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Study of End-Tidal Co2 Tension at 1nsufflation And Exsufflation of CO_2 during Laparoscopic Surgery

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Abstract: *This study was conducted to know the changes in the $ETCO_2$ values with different baseline value & to obtain a safe value prior to insufflation so that the normocarbia is maintained throughout the procedure.*
It was conducted on 60 patients divided into 3 groups. The control having the baseline group value of 31 - 35 mmHg & the study groups one with slight hypocarbia 25-30 mmHg being achieved prior to insufflation & the other with the baseline value of 36 - 40 mmHg were compared with the control group.
The study included only ASA I patients & were subjected to any of the following laparoscopic surgeries - laparoscopic cholecystectomy, laparoscopic mesh repair of incisional hernias & laparoscopic gynecological surgeries. Standard anesthesia technique was chosen for all & the similar drugs were given to all of them including premedication, induction, muscle relaxation & the volatile agents. The patients are either put in trendelenberg position or reverse trendelenberg position.
The observation made of the study are, there is significant rise in value of $ETCO_2$ when the initial $ETCO_2$ before insufflation is maintained at 36 - 40 mmHg. $ETCO_2$ can be maintained near to the preinsufflation value when slight hypocarbia is maintained prior to insufflation.
In conclusion this study shows that in healthy person's normocarbia can be maintained by using moderate hyperventilation of about 14% to achieve a baseline value of 25 - 30 mmHg prior to insufflation.

Keywords: *End-Tidal CO_2 tension, Hypercarbia ,Insufflation, Laparoscopy,Pneumoperitoneum*

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I. Introduction

Laparoscopy is a relatively modern procedure that involves the insufflation of the abdomen by a gas so that the endoscope can visualize the infra abdominal contents without being in contact with the viscera or tissue. It started as a gynecological procedure in the mid -fifties and developed into other surgical specialties and targeted more sick patients. With the advantages of shorter hospital stay, more rapid return to normal activities, less pain, small incision and less postoperative ileus compared with traditional open cholecystectomies laparoscopic cholecystectomy started to enjoy ever increasing popularity. Carbon dioxide (CO_2) is the preferred gas for the creation of pneumoperitonium because it is inexpensive, highly soluble, chemically stable, rapidly eliminated, physically inert, suppresses combustion. CO_2 is a normal product of human cellular metabolism and at physiological level less toxic. With this in background one should be aware of the physiological alterations caused by CO_2 insufflation and elevated intraabdominal pressure (IAP).

The insufflation of CO_2, and the position of the patient during the surgery causes elevation in the intra abdominal pressure and undesirable cardiorespiratory effects. During pneumoperitoneum, CO_2 is very rapidly absorbed from the peritoneal cavity into the circulation. The absorption depends upon the diffusibility of the insufflating gas, the surface area of the cavity and the perfusion of the cavity. As diffusibility of CO_2 is high, large quantity of CO_2 is absorbed and thus marked increase in Partial Pressure of arterial Carbon dioxide ($PaCO_2$) is expected to occur. But the limited rise of $PaCO_2$ actually observed can be explained by the impaired local perfusion due to increased IAP. Also excessive CO_2 accumulates in the body to be eliminated during the recovery phase. During pneumoperitoneum, the CO_2 eliminated in the expired gas contains both the metabolic and absorbed CO_2 from the peritoneal cavity. The rate of CO_2 eliminated in the expired gas is not a match for the real rate of metabolic production and absorbed CO_2, from the peritoneal cavity.

Laparoscopy is ideally performed under general anesthesia. The technique may be associated with numerous and diverse potentially serious problems such as subcutaneous emphysema, pneumothorax, pneunomediastinum, hypercarbia and hypoxemia. These changes may produce cardiac dysrhythmias, hypotension and acid base disturbances. It is thus important to know the CO_2 homeostasis in patients undergoing laparoscopic surgeries. There are contradictory reports regarding $ETCO_2$ changes following insufflation. One report has recommended increase in the minute ventilation by 12-16% in order to maintain $ETCO_2$ and thus $PaCO_2$ to preinsufflation level. Some reports have demonstrated increase in $PaCO_2$ despite increase in minute ventilation. And few reports noted no significant changes in $ETCO_2$ though the ventilation was kept constant. These changes were attributed to different baseline values of $ETCO_2$ prior to insufflation or to estimation of increase following insufflation before the maximum level is reached. Keeping the above factors in mind this study has been carried out to evaluate changes in $ETCO_2$ during insufflation and creation and removal of pneumoperitoneum during laparoscopic surgeries.

