Original Article

The Effects of Co2 Pneumoperitoneum on End Tidal CO_2 ($ETCO_2$), Arterial Blood Pressure and Heart Rate During Laparoscopic Cholecystectomy Under General Anaesthesia

1. Noor Hussain 2. Dure Shahwar 3. Basharat Ali Khan 4. Rehan Masood

1. Prof. of Anesthesia, FUMC, Rawalpindi 2. Assoc. Prof. of Surgery, FUMC, Rawalpindi 3. Assoc. Prof. of Surgery, FUMC, Rawalpindi 4. Asstt. Prof. of Surgery, FUMC, Rawalpindi

ABSTRACT

Objective: To analyze the effects of CO2 pneumoperitoneum at 13 to 15 mmHg intra-abdominal pressure on end tidal CO2 (ETCO2), arterial blood pressure and heart rate during laparoscopic cholecystectomy under general anaesthesia with controlled mechanical ventilation.

Design: Prospective Descriptive Study.

Place and Duration of Study: The study was conducted at the department of anaesthesia Fauji Foundation Hospital Rawalpindi from August 2005 to September 2006.

Patients and Methods: Fifty consecutive ASA grade-1 patients, 47 females, 3 males and aged 35-65 years undergoing laparoscopic cholecystectomy were included in the study. All patients received a standardized balanced anaesthetic in which 0.7 - 1 % isoflurane was used during maintenance. Ventilation was controlled and minute volume was kept at 100 ml/kg/min. Pneumoperitoneum was created with CO2 at a flow rate of 10 liter/min and 13 to 15 mmHg intra abdominal pressure was maintained during the operation. End tidal CO2 (ETCO2), heart rate and non invasive systolic, diastolic and mean arterial blood pressure was recorded immediately before intra abdominal CO2 insufflation and then after 5 minutes interval during the period of CO2 pneumoperitoneum. Study period started immediately before intra-abdominal CO2 insufflation till about 60 minutes of surgical procedure.

Results: The end tidal CO2 (ETCO2) levels progressively increased to reach a plateau 36mmHg 20 minutes after the beginning of intra abdominal CO2 insufflation. The end tidal CO2 (ETCO2) levels increased to 21 % of base line (from 30 to 36 mmHg) during CO2 pneumoperitoneum for laparoscopic cholecystectomy under controlled mechanical ventilation at minute volume 100 ml/kg/min. The systolic, diastolic and mean arterial blood pressure increased to 12% to 17% of the baseline during CO2 pneumoperitoneum at 13-15 mmHg intra abdominal pressure. There was no significant change in heart rate.

Conclusion: CO2 pneumoperitoneum produces rise in end tidal CO2 (ETCO2) levels and hemodynamic alterations proportional to the increased intra abdominal pressure during laparoscopic surgery under general anaesthesia with controlled mechanical ventilation.

Key Words: General anaesthesia, laparoscopic cholecystectomy, CO2 Insufflation, Pneumoperitoneum, end tidal CO2, Blood Pressure, Heart Rate.

INTRODUCTION

Laparoscopic cholecystectomy requires abdominal insuffulation with CO2 and an increase in the intra abdominal pressure to 15 mmHg (1). The physiologic affects of pneumoperitoneum include systemic absorption of CO2, haemodynamic and physiological alteration in a variety of organs due to increased intra abdominal pressure. CO2 absorption across the peritoneal surface and into systemic circulation can result in hypercarbia and eventual systemic acidosis. Hypercarbia and acidosis can cause hemodynamic changes by direct action on the cardio vascular system and by an indirect action through sympathoadrenal stimulation (2, 3). To prevent hypercapnia, close intra operative monitoring of end tidal CO2 (ETCO2) or arterial partial pressure of CO2 (PaCO2) is therefore essential. ETCO2 level is an easily accessible monitoring parameter than PaCO2. The increased intra abdominal pressure during pneumoperitoneum has been shown to result in hemodynamic alteration and changes in femoral venous flow, renal, hepatic and cardio pulmonary function (4, 5). Longer the operative time during laparoscopic surgery , longer will be the exposure of the host to the adverse physiologic effects of pneumoperitoneum. Appropriate anaesthetic techniques and monitoring facilitate surgery and allow early detection and reduction of complications.

