Original Article

Influence of intra-abdominal pressure on ventilatory mechanical power delivery and respiratory driving pressure during laparoscopic cholecystectomy: A prospective cohort study

Unnikannan Shaji, Gaurav Jain, Debendra Kumar Tripathy, Navin Kumar[1], Nilotpal Chowdhury[2]

Departments of Anaesthesiology, [1]Surgery, [2]Pathology, All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India

Abstract

Background and Aims: Pneumoperitoneum creation for laparoscopic surgery increases the intraabdominal pressure and causes alveolar atelectasis. We investigated the influence of an increase in intra-abdominal pressure (IAP) on ventilatory mechanical power (MP) delivery during pneumoperitoneum creation for laparoscopic cholecystectomy.
Material and Methods: In a prospective cohort design, we enrolled 42 patients undergoing laparoscopic cholecystectomy. During pneumoperitoneum creation, the IAP was sequentially raised to three predefined IAP levels (8, 11 and 14 mmHg), keeping identical ventilatory settings (timepoints T1, T2, and T3). After that, positive end-expiratory pressure (PEEP) was sequentially raised from 5 to 8 to 11 cmH_2O (timepoint T4 and T5). The primary outcome included ventilatory MP delivery at each timepoint. Other variables included respiratory driving pressure (DP), airway resistance (AR), and respiratory compliance (RC).
Results: The MP increased linearly with a rise in IAP from T1 to T3 ($r = 0.71$, $P < 0.001$); the MP increased by 0.19 per unit rise in IAP (effect size 0.90, $P < 0.001$). A similar positive correlation was also observed between DP and IAP from T1 to T3 ($r = 0.73$, $P < 0.001$); the DP increased by 0.72 per unit rise in IAP (effect size 0.89, $P < 0.001$). The MP increased significantly on increasing PEEP from T3 to T5, while the DP decreased concomitantly ($P < 0.001$). The AR increased significantly from T1 to T3, while RC decreased concomitantly; vice-versa was observed at T4 and T5 ($P < 0.001$).
Conclusions: The ventilatory MP delivery rises linearly with an increase in IAP. Targeting an IAP-guided MP level could be an attractive approach to minimize lung injury.

Keywords: Laparoscopic surgery, lung injury, pneumoperitoneum, positive end-expiratory pressure, positive pressure ventilation

Introduction

During the delivery of ventilatory breath, a significant quantum of energy is transferred to the respiratory system to overcome the airway resistance (AR) to gas movement (flow-resistive work), negate the respiratory elastic recoil, and expand the thoracic and pulmonary wall (tidal volume [TV]-associated work).[1] This delivered energy per unit of time is called mechanical power (MP; J/min). A fraction of this energy is directly transferred to the lung parenchyma, deforming the anchored tissue architecture. Thus, higher delivered MP leads to lung injury. Pneumoperitoneum creation during laparoscopic surgery increases the intra-abdominal pressure (IAP). It causes cranial displacement of diaphragm, resulting in increased intrathoracic pressure, decreased lung compliance, basal lung tissue compression, alveolar

Address for correspondence: Dr. Gaurav Jain,
Department of Anaesthesiology, All India Institute of Medical Sciences, Virbhadra Marg, Rishikesh, Uttarakhand - 249 203, India.
E-mail: icubhu@gmail.com

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atelectasis, and impaired gaseous exchange.[2] These effects are compounded by positive pressure ventilation due to preferential ventilation of apical lung regions. Therefore, the ventilator needs to deliver higher energy during laparoscopic surgery to compensate for associated atelectasis and optimize the ventilatory pattern at the expense of increased airway pressures. It should ultimately lead to higher MP delivery to the respiratory system and, thus, possible lung injury. Therefore, targeting a lower MP may help clinicians plan a lung-protective ventilation strategy.[3,4] There is paucity of studies focusing on targeted MP based on IAP values to minimize lung injury. We hypothesized that an increase in IAP due to pneumoperitoneal insufflation in patients undergoing laparoscopic cholecystectomy should correlate linearly with higher ventilatory MP delivery during positive pressure ventilation. We investigated the influence of increased IAP on ventilatory MP delivery during pneumoperitoneum creation for laparoscopic cholecystectomy, at predefined ventilator settings. We also determined the impact of IAP and positive end-expiratory pressure (PEEP) on respiratory driving pressure (DP), AR, and respiratory compliance (RC) as secondary objectives.