II. Aims

To study the changes in the End Tidal CO_2 tension during various steps in laparoscopic surgeries and to obtain an ideal baseline $ETCO_2$value so that the normocarbia is maintained throughout the laparoscopic surgery.

III. Objectives

1. To record a baseline value of $ETCO_2$ after the induction of anaesthesia and prior to CO_2, insufflation by monitoring through capnograph.
2. To record the $ETCO_2$ values immediately after insufflation and every 10 min thereafter and immediately after exsufflation.
3. To study whether the alteration in the baseline value results in significant alteration in maximum value of $ETCO_2$ reached during intraperitoneal CO_2 insufflation, throughout the surgery and to study the $ETCO_2$ changes during exsufflation.

IV. Material & Methods

The study was conducted on 60 patients who were undergoing either Laparoscopic upper abdominal surgeries or laparoscopic gynecological surgeries, which involves the intraperitoneal insufflation of CO_2, the duration of which were expected to prolong > 40 mints. Upper abdominal surgeries like cholecystectomy and mesh repair of incisional hernias & the gynecological surgeries were laparoscopic assisted vaginal hysterectomy or ovarian cyst removal were included. All the patients were in ASA 1 without any systemic illness. Written informed consent was taken from all. On arrival in OT patients were allocated to either group A or B or C. Group B is taken as a Control group and the baseline $ETCO_2$ after the induction of anesthesia and before the CO_2 insufflation is kept between 31-35 mmHg. In group A and C, which are the study groups, the baseline $ETCO_2$ value is maintained between 25 - 30 mmHg and 36-40 mmHg respectively. In group A there were 15 patients for Laparoscopic cholecystectomy and 2 for mesh repair of incisional hernias & 3 for laparoscopic gynecological surgeries which included 2 LAVE & 1 ovarian cyst removal. In group B 13 were for Laparoscopic cholecystectomy & 6 for LAVH. In group C, 10 were for Laparoscopic cholecystectomy and 10 for gynecological surgery.

All patients were premedicated with Atropine, Pethidine, Ranitidine, & Metoclopramide in appropriate doses according to body weight just prior to surgery. Standard anesthesia with Thiopentone, followed by intubation with Suxamethonium, & maintenance with oxygen, N_20 & Halothane & adequate muscle relaxation maintained with Vecuronium.The $ETCO_2$ values, Heart Rate, Blood pressure, SPO_2 & Airway pressure were recorded as per Appendix A.

The observations were made as follows.

- Baseline value prior to insufflation after steady ventilation for about 10 -15 mints after induction.
- 5 mints after the insufflation
- Every 10 mints interval during the procedure
- Immediately following exsufflation

The first value that is a baseline value of $ETCO_2$ is set by adjusting the Minute Ventilation and the Respiratory Rate before the abdomen is insufflated with CO_2 as per the groups allocated. The purpose of this study is to study the trend of $ETCO_2$ throughout the procedure and to compare the changes with different baseline values and to have an optimum baseline $ETCO_2$ value. The study considers abandoning during the procedure when the safe upper limit of $ETCO_2$ is crossed which is taken as 50 mmHg in hemodynamically stable patients.

