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Research Paper

Effect of anaesthetic use of nitrous oxide on carbon dioxide elimination in laparoscopic surgery

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ABSTRACT:-
Background:- Inefficient elimination of CO_2 during laparascopy leads to hypercapnia even in the absence of abnormal $EtCO_2$. When N_2O is used in anaesthetic management large volume of N_2O are released during the first 5-10 min. of recovery. This outpouring of large volumes of N_2O dilutes alveolar CO_2 and may decrease respiratory drive and ventilation. This study was designed to observe changes in CO_2 elimination pattern with N_2O.

Materials and Methods:-60 consented patients of ASA grade 1 and 2 for elective laparoscopic surgeries were enrolled. They were divided into group 1 without N_2O in anesthetic mixture and group 2 with N_2O. Vital parameters and $EtCO_2$ monitoring was done at the time of desufflation for 15 min. R-R, Tidal volume and Machine was kept constant.

Results:-In group 1 significant increase of $EtCO_2$ was seen on 4th min returns to preinsufflation values at 8th min. In group 2 increase was seen at 8th min, returned to pre insufflations value at 14th min. significant increase of heart rate, systolic and diastolic BP was seen corresponding to the $EtCO_2$ levels.

Discussion:- Flink BR reported, outpouring of large volume of nitrous can produce diffusion anoxia, may cause hypoxic by displacing oxygen and diluting alveolar CO_2.

Conclusions:- Avoidance of N_2O in anaesthetic mixture results in faster CO_2 elimination in laparoscopic surgery and prevents hycpercarbia and its related heamodynamic changes. Because hypercapnia can exist even in absence of abnormal $EtCO_2$, $PaCO_2$ values in post OP period can give clear picture of severity of hypercapnia with N_2O.

KEYWORDS:-CO_2, N_2O, Laparoscopy, CO_2 elimination.

I. INTRODUCTION

Laparoscopic surgeries requires only small limited incisions very short hospital stay and faster recovery time, thereby allowing patients to return to routine activities much sooner.[1]Carbon dioxide is ready availability, low cost and proven efficacy, carbon dioxide (CO_2) has evolved as the insufflations gas of choice for laparoscopic surgery.[1]

CO_2diffusibility is high, absorption of large quantities of CO_2 into blood and the subsequent marked increase in partial pressure of carbon dioxide ($PaCO_2$) would be expected to occur.During deflation, CO_2 that is accumulated in collapsed peritoneal capillary vessels reaches systemic circulation leading to transient increase in $PaCO_2$ and end tidal carbon dioxide ($EtCO_2$).[2] During recovery from anaesthesia, washout of high concentration of nitrous oxide can lower alveolar concentration of oxygen and carbon dioxide, a phenomenon called diffusion hypoxia. The resulting alveolar hypoxia can cause hypoxemia also, alveolar hypocarbia can depress respiratory drive which may exacerbate hypoxemia.[3]Reduce washout of CO_2 leads to hypercapnea and acidosis, but $EtCO_2$ levels may remain normal. The resulting rise in $PaCO_2$ is unpredictable particularly in

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patients with severe pulmonary disease and therefore $EtCO_2$ may not be a reliable index of $PaCO_2$ during CO_2 insufflation. The actual disparity between $EtCO_2$ and $PaCO_2$ values can be confirmed by Arterial Blood Gas analysis. If addition of Nitrous oxide further delays elimination of CO_2, further rise in $PaCO_2$ can lead to worsening of clinical condition.

In the present study we are going to observe whether use of nitrous oxide in management of anaesthesia has effect on elimination of CO_2. By monitoring $EtCO_2$ trend throughout the various phases of laparoscopic surgery, evaluation of effect of nitrous oxide on CO_2 elimination will be done.

II. MATERIALS AND METHOD

This study was conducted as prospective randomized trial study. Total of 60 patients posted for laparoscopic surgeries were included in the study and were divided into 2 groups having 30 patients each. Written informed consent were obtained standard aneasthetic technique for general aneasthesia was used.

In group I maintenance is with 100% O_2, isoflurane (1.5MAC) and epidural top ups where epidural catheter were put.In group II maintenance was with O_2 (60%) + N_2O(40%) + isoflurane (1.5 MAC).