Material and Methods

After institutional ethical approval and written informed consent were obtained, we included American Society of Anesthesiologists (ASA) grade I–II patients of either sex, aged 18–65 years, undergoing elective laparoscopic cholecystectomy under general anesthesia (GA), admitted between Feb 2021 and March 22, in this prospective, outcome assessor–blinded, cohort trial. We followed all ethical principles for medical research involving human subjects as per the Helsinki Declaration 2013. We excluded those with known cardiorespiratory/renal/neuromuscular disease, body mass index >30 kg/m^2, contraindication to PEEP like increased intracranial pressure or hypotension, pregnant and breastfeeding females, and those who had undergone chest/open abdominal surgery previously.

Patients were shifted to the operation theater, a multipara monitor was placed, and an intravenous (IV) line was secured. The baseline parameters were recorded. After preoxygenation (3 min), GA was induced with fentanyl (2 µg/kg IV), propofol (2 mg/kg IV), and vecuronium (0.10 mg/kg IV), and the trachea was intubated with appropriate-sized tracheal tube. The tube position was confirmed by end-tidal carbon-dioxide ($EtCO_2$) tracings. Mechanical ventilation was initiated on volume-controlled mode at a TV of 7 ml/kg of predicted body weight, Inspiratory: Expiratory ratio of 1:2, fresh gas flow of 2 l/min, PEEP of 5 cmH_2O, fractional inspiratory oxygen saturation (FiO_2) of 0.4 further titrated to achieve SpO_2 ≥94%, and a respiratory rate (RR) of 12/min further adjusted to achieve an $EtCO_2$ range of 35–38 mmHg. The upper limits of peak airway pressure (P_{peak}) and plateau pressure (P_{plat}) were kept at 35 and 30 cmH_2O, respectively. Anesthesia was maintained with sevoflurane at a minimum alveolar concentration (MAC) of 1.7, titrated further to keep the mean arterial pressure within ±20% of the baseline value. It followed surgical painting–draping and pneumoperitoneum creation through an insufflator (EleVision HD1 System; Medtronic, Dublin, Ireland) via a Veress needle under supine patient position. The IAP was gradually raised to 8 mmHg, and the outcome parameters were recorded after 2 min of stable IAP levels (Timepoint: T1). After that, the IAP was increased to 11 mmHg (Timepoint: T2) and 14 mmHg (Timepoint: T3) and the outcome variables were recorded at each level using a similar methodology. Once the IAP stabilized at 14 mmHg, PEEP was raised to 8 cmH_2O (Timepoint: T4) and then to 11 cmH_2O (Timepoint: T5); the corresponding outcome variables were recorded after 2 min of stabilization at each level. After that, the IAP was kept at 14 mmHg, PEEP was reduced to 5 cmH_2O, and the surgery was allowed to proceed. The primary variable included "MP," defined as energy transferred to the respiratory system by the mechanical ventilator per unit of time. It was calculated as (minute

