

Experimental Intra-abdominal Hypertension Influences Airway Pressure Limits for Lung Protective Mechanical Ventilation.

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ABSTRACT

Background:

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) may complicate monitoring of pulmonary mechanics due to their impact on the respiratory system. However, recommendations for mechanical ventilation of patients with IAH/ACS and the interpretation of thoraco-abdominal interactions remain unclear. Our study aimed to characterize the influence of elevated intra-abdominal pressure (IAP) and positive end-expiratory pressure (PEEP) on airway plateau pressure (P_{PLAT}) and bladder pressure (P_{BLAD}).

Methods:

Nine (n=9) deeply anesthetized swine were mechanically ventilated via tracheostomy: volume-controlled mode at tidal volume = 10 ml/kg, frequency=15, Inspiratory:Expiratory ratio=1:2 and PEEP of 1 and 10 cmH₂O (PEEP₁ and PEEP₁₀, respectively). A tracheostomy tube was placed in the peritoneal cavity and different levels of IAP (5, 10, 15, 20 and 25 mmHg) were applied utilizing a CPAP system. Bladder pressure and airway pressure measurements were recorded after 10 minutes of stabilization at each level of IAP. Measurements were performed during both PEEP₁ and PEEP₁₀.

Results:

P_{BLAD} increased as experimental IAP rose ($y=0.83x+0.5$, $R^2=0.98$; $p < 0.001$ at PEEP₁). Minimal underestimation of IAP by P_{BLAD} was observed (-2.5 ± 0.8 mmHg at IAP of 10-25 mmHg). Applying PEEP₁₀ did not significantly affect the correlation between experimental IAP and P_{BLAD} . Approximately ~50% of the P_{BLAD} (in cmH₂O) was reflected by changes in P_{PLAT} regardless of the PEEP level applied. Increasing IAP did not influence hemodynamics at any level of IAP generated in our study.

Conclusion:

With minimal underestimation, P_{BLAD} measurements closely correlated with experimentally regulated IAP independent of the PEEP level applied. For each PEEP level applied, a constant proportion (~50%) of measured P_{BLAD} (in cmH₂O) is reflected on P_{PLAT} . A higher safety threshold for P_{PLAT} should be considered in the setting of IAH/ACS as the clinician considers changes in V_T . A strategy of reducing tidal volume to cap P_{PLAT} may not be warranted in the setting of increased IAP.

Key words:

Intra-abdominal hypertension, abdominal compartment syndrome, plateau airway pressure, bladder pressure, positive end-expiratory pressure.

Introduction

Deleterious rise in intra-abdominal pressure (IAP) is commonly encountered among both surgical and medical patients who are critically ill. The incidence of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) varies according to the studied patient population. For example, some studies that include critically ill patients with associated traumatic injuries have reported an incidence ranging from ~2 to 70% for IAH and from ~0.5 to 36% for ACS [1-4]. In 2005, the World Society of Abdominal Compartment Syndrome (WSACS) developed consensus definitions outlining standards for IAP measurements as well as diagnostic criteria for IAH (IAP \geq 12 mmHg) and ACS (IAP \geq 20 mmHg or IAH associated with new organ dysfunction/failure) [1-2].

IAH and ACS have been recognized as important causes of morbidity and mortality in the setting of trauma, perioperative medicine, post-resuscitation states, and critical care medicine [1-3]. A well structured group of categorized risk factors (CRF) for IAH/ACS has been defined [2], which includes: conditions associated with diminished abdominal wall compliance, increased intra-luminal or total abdominal content, capillary leak syndrome or massive fluid resuscitation [2]. Estimation of IAP by standardized bladder pressure (P_{BLAD}) monitoring has been recommended by the WSACS as an important step within the IAH-assessment algorithm [2].

While the algorithm for diagnosis and management of IAH/ACS has proven effective in improving survival [2,5], recommendations for mechanical

ventilation of patients with IAH/ACS remain unclear. Critical care practitioners utilize a mechanical ventilation strategy with lower tidal volume (V_T) of ~ 6 mL/Kg of predicted body weight (PBW) and plateau airway pressure (P_{PLAT}) not exceeding 30 cmH₂O which has resulted in decreased mortality and shorter duration of mechanical ventilation, not only among patients with acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) but also in those with normal lungs [6-9]. However, in the setting of IAH/ACS, transmission of IAP to P_{PLAT} could complicate regulation of V_T to avoid deleterious levels of P_{PLAT} (>30 cmH₂O).