The purpose of study is to determine the effects of CO2 pneumoperitoneum at 13 to 15 mmHg intra-abdominal pressure on end tidal CO2 (ETCO2), arterial blood pressure and heart rate during laparoscopic cholecystectomy under general anaesthesia with controlled mechanical ventilation.

PATIENTS AND METHODS

Fifty consecutive patients (47 females and 3 males) of ASA Grade -1 with an age range of 35 to 65 years and weighing between 50-80 Kg, undergoing cholecystectomy were included in the study (table-1) . Patients older than 65 years, those with morbid obesity and ASA grade II and above were excluded from the study.

All patients were premedicated with diazepam 10mg orally before midnight and were kept NPO after midnight. Intravenous line was established with 18 gauge cannula on the dorsum of hand. 3mg midazolam was given IV 5mins before induction of anaesthesia. Induction of anaesthesia was with intravenous 0.1mg/kg nalbuphine and thiopentone sodium 5mg/kg. Tracheal intubation was facilitated by IV atracurium 0.5mg/kg. Anaesthesia was maintained with 50% nitrous oxide in oxygen, 0.7 – 1 % isoflurane and intermittent bolus of Atracurium as required. Blease anaesthesia machine and Blease 6500 anaesthesia ventilator with close circuit was used. The ventilatory system was set to maintain a minute volume of 100ml/kg/min, an inspiratory to expiratory ratio of 1:2 and ventilatory frequency of 14 bpm. Monitoring included pulse rate, continuous ECG, intermittent NIBP, pulse oximetery(SPO2) and capnography (ETCO2). The cardiac monitor Welch Allyn was used for measuring these parameters.

Patients were placed in a supine position on operating table. Open pneumoperitoneum was created with carbon dioxide at a flow rate of 10 liters per minute and 13 to 15 mmHg intra-abdominal pressure was maintained. Reverse trendelenburg 15 to 20 degrees tilt was made in the beginning of laparoscopic procedure. The average duration of surgery was about one hour.

One liter of Ringer's lactate was given intravenously during the operation. At the end of surgery the neuromuscular blocking agent was antagonized with a combination of Neostigmine 0.05 mg/kg and Atropine 0.02 mg/kg, patients were transferred to the post operative ward and discharged on second post operative day.

Collection of Data

Cardiac monitor was set to record ETCO2, SPO2, heart rate and non invasive systolic, diastolic and mean arterial blood pressure immediately before intra abdominal carbon dioxide insufflation and then after every 5 minutes interval during the period of CO2 pneumoperitoneum in all patients during surgery. The data collection started 10- 20 minutes after induction of anaesthesia, just before intra-abdominal CO2 insufflations. The record of blood pressure, heart rate and end tidal CO2 before induction, after induction of anaesthisa for 15 minutes and during recovery period was not included in the study. The print of the recorded data was taken from the cardiac monitor at the end of each operation.

Statistical Analysis

Data was analyzed using SPSS version 10.0. Mean, standard deviation, standard error of mean (SEM) and percentages were calculated. The statistical analysis was done by students' t-test and p-value less then 0.05 was considered significant.

Table 1: Patients Characteristics (n=50)

Total no. of patients	50
Age (years) mean ± SD	49 ± 7.9
Female/ male ratio	47/3 / 94:6
Weight (kg) mean ± SD	67 ± 7.5
American Society of Anaesthesiologist 's physical status (ASA grade)	ASA Grade I

RESULTS

Effect oN End Tidal CO2 (PETCO2)

The PETCO2 levels increased to 10 % (33 mmHg), 17% (35 mmHg) and 21% (36 mmHg) respectively by baseline (30 mmHg) after 5 mins, 10 mins, and 20 mins beginning of intra abdominal CO2 insufflation. (Table 2, Fig 1).

The PETCO2 levels progressively increased to reach a plateau 36 mmHg 20 mins after the beginning of intraperitoneal CO2 insufflation. The PETCO2 levels remained 21% elevated during the period of peritoneal insufflation. This 21% increase in PETCO2 levels from 30 mmHg at base line to 36 mmHg were observed during CO2 pneumoperitoneum under controlled mechanical ventilation, at a constant minute ventilation 100ml/kg/min and 13-15 mmHg fixed intra abdominal pressure.

