275,**NS**
HRCO30	92.17	11.53	0.0001,**S**	96.03	14.16	0.0001,**S**	0.251,**NS**

5) Comparison of Mean Post-insufflation End Tidal Carbon Dioxide ($EtCO_2$) in two groups and at post-desufflation ($ETCO_2PD$)

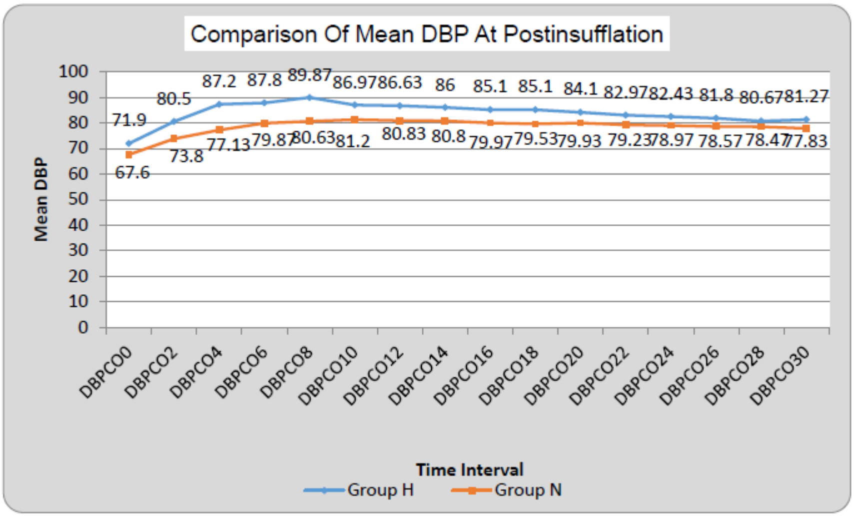
Time Interval	Group H			Group N			p-value
	Mean	SD	p-value	Mean	SD	p-value	
ETCO0	31.23	1.68	-	31.27	1.20	-	0.930,**NS**
ETCO5	34.83	2.13	0.0001,**S**	34.47	1.46	0.0001,**S**	0.440,**NS**
ETCO10	37.67	1.99	0.0001,**S**	36.67	1.52	0.0001,**S**	0.033,**S**
ETCO15	40.30	1.56	0.0001,**S**	39.30	1.39	0.0001,**S**	0.011,**S**
ETCO20	42.07	1.17	0.0001,**S**	40.77	1.17	0.0001,**S**	0.0001,**S**
ETCO25	43.03	1.00	0.0001,**S**	42.53	1.20	0.0001,**S**	0.084,**NS**
ETCO30	44.43	0.97	0.0001,**S**	43.77	0.97	0.0001,**S**	0.010,**S**
ETCO2PD	42.03	1.00	0.0001,**S**	41.77	0.97	0.0001,**S**	0.299,**NS**

Graphs :

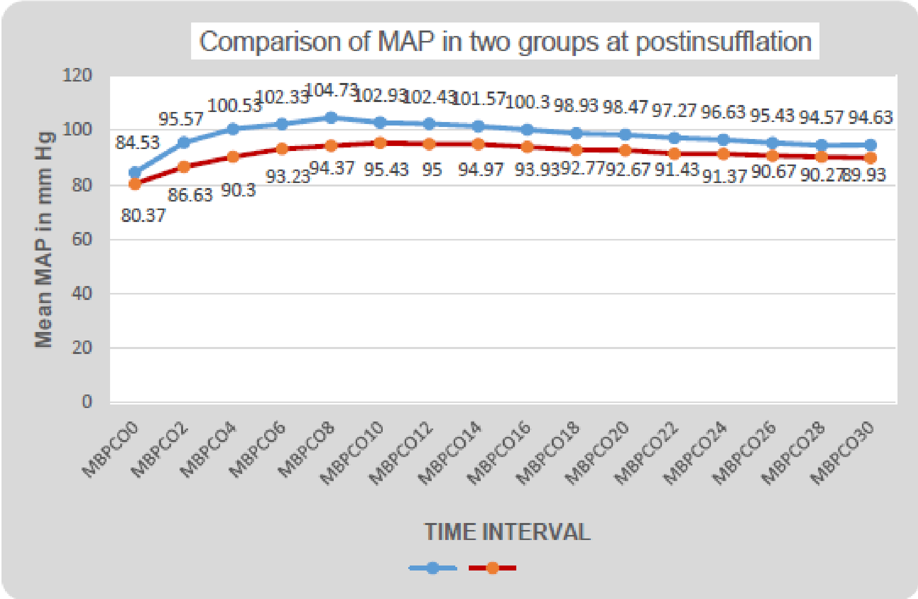
1) Comparison of Mean post-insufflation Systolic Blood Pressure (SBP) in two groups at various time interval