To help clarify the effect of increased IAP on standard critical care ventilation parameters, our study was designed to confirm the accuracy of P_{BLAD} in estimating a range of experimentally regulated IAP values, to evaluate the effect of different PEEP levels on bladder pressure measurements in this model, and to evaluate transmission of IAP to airway pressure during passive mechanical ventilation. Implications of our findings for mechanical ventilation strategy in the setting of IAH will be discussed.

Materials and Methods

This protocol was approved by the Animal Care and Use Committee of Regions Hospital (St. Paul, MN). Nine (n=9) young healthy Yorkshire pigs (mean weight 46.2 ± 6.2 kg) were premedicated with intramuscular telazol/xylazine (2.2 and 6.6 mg/kg, respectively) and after tracheostomy, received a continuous flow (0.08-2 L/min) of Isoflurane-50% + nitrous oxide inhalational mixture. No neuromuscular blocking agents were used. The preparation included a femoral venous catheter, carotid arterial line, pulmonary artery catheter, tracheostomy, and midline suprapubic cystostomy. The peritoneal cavity was accessed by surgical placement of a gas-tight tracheostomy tube (Covidien® Shiley™ Trach tube 7 – Mansfield, MA) and its intra-peritoneal position was confirmed by direct visualization using an optical fiberscope. A continuous positive airway pressure (CPAP) circuit was then connected to the abdominal tracheostomy tube. Using a 'y-shaped' connector, a tube extension was added to the system and then immersed in a graduated flask filled with water; the pressure applied to the peritoneal cavity was regulated by immersing the tube opening in a column of water, and by adjusting its depth to achieve the desired IAP value. To validate IAP, we measured bladder pressure via a standard fluid filled transducer system as recommended by the World Society of the Abdominal Compartment Syndrome (25mL normal saline vesical instillation) [1-2] and monitored the intra-peritoneal pressure by connecting the tracheostomy tube to an auxiliary pressure port of the mechanical ventilator. Inhalational anesthesia was slowly discontinued over approximately 30 min and replaced by a titrated IV drip

infusion of Telazol®, ketamine, and xylazine to maintain deep anesthesia, adjusted as indicated by intermittent reflex testing and continuous bi-spectral analysis (BIS) to assure that no breathing efforts could be observed in the airway pressure tracing [10]. Pigs were then ventilated using the Engström Carestation™ (GE Healthcare, Madison, WI): volume-controlled mode, square wave flow, tidal volume (V_T) = 10 ml/kg, frequency titrated to an $P_{ET}CO_2$ of 30-40 mmHg, I:E of 1:2, PEEP of 1 or 10 cmH₂O, no inspiratory pause, and FIO_2 of 0.5. At the end of each experiment, animals were euthanized by rapid injection of Euthasol®.

Experimental protocol

End-inspiratory plateau airway pressure (P_{PLAT}) and bladder pressure (P_{BLAD}) were evaluated across experimentally controlled levels of IAP and PEEP. Ventilator settings remained unchanged throughout the experimental protocol, except for recruitment maneuvers and PEEP. Before and after each change of IAP generated during ventilation with PEEP of 1 or 10 cmH₂O, recruitment maneuvers were performed, using 10 breaths of pressure-controlled ventilation (PCV) with inspiratory pressure of 40 cmH₂O and PEEP of 20 cmH₂O (PCV of 40/20). Because a minimum PEEP of 1 cmH₂O (PEEP₁) is a technical requirement for determining additional measurements (e.g. functional residual capacity-FRC) when using Engström Carestation™ mechanical ventilator, PEEP of 1 cmH₂O served as the end-expiratory airway pressure baseline. PEEP of 10 cmH₂O (PEEP₁₀) was selected due to its common use in clinical mechanical ventilation settings and its generous distending effect on normal lungs [11]. These two different lung stress conditions (PEEP₁ and PEEP₁₀) were used when

evaluating the effects of increased IAP on P_{PLAT} , and also to assess the potential influence of PEEP on bladder pressure measurements.