The 21% (36 mmHg) increase in PETCO2 levels by base line (30 mmHg) were statistically significant ($P< 0.05$)

Effect on Arterial BP and Heart Rate

he systolic, diastolic and mean arterial blood pressure increased to 13 – 14% (132 mmHg systolic, 88 mmHg diastolic, 102 mmHg mean) after 10 minutes, and after 20 to 30 minutes 16 -17% by baseline after beginning of intra abdominal CO2 insufflation. After 40 minutes systolic, diastolic and mean arterial BP remained 10 – 12% elevated by base line (117 mmHg systolic, 77 mmHg diastolic, 90mmHg mean) during laparoscopic surgery (Table 3. Fig 2).This increase in arterial BP was statistically significant ($P < 0.05$)

The heart rate slightly increased from 86 beats per minute to 90 beats per minute (Table 4). No significant increase in heart rate was detected during CO2 pneumoperitoneum for laparoscopic cholecystectomy.

Table 2: ***Changes in PETCO2 during CO2 Pneumo-peritoneum for laparoscopic Cholecystectomy(n=50)***

	Time	Petco2 level Mmhg	% >	P-Value
Before beginning of intra-abdominal CO2 insufflation	0 min	30.13 ± 0.87		
After beginning of intra-abdominal CO2 insufflation	5min	33.00 ± 0.95	9.51%	P<.05
	10 min	35.30 ± 1.06	17.15%	P<.05
	15 min	36.20 ± 1.02	20.13%	P<.05
	20 min	36.47 ± 0.99	21.02%	P<.05
	25 min	36.60 ± 0.99	21.46%	P<.05
	30 min	36.57 ± 0.97	21.35%	P<.05
	35 min	36.57 ± 0.98	21.35%	P<.05
	40 min	36.60 ± 0.97	21.46%	P<.05
	45 min	36.57 ± 0.98	21.35%	P<.05
	50 min	36.50 ± 0.97	21.46%	P<.05
	55 min	36.57 ± 0.98	21.35%	P<.05
	60 min	36.60 ± 0.97	21.46%	P<.05

**Minute ventilation was kept constant at 100 ml \kg\min and intra-abdominal pressure 13-15 mmHg during study.*
** Data are Mean ± SEM, P < 0.05 as compared with time 0.*

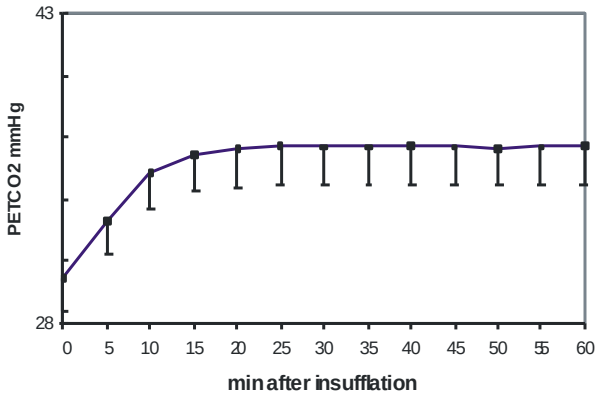

Figure 1: The Graph showing changes in PETCO2 during CO2 Pneumoperitoneum for laparoscopic Cholecystectomy(n=50)

Table 3: Changes in Arterial Blood Pressure during CO2 Pneumoperitoneum for laparoscopic Cholecystectomy (n=50)