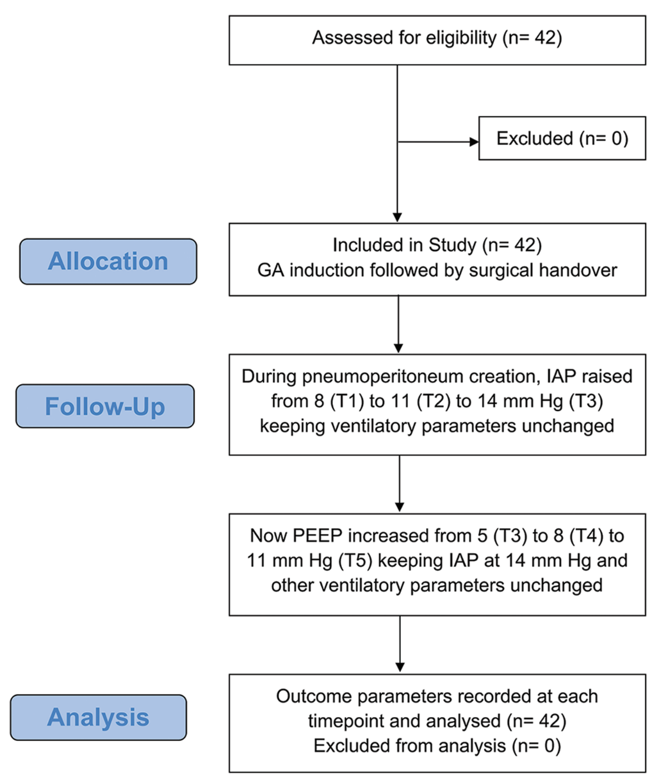

Figure 1: STROBE flowchart of patient's selection. GA = general anesthesia, T1–T5 = timepoints

ventilation × [P_{peak} + PEEP + flow rate/6])/20 and noted at all studied timepoints.[5] The secondary variables included DP, RC, and AR and were recorded at the same timepoints. "DP" was defined as the amount of cyclic pulmonary parenchymal deformation imposed on ventilated, preserved lung units and calculated as (P_{plat} − PEEP).

Statistical analysis

The sample size was estimated by G power statistical software (3.1.9.1®). Considering an uncorrected alpha error of 0.025 (corresponding to Bonferroni's correct alpha error of 0.05), beta error of 0.20, and an effect size of 0.5 (pilot observation), the sample size needed for a paired *t*-test analysis of MP data at all studied timepoints was 41. Considering a 2% dropout, we included 42 patients. Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) 23.0. The outcome variables were analyzed by bivariate correlation, scatter plot, and one-way analysis of variance (ANOVA) test; *post hoc* analysis was done using Bonferroni's method. To further analyze the impact of IAP on MP or DP and to identify the covariates, all outcome data were also analyzed by repeated measures ANOVA test through linear mixed-effect modeling, using R statistical software (4.2.1®) with "lmer 4," "lmer Test," and "effect size" packages. We tested different models for best fit and selected converging models with lower Akaike information criteria (AIC) value. The assumptions were checked by Pearson residual plot against the fitted values and by normal residuals plot. The effect size for IAP as a predictor for MP or DP was estimated by "partial eta^2" and "partial omega^2" values. A *P*-value < 0.05 was considered significant.

Results

We included 42 eligible patients over 14 months, with no dropouts [Figure 1]. A total of 210 values were obtained over T1–T5 timepoints. The patient demographic profile is summarized in Table 1. The majority of patients were within the 30–50 years age group (median age: 42 years, interquartile range [IQR]: 32.75–49), predominantly females (34, 81%), ASA grade I (28, 66.7%) patients, with hypertension being the significant comorbidity. The surgical procedure went