After randomizing the initial PEEP level (PEEP₁ or PEEP₁₀), the abdomen was insufflated (with air) to constant pressures of 0, 5, 10, 15, 20 and 25 mmHg (0, 7, 14, 20, 27 and 34 cmH₂O, respectively) in a randomized order and by using the previously described abdominal CPAP system. Afterward, all conditions were repeated in the same fashion at the remaining level of PEEP (PEEP₁ or PEEP₁₀, according to the randomization).

For each combination of IAP and PEEP, end-inspiratory plateau airway pressure (P_{PLAT}) was recorded during an end-inspiratory pause of 1.5 seconds. Bladder pressure was recorded after a 10 minutes stabilization period. Additionally, a simultaneous verification of the IAP generated by the abdominal CPAP system was performed as previously described. Baseline conditions were re-established (IAP=0 mmHg) after every IAP level was applied, and re-calibration of the bladder pressure sensor with verification of the fidelity of its pressure tracing was performed between the different IAP levels. Heart rate (HR), mean arterial pressure (MAP), and oxygen saturation (SatO₂) were monitored during the entire experiment and cardiac output (CO) was measured by thermodilution. All hemodynamic data and bladder pressure measurements were recorded 10 minute after every IAP level was established.

Statistical Analysis

Our dependent variables of interest were: (1) end-inspiratory P_{PLAT} , (2) bladder pressure. Each of these variables was described using means and standard errors

at each combination of the two independent variables of the study (IAP and PEEP). A linear model provided a close approximation to the relationship between bladder pressure and IAP – first aim of the study. The second aim of the study (to characterize the effects of IAP and PEEP upon end-inspiratory P_{PLAT}) was met by using a graphical depiction of the average values observed for the dependent variables across IAP, separately for each level of PEEP. Bladder pressure (P_{BLAD}) was expressed as a function of: PEEP, IAP, and the interaction between these pressure levels.

Results:

P_{BLAD} increased as experimental IAP elevated ($y=0.83x+0.5$, $R^2=0.98$; at PEEP1) $p < 0.001$ (figure 1). Minimal underestimation (mean $P_{\text{BLAD}}-IAP$) of IAP by P_{BLAD} was observed at PEEP1 when $IAP \geq 5$ mmHg. This difference between P_{BLAD} and IAP was more evident with experimental IAP values of 10 to 25 mmHg, with a mean difference of -2.5 ± 0.8 mmHg across that range (figure 1).

Applying PEEP10 did not significantly affect the correlation between experimental IAP and P_{BLAD} (figure 2). Predicted equations for y-variable P_{BLAD} as a function of the x-variable IAP were: $y=0.83x+0.5$, $R^2 = 0.98$ at PEEP1; and $y=0.8x+0.7$, $R^2 = 0.97$ at PEEP10 (figure 2). As can be seen by both the figure 2 and the linear model equations, there is no observable effect of PEEP upon the relationship between P_{BLAD} and experimental IAP. As applied, PEEP does not affect the slope or the intercept of the linear relationship between IAP and P_{BLAD} .

To extend the clinical applicability of our observations regarding the relationship between IAP and P_{PLAT} , we characterized the behavior of P_{PLAT} as a function of IAP as estimated by P_{BLAD} (Figure 3). For each level of PEEP, P_{BLAD} values in mmHg were converted to cmH_2O ($1 \text{ mmHg} = 1.36 \text{ cmH}_2\text{O}$). With P_{PLAT} and P_{BLAD} expressed in the same units (cmH_2O), we found strong correlation at both PEEP1 ($y=0.57x+10.8$, $R^2 = 0.97$) and PEEP10 ($y=0.51x+16.5$, $R^2 = 0.96$). Approximately $\sim 50\%$ of the P_{BLAD} (in cmH_2O) was transmitted to P_{PLAT} regardless of the PEEP level applied. (figure 3).

Increasing IAP did not influence measured variables of cardiovascular status (HR, MAP, Sat O₂, and CO) at any level of IAP generated in our study (table 1).