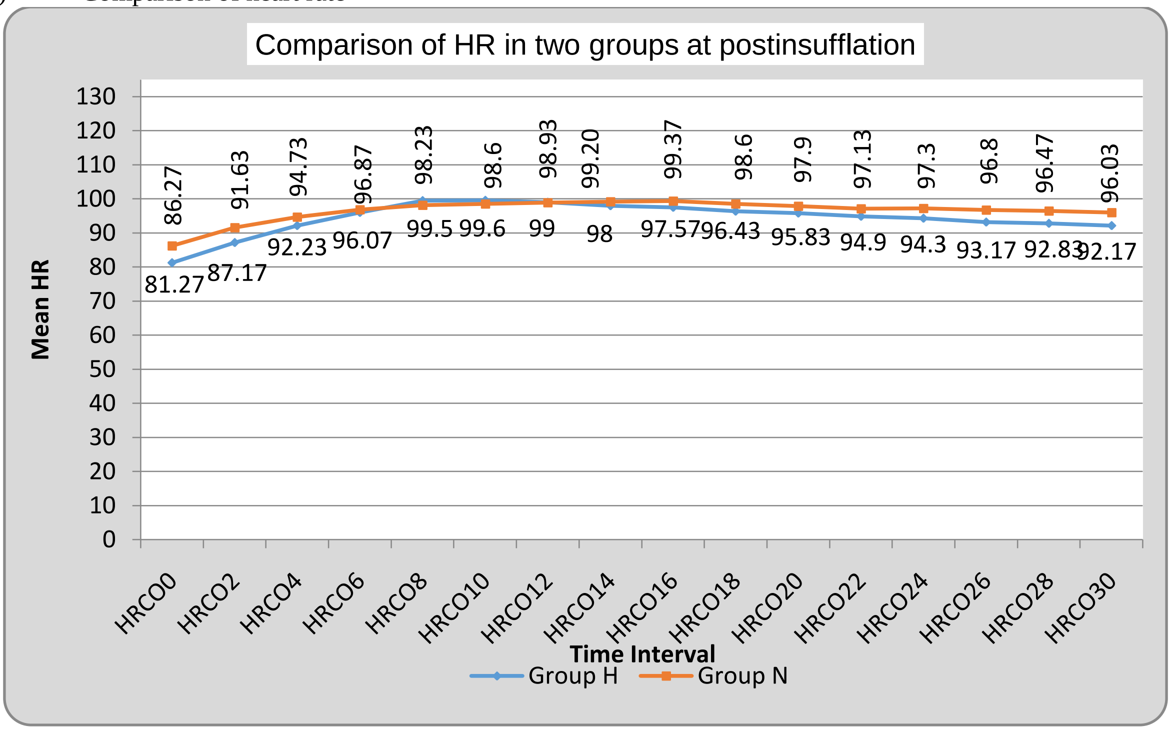
2) Comparison of Mean post-insufflation Diastolic Blood Pressure (DBP) in two groups at various time interval