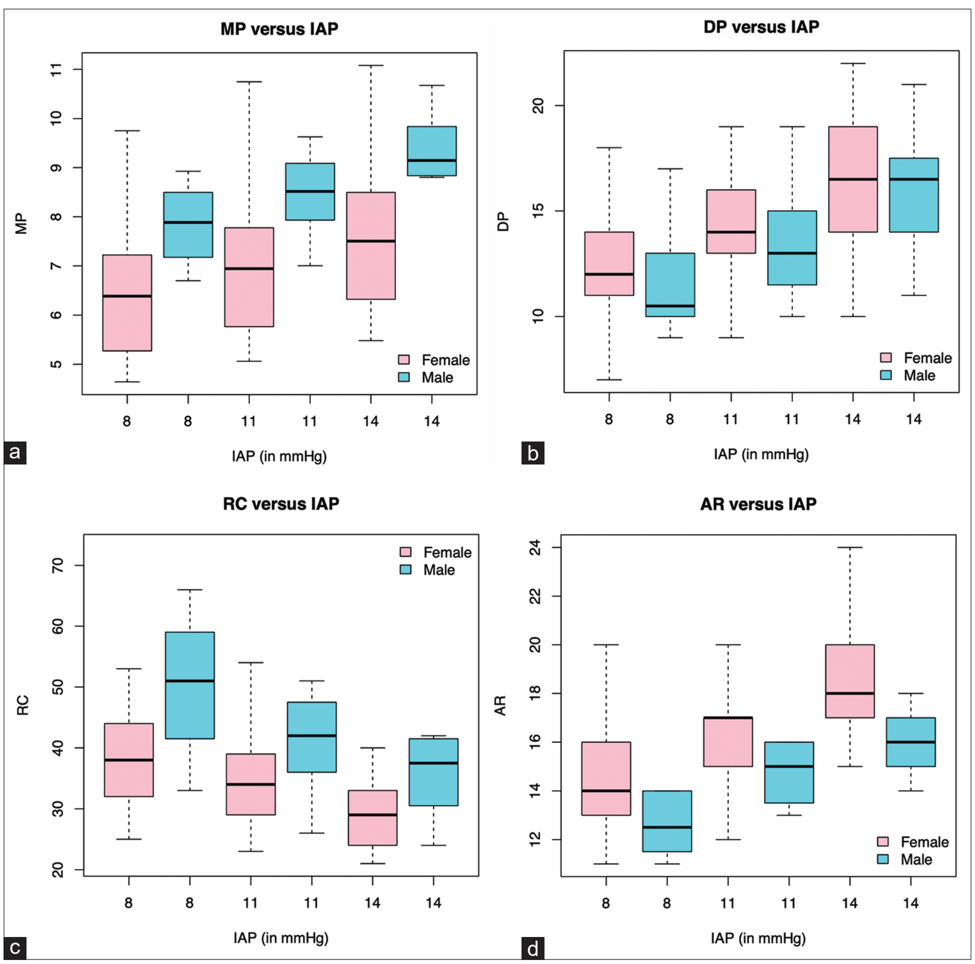

Figure 2: Box plot diagram showing mechanical power (a), driving pressure (b), respiratory compliance (c), and airway resistance. (d) Changes from T1 to T3 timepoints according to gender distribution

uneventful, with a median duration of 72.30 min (IQR: 63.45–81.15) [Table 1].

MP and DP increased significantly with a rise in IAP from T1 to T3 ($P < 0.001$); the increase in MP and DP occurred to a greater extent in males [Figure 2, Table 2]. The MP values were higher for males, while the DP values were elevated in females [Figure 2]. On bivariate analysis, the increase in MP had a positive linear correlation with a rise in IAP from T1 to T3 ($r = 0.71$, $P < 0.001$) [Figure 3]. A similar correlation was also observed between the increase in DP and IAP from T1 to T3 ($r = 0.73$, $P < 0.001$) [Figure 3]. A decline in RC, but an increase in AR was observed from T1 to T3; the decline in RC was more in males, while the increase in AR was more in females [Table 2, Figure 2]. On increasing PEEP from T3 to T5, an increase in MP and RC and a decline in DP and AR were observed ($P < 0.001$) [Table 2].

On linear mixed-effect modeling, with MP or DP as a dependent variable and patient as a random factor, we considered gender as the only add-on predictor. Including other baseline parameters resulted in unstable nonconverging models, so they were excluded from analysis. After applying different models for fitting data, we included three models as follows: model 1: IAP as categorical factor (univariate analysis), model 2: IAP as a continuous predictor (univariate analysis), and model 3: IAP as a continuous predictor and sex as another predictor (multivariate analysis). The univariate analysis for MP as a dependent variable indicated that IAP was a better fit as a continuous predictor (AIC [model 2]: 176.79) rather than a categorical one (AIC [model 1]: 179.58). The MP increased significantly ($P < 0.001$) by 0.19 per unit rise in IAP (mmHg) over and above the intercept value of 5.10 [model 2, Table 3]. Multivariate analysis indicated an even better fit (AIC: 169.49); both male gender and rise in IAP significantly predicted concomitant rise in MP ($P < 0.001$). In model 3, the MP increased by 0.19 per unit rise in IAP (mmHg) over and above the intercept value of 4.80, while male gender additionally increased the *MP* value by 1.56 [Table 3]. The partial