Discussion:

The primary findings of our study can be summarized: 1) P_{BLAD} tracked experimentally-regulated IAP with minimal underestimation. 2) Changes in PEEP did not influence the relationship between IAP and P_{BLAD} in our model. 3) For each combination of fixed tidal volume and PEEP, elevating IAP progressively increased plateau airway pressure (P_{PLAT}) by approximately ~50% of the applied IAP value. Although there are differences among IAP-generation techniques and the potential effect on the diaphragm due to increased IAP, our results support the work of other investigators describing the influence of IAP on hemodynamics and respiratory mechanics [12-14].

Our results emerged from a protocol designed to determine the accuracy of P_{BLAD} in estimating a range of precisely regulated IAP values using an air-based model that directly insufflated the peritoneal cavity and allowed uniform pressure distribution. Using this model, bladder pressure measurements (as recommended by the WSACS [1-2]) minimally underestimated experimental IAP. Differences between P_{BLAD} and IAP were more evident with IAP values of 10 mmHg and above (figure 1). By choosing an air-based technique, a uniform distribution of pressure inside the peritoneal cavity is generated, thereby ensuring a consistent impact on diaphragmatic configuration and function [15-16].

In our study, changing PEEP values did not affect the relationship between experimental IAP and measured P_{BLAD} . Some reports regarding the bi-directional interaction between the abdominal and thoracic compartments have emphasized

the effect of PEEP on IAP measurements and intra-abdominal perfusion pressure in the setting of IAH [17-22]. Our results demonstrate that changing PEEP from 1 to 10 cmH₂O does not affect the measured P_{BLAD} across a wide and clinically significant range of IAP values. Consistent with our experience, Jakob et al [18] reported that removing PEEP and maintaining “extra-abdominal” pressure (7 Kg weight) showed no effect upon P_{BLAD} [18]. Our study extends their findings and replicates a scenario closer to the actual clinical setting by using an IAP-generation method that allowed a uniform effect on the diaphragm [15-16,23].

To “isolate” the effect of PEEP on P_{BLAD} measurements, we implemented our protocol in healthy animals. Results from studies evaluating PEEP vs. IAP relationship in the setting of lung injury have differed quantitatively from ours [21-22]. For example, Verzilli and colleagues [22] found that among ALI/ARDS patients with concomitant IAH, PEEP affects IAP when the P_{BLAD} value is >12mmHg [22]. In contrast, our study supports a negligible effect of PEEP on measured IAP in the setting of IAH/ACS without lung injury (figure 2).

In a recent study, Regli et al [24] examined the effect of matching PEEP to the IAP on cardio-pulmonary parameters. In their preparation, a latex balloon was placed in the peritoneal cavity and inflated to generate different IAP levels; pressures inside the balloon were monitored for IAP measurements [24]. Matching PEEP to IAP values reversed IAH-induced reduction of lung volume, reduced cardiac output, but did not improve arterial oxygen tension. In this study, P_{PLAT} was expressed as a function of PEEP and IAP ($P_{PLAT} = 0.27 \text{ IAP}_{\text{cmH}_2\text{O}} + 1.03 \text{ PEEP} + 5.8$) which implies that ~27% of applied IAP (in cmH₂O) was transmitted to P_{PLAT} [24]. In our study, when analyzing the relationship between

measured P_{BLAD} and P_{PLAT} , we found that approximately 50% of the applied IAP (in cmH_2O) was transmitted to P_{PLAT} in the presence of both PEEP of 1 and 10 cmH_2O (figure 3). Using an intra-peritoneal air-filled balloon and applying higher PEEP levels, may explain the differences in IAP transmission between our findings and previous reports.

Our results regarding the effect of increased IAP on P_{PLAT} support and extend those reported by Torquato [12], Jakob [18], Regli [24] and colleagues. However, our model used regulated IAP and direct P_{BLAD} measurements instead of extra-abdominal weight or intra-balloon pressure monitoring. A more consistent distribution of gas used to raise IAP in our model may contribute to our results (figure 3).

Limitations

Limitations of this study must be acknowledged. The IAP-generation method we implemented is clearly different from pathologic conditions associated with ascites or edematous tissue, which could affect the transmission fraction of the experimentally regulated IAP to the bladder compartment. While compressibility of air and compliance of intra-abdominal contents may influence transmission of experimental IAP to the bladder compartment, our method allowed rapid return to baseline conditions ($\text{IAP} = 0 \text{ mmHg}$) and ensured uniform distribution of IAP on diaphragm [15-16,23]. The correlation between experimental IAP and P_{BLAD} supports the reliability of our technique in generating a range of clinically significant levels of IAP.