	Time	Systolic BP Mmhg	% >	Diastolic BP Mmhg	% >	Mean BP Mmhg	% >	P-Value
Before beginning of intra-abdominal CO2 insufflation	0 min	117.33± 2.49		77.29± 1.68		89.92± 1.70		
After beginning of intra-abdominal CO2 insufflation	5min	124.00± 4.26	5.06%	82.57± 2.89	7.06%	96.71 ± 3.24	7.55%	P<.05
	10 min	131.96± 5.37	12.46%	88.50± 3.35	14.50%	102.00± 3.99	13.44%	P<.05
	15 min	134.25± 5.24	14.42%	89.71± 3.06	16.06%	104.63± 3.71	16.36%	P<.05
	20 min	136.54± 5.10	16.37%	89.92± 2.86	16.33%	14.92 ± 3.41	16.68%	P<.05
	25 min	137.88± 4.28	17.51%	90.54± 2.82	17.14%	106.71± 3.31	18.67%	P<.05
	30 min	135.75± 4.55	15.70%	89.71± 2.95	16.06%	105.29± 3.39	17.10%	P<.05
	35 min	133.67± 4.24	13.92%	88.29± 2.48	14.23%	103.67± 3.06	15.29%	P<.05
	40 min	131.58± 3.23	12.14%	87.63± 2.18	12.37%	101.50± 2.51	12.88%	P<.05
	45 min	130.00± 2.91	10.80%	85.79± 2.15	11.00%	101.04± 2.23	12.37%	P<.05
	50 min	127.38± 2.61	8.56%	84.63± 1.88	9.49%	100.33± 2.15	11.58%	P<.05
	55 min	129.83± 2.51	10.65%	85.71± 1.89	10.89%	99.88 ± 2.04	11.08%	P<.05
	60 min	129.58± 2.67	10.44%	84.96± 1.98	9.92%	100.38± 2.12	11.63%	P<.05

*Intra abdominal pressure was kept 13-15 mmHg during study.
*Data are Mean ± SEM, P < 0.05 as compared with time 0.

