

# Hemodynamic Changes During Laparoscopic Cholecystectomy Using Different Intra-Abdominal Pressures

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## ABSTRACT

**Background-**Biliary diseases, which have been there for a long time, account for a large percentage of digestive system ailments all over the world. Cholelithiasis is the most common of them, producing generalised illness and necessitating surgical intervention for complete recovery. The goal of the study was to examine hemodynamic changes in patients having laparoscopic cholecystectomy with varying intra-abdominal pressures caused by carbon dioxide insufflation.

**Methods-**The patients were assigned to one of three groups, each of which maintained varying intra-abdominal pressures (8–10 mmHg, 11–13 mmHg, and 14 mmHg and above). Heart rate, non-invasive blood pressure (systolic and mean), and end-tidal carbon dioxide were the baseline parameters examined. All parameters were measured at various intervals, including immediately during CO<sub>2</sub> insufflation, 5 minutes, 10 minutes, 20 minutes, and 30 minutes after CO<sub>2</sub> insufflation, and every 10 minutes if the surgery lasted longer than 30 minutes, at insufflation, and 10 minutes after CO<sub>2</sub> exsufflation. The Pedius Drager Ventilator was used to ventilate the patients, with a tidal volume of 8–10 ml/kg and a respiratory rate of 12–14 breaths/min. Patients were placed in a reverse Trendelenburg position (head up) at 15 degrees during surgery. The obtained results were statistically assessed and analysed. The qualities at the start were determined to be comparable.

**Results-**The mean and standard deviation of hemodynamic variables were reported. Analysis of Variance and the unpaired student t-test was used to determine statistical significance between groups (two tailed). The Bonferroni test was used to make inter-group comparisons. A p-value of less than 0.05 was deemed statistically significant. The mean heart rate increased during CO<sub>2</sub> insufflation in all three groups (baseline 84.0812.50, 87.9615.73, and 86.9217.00, respectively), and the rise in heart rate continued until exsufflation, after which it decreased, and the heart rates were comparable with the baseline at 10 minutes after exsufflation. The difference in mean heart rate between I and III was statistically significant at 10, 20, and 30 minutes after CO<sub>2</sub> insufflation, and at 10 minutes after CO<sub>2</sub> exsufflation [p<0.05]. The difference in systolic blood pressure at 10, 20, 30 minutes after CO<sub>2</sub> insufflation, at exsufflation, and 10 minutes after exsufflation [p=00.0001] and mean arterial pressure at 5, 10, 20, 30 minutes after CO<sub>2</sub> insufflation, at exsufflation, and 10 minutes after exsufflation [p=00.0001] was statistically significant in the inter-group comparison between I and III. The difference in EtCO<sub>2</sub> between Group I and Group III, as well as between Group II and Group

III, was highly significant statistically immediately after insufflation, and the same trend continued until the end of surgery and even 10 minutes after exsufflation [ $p=0.001$ ].

**Conclusion-**The study found that laparoscopic cholecystectomy causes significant intraoperative hemodynamic alterations, with the majority of pathophysiological changes connected to the cardio-vascular system and induced by CO<sub>2</sub> insufflation. When compared to low intraabdominal pressure, a high intraabdominal pressure caused by CO<sub>2</sub> insufflation is associated with more hemodynamic fluctuations and increased peritoneal CO<sub>2</sub> absorption. As a result, low-pressure pneumoperitoneum is feasible for laparoscopic cholecystectomy and minimises the adverse hemodynamic effects of CO<sub>2</sub> insufflation.

**Keywords-**Laparoscopic cholecystectomy. Insufflation. Hemodynamic. Intra-abdominal pressure

## INTRODUCTION

The first mention of laparoscopy can be found in the Bible. The peritoneal cavity was the focal point in ancient times, with the umbilicus signifying life and the liver representing the "cradle of the soul." [1].

Early in the twentieth century, the first endoscopic exams of the peritoneal cavity were performed. George Kelling, a German surgeon, invented the name celioscopy after using a cystoscope to inspect the intra-abdominal viscera of a dog after insufflating the peritoneal cavity with air in 1901. In Sweden, Jacobeus performed the first human celioscopy in 1910 [2]. In 1982, general surgeons undertook the first laparoscopic surgery, liver biopsies [3]. Mouret conducted the first laparoscopic cholecystectomy on a person in France in 1987 [4].