Clinical implications

Our results support the value of P_{BLAD} measurements in estimating a range of experimentally regulated IAP levels and confirm the clinical relevance of this measurement technique. Although attributable to the implemented air-based model for IAP-generation, minimal underestimation of IAP by P_{BLAD} (2-3 mmHg) should be considered when IAP is within the range of 10-25 mmHg. P_{BLAD} measurements appear insensitive to changes in PEEP in a clinically relevant range.

Regarding the effect of IAP upon P_{PLAT} , in the absence of lung injury or active breathing, our observations suggest that once IAP is estimated by P_{BLAD} and converted to cmH_2O , approximately 50% of that pressure is transmitted to the thorax and reflected in P_{PLAT} . Knowing that an effect of increased IAP may be reflected in measured P_{PLAT} (~50% of transmission); the practitioner should interpret P_{PLAT} with caution when considering reductions in V_{T} to prevent hazardous lung pressure exposure. Assessment of the “true P_{PLAT} ” should be performed when treating patients with IAH/ACS. Implementation of conservative V_{T} values based on airway pressure limits to maintain $P_{\text{PLAT}} < 30 \text{ cmH}_2\text{O}$ could cause hypoventilation and tidal opening and collapse potentially avoidable by adequate changes in V_{T} and PEEP.

Although rarely implemented in the clinical setting, monitoring esophageal pressure and calculating transpulmonary pressure have been proposed as a guide to adjust mechanical ventilation in patients with ALI/ARDS [25]. This technique could also be helpful in further assessing the effects of elevated IAP on lung inflation pressures. Our findings, however, suggest the

necessity for re-interpretation of standard airway pressure limits, routinely used in clinical practice for lung protective mechanical ventilation in the setting of IAH/ACS.

Conclusions

While insensitive to changes in PEEP, bladder pressure measurements closely correlate with experimentally regulated IAP over a range of 0-25 mmHg. For each PEEP level applied, approximately 50% of bladder pressure (in cmH₂O) is reflected on the P_{PLAT} at this tidal volume. A higher threshold for P_{PLAT} could be acceptable in the setting of IAH/ACS as the clinician considers changes in V_T. Replication of our experiment in the setting of lung injury will extend its applicability to the clinical setting.

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FIGURE LEGENDS

Figure 1. Minimal underestimation of experimental IAP by bladder pressure measurements: P_{BLAD} consistently increased as experimental IAP elevated. Minimal underestimation (mean $P_{\text{BLAD}} - \text{IAP}$) of IAP by P_{BLAD} was observed at PEEP₁ when $\text{IAP} \geq 5$ mmHg. Mean values with standard errors are illustrated. *IAP (intra-abdominal pressure), P_{BLAD} (measured bladder pressure), PEEP (positive end-expiratory pressure).*

Figure 2. Changes in PEEP did not affect the relationship between experimental IAP and measured P_{BLAD} : Changing PEEP from 1 to 10 cmH₂O did not affect measured P_{BLAD} across a wide and clinically significant range of IAP values. Mean values with standard errors are illustrated. *IAP (intra-abdominal pressure), P_{BLAD} (measured bladder pressure), PEEP (positive end-expiratory pressure).*

Figure 3. Relationship of airway plateau pressure to measured P_{BLAD} : Approximately 50% of measured P_{BLAD} (in cmH₂O) corresponds to changes in P_{PLAT} with tidal volume held constant at both PEEP₁ and PEEP₁₀. Mean values with standard errors are illustrated. *IAP (intra-abdominal pressure), P_{PLAT} (airway plateau pressure), PEEP (positive end-expiratory pressure).*

Figure 1.