Table 1: Characteristics of included patients (*n*=42)

Variables	Value
Age (years)	40.93±11.04
Sex (females)	34 (81%)
Height (cm)	162.78±5.41
Weight (kg)	60.21±8.27
ASA grade I/II	28 (66.7%)/14 (33.3%)
Indication for surgery	
Acute cholecystitis	3 (6.6%)
Chronic cholecystitis	39 (93.4%)
Systolic blood pressure (mmHg)	127.24±14.84
Diastolic blood pressure (mmHg)	70.02±10.28
Heart rate (beats/min)	83.40±10.26
Respiratory rate (per min)	14.81±0.89
Body temperature (°C)	37.08±0.23
Duration of surgery (min)	72.30 (63.45–81.15)

Data presented as mean±standard deviation, number (percentage), median (interquartile range). ASA=American Society of Anesthesiologists

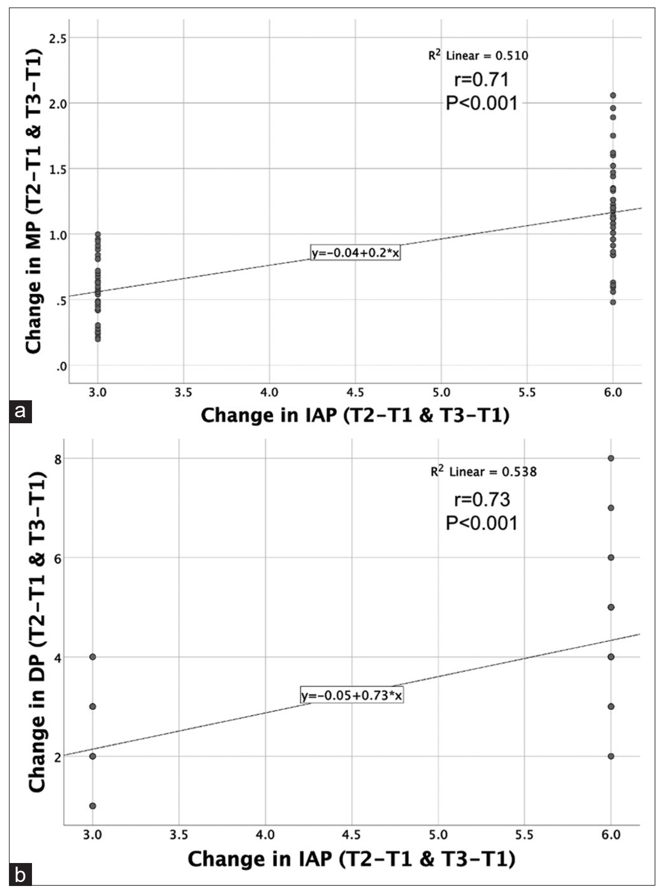

Figure 3: Scatter plot showing correlations between change in (a) MP and IAP (b) DP and IAP across various timepoints (T2–T1 and T3–T1 timepoints). DP = driving pressure, IAP = intra-abdominal pressure, MP = mechanical power

Table 2: MP, DP, RC, and AR at all predefined IAP and PEEP levels (*n*=42)

Parameters	T1	T2	T3	T4	T5	*P*
MP (J/min)	6.66±1.31	7.72±1.41	7.83±1.52	9.17±1.72	10.58±1.84	<0.001
DP (cmH_2O)	12.02±2.51	14.17±2.59	16.36±2.82	15.40±2.73	14.79±2.31	<0.001
RC (l/cm H_2O)	41.50±10.91	35.60±7.70	31.02±6.98	33.50±7.48	35.52±8.28	<0.001
AR (cmH_2O/l/s)	14.40±2.50	16.36±2.18	18.00±2.43	17.12±2.15	16.19±2.32	<0.001