In recent decades, the use of laparoscopic surgery in general surgery has grown in popularity. Patients like the small, limited incisions, and they benefit from the faster healing time. By minimising the length of postoperative hospital stays and the requirement for postoperative analgesia, health costs can be reduced [5]. The advantages of laparoscopic surgery explain why it is becoming more popular. Laparoscopic surgery, on the other hand, necessitates extensive intraoperative physiological changes, some of which are unique to these procedures. Patient posture, injection of exogenous insufflation gas, CO<sub>2</sub>, and increased intra-abdominal pressure due to pneumoperitoneum cause physiological alterations during laparoscopic surgery [6]. During a laparoscopic cholecystectomy, the patient is placed in a reverse Trendelenburg posture to allow the viscera to gravitate away from the surgical site. It helps to breathe and is thought to be beneficial to breathing [5]. However, it lowers mean arterial pressure and cardiac output by lowering venous return, right atrial pressure, and pulmonary capillary wedge pressure [7].

Hypercarbia and an increase in end-tidal CO<sub>2</sub> could be caused by CO<sub>2</sub> absorption from the peritoneal cavity [8]. Severe hypercarbia lowers left ventricular performance and has a negative inotropic effect on the heart [9]. Increased intra-arterial pressure, CO<sub>2</sub> absorption, temperature change, and a neuro-hormonal stress response are all effects of the pneumoperitoneum. Increased intra-abdominal pressure has an impact on all of the body's major systems, resulting in severe hemodynamic and ventilatory changes [6]. Increased intra-abdominal pressure obstructs venous and arterial blood flow in the infra-diaphragmatic area. It may also push the diaphragm into the chest cavity, reducing total lung capacity and functional residual capacity and exacerbating the acid-base imbalance. With an increase in ventricular stroke work and heart rate, cardiac output decreases. The pressure in the upper body is also increased by pressure on the abdominal aorta. Within 5 minutes of gas insufflation, the ventilatory and circulatory alterations are noticeable. Significant pathophysiologic effects are linked with pressures more than 15 mmHg, however they are reversible during a 2-hour period [10].

The amount of hemodynamic changes associated with pneumoperitoneum formation is determined by intra-abdominal pressure, CO<sub>2</sub> absorption volume, patient intravascular volume, ventilatory technique, and surgical conditions [11]. Subcutaneous or mediastinal emphysema, pneumothorax, hypoxemia, hypotension, CO<sub>2</sub> embolism, cardiovascular collapse, and cardiac arrhythmias are all common consequences of pneumoperitoneum formation [11].

Several investigations have found that low intra-abdominal pressure lowers the occurrence of hemodynamic and ventilatory abnormalities, resulting in modest and temporary organ dysfunction, and lessens the likelihood of physiological alterations becoming problems [12]. The study has been undertaken in an attempt to compare the hemodynamic changes in a patient undergoing laparoscopic cholecystectomy using different preset intra-abdominal pressures created due to carbon dioxide insufflation.

## MATERIALS AND METHODS

The study comprised patients with ASA I and II of either sex and ages ranging from 18 to 60 years who were scheduled to have an elective laparoscopic cholecystectomy. The patients were assessed and given a thorough general physical and systemic examination.

The study excluded patients with uncontrolled medical conditions such as hypertension, coronary artery disease, diabetes mellitus, COPD, and asthma. The study excluded patients with substantial portal hypertension, uncorrectable coagulopathies, probable gallbladder cancer, cirrhosis, and widespread peritonitis. The patients were assigned to one of three groups, each with a distinct degree of intra-abdominal pressure maintained by CO<sub>2</sub> insufflation throughout the surgical intervention.