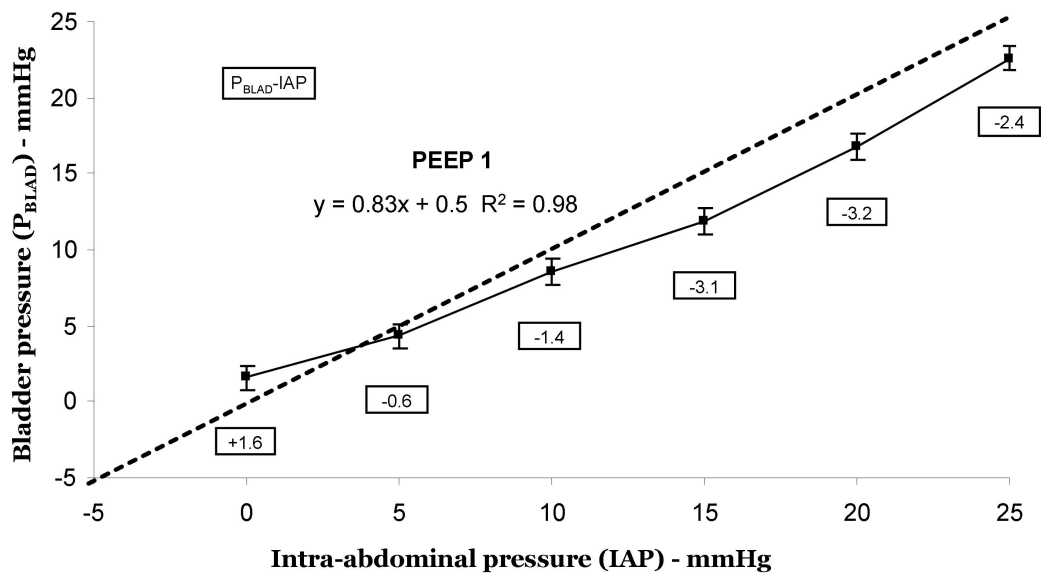


Figure 2.

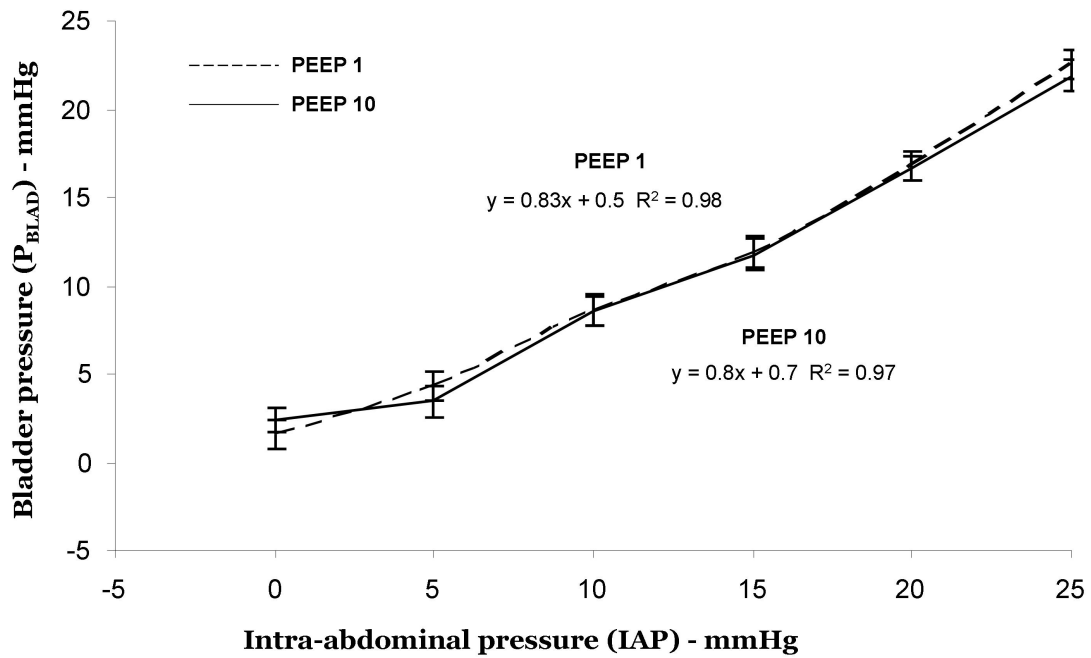


Figure 3.