Pneumoperitoneum was created by CO_2 insufflation, intra abdominal pressure (IAP) was maintained less than 15 mm of Hg.Keeping the respiratory rate in the range of 12-16 per minute, hemodynamic monitoring along with $EtCO_2$ were monitored continuously and recorded at baseline, every 10 minutes for the first 60 minutes, later every 15 minutes up to time of defflation and every min till it returns to normal after deflation.

Throughout the procedure we maintained the IAP< 15 mm of Hg. Same machine were used for all the cases, sodalime was checked for any case prior to the beginning of ventilation.In patient posted for laparoscopic cholecystectomy epidural catheter was put prior to induction. Top up were given timely in such a way that it did not overlapped with deflation time.

III. RESULT

It is an observational study and comprised of 60 patients posted for laparoscopic surgeries over a span of 2 years. Analysis of the data was carried out using statistical test of significance unpaired t-test was used for continuous variables and chi-square test was used for non continuous variables. All the cases that are converted to open surgeries were not considered in the study. All the patients in my study are between age 20-50yrs ASA I & II and female patients are more than the male patients.

Minute	Group-1 (Without N_2O)		Group-2 (With N_2O)		p-Value
	Mean	SD	Mean	SD	
1	29.03	2.53	29.5	3.87	0.58362
2	30.23	3.69	32.43	4.26	0.03481
3	32.9	4.5	34.4	3.68	0.15129
4	35.6	4.75	37.87	2.49	0.02138
5	35.87	3.77	38.23	1.71	0.00327
6	35.27	4.12	38.8	2.09	0.00014
7	33.97	3.42	40	1.43	<0.001
8	32.63	3.18	35.87	2.09	<0.001
9	31.64	3.15	32.19	2.93	0.54717
10	29.83	2.76	30.81	1.69	0.17191
11	29.15	2.67	30.19	2.95	0.33166
12	30	1.63	29.73	2.61	0.81545
13	29	1.41	29.29	3.09	0.86117

Table 1: $EtCO_2$ Trend after desufflation

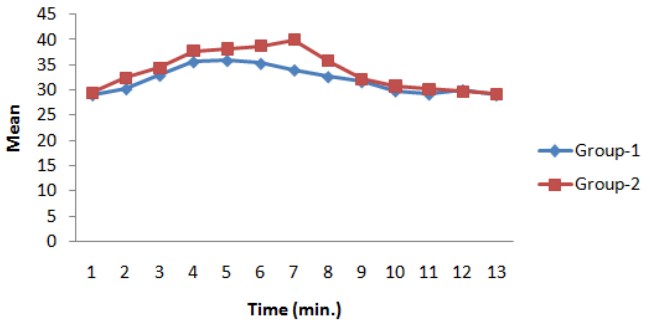

Table 1 presents the results $EtCO_2$ monitoring after desufflation in group 1(without nitrous oxide) significant increase in $EtCO_2$ was seen at 4^{th} min (35.6±4.75) and remain high for 5^{th} (35.87±3.77), 6^{th} (35.27±4.21) 7^{th} min (33.97±3.42) reached to pre-insufflation value at 10^{th} min (29.83±2.67) as compared to group 2 (with nitrous oxide) significant increase is seen at 7^{th} min (40±1.43) remain high for 8^{th} min (35.87±2.09), 9^{th} min (32.19±2.93) reached to pre-insufflation values at 14^{th} min (29.5±1.91). By using unpaired t-test of significance, having p values were <0.02138 and<0.001 (highly significant) at 4^{th} min. and 7^{th} min. The difference in $EtCO_2$ in group 1 and group 2 at 4^{th} min. and 7^{th} min. was highly significant.

Minute	Group-1 (Without N_2O) Mean	SD	Group-2 (With N_2O) Mean	SD	p-Value
1	73.4	9.03	69.38	5.37	0.04323
2	73.8	9.02	71.03	6.03	0.17039
3	76.97	9.88	73.23	7.03	0.09843
4	78.37	9.45	77.53	8.75	0.72494
5	79.4	9.35	84.33	6.43	0.01868
6	80.4	10.69	85.93	7.04	0.02167
7	79.53	10.49	88.07	7.64	0.00067
8	78.03	8.45	86.93	7.88	0.0001
9	75.35	8.05	85.3	6.55	<0.001
10	74.24	7.54	84.03	6.6	<0.001
11	71.1	7.03	80.32	6.93	<0.001
12	72.1	6.94	78.52	6.74	0.02176
13	72.5	6.99	76.42	6.69	0.18769
14	72	6.93	74.67	7.38	0.43937
15	72.5	7.42	75.22	7.15	0.50021