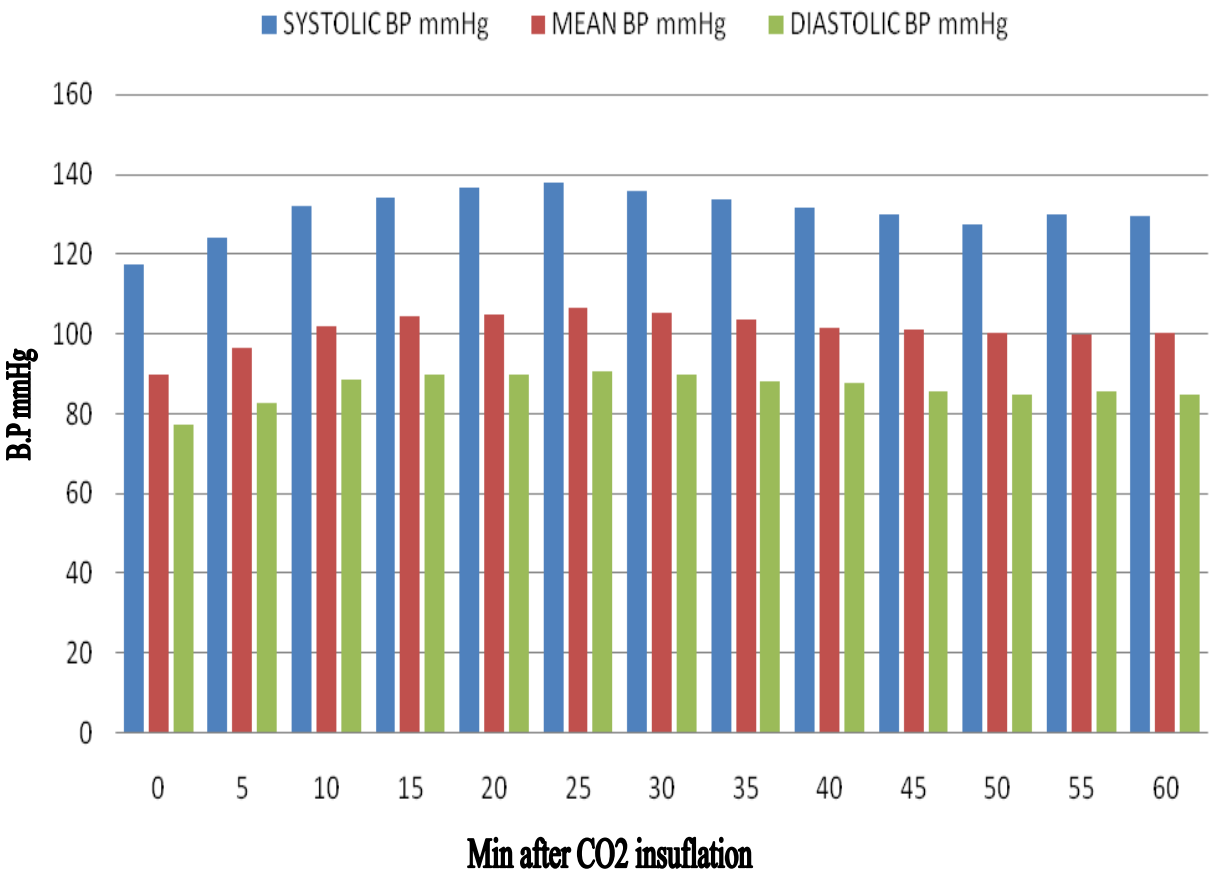

Figure 2: The Graph showing Changes in Arterial Blood Pressure during CO2 Pneumoperitoneum for laparoscopic Cholecystectomy (n=50)

Table 4: ***Changes in Heart Rate during CO2 Pneumo-peritoneum for laparoscopic Cholecystectomy (n=50)***

	Time	Heart Rate /min	% Change
Before beginning of intra-abdominal CO2 insufflation	0 min	86.58± 2.73	
After beginning of intra-abdominal CO2 insufflation	5min	86.29± 2.30	-0.34%
	10 min	87.17± 2.94	0.67%
	15 min	87.83± 2.83	1.44%
	20 min	91.00± 3.06	5.10%
	25 min	90.42±3.06	4.43%
	30 min	91.17± 3.39	5.29%
	35 min	89.67± 3.59	3.56%
	40 min	90.38± 3.41	4.38%
	45 min	90.38± 3.55	4.38%
	50 min	87.75± 3.47	1.35%
	55 min	86.67± 3.09	0.10%
	60 min	87.21± 3.19	0.72%

*Data are Mean ± SEM.

DISCUSSION

Effects on Etco2

The CO2 pneumoperitoneum produces increased intra abdominal pressure and increase in end tidal CO2 levels due to CO2 absorption from peritoneal cavity (6). During uneventful CO2 – pneumoperitoneum, PETCO2, and PaCO2 progressively increases to reach a plateau 15 to 30 min after beginning of CO2 insufflation in patients under controlled mechanical ventilation during gynaecological laparoscopy in the Trendelenburg position or laproscopic cholecystectomy in head up position (7).The mean Pa CO2 and PETCO2 gradients do not change significantly during peritoneal insufflation of CO2.In our study PETCO2 levels increased progressively to 10% (33mmHg) of baseline (30mmHg) after 5 min,17% (35mm Hg) after 10 min and plateau 21% (36mm Hg) after 20 min beginning of intra abdominal CO2 insufflation. The 21% increase in ETCO2 levels remained elevated during period of intraabdominal CO2 insufflation. The 21% increase in ETCO2 (36mm Hg) of base line (30mmHg) is found during CO2 pneumoperitoneum for laparoscopic

cholecystectomy under controlled mechanical ventilation at a constant minute ventilation 100ml/Kg/min and 13-15mmHg intra-abdominal pressure (Fig.1. and Table.2). The results of our study correlate with the previous study.

Absorption of CO2 across the peritoneum is normally eliminated through the lungs because of its high aqueous solubility and difusibility. If intraoperative ventilation is impaired, CO2 absorption can result in hypercapnia and acidosis (8).

To prevent hypercapnia, close intraoperative monitoring of end-tidal CO2 (ETCO2) or arterial partial pressure of CO2 (PaCO2) is therefore essential. End tidal CO2 (ETCO2) is most commonly used as a non-invasive substitute for PaCO2 in evaluating the adequacy of ventilation during laparoscopic surgery. The PaCO2 usually correlates with the ETCO2, except in patients with cardiopulmonary compromise and associated ventilation perfusion mismatch. A direct estimation of PaCO2 may become necessary in such patients (9). In patients undergoing laparoscopic adjustable gastric banding, Demiroluk et al reported an increase in PaCO2 levels from 34mm Hg at baseline to 42mm Hg after abdominal insufflation. In a study of laparoscopic versus open GBP (gastric by pass), ETCO2 levels were found to increase by 14% of baseline (from 35 mm Hg to 40 mmHg), PaCO2 levels increased by 10% of baseline (from 38 mmHg to 42 mm Hg) during laparoscopic GBP (10). In our study ETCO2 levels are increased by 21% of baseline (from 30 mm Hg to 36 mm Hg) during laparoscopic cholecystectomy. (Fig.1, Tale 2.)