**Table 1 Mean heart rate (min<sup>-1</sup>) in groups I, II, and III**

Stage	Group I Mean±SD	Group II Mean±SD	Group III Mean±SD	F value	P value	Result
Immediately during insufflation	94.48±11.57	98.44±11.78	97.52±17.20	0.566	0.570	N.S.
5min after CO <sub>2</sub> insufflation	95.20±11.19	97.56±11.45	101.20±8.23	1.163	0.318	N.S.
10min after CO <sub>2</sub> insufflation	96.12±11.25	97.36±11.82	106.28±14.82	4.737	0.011	SIG.
20min after CO <sub>2</sub> insufflation	96.45±10.83	94.33±12.54	105.25±10.19	5.205	0.008	SIG
30min after CO <sub>2</sub> insufflation	97.50±10.83	– <sup>a</sup>	112.40±12.19	4.56 <sup>b</sup>	0.0001	HS
At exsufflation	92.16±10.32	88.28±13.23	104.52±12.79	12.110	0.0001	HS
10min after exsufflation	87.88±10.97	86.56±11.67	100.56±12.14	11.080	0.0001	HS

a. In this group, only one patient's surgery continued till 30min with a value-96

b. bUnpaired Student's t test (two tailed) used to assess difference. (F0 ANOVA)

Group I Intra-abdominal pressure was maintained between 8 and 10 mmHg.

Group II Intra-abdominal pressure was maintained between 11 and 13 mmHg.

Group III Intra-abdominal pressure was maintained at 14 mmHg and above.

In the operation theatre after attaching monitors to the patient, the following base line parameters were monitored.

- Heart rate
- Noninvasive blood pressure (Systolic and mean)
- End-tidal carbon dioxide

All the above-mentioned parameters were monitored at various intervals, that is,

- Immediately during insufflation.

- 5 min after CO2 insufflation
- 10 min after CO2 insufflation
- 20 min after CO2 insufflation
- 30 min after CO2 insufflation
- After every 10 min if surgery exceeds 30 min
- At exsufflation
- 10 min after CO2 exsufflation

**Table 2 Mean systolic blood pressure (mmHg) in groups I, II, and III**

Stage	Group I Mean±SD	Group II Mean±SD	Group III Mean±SD	F value	P value	Result
Immediately during insufflation	124.12±9.98	132.72±6.68	128.12±13.50	4.254	0.0179	SIG
5 min after CO2 insufflation	125.96±9.48	134.92±6.56	136.28±10.27	9.872	0.0001	HS
10 min after CO2 insufflation	127.36±9.42	135.76±7.16	140.32±8.89	14.784	0.0001	HS
20 min after CO2 insufflation	128.75±9.78	140.88±8.21	143.20±8.12	15.557	0.0001	HS
30 min after CO2 insufflation	128.85±7.50	–a	143.60±7.79	6.63 <sup>b</sup>	0.0001	HS
At exsufflation	124.88±9.37	136.72±5.77	136.44±7.12	19.896	0.0001	HS
10 min after exsufflation	123.00±9.23	125.68±7.61	133.56±6.57	12.118	0.0001	HS

<sup>a</sup>In this group, only one patient's surgery continued till 30 min with a value-130

<sup>b</sup> Unpaired Student's *t* test (two tailed) used to assess difference. (F0ANOVA)

Patients were ventilated with Pedius Drager Ventilator keeping tidal volume 8–10 ml/kg and respiratory rate 12–14 breaths/min. During surgery, patients were placed in reverse Trendelenburg position (head up) at 15 ° and right side of table elevated in order to have gut loops away from the site of surgery.

The results obtained were evaluated statistically and analyzed.

## RESULTS

In all three groups, the mean heart rate increased immediately at insufflation, 5, 10, 20, and 30 minutes later, and dropped at exsufflation and 10 minutes later. At 10 and 20 minutes after CO2 insufflation, the difference in mean heart rate was statistically significant, and at 30 minutes after CO2 insufflation, at exsufflation, and 10 minutes after exsufflation, it was extremely significant (Table 1).

In all three groups, mean systolic blood pressure increased immediately at insufflation, 5, 10, 20, and 30 minutes later, and reduced at exsufflation and 10 minutes later. The difference was statistically significant during insufflation and was highly significant at 5, 10, 20, and 30 minutes after CO2 insufflation, as well as during exsufflation and 10 minutes after exsufflation (Table 2).

