Brief Communication

Changes in respiratory mechanics during extraperitoneal insufflation in inguinal hernia surgery

INTRODUCTION

Most laparoscopic surgeries involve intraperitoneal insufflation gas in the abdominal cavity, while the extraperitoneal approach is commonly used for various laparoscopic procedures such as inguinal hernia repair, prostatectomy and varicocelectomy.[1]

The adverse respiratory and cardiovascular changes seen during intraperitoneal insufflation have been extensively studied.[2-5] There are several reports on the haemodynamic effects of extraperitoneal insufflation, but there is sparse data to evaluate the effect of extraperitoneal insufflation on changes in respiratory mechanics.[1,6]

METHODS

After institutional ethics committee approval and written informed consent from the patients, 100 male patients, belonging to the American Society of Anesthesiologists Physical Status I and II in the age group of 18–75 years (body mass index <30 kg/m^2) undergoing TEP were included in this prospective cohort study.

After arrival in the operation theatre, intravenous access was obtained. Monitoring included noninvasive blood pressure, electrocardiogram, oxygen saturation, end-tidal CO_2 ($EtCO_2$) and neuromuscular monitoring.

Anaesthesia was induced with midazolam 1 mg, fentanyl 2 μg/kg and propofol 2 mg/kg intravenously. Vecuronium bromide 0.1 mg/kg intravenously was used for muscle relaxation followed by endotracheal intubation. Anaesthesia was maintained with 1-2% sevoflurane in 50% O_2 and N_2O gas mixture. Intraoperative analgesia was provided with incremental doses of intravenous fentanyl 1 μg/kg and muscle relaxation maintained with intermittent boluses of vecuronium bromide 0.02 mg/kg intravenously.

Ventilation was performed using the Penlon AV 900 ventilator with a rebreathing circuit incorporating a CO_2 absorber. A continuous fresh gas flow of 1.6 L/min (0.8 L O_2 and 0.8 L N_2O), an inspiratory to expiratory ratio of 1:2 and zero end-expiratory pressure was applied. The tidal volume was set at 8 ml/kg and respiratory rate adjusted to maintain $EtCO_2$ between 35 to 45 mmHg throughout the duration of surgery.

We designed this prospective, observational cohort with the primary objective of studying the dynamic effects of extraperitoneal CO_2 insufflation in patients undergoing elective laparoscopic extraperitoneal inguinal hernia repair (total extraperitoneal [TEP]) with respect to changes in respiratory mechanics, namely, dynamic respiratory compliance (Cdyn) and also airway resistance (R), work of breathing (WOB), peak inspiratory pressure (PIP) and minute ventilation (MV). The secondary objective was to evaluate the changes in haemodynamics i.e., heart rate (HR) and mean arterial blood pressure (MAP).

The respiratory mechanics were monitored using a side stream spirometry device (respiratory mechanics module, GE medical system, Milwaukee USA) which displayed the readings of Cdyn, WOB, R, PIP and MV.

The insufflation pressure of CO_2 was limited to 12 mmHg and maintained throughout the surgery with the patient in neutral position.

The recordings (Cdyn, WOB, R, PIP, MV, HR, SBP, DBP and MAP) were made 5 min after tracheal intubation, every 10 min after insufflation, at the time of deflation, 5 min after deflation and 5 min after tracheal extubation. At the end of surgery, the neuromuscular block was reversed with intravenous neostigmine methylsulphate 50 μg/kg and glycopyrrolate 10 μg/kg.

The statistical analysis was performed using the SPSS program for Windows, version 17.1 (SPSS, Chicago, Illinois, USA).

RESULTS

A total of 100 patients were included in the study. Eighteen patients were excluded post-recruitment from the analysis as they developed significant intraperitoneal CO_2 insufflation during surgery. The average age (53.3 ± 15.49 years), weight (70.87 ± 10.32 kg). Amount of CO2 insufflation (50.71 ± 23.67 L) were noted. We noted three patients with subcutaneous emphysema of chest wall, not extending to the neck, after surgery. The respiratory parameters

observed during the study are shown in Table 1, and the haemodynamic values are provided in Table 2.

DISCUSSION

The results from our study demonstrate that after extraperitoneal insufflation, the dynamic lung compliance decreased by about 37% from baseline after 10 min of CO_2 insufflation ($P < 0.01$). After 5 min of deflation, the compliance did not return to the baseline value ($P < 0.01$). The decreased lung compliance and increased PIP reflect reduced diaphragmatic excursion and premature closure of small airways resulting in reduced functional residual capacity.