During pneumoperitoneum, appropriate ventilatory changes should be performed to eliminate the increased CO2 load and prevent systemic acidosis. Ventilatory changes consist of increasing the minute ventilation. Dumont et al reported that minute ventilation increased by 21% to limit the rise in ETCO2 in patients undergoing laparoscopic gastroplasty (11).

Elimination of the increased CO2 load is performed primarily through the lungs. The total volume of exhaled CO2 (VCO2) during pneumoperitoneum is therefore an indirect method to quantify the amount of CO2 absorbed during laparoscopy. In a study of nonobese patients, Tan et al estimated that the volume of CO2 absorbed from the peritoneal cavity ranged from 38 to 42 ml/min during laparoscopy, which represented a 30% increase in the CO2 load (12).

Effect on Blood Pressure and Heart Rate:

In our study 13-14% increase in systolic, diastolic and mean arterial blood pressure by base line is found 10 minutes after beginning of intra-abdominal CO2 insufflation. The maximum 16-17% increase in systolic, diastolic and mean arterial blood pressure by baseline is observed between 20-30 minutes after beginning of intra abdominal CO2 insufflation. After 40 minutes 10% to 12% increase in systolic, diastolic and mean arterial blood pressure is observed during the period of CO2 pneumoperitoneum. (Table 3. Fig 2.). The intra-abdominal pressure is maintained at 13-15mm Hg during CO2 pneumoperitoneum. Cardiovascular changes have been characterized by many clinical studies. Most of these studies reported increased systemic and pulmonary vascular resistance and reduction of cardiac index when laparoscopy was performed at about 15mm Hg and head up tilt 10 degree. Joris et al (13) using invasive monitoring, observed a significant increase in mean arterial pressure (35%) after peritoneal insufflations, along with an increase of systemic vascular resistance (65%).

The insufflation of gas into the peritoneal cavity can provoke arrhythmias. Their incidence is as high as 14-27% of laparoscopies (14) which is higher than open surgery. In our study no change or slight increase in heart rate is observed (Table 4). Sinus bradycardia is found only in one patient immediately after the beginning of intra-abdominal CO2 insufflation. The incidence of arrhythmias is very low in our study. The anaesthesia technique and agents used during the study has minimum effect on haemodynamics. The blood pressure and heart rate is recorded 20 minutes after the induction of anaesthesia when adequate level of depth of anesthesia is achieved. Haemodynamic alterations occur only when the PaCO2 is increased by 30% above the normal levels. Mild hypercapnia causes sympathetic stimulation which results in trachycardia, increased systemic vascular resistance, systemic arterial pressure, central venous pressure and cardiac out put (15). An increase in the intra-abdominal pressure is most important factor contributing to circulatory instability during laparoscopy (16). Reflex increase in vagal tone due to excessive stretching of the peritoneum may produce bradycardia. The threshold pressure that has minimum effects on haemodynamic functions is < 12mm Hg. If the inflation pressures are increased > 15mm Hg, the insufflated CO2 compresses both venous capacitance and the arterial resistance vessels. This produces rise in the systemic vascular resistance and pulmonary vascular resistance leading to an increased after load. The mean arterial blood pressure rises and cardiac out put falls (25-35%) (17).

These haemodynamic changes are well tolerated by healthy individuals, but may have deleterious consequences in patients with cardio vascular disease (18-20).

CONCLUSION

Altered physiology has been demonstrated during laparoscopic cholecystectomy CO2 pneumoperitoneum produces rise in end tidal CO2 (ETCO2) levels and increase in arterial blood pressure during laparoscopic

surgery under general anaesthesia with controlled ventilation at 13-15 mmHg intra abdominal pressure. Heart rate increases slightly or there is no change in heart rate. The increase in end tidal CO2 (ETCO2) levels and hemodynamic changes are proportional to the increased intra abdominal pressure.

Maintaining intra abdominal pressure under 15mmHg reduces the incidences of these changes leading to minimal and transient consecutive organ dysfunction and without consequences for the outcome. It is the merit of the anesthesiology team to not let the pathophysiological changes transform into complications, and the surgeon has to be aware that a low insufflation pressure diminishes the pathophysiological responses and avoids most of the complications.