Stage	Group I Mean±SD	Group II Mean±SD	Group III Mean±SD	F value	P value	Result
Immediately during insufflation	95.08±6.77	101.00±5.08	99.96±8.66	5.101	0.0084	SIG
5min after CO2 insufflation	96.56±6.86	102.80±5.72	104.80±7.79	9.848	0.0001	HS
10min after CO2 insufflation	97.44±6.51	103.80±6.42	108.00±7.05	15.869	0.0001	HS
20min after CO2 insufflation	98.75±6.18	105.88±5.13	110.60±6.31	20.265	0.0001	HS
30min after CO2 insufflation	98.95±6.13	— <sup>a</sup>	113.20±4.43	9.88 <sup>b</sup>	0.0001	HS
At exsufflation	95.36±5.98	104.48±4.96	105.64±6.51	23.103	0.0001	HS
10min after exsufflation	93.56±5.89	95.68±5.39	101.72±6.30	12.987	0.0001	

a. In this group, only one patient's surgery continued till 30 min with a value of 105

b. Unpaired Student's t test (two-tailed) used to assess difference. (F0 ANOVA)

In all three groups, mean arterial pressure increased during insufflation, 5, 10, 20, and 30 minutes later, and reduced during exsufflation and 10 minutes afterward. The difference was statistically significant during insufflation and highly significant 5, 10, 20, and 30 minutes after CO<sub>2</sub> insufflation, as well as during exsufflation and 10 minutes after exsufflation (Table 3).

End-tidal CO<sub>2</sub> increased quickly after insufflation, and the increase in EtCO<sub>2</sub> persisted as the duration of CO<sub>2</sub> insufflation increased; even 10 minutes after exsufflation, the mean values in all three groups were greater than the baseline. At 5, 10, 20, and 30 minutes after CO<sub>2</sub> insufflation, the difference was statistically significant at exsufflation and 10 minutes after exsufflation (Table 4).

The intergroup comparison of mean heart rate was statistically significant at 10, 20, and 30 minutes after CO<sub>2</sub> insufflation, which continued at exsufflation and 10 minutes after CO<sub>2</sub> exsufflation, whereas the intergroup comparison of mean systolic blood pressure was statistically significant at 5, 10, 20, and 30 minutes after CO<sub>2</sub> insufflation, at exsufflation, and 10 minutes after CO<sub>2</sub> exsufflation (Table 5).

Mean arterial pressure was statistically significant during insufflation and highly significant 5, 10, 20, and 30 minutes after CO<sub>2</sub> insufflation, at exsufflation, and 10 minutes after exsufflation, whereas end-tidal CO<sub>2</sub> was statistically significant immediately after insufflation and highly significant 5, 10, 20, and 30 minutes after insufflation, at exsufflation, and 10 minutes after exsufflation (Table 5).

Intergroup comparisons of mean heart rate were statistically significant at 10 and 20 minutes after CO<sub>2</sub> insufflation, and continued at exsufflation and 10 minutes after CO<sub>2</sub> exsufflation, whereas mean systolic blood pressure comparisons were statistically significant at 10 minutes after CO<sub>2</sub> exsufflation (Table 6).

The intergroup comparison of mean arterial pressure was statistically significant at 20 minutes after insufflation and highly significant at 10 minutes after exsufflation, whereas the intergroup comparison of end-tidal CO<sub>2</sub> was significant after insufflation and highly significant at 5, 10, 20, and 30 minutes after insufflation, at exsufflation, and at 10 minutes after exsufflation (Table 6).

## DISCUSSIONS

*Analysis of Heart Rate* The mean heart rate increased in all three groups during CO<sub>2</sub> insufflation, and the rise remained until exsufflation, after which it declined, and the heart rates were comparable with the baseline at 10 minutes following exsufflation (Table 1).