Data presented as mean±standard deviation. A P<0.05 is considered significant. AR=airway resistance, DP=driving pressure, IAP=intra-abdominal pressure, MP=mechanical power, PEEP=positive end-expiratory pressure, RC=respiratory compliance

Table 3: Univariate and multivariate regression analyses of IAP and sex distribution as predictors of mechanical power and driving pressure (*n*=42)[a]

Group	Random effects	Fixed effects				
	Variance±SD	Estimate	SE	df	*t*	*P*
Model 2 (MP)						
Patients	1.96±1.40	-	-	-	-	-
Residuals	0.04±0.20	-	-	-	-	-
MP (intercept)	-	5.10	0.23	52.38	22.09	<0.001
IAP (T1–T3)	-	0.19	0.01	83	26.74	<0.001
Model 3 (MP and sex)						
Patients	1.62±1.27	-	-	-	-	-
Residuals	0.04±0.20	-	-	-	-	-
MP (Intercept)	-	4.81	0.23	50.89	20.62	<0.001
IAP (T1–T3)	-	0.19	0.01	83	26.74	<0.001
Male sex	-	1.56	0.50	39.99	3.10	0.003
Model 2 (DP)						
Patients	6.43±2.53	-	-	-	-	-
Residuals	0.56±0.75	-	-	-	-	-
DP (Intercept)	-	6.24	0.49	86.87	12.55	<0.001
IAP (T1–T3)	-	0.72	0.03	83	26.58	<0.001
Model 3 (DP and sex)						
Patients	6.54±2.56	-	-	-	-	-
Residuals	0.56±0.75	-	-	-	-	-
DP (Intercept)	-	6.35	0.53	76.73	11.85	<0.001
IAP (T1–T3)	-	0.72	0.03	83	26.58	<0.001
Male sex	-	−0.58	1.02	40	−0.57	0.569

A P<0.05 is considered significant. [a]Linear mixed-effect model was used with patients as the random variable, constituting a random effect on the intercept. df=degree of freedom, DP=driving pressure, IAP=intra-abdominal pressure, MP=mechanical power, SD=standard deviation, SE=standard error

eta^2 and partial omega^2 values for IAP as a predictor for MP were 0.90 (95% confidence interval [CI]: 0.86–0.92) and 0.89 (0.85–0.92), respectively. Similarly, univariate analysis for DP showed equivalent AIC values for model 1 (445.96) and model 2 (445.50). In model 2, the DP increased significantly ($P < 0.001$) by 0.72 per unit rise in IAP (mmHg) over and above the intercept value of 6.24. Multivariate analysis also had an equivalent fit (AIC: 445.30), with no effect of gender on MP (*P*: 0.569) [Table 3]. The partial eta^2 and partial omega^2 value for IAP as a predictor for MP was 0.89 (0.85–0.92). The model assumptions for both MP and DP were found to represent an adequate fit, as examined by Pearson residuals against the fitted values and by checking the approximate normality of residuals [Figure 4].