V. Observation And Analysis

Table-1: The age and weight distribution of the patients in the study is as follows:-

	A	B	C
Mean age (yrs)	38.5	32.9	39.7
Mean Weight (Kg)	55	50	52

Table -2 Different types of surgeries were undertaken in the study. They were as follows

Surgeries	A	**B**	C
Laparoscopic cholecystectomy	15	13	10
Laparoscopic Mesh Repair	2	0	0
Laparoscopic Assisted Vaginal Hysterectomy or Ovarian cyst removal	3	6	10

Table-3 Mean duration of the surgeries performed

	A	**B**	C
Mean duration	74 min	76.36 min	60 min

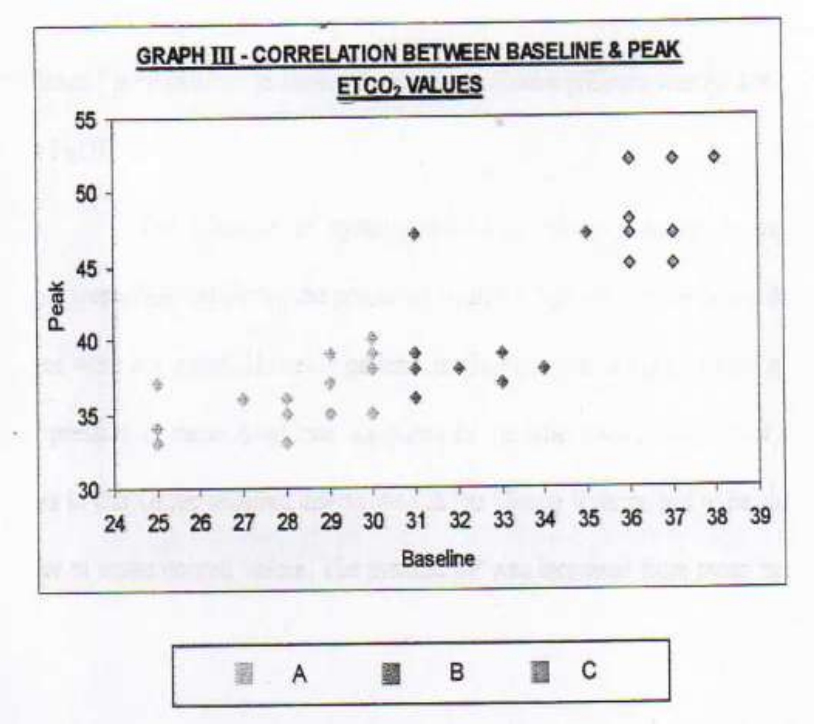
One patient in Group B, who was undergoing Laparoscopic Cardiomyotomy for Achalasia Cardia developed Subcutaneous Emphysema and persistent hypercarbia followed by cardiac arrhythmia & the study was abandoned. Another patient in the same Group who was for laparoscopic removal of the ovarian cyst developed persistent hypercarbia which went above the safe upper limit (about 53 mmHg) with marked tachycardia & raise in blood pressure was also abandoned. In Group C, 5 out of 20 studies could not be completed because of the complications like hypercarbia leading to increased sympatho adrenaI stimulation which manifested as either tachycardia or raised blood pressure or arrhythmias & the patients had to be subsequently hyperventilated till the $ETCO_2$ values came down. In this Group the patients were hypoventilated (mean Minute Ventilation being 4.67 L/min) after the induction and before the CO_2 insufflation, to maintain the baseline $ETCO_2$ value between 36 - 40 mmHg. The peak $ETCO_2$ reached was very high which invariably led to intervention during the study. Tachycardia and raised blood pressure was noticed in the remaining patients also during the study. When after induction & prior to abdominal insufflations, a slight hypocarbia was attained by hyperventilation (by about 14%) .the peak $ETCO_2$ reached was about 36.7 mmHg.

Whereas when normocarbia was maintained prior to insufflation as in the control Group B the peak $ETCO_2$ value was around 40 mmHg after 30 min. No further significant increase was observed when the duration of laparoscopy was extended beyond this time. This study also showed there was a slight increase in the $ETCO_2$, following exsufflation. The result was subjected to paired t-test and the p value was < 0.001 which is significant.