**Table 4 End-tidal CO<sub>2</sub>(mmHg) in groups I, II, and III**

Stage	Group I Mean±SD	Group II Mean±SD	Group III Mean±SD	F value	P value	Result
Immediately during insufflation	31.96±2.74	32.28±3.02	34.68±3.30	6.014	0.003	SIG.
5min after CO <sub>2</sub> insufflation	32.92±2.76	33.12±2.92	37.92±2.78	25.114	0.0001	H.S.
10min after CO <sub>2</sub> insufflation	33.56±2.69	34.08±3.01	41.04±2.55	57.196	0.00	H.S.
20 min after CO <sub>2</sub> insufflation	34.20±2.70	35.88±2.54	42.80±1.85	72.145	0.00	H.S.
30min after CO <sub>2</sub> insufflation	37.00±2.58	— <sup>a</sup>	43.80±1.09	12.13 <sup>b</sup>	0.0001	H.S.
At exsufflation	33.00±2.95	33.84±2.96	40.12±1.92	53.434	0.00	H.S.
10min after exsufflation	32.04±2.92	32.20±3.16	37.52±2.12	31.647	0.0001	H.S.

a. In this group, only one patient's surgery continued till 30 min with a value -39.

b. Unpaired Student's t test (two tailed) used to assess difference. (F0 ANOVA)

**Table 5: Inter group comparison (group I v/s III) of mean heart rate, systolic blood pressure, mean arterial pressure, and end-tidal CO<sub>2</sub> Bonferroni test**

Stage	Mean Heart Systolic Blood	Mean End- Tidal Rate	Pressure	Pressure CO <sub>2</sub>
Immediately during insufflation	—	0.538	0.048	0.006
5min after CO <sub>2</sub> insufflation	0.403	0.0003	0.0001	0.0001
10min after CO <sub>2</sub> insufflation	0.001	0.0001	0.0001	0.0001
20min after CO <sub>2</sub> insufflation	0.04	0.0001	0.0001	0.00
30min after CO <sub>2</sub> insufflation	0.0001	0.0001	0.0001	0.0001
At exsufflation	0.0007	0.0001	0.0001	0.0001
10min after exsufflation	0.0007	0.0001	0.0001	0.0001

This increase in heart rate is related to decreased venous return, which reduces cardiac output and causes a compensatory increase in heart rate, as well as hypercarbia caused by CO<sub>2</sub> insufflation, which causes sympathetic activation due to catecholamine release [13, 14].

At 10, 20, and 30 minutes after CO<sub>2</sub> insufflation, the intergroup comparison of mean heart rate between groups I and III was statistically significant, which maintained at exsufflation and 10 minutes after CO<sub>2</sub> exsufflation (Table 5). At 10 and 20 minutes after CO<sub>2</sub> insufflation, at exsufflation, and 10 minutes after CO<sub>2</sub> exsufflation, there was a statistically significant

difference between groups II and III (Table 6).

When the other two groups (groups I and II) were compared with the high CO<sub>2</sub> pressure group, the difference in heart rate was statistically significant following CO<sub>2</sub> insufflation (group III). Because of the high CO<sub>2</sub> pressure employed, this considerable rise in heart rate can be explained by enhanced sympathetic activation and more impaired venous return in group III patients [12, 15].

*Analysis of Systolic Blood Pressure* The mean systolic blood pressure increased in all three groups during CO<sub>2</sub> insufflation, 5, 10, 20, and 30 minutes after CO<sub>2</sub> insufflation, but declined during CO<sub>2</sub> exsufflation and 10 minutes after CO<sub>2</sub> exsufflation (Table 2). The rise in systolic blood pressure following CO<sub>2</sub> insufflation can be explained by a reflex increase in systemic vascular resistance in reaction to abdominal distension, an increase in afterload to the heart, and sympathetic effects of CO<sub>2</sub> absorbed from the peritoneal cavity [16–17]. The reversal of CO<sub>2</sub> pneumoperitoneum effects causes a drop in systolic blood pressure after exsufflation. At 10 minutes after exsufflation, the intergroup comparison between groups II and III revealed a statistically significant difference (Table 6). However, at 5, 10, 20, and 30 minutes after CO<sub>2</sub> insufflation, at exsufflation, and 10 minutes following exsufflation, the intergroup comparison between groups I and III revealed statistically significant differences (Table 5). This significant difference between the low-pressure (group I) and high-pressure (group III) groups after insufflation can be explained by more abdominal distention in the latter, which leads to a significant increase in systemic vascular resistance and afterload to the heart, as well as sympathetic effects of CO<sub>2</sub> [12, 18, 19].