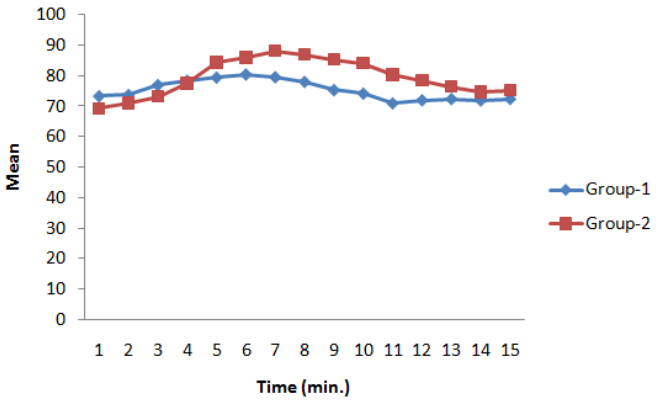

Table 2: Pulse rate after desufflation

Significant rise of heart rate at 5th min (79.4±9.35) comes back to baseline at 10th min (74.24±7.54) whereas in group 2 significant increase of heart rate is seen at 5th min (84.33±6.43) comes back to baseline at 15th min (75.22±7.15), showing increase of heart rate with the corresponding rise of $EtCO_2$.

On comparing the heart rate between the two groups at different time intervals, the difference in heart rate was statistically highly significant at 5th minutes intervals (p value <0.01868).

Minute	Group-1 (Without N_2O) Mean	SD	Group-2 (With N_2O) Mean	SD	p-Value
1	105.87	15.16	104.4	12.04	0.67898
2	101.8	20.96	104.8	11.91	0.498035
3	118.43	15.83	122.47	13.29	0.29199
4	124.33	14.83	128.57	13.35	0.25192
5	127.4	16.3	134.33	10.74	0.05808
6	129.37	15.56	137.33	9.54	0.02083
7	126.93	15.07	139.5	10.83	0.00045
8	121.63	14.42	139.97	11.9	<0.001
9	118.63	12.49	136.23	9.85	<0.001
10	114.67	12.67	130.17	23.17	0.00231
11	114.28	11.78	133.07	6.55	<0.001
12	110.71	16.52	129.3	5.93	0.02445
13	105	7.07	127.41	7.1	0.12032
14	105	7.07	124.96	6.74	0.13645

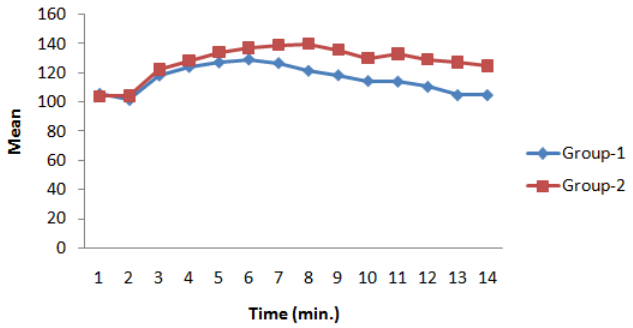

Table 3: Systolic BP trend after desufflation

Significant increase of systolic BP is seen at 6th min (129.37±15.56) reached to pre in suffflation value at 14th min (105 ±7.07) However in group 2 patients significant increase of systolic BP is seen at 6th min (137.33±9.54) and remains high for 7th 8th 9th 10th min and reached to baseline value at 15th min (126.71±5.12).

On comparing the systolic BP between the group at different time interval, the difference in systolic BP was statistically highly significant at 6th min. interval (p value <0.02083).

Minute	Group-1 (Without N_2O) Mean	SD	Group-2 (With N_2O) Mean	SD	p-Value
1	68.47	7.93	67.2	7.32	0.52533
2	67.67	7.37	67.67	7.32	0.999
3	77.47	10.92	80.33	9.03	0.27164
4	81.13	10.35	83.9	9.2	0.27412
5	83.33	10.48	86.77	7.99	0.15546
6	82.63	10.51	89.03	6.51	0.0058
7	81.23	9.93	89.4	6.94	0.00043
8	78.5	8.9	90.47	6.78	<0.001
9	75.5	8.31	86.9	7.66	<0.001
10	72.6	8.39	84.2	8.66	<0.001
11	73.56	9.64	82.53	8.08	0.00235
12	74.57	11.93	79.93	7.26	0.29105
13	65	7.07	78.24	7.17	0.21066
14	65	7.07	77.81	6.8	0.21792