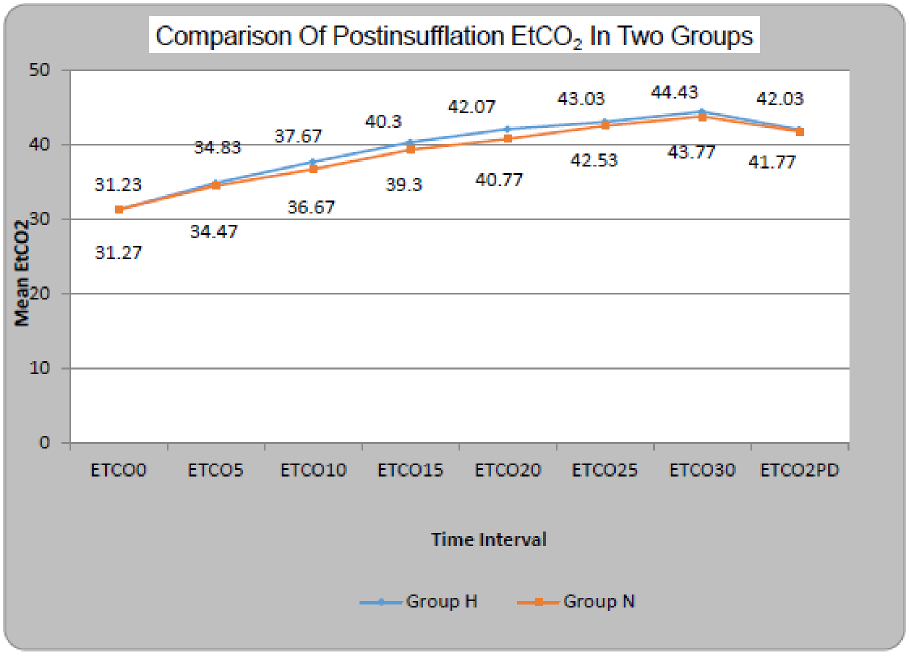
3) Comparison of Mean post-insufflation Mean Arterial Blood Pressure (MBP) in two groups at various time interval.