A preliminary study conducted on nine patients by Bordes *et al*. reported a significant decrease in end expiratory lung volume from 2115 ± 635 ml to 1716 ± 444 ml and thoracopulmonary compliance from 49.5 ± 6.3 to 40.1 ± 5 ml/cmH_2O at 5 min after extraperitoneal insufflation, which is similar to our observations. They also observed that end-expiratory lung volume increased during application of 10 cmH_2O PEEP which also homogenised ventilation distribution.[7]

These findings led us to assume that extraperitoneal insufflation effects are similar to those of pneumoperitoneum where there was also increase in the abdominal cavity pressure accompanied with cranial movement of the diaphragm. CT-scan study performed during extraperitoneal insufflation may help in corroborating this mechanism, as previously reported by Andersson during intraperitoneal insufflation in patients undergoing laparoscopic cholecystectomy.[8]

The cephalad displacement of the diaphragm and decreased pulmonary compliance may impair respiratory muscle efficiency and increase the work of breathing, as has been observed in our study.

After CO_2 insufflation, the MV required to maintain $EtCO_2$ between 35-45 mmHg was achieved by increasing the respiratory rate. These findings emphasise that there is a significant absorption of CO_2 after extraperitoneal insufflation.

We noted a significant increase in HR and MAP till 20 min after insufflation which settled thereafter. This can be explained by the increased sympathetic output as well as initial increase in preload and cardiac output following CO_2 insufflation. Our results

Table 1: Respiratory parameters

Time points	Compliance (ml/cmH_2O)	Resistance (cmH_2O/l/s)	WOB (J/min)	PIP (cmH_2O)	MV (l)
5 ATI	75.38±36.27	12.60±7.49	0.73±0.26	12.15±3.03	5.85±1.99
10 A INSU	45.90±20.77*	13.43±4.17	0.95±0.26*	15.90±3.20*	5.41±1.03*
20 A INSU	40.46±17.06*	13.17±3.24	1.00±0.27*	16.81±3.13*	5.85±1.21
30 A INSU	36.88±15.60*	13.24±3.22	1.06±0.31*	17.72±3.08*	6.35±1.42
40 A INSU	37.21±19.10*	14.07±3.75*	1.08±0.38*	18.16±3.29*	6.69±1.34*
A DEF	55.68±29.86*	11.51±3.65	0.85±0.30*	15.44±3.51*	6.79±1.38*
5 A DEF	58.23±31.83*	11.23±4.14	0.86±0.31*	15.10±3.38*	6.70±1.52*

*Statistically significant values compared to corresponding baseline value which is 5 ATI in this table. Data presented as mean±SD. A $P<0.05$ was considered statistically significant. 5 ATI – 5 min after tracheal intubation; 10 A INSU – 10 min after insufflation; 20 A INSU – 20 min after insufflation; 30 A INSU – 30 min after insufflation; 40 A INSU – 40 min after insufflation; A DEF – Just after deflation; 5 A DEF – 5 min after deflation; WOB – Work of breathing; PIP – Peak inspiratory pressure; MV – Minute ventilation; SD – Standard deviation

Table 2: Haemodynamic parameters

Time points	Heart rate (beats/min)	SBP (mmHg)	DBP (mmHg)	MAP (mmHg)
PI	78.48±15.44	142.99±17.62	82.87±11.00	104.93±13.87
5 ATI	74.33±15.28	122.66±22.75	74.52±13.06	91.18±15.96
10 A INSU	68.94±13.59*	137.39±24.78*	85.46±13.78*	105.18±17.01*
20 A INSU	69.05±12.20*	131.69±22.92*	81.17±12.27*	99.40±16.15*
30 A INSU	71.78±12.62	125.43±20.40	78.82±12.52	95.49±14.83
40 A INSU	71.20±12.34	125.45±20.40	77.16±10.69*	94.59±13.99
A DEF	70.53±12.61*	125.44±18.87	75.79±10.80	93.85±14.61
5 A DEF	72.79±13.86	125.91±19.72	76.81±12.57	94.05±14.44
5 A EXT	84.00±14.55*	150.44±18.82*	83.51±10.81*	103.83±13.48*

*Statistically significant values compared to corresponding baseline value which is 5 ATI in this table. Data presented as mean±SD. A $P<0.05$ was considered statistically significant. PI – Pre-induction; 5 ATI – 5 min after tracheal intubation; 10 A INSU – 10 min after insufflation; 20 A INSU – 20 min after insufflation; 30 A INSU – 30 min after insufflation; 40 A INSU – 40 min after insufflation; A DEF – Just after deflation; 5 A DEF – 5 min after deflation; 5 A EXT – 5 min after tracheal extubation; MAP – Mean arterial pressure; DBP – Diastolic blood pressure; SBP – Systolic blood pressure; SD – Standard deviation

indicate a significant rise in MAP in contrast to a study by Wright *et al*. where no changes were reported in the mean arterial pressure.[1]

The limitation of our study is that we have observed the effects of extraperitoneal insufflation in neutral position. However, as head down position and insufflation pressure of more than 12 mmHg for TEP may be practiced by a majority of surgeons, these changes may get exaggerated and have further deleterious effects on respiratory mechanics and haemodynamic parameters.

CONCLUSION

Extraperitoneal insufflation causes significant impairment in respiratory mechanics and haemodynamics. As the TEP approach has gained ascendance, our results call for further research in ways to minimise its consequences in patients with reduced cardiopulmonary reserve.

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Conflicts of interest
There are no conflicts of interest.

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