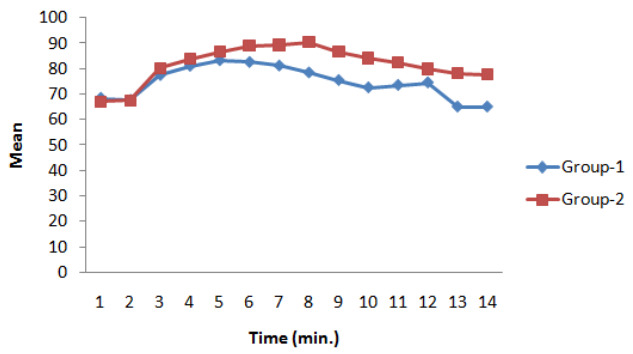

Table 4: Diastolic BP trend after desufflation

Significant increase of diastolic BP rise is seen at 6^{th} min (82.63±10.51) reached to baseline value at 13^{th} min (65±7.07) in group 1 patients. However in group 2 patients significant increase of diastolic BP is seen at 6^{th} min (89.03±6.51) remains high for 7^{th} 8^{th} 9^{th} 10^{th} min. reached to baseline value at 14^{th} min (77.81±6.8).

On comparing the diastolic BP between the group at different time interval, the difference in diastolic BP was statistically highly significant at 6^{th} min. interval (p value <0.0058).

IV. DISCUSSION

The major factor which causes path physiological changes, principally in heamodynamics and respiration during laparoscopic surgery is pneumoperitoneum by CO_2 insufflation. Hypercapnia may result from CO_2 wash out disproportionate to increased CO_2 absorption. Deaths during gynaecologic laparoscopic procedures have been attributed to hypoventilation and CO_2 retention. Apart from this respiratory changes because of raised IAP and decreased diaphramagtic movements and ventilation perfusion mismatched with increased dead space leads to decreased CO_2 elimination from the lungs.[4]

Rackaw H et. al in their study that stated that" the movement of N_2O through the alveolar space washes out and dilutes alveolar CO_2, thereby lowering $PaCO_2$. A decrease in ventilator drive with hypoventilation would follow". Excretion of carbon dioxide is dependent on the rates of change of alveolar and mixed venous CO_2, which in turned is governed by cardiac output, alveolar ventilation and the respiratory quotient.[5]

All cases were done with isoflurane as inhalational agents to maintain the uniformity of recovery, but actual uniform maintenance of end tidal conc. of isoflurane is not possible without respiratory gas monitoring. Maximum care was taken to maintain adequate plane of anaesthesia without overuse of isoflurane. Variation of uptake, distribution and elimination of inhalational agent can affect the haemodynamic monitoring to some extent but it does not have effect on $EtCO_2$ changes. So there is less possibility of changes in $EtCO_2$ trend due to this slight variation of inhalational agents.

Mild hypercapnia may not produce significant heamodynamic effects whereas moderate to severe hypercapnia of 50-70 mmHg may decrease cardiac output, stoke volume, systemic blood pressure, and serum pH. Endogenous catecholamine are also released, resulting in increase in heart rate, systemic blood pressure and pulmonary artery pressure.[6] In our study in group 1 maximum increase in heart rate, systolic and diastolic pressure was seen at 5^{th} and 6^{th} min. which corresponds to the significant rise of $EtCO_2$ levels. And in group 2 maximum increases in systolic, diastolic blood pressure and heart rate was seen at 5^{th} and 6^{th} min. and stays high for longer time in compare to group 1and also corresponds to the significant rise of $EtCO_2$ levels.

There were no major alterations in electrocardiogram. Electrocardiogram (ECG) showed sinus rhythm in both the groups throughout the procedure except tachycardia. We monitor the $EtCO_2$ readings every one min after desufflation of CO_2 and we found out that the significant rise of $EtCO_2$ at desufflation in group 1 is at 4^{th} min. (35.6±4.75) and reached to pre-insuflation values at 10^{th} min. (29.83±2.76). And in group 2 significant rise of $EtCO_2$ is seen at 7^{th} min (40±1.43) and reached to pre-insuflation values at 14^{th} min. (29.5±1.91).