*Analysis of Mean Arterial Pressure* The mean arterial pressure increased in all three groups during CO<sub>2</sub> insufflation, and the rise in mean arterial pressure continued as the pneumoperitoneum duration lengthened. At exsufflation and 10 minutes after exsufflation, mean arterial pressure dropped (Table 3). With CO<sub>2</sub> insufflation, mean arterial pressure rises due to increased systemic vascular resistance, sympathetic effects of CO<sub>2</sub> absorbed from the peritoneal cavity, and the release of humoral mediators as a result of increased intra-abdominal pressure [13, 16]. The drop in mean arterial pressure after exsufflation could be due to the effects of CO<sub>2</sub> pneumoperitoneum reversing.

**Table 6 Intergroup comparison (group II v/s III) of mean heart rate, systolic blood pressure, mean arterial pressure, and end-tidal CO<sub>2</sub> Bonferroni test**

Stage	Mean Heart Systolic Blood	Mean End-Tidal Rate	Pressure	PressureCO <sub>2</sub>
Immediately during insufflation	–	0.370	–	0.019
5min after CO <sub>2</sub> insufflation	–	–	0.915	0.0001
10min after CO <sub>2</sub> insufflation	0.001	0.190	0.087	0.0001
20min after CO <sub>2</sub> insufflation	0.01	–	0.052	0.0001
30min after CO <sub>2</sub> insufflation	–	–	–	–
At exsufflation	0.001	–	–	0.0001
10min after exsufflation	0.001	0.002	0.001	0.0001

At 20 minutes after CO<sub>2</sub> insufflation and 10 minutes after CO<sub>2</sub> exsufflation, there was a substantial difference between groups II and III (Table 6). Table 5 shows substantial statistical

differences between groups I and III at insufflation, 5, 10, 20, and 30 minutes after CO<sub>2</sub> insufflation, at exsufflation, and 10 minutes after exsufflation (Table 5) [15, 20].

*Analysis of EtCO<sub>2</sub>* End-tidal CO<sub>2</sub> increased in all three groups immediately after insufflation, and the increase in EtCO<sub>2</sub> persisted as the length of CO<sub>2</sub> insufflation extended until exsufflation. The mean values in all three groups were greater than the baseline 10 minutes after exsufflation (Table 4). The difference in EtCO<sub>2</sub> between groups I and III (Table 5) and group II and III (Table 6) was highly significant statistically immediately after insufflation, and the same trend continued until the end of operation and even 10 minutes after exsufflation.

These findings reveal that when groups I and II were compared to group III (high pressure group), there was a significant difference at all phases of operation following CO<sub>2</sub> insufflation [20, 21]. The increase in EtCO<sub>2</sub> following CO<sub>2</sub> insufflation is explained by CO<sub>2</sub> absorption due to a greater CO<sub>2</sub> tension gradient between the pneumoperitoneum and the blood perfusing the peritoneum. The large pressure gradient and greater CO<sub>2</sub> absorption can explain the higher EtCO<sub>2</sub> levels at the end of operation.

## CONCLUSIONS

The study resulted in the following conclusions. Intraoperatively, laparoscopic cholecystectomy causes considerable hemodynamic alterations. The majority of pathophysiological alterations are generated by CO<sub>2</sub> insufflation and are related to the cardiovascular system. When compared to low intra-abdominal pressure, high intra-abdominal pressure caused by CO<sub>2</sub> insufflation is related with higher hemodynamic oscillations and increased peritoneal CO<sub>2</sub> absorption. Laparoscopic cholecystectomy induces considerable hemodynamic alterations even in ASA grade I and II patients. Although these physiological changes do not usually necessitate intervention, they do necessitate constant intraoperative monitoring. Low-pressure pneumoperitoneum is appropriate for laparoscopic cholecystectomy because it reduces CO<sub>2</sub> insufflation's unfavourable hemodynamic effects.