Discussion

An increased IAP during laparoscopic surgery is reflected as raised intrathoracic pressure and reduced chest wall/lung compliance.[2] Though GA induction reduces its effect on chest wall mechanics, intrathoracic pressure and lung mechanics are still adversely affected. Increased IAP also potentiates pulmonary edema by reducing the lymphatic outflow from lungs.[6] Our analysis shows that an increase in IAP results in a concomitant linear rise in MP at a strong effect size: the MP increased by 0.19 per unit rise in IAP (mmHg) over and above the baseline value of 5.1. Though the observed MP remained well below the risk limit for lung injury, the influence of IAP on MP could be greater in patients with cardiorespiratory disease, limited lung reserve, airway reactivity, or central obesity.[7] A varying head-down body position during pneumoperitoneum augments the diaphragmatic shift and potentiates its adverse effects.[8] A consistently elevated IAP causes a significant alteration in lung mechanics and is known to cause lung injury even with a few hours of mechanical ventilation.[9] The above conditions may result in a higher baseline MP value, with an amplified increase in MP with a rise in IAP during pneumoperitoneum, thus posing a risk for lung injury. A linear correlation between IAP and MP may, however, aid in optimizing intraoperative MP delivery by titrating IAP, if baseline and final M*P* values during pneumo-insufflation are known. Moreover, cogitating the strong impact of IAP on MP, the current formulae for MP calculation should also take into account the impact of IAP. The overall effect of the above conditions and IAP titration are subjected to future trials. We also observed that male gender additionally increased MP by 1.56. It could be related to the gender-based difference in baseline abdominal/chest wall compliance or fat distribution.[10]

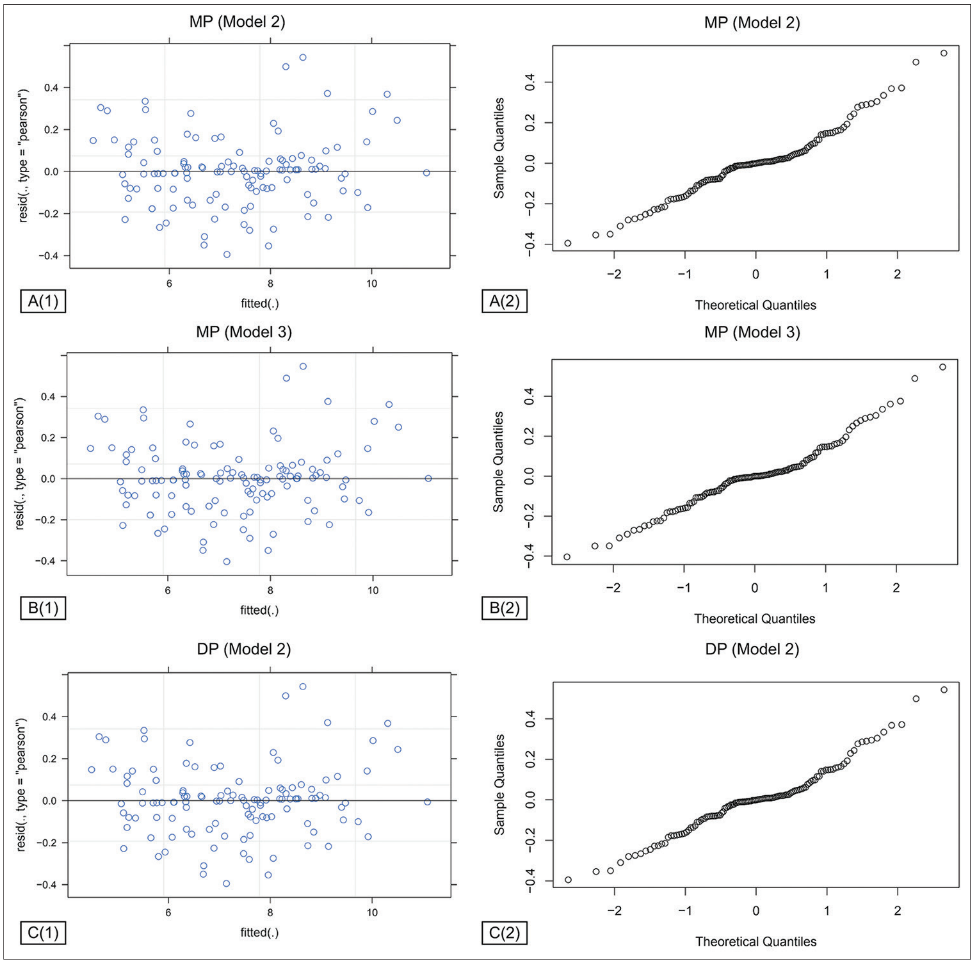

Figure 4: Scatter plot of Pearson residuals (1) and normality plot of residuals (2). (A) Mechanical power (model 2), (B) mechanical power (model 3), (C) driving pressure (model 2)