Table – 4 The mean $ETCO_2$ changes recorded in all the three groups are as follows :-

	A	**B**	**C**
Baseline values prior to insufflations	27.88	32.5	37
Immediately after insufflations	29.76	33.5	39.4
5 min	32.43	35.5	43.00
10 min	34.37	37.28	45.6
20 min	34.55	39.00	47.16
30 min	36.75	40.00	47.11
40 min	36.49	39.52	46.75
50 min	36.57	39.27	46.91
60 min	36.50	40.12	47.0
70 min	36.62	39.75	47.99
80 min	36.90	39.5	48
90 min	37.02	39.97	-
100 min	36.88	40.04	-
After Exsufflation	41.66	42.42	50.6

Fig-1 shows correlation between the baseline value and the peak value of $ETCO_2$ reached



The changes in the airway pressures in the study groups were significant when compared with the control group. In Group A the airway pressure was significantly lower ($p < 0.008$). The rise of airway pressure was 23% of the preinsufflation value (17 - 21 cm H_2O) whereas in Group C the airway pressure increased by 61% from the baseline value (13 - 21.16 cm H_2O) which is statistically significant ($p < 0.0003$). In Group B, the rise in airway pressure was by 40% (14 - 20 cm H_2O).

The changes in systolic & diastolic blood pressure & heart rate changes were observed during the procedure in all the 3 groups. It was found that the changes were not much. However patients in Group C had a higher mean systolic blood pressure & mean heart rate compared to the other two groups. Most of the patients in this Group required intervention & the Minute Volume had to be changed in order to attain normal values. The systolic BP was increased from mean baseline value of 102 mmHg to 143.57 mmHg (39%). In the other two Groups A & B. there was not much changes from the baseline values. The systolic BP increased by 11% in Group A & 13% in Group B.

Table -5 mean changes in systolic blood pressure

	A	**B**	C
Baseline	121.5	119.46	102.50
After Insufflation	125.75	121.58	140.10
05	131.78	131.90	142.28
10	134.13	139.50	143.37
20	135.21	135.61	143.14
30	131.07	130.08	143.57
40	135.53	127.38	139.00
50	137.46	129.91	144.33
60	130.85	126.71	142.3
70	139.65	124.76	140.6
80	134.84	128.5	142
90	137.2	127.8	0
100	135.8	125	0
After Exsufflation	130.7	121.88	138.7

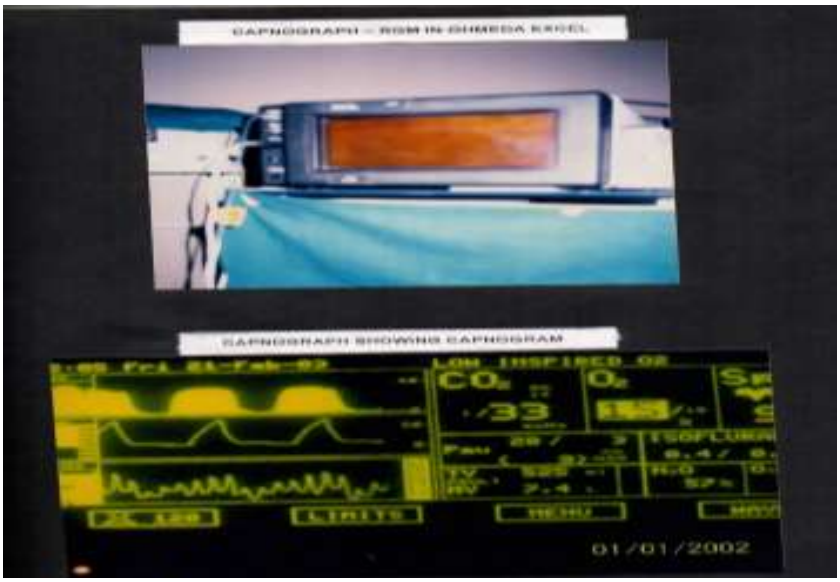
Table –6mean changes in diastolic blood Pressure

	A	**B**	C
Baseline	79.4	79.88	78
After Insufflation	87.7	84.33	94
5	90.83	90.07	92.14
10	87.88	91.2	87.33
20	89.72	91.8	91
30	89.9	89.5	86.83
40	84.9	89.7	88.2
50	85.88	90.4	89.33
60	86.33	94	90.2
70	86.6	92	91.2
80	90.33	85.2	90.7
90	88.72	86.7	0
100	89.2	88.3	0
After Exsufflation	85.5	85.5	93.33
After Insufflation	87.7	84.33	94