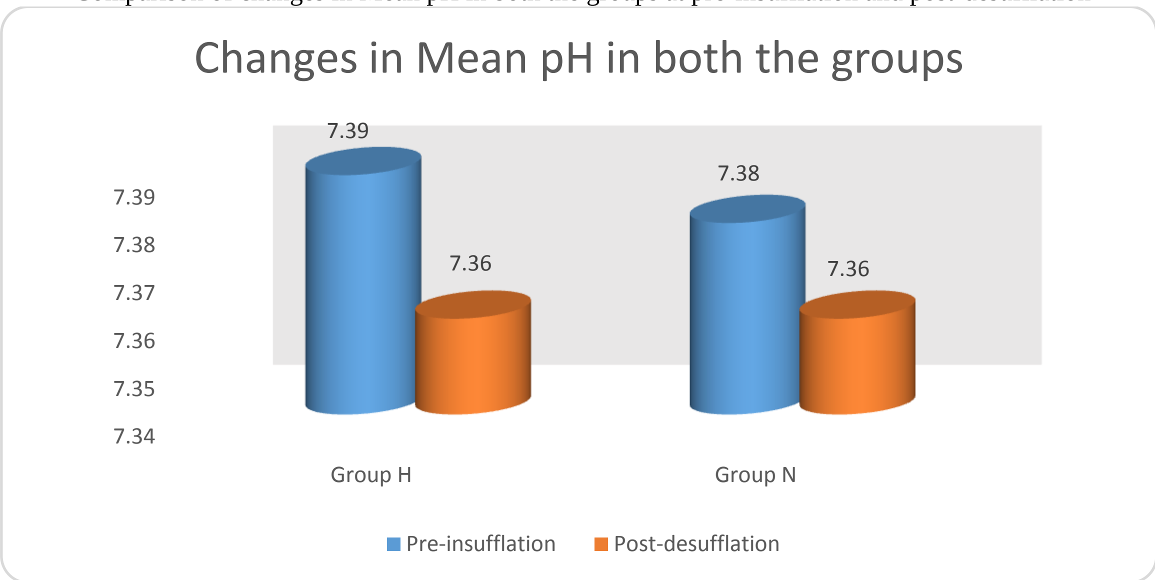
4) Comparison of heart rate



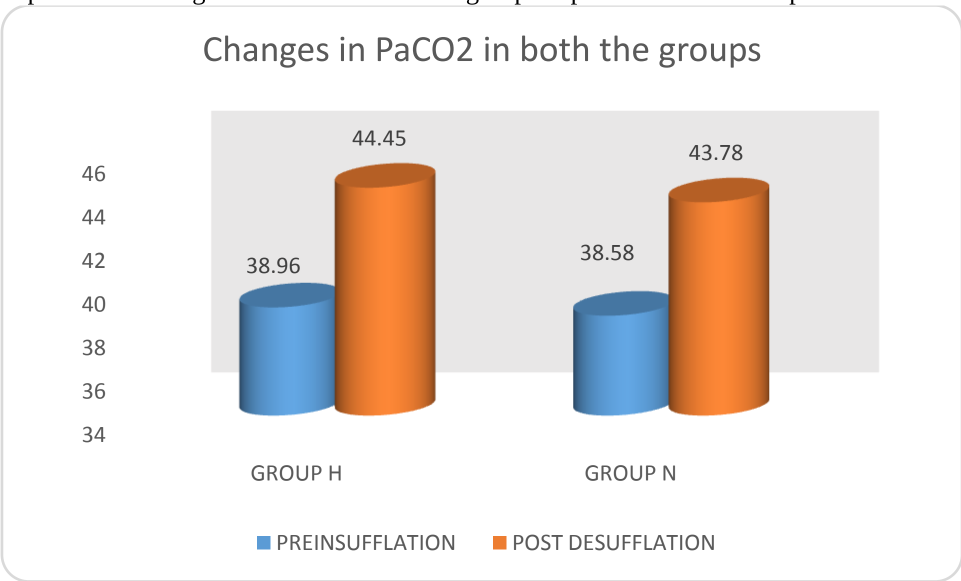
5) Comparison of Mean Post-insufflation End Tidal Carbon Dioxide (EtCO2) in two groups and at post-desufflation (ETCO2PD)



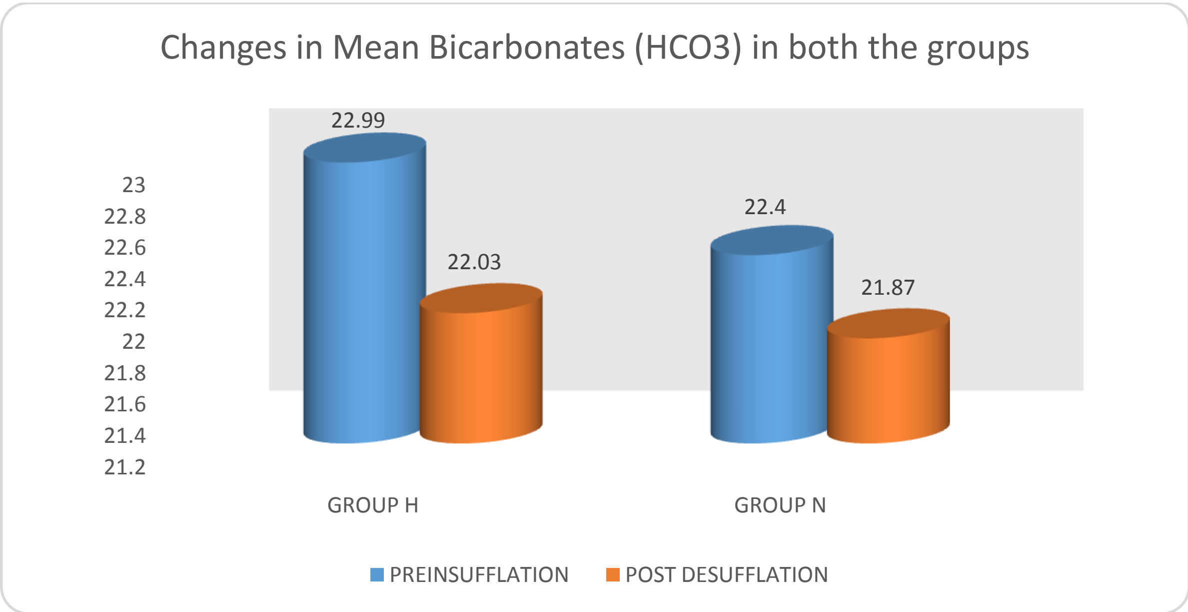
6) Comparison of changes in Mean pH in both the groups at pre-insufflation and post-desufflation



7) Comparison of changes in $PaCO_2$ in both the groups at pre-insufflation and post-desufflation



8) Comparison of changes in Mean Bicarbonates (HCO_3) in both the groups at pre-insufflation and post-desufflation.



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