The application of PEEP during laparoscopy is believed to maintain the lung volume by reducing the cyclic alveolar collapse between each ventilatory breath. A higher targeted PEEP strategy has been recently shown to optimize alveolar ventilation and concomitantly reduce the increased transpulmonary DP due to increased IAP during laparoscopic surgery.[11] We observed that MP delivery increased further on increasing the PEEP from 5 to 11 cmH_2O over the three predefined timepoints (T3–T5), keeping other settings unchanged. Collino *et al*.[1] observed similar results in a piglet under prone ventilation showing a linear increase in MP with an increase in IAP, over and above the PEEP value of 8 cmH_2O. It indicates that PEEP should also be targeted in the context of MP delivery, as higher PEEP may result in higher MP delivery, consequent lung strain, pleural effusion through lymphatic compression, and compromised right ventricular function, especially in high-risk groups. A quest for optimal PEEP during pneumoperitoneum requires further trials.

A similar linear rise in DP was observed on increasing the IAP at a strong effect size; the DP increased by 0.72 per unit rise in IAP (mm Hg) over and above the baseline value of 6.24. With relation to gender, a higher RC explains the lower DP values in males, though gender had no impact on DP as a predictor variable. A reduction in D*P* values was observed on increasing PEEP from 5 to 11 cmH_2O (T3–T5). Mazzinari *et al*.[11] also showed that transpulmonary DP rises with an increase in IAP. Moreover, application of targeted PEEP (2 cmH_2O above IAP) in this study alleviated the increase in DP, an effect that was greater at higher IAP levels. However, the clinical implication of rise in MP but decline in DP on increasing PEEP remains uncertain. We noted that increase in IAP leads to a decline in RC, but a concomitant increase in AR (T1–T3). The decline in RC is an expected result, as increased IAP reduces chest wall compliance and increases lung atelectasis.[12] The AR is inversely proportional to the airway radius by a power of four. During expiration, increased intrathoracic pressure and loss of elastic lung recoil leads to narrowing of smaller airways and consequent increase in AR.[13] In the current study, similar physiology could have contributed to a rise in AR on increasing the IAP. This extrapolation is further signified by reduction in AR and rise in RC on increasing PEEP at T4 and T5. The increased PEEP improved the alveolar and smaller airway patency, which reduced AR and increased RC.[14,15]

This study has some strengths. First, all timepoints in the stepwise study protocol were practically achievable and precisely followed in all included patients. Second, stringent exclusion criteria resulted in a clear predefined analysis, limiting any possible biasness. Third, detailed repeated measures ANOVA using linear mixed-effect modeling was performed to include intra- and interpatient variability in the estimates and IAP was also analyzed as a continuous variable, not just a categorical one.

This study has a few limitations. First, we used a surrogate formula for calculating the MP. Although it has been proven to be accurate, results obtained by using other proposed formulae may still affect the observed relationship. However, considering a strong effect size, this study adds to current understanding of the effect of increased IAP on MP delivery by the ventilator during invasive ventilation. Second, due to financial constraints, we could not measure transpulmonary DP at the studied timepoints during pneumo-insufflation. A real-time capture of transpulmonary DP may add to the current understanding of the complex relationship between IAP and individual components of pulmonary mechanics. Third, we could not consider factors like social class, ethnicity, intraoperative time-dependent changes, and postoperative complications. We perceive our results as evidence of studied hypothesis with power estimates of a limited sample.

Conclusion

Ventilatory MP delivery increased linearly through three incremental IAP levels during pneumoperitoneum creation in patients undergoing laparoscopic cholecystectomy. We extrapolate that targeting an IAP-guided MP level could be an attractive approach to minimize lung injury, and this could be a subject of future trials.

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Nil.

Conflicts of interest
There are no conflicts of interest.

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