Table-7mean changes in heart rate

	A	B	C
Baseline	80.45	79.88	101.2
After Insufflation	88	98.66	104.8
5	91.18	99.5	105.5
l0	88.83	98.33	103.6
20	90.46	98.16	103.4
30	91.2	99.27	104
40	88.92	89.6	95
50	90.5	93.75	99
60	89.88	94	104.6
70	88.16	94	102
80	92.33	88.66	103
90	81.58	89.87	0
100	88.7	92.7	0
After Exsufflation	90.0	93.7	102

Fig 2: showing Capnograph



VI. Discussion

Insufflation of CO_2 in to the peritoneal cavity creates a high CO_2 tension gradient across the peritoneum between the pneumoperitoneum & the blood perfusing the peritoneum. CO_2being a highly diffusible gas can be readily absorbed into the blood stream resulting in significant hypercarbia. One study has demonstrated 30% increase in CO_2 load due to absorption from the peritoneal cavity [1] It is known that CO_2 pneumoperitoneum can have significant effects on cardiovascular and respiratory functions that may be deleterious and that these effects appear to be more prominent in patients with preexisting cardiorespiratory

diseases [2]. While it is known that there is significant CO_2 absorption from the peritoneal cavity and that hemodynamic changes occur ,a complete description of CO_2 absorption and its subsequent systemic effects has not been fully elucidated.[1,3,4,5]

Peritoneal space is a closed, collapsible body cavity that normally contains only a little serous fluid. Following carboperitoneum there are two immediate effects. First, an increase in Intra Abdominal Pressure & secondly, there is gaseous interchange between the peritoneal gas and the blood perfusing the cavity. The $ETCO_2$ vs. time curve shows that the $ETCO_2$ increases with time initially to reach a steady state in about 15 min after the onset of insufflation [6]. In a study by Baraka the steady state was at about 40 min [7]. However in this study the steady state was obtained at 30 min following the CO_2, insufflation. The plateau suggests that the CO_2 absorbed has reached equilibrium with that removed by the ventilation. Thereafter the CO_2 begins to accumulate in the body stores. Lister M.D [8] in his study shows that this plateau is not simply time related. He demonstrates that CO_2 absorption seems to reach a maximum at relatively low intraperitoneal CO_2 insufflation pressure & does not increase significantly beyond this despite further increases in intraperitoneal CO_2 pressure. This can be explained by the Fick s law of Diffusion for CO_2.

$$\theta CO_2 = \frac{D\ A\ (PpCO_2 - PBCO_2)}{d}$$

θCO_2 is CO_2 flux, D is diffusibility of CO_2, which is not a variable term. A is the area of peritoneum exposed to CO_2. At low insufflation pressures of 10 mmHg the peritoneum is fully distended. Therefore A increases substantially at low intraperitoneal pressure when CO_2 maximally gets absorbed. Any further increase in this pressure cause only increase in tension in the abdominal wall & thus no further increase in CO_2 absorption occurs. d is the anatomic distance between the peritoneal surface & the adjacent capillary bed. The capillaries just below the peritoneal surface collapse as the intraperitoneal pressure rises above the capillary hydrostatic pressure, diverting blood flow away from the peritoneal surface. This would increase the distance and further limit increases in CO_2 absorption. $PpCO_2$ is the partial pressure of CO_2 in the peritoneum and $PBCO_2$ is the partial pressure of CO_2 in the blood.

This study by Lister M.D. indicates that the CO_2 absorption & $ETCO_2$ is not linearly related to intraperitoneal insufflation pressure. He showed that though CO_2 elimination did not increase significantly as intraperitoneal insufflation pressure increases about 10 mmHg, the dead space continued to increase at high intraperitoneal pressure. Therefore the increase in the partial pressure of arterial CO_2 as intraperitoneal pressure increases from 0 - 10 mmHg largely is accounted for by the increase in CO_2 absorption and elimination, whereas increase in PaCO2 as insufflation pressure increases from 10 - 25 mmHg is largely accounted for by the increase in dead space.