## REFERENCES

1. Sgambati SA, Ballantyne GH (1996) History of minimally invasive colorectal surgery. In: Jager RM, Steven D, Wexner SD (eds) Laparoscopic colorectal surgery. Churchill Livingstone, New York, pp 13–22
2. Haubrich WS (1987) History of endoscopy. In: Sivak MV (ed) Gastroenterologic endoscopy. WB Saunders, Philadelphia
3. Lightdale CJ (1982) Laparoscopy and biopsy in malignant liver disease. *Cancer* 11:2672
4. Dubois F, Icard P, Berthelot G (1990) Celioscopic cholecystectomy: preliminary report of 36 cases. *Ann Surg* 211:60
5. Marco AP, Yeo CJ, Rock P (1990) Anaesthesia for patient undergoing laparoscopic cholecystectomy. *Anaesthesiology* 73:1268–1270
6. Jayashree S, Kumar VP (2003) Anaesthesia for laparoscopic surgery. *Indian J Surg* 65:232–240
7. Cunningham AJ, Turner J, Rosenbaum S, Rafferty T (1993) Transoesophageal echocardiographic assessment of hemodynamic functions during laparoscopic cholecystectomy. *Br J Anaesth* 70:621–625
8. Magno R, Medegrad A, Bengtsson R, Tronstad SE (1979) Acid base balance during laparoscopy. The effects of intraperitoneal insufflation of CO<sub>2</sub> and nitrous oxide on acid base balance during controlled ventilation. *Acta ObstetGynecolScand* 58:81–85
9. Safran D, Sgambatis S, Orlando R (1993) Laparoscopy in high risk cardiac patients. *Surg Gynecol Obstetrics* 176:548–554
10. Evitt MA, Singh K (2007) Physiology of minimal access surgery. In: *Pediatrics. minimal access surgery*, 15 Nov

11. Cunningham AJ, Brull S (1993) Laparoscopic cholecystectomy: anaesthetic implication. *AnaesthAnalg* 76:1120–1133
12. Gutt CN, Oniu T, Mehrabi A, Schemmer P, Kashfi A, Kraus T (2004) Circulatory and respiratory complications of carbon dioxide insufflation. *Dig Surg* 21:95–105
13. Dorsay DA, Greene FL, Baysinger CL (1995) Hemodynamic changes during laparoscopic cholecystectomy monitored with transesophageal echocardiography. *Surg Endosc* 9:128–134
14. Berg K, Wilhelm W, Grundmann U, Ladenburger A, Feifel G, Mertzluft F (1997) Laparoscopic cholecystectomy—effect of position changes and CO<sub>2</sub> pneumoperitoneum on hemodynamic, respiratory and endocrinologic parameters. *ZentralblChir* 122:395–404
15. Dexter SPL, Vucevic M, Gibson J, McMahan MJ (1999) Hemodynamic consequences of high- and low-pressure cap- noperitoneum during laparoscopic cholecystectomy. *Surg Endosc* 13:376–381
16. Critchley LAH, Gin T (1993) Hemodynamic changes in patients undergoing laparoscopic cholecystectomy: measurement by trans- thoracic electrical bioimpedance. *Br J Anaesth* 70:681–683
17. Chopra G, Singh DK, Jindal P, Sharma UC, Sharma JP (2008) Haemodynamic, end-tidal carbon dioxide, saturated pressure of oxygen and electrocardiogram changes in laparoscopic and open cholecystectomy: A comparative clinical evaluation. *The Internet Journal of Anesthesiology* 16:1
18. Korkmaz A, Alkis M, Hamamci O (2002) Hemodynamic changes during gaseous and gasless laparoscopic cholecystectomy. *Surg Today* 32:685–689
19. Sood J, Jayaraman L, Kumra VP, Pradeep C (2006) Laparoscopic approach to pheochromocytoma: is a lower intraabdominal pres- sure helpful. *AnaesthAnalg* 102:637–641
20. Rishimani AS, Gautam SC (1996) Hemodynamic and respiratory changes during laparoscopic cholecystectomy with high and re- duced intraabdominal pressure. *Surg LaparoscEndosc* 6:201–204
21. Baraka A, Jabbour S, Hammoud R, Aouad M, Najjar F, Khoury G, Sibai A (1994) End-tidal carbon dioxide tension during laparo- scopic cholecystectomy: correlation with the baseline value prior to carbon dioxide insufflation. *Anaesthesia* 49:304–306