The most important factor for the increase in $EtCO_2$ seen during laparoscopic surgery is absorption of CO_2 from peritoneum. Apart from this, respiratory changes of raised IAP and decreased diaphragmatic movements and ventilation perfusion mismatched with increased dead space lead to decreased CO_2 elimination from the lungs. This increased arterial alveolar CO_2 gradient in circulation.[2]

Christine E. Mullet et. al in her studies found that "after cessation of CO_2 insufflation, the time required to return to preinsufflation values was about 10 min. in patients having intraperitoneal insufflation. In patients having pelviscopy, values of $EtCO_2$ and $PaCO_2$ measured 45min after cessation of CO_2 insufflation was still significantly higher than the preinsufflation values.[7]

Maria F. Marfin-Cancho et. al mentioned in their article that CO_2 spreads in to the blood and it's eliminated during the expiration. But it's also accumulated in the fat tissue and muscle, which explains the high carbon dioxide expirated rate during some minutes after the end of insufflations.[8]

Hypercapnia may be associated with pulmonary atelactesis, reduced functional residual capacity, increased airway pressure, and increased CO_2 absorption from the peritoneum and decreased diaphragmatic excursion caused either by positional change or increased abdominal pressure. The accompanying cardiovascular changes include hypertension related to augmentation of preload or increased systemic and pulmonary resistance,

hypotension caused by impaired venous return from the inferior vena cava compression, arrhythmias due to stress, high plasma catecholamine levels, a vagal mediated reflex from the peritoneal stretch or increased sympathetic tone. These respiratory and circulatory pertuberations can be tolerated by physiologic adjustments in healthy individuals but may pose potential problems in patients with impaired cardiopulmonary reserve.[6]

The principal effects of pre existing lung disease are to exacerbate the hypercarbia and acisosis associated with the CO_2 pneumoperitoneum. Although the increased abdominal pressure contributes to these alterations, it is probably the CO_2 gas itself that is the main cause of the dramatic hypercarbia and acidosis noted in these patients. Chronic obstructive pulmonary disease (COPD) patients with chronic carbon dioxide retention, at baseline, have nearly or fully saturated their body's CO_2 storage sites, such as the bone and skeletal muscle, and have a limited ability to accomoadate additional CO_2 during a laparoscopic procedure. These individuals often manifest an exaggerated hypercarbia when compared to patient with normal lung function and require more time to eliminate the CO_2 through their lungs after desufflation.[9]

After desufflation, plane of aneasthesia was maintained and $EtCO_2$ monitoring was done till it comes back to normal. Decision of reversal was taken after witnessing normal $EtCO_2$.

V. SUMMARY

Hypercapnia and acidosis are two physiological changes in response to carbon dioxide (CO_2) pneumoperitoneum during laparoscopic surgery. $EtCO_2$ monitoring after desufflations shows that in groups without nitrous oxide, significant increase seen at 4^{th} min reached to pre-insufflation value at 10^{th} min. as compared to group 2 with nitrous oxide, significant increase is seen at 7^{th} min. reached pre-insufflation values at 14^{th} min. This shows that CO_2 elimination is faster in groups without nitrous oxide whereas in groups with nitrous oxide elimination is delayed indicating an increase in $PaCO_2$.Delayed release of stored CO_2 from various tissue compartment after discontinuation of CO_2 pnuemoperitoneum should be kept in mind and resolved with caution .The haemodynamic changes between the two groups shows that in group 1 patients there was significant rise of heart rate at 5^{th} min reached to baseline at 10^{th} min. whereas in group 2 significant increase of heart rate is seen at 5^{th} min. reached to baseline at 15^{th} min., showing increase of heart rate with the corresponding rise of $EtCO_2$ BP reached to normal within 10^{th} min. whereas in group 2 heart rate and BP remain high for 15^{th} min.This study was conducted in healthy young adults. So there is possibility that, these differences in CO_2 elimination can get aggravated in elderly patients and patients with compromised cardio respiratory status.

VI. CONCLUSION

These results highlights the fact that laparoscopy induces significant haemodynamic changes even in healthy patients and create increase in blood CO_2 levels at the time of desufflation leading to increase in systemic vascular resistance and increase in mean arterial pressure and heart rate.

Whereas these cardiovascular changes may not be hazardous in healthy patients, special care and monitoring is mandatory for patients with impaired cardio pulmonary function. In these patients postoperative benefits of laparoscopy should be balanced against intraoperative risk. We conclude that nitrous oxide does interfere in carbon dioxide elimination. This effect can be variable according to cardiopulmonary status of patient and can influence the post-operative haemodynamics and acid-base balance.

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