The concept of increase in partial pressure of arterial CO_2, by the increased dead space during laparoscopic surgery is correlating with the studies conducted by Alexander et al during 1970s. Respiratory impedance measurements done by Alexander et al have shown marked elevation of the diaphragm. In addition to definite diaphragmatic splinting there is a cephalad shift of diaphragm [9]. Transmission of increased intraabdominal pressure into the thoracic cavity reduces the lung compliance. This causes an increase in the Peak Airway Pressure (PAW) with the resultant rise in the dead space. Therefore he hypothesized that the consistent rise in $PaCO_2$ accompanied by fall in arterial PH was due to CO_2 absorption from the peritoneal cavity as well as due to increase in the dead space, due to the raised airway pressures which could also have contributed to hypoventilation.

This study is inconsistent with our study which shows that in Group A where patients were hyperventilated before insufflation the rise in the airway pressure was minimum (17 - 21 cm H_20) and in Group C when hvpoventilation was done prior to insufflation there was significant rise in airway pressures (13 - 21.16 cm H_20) which could have contributed to further rise in dead space and CO_2 accumulation. In Group B, the airway pressure increased from 14-20 cm H_{2O} which is significant.

Christine E. Mullet has shown that this CO_2 diffusion & absorption is dependent upon the site of the CO_2, insufflation and not on the duration of CO_2 insufflation [6]. He demonstrated that a plateau was reached within 15 - 20 min in patients having intraperitoneal CO_2 insufflation whereas the $ETCO_2$ continued to increase slowly up to the end of the insufflation during pelviscopy where extraperitoneal CO_2 insufflation was done. Thus beyond 15 minutes of laparoscopy, the rate of CO_2 diffusion during intraperitoneal CO_2 insufflation, is no longer related to the duration of insufflation whereas the extraperitoneal insufflation may require more time to reach a steady state due to the nature of the exchange area in contact with CO_2 and secondly there may be continued recruitment of more gas exchanging area caused by the continued dissection of extraperitoneal space.

A E Cameron and A P Kendall in their study showed that neither there exists a relationship between the $ETCO_2$ rise & the total volume of CO_2 insufflated [10,11]. This is not surprising since the volume insufflated only reflect the size of the leak around the instrument insertion site. It has been shown that once

pneumoperitoneum has been established a flow of 200 ml/min is required to maintain the pneumoperitoneum & only 20 - 30 ml/min is absorbed from the peritoneal cavity the remaining being lost to the atmosphere due to a constant leak.

Many studies have shown increase in the Minute Ventilation to maintain the maximum $ETCO_2$close to the preinsufflation level. Some have increased Minute Ventilation by 12 - 16% while some studies have shown increase upto 25% [7]. This study has shown an increase of Minute Ventilation by 14% to maintain $ETCO_2$ at 36 mmHg. This increase in MV was achieved before CO_2 insufflation. Thus by keeping the preinsufflation value of $ETCO_2$ in the hypocapnic range (25 - 30 mmHg) as in Group A, the peak $ETCO_2$ and thus the partial pressure of arterial CO_2 can be maintained at near normocapnic value. Also there was no adverse effects of hypocarbia observed during the study. Alexander et al in his study showed the rise of $ETCO_2$ from 27 - 35 mmHg. This is in agreement with our study where $ETCO_2$ values graduated from 27.88 - 36.7 mmHg. It was also observed that there was a slight increase in $ETCO_2$ after exsufflation. This can be explained by essential CO_2 resorption immediately after the decrease of intraabdominal pressure [12]& also by the increased metabolic CO_2 production soon after the release of IAP which is indicated by increased oxygen consumption at that phase of the procedure [13,14].

In conclusion this study shows that in healthy person's normocarbia can be maintained by using moderate hyperventilation of about 14% to achieve a baseline value of 25 - 30 mmHg prior to insufflation.

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