REFERENCES

1. Ninh T. Nguyen, and Bruce M. Wolfe. The physiological effects of Pneumoperitoneum in the Morbidly Obese. Ann Surg 2005; 241(2): 219 – 226.
2. Hsieh CH: Laparoscopic cholecystectomy for patients with chronic obstructive pulmonary disease. Laparoendosc Adv Surg Tech [A] 2003; 13:5 – 9.
3. Neudecker J, Sauerland S, Neugebauer E, Bergamaschi R, et al. The EAES clinical practice guidelines on pneumoperitoneum for laparoscopic surgery. Surg Endosc 2002; 16: 1121 – 1143.
4. Jakimowics J, Stultiens G, Smulders F. Laparoscopic insuffulation of the abdomen reduces portal venous flow. Surg Endosc 1998; 12: 129 – 132.
5. Hirvonen EA, Poikolainen EO, Paakkonen ME, et al. The adverse hemodynamic effects of anaesthesia, head-up tilt, and carbon dioxide pneumoperitoneum during laparoscopic cholecystectomy. Surg Endosc 2000;14:272 – 277.
6. Sood J, Kumra VP. Anaesthesia for laparoscopic surgery. India J Surg 2003; 65:232-240.
7. Miller R D (ed) Anaesthesia, 5th ed, p. 2004. New Churchill Livingstone, 2000.
8. Lindgren L, Koivusalo AM, Kellokumpu I. Conventional pneumoperitoneum compared with abdominal wall lift for laparoscopic cholecytectomy. Br J Anaesth 1995;75:567-572.
9. Fitgerald SD, Andrus CH, Baudendistal LJ, Kaminski DL, Hypercarbia during carbon-dioxide pneumoperitoneum. Am J Surg 1992; 163: 186-190.
10. Demiroluk S, Salihoglu Z, Zengin K, et al. The effects of pneumoperitoneum on respiratory mechanics during bariatric surgery. Obes Surg. 2002; 12:376-379.
11. Dumont L, Mattys M, Mardirosoff C, et al. Changes in pulmonary mechanics during laparoscopic gastroplasty in the morbidly obese patient. Acta Anathesiol Scand 1997;41:408-413.
12. Tan PL, Lee TL, Tweed WA. Carbon dioxide absorption and gas exchange during pelvic laparoscopy. Can J Anaesth 1992;39:677-681.
13. Joris JL, Noirot DP, Legrand MJ, Jacquet NJ, Lamy ML: Hemodynamic changes during laparoscopic cholecystectomy. Anesth Analg 1993;75:1067-1071.
14. Magrina JF: Complications of laparoscopic surgery. Clin Obstet Gynecol 2002;45:469-480.
15. Marshall RL, Jebson PJ, Davie IT, Scott DB. Circulatory effects of carbon dioxide insufflation of the peritoneal cavity for laparoscopy. Br J Anaesth 1972;44:680-684.
16. Dexter SP, Vucevic M, Gibson J, McMahon MJ. Hemodynamic consequences of high and low pressure capnoperitoneum during laparoscopic cholecystectomy. Surg Endosc 1999;13:376-381.
17. Johannsen G, Andersen M, Juhl B. The effect of general anaesthesia on the haemodynamic events during laparoscopy with CO2 insufflation. Acta Anaesthesiol Scand 1989;33:132-136
18. Westerband A, Van DE, Water JM, Amzallag M., Lebowitz PW, Nwasokwa ON. Cardiovascular changes during laparoscopic cholecystectomy. Surg Gynecol Obstet 1992;175:535-538.
19. Wahba RWM, Beiqe F, Kleiman SJ: Cardiopulmonary function and laparoscopic cholecystectomy . Can J Anaesth : 1995 ; 42:51.
20. Westerband A, Van De Water JM , Amzallag M: Cardiovascular changes during laparoscopic cholecystectomy. Surg Gynaecol Obstet 1992; 39:A132.

Address for Corresponding Author:
Brig. (R) Noor Hussain,
Professor of Anesthesia,
Foundation University Medical College,
Rawalpindi.