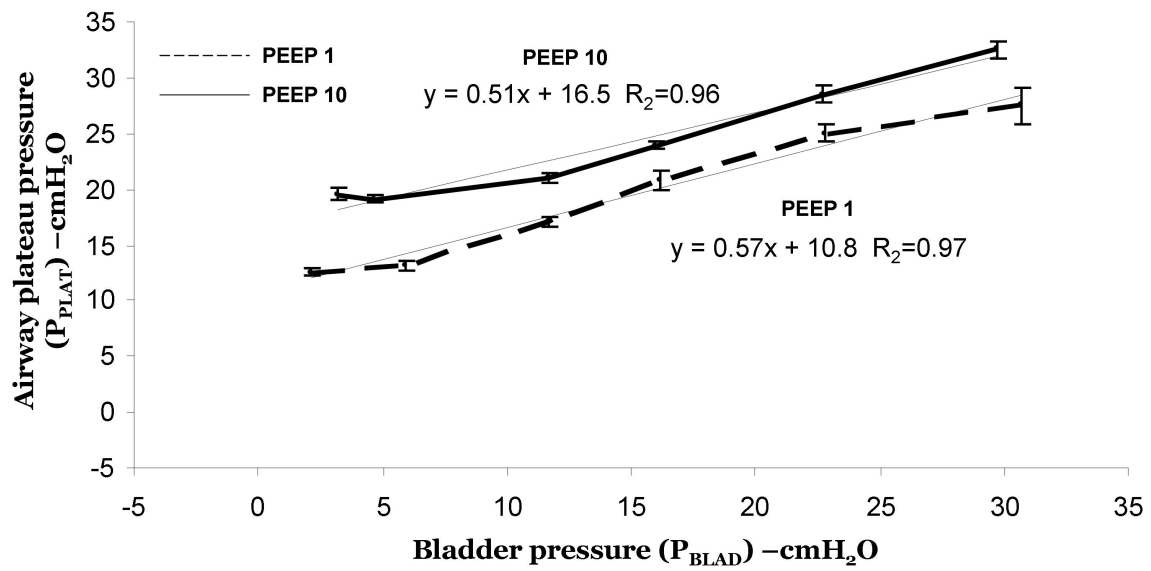


Table 1. Hemodynamics and bladder pressure measurements.

IAP	0 mmHg (0 cmH ₂ O)		5 mmHg (7 cmH ₂ O)		10 mmHg (14 cmH ₂ O)	
	1	10	1	10	1	10
PEEP						
HR	58.3 ± 6.4	64.4 ± 7.8	58.9 ± 8.2	63.7 ± 8.9	61.4 ± 18.9	61.4 ± 20.5
SAP	111.2 ± 19.9	96.5 ± 19.2	114.4 ± 22.2	102.4 ± 17.1	117.1 ± 23.6	110 ± 16.8
DAP	69.3 ± 11.7	61.1 ± 14.7	70.3 ± 12.6	62.8 ± 14.6	71.6 ± 13.7	66.9 ± 14.7
MAP	87.7 ± 13.3	74.6 ± 18.4	89.5 ± 14.7	78.8 ± 15.9	91.5 ± 15.1	84.3 ± 15.8
CO	3.6 ± 0.7	2.8 ± 0.6	3.7 ± 0.6	2.8 ± 0.5	3.7 ± 0.6	2.9 ± 0.8
P bladder	1.6 ± 2.3	2.4 ± 2.1	4.3 ± 2.2	3.4 ± 2.6	8.5 ± 2.4	8.6 ± 2.4
IAP	15 mmHg (20 cmH ₂ O)		20 mmHg (27 cmH ₂ O)		25 mmHg (34 cmH ₂ O)	
	1	10	1	10	1	10
PEEP						
HR	59.6 ± 8.8	61.1 ± 15.6	62.9 ± 16.6	62 ± 21.6	58.5 ± 7.5	63.6 ± 16.2
SAP	115.8 ± 21.9	118.5 ± 19.5	112.9 ± 20.9	112.5 ± 16.3	110.5 ± 22.7	116.7 ± 11.6
DAP	71.9 ± 12.3	75.7 ± 13.5	71.8 ± 12.1	70.1 ± 14.9	70.9 ± 14.1	74.6 ± 8.3
MAP	90.6 ± 14.8	93.1 ± 14.6	89.4 ± 14.6	87.1 ± 15.7	86.6 ± 15.9	90.6 ± 8.4
CO	3.8 ± 0.9	3.3 ± 0.6	3.6 ± 0.9	3 ± 0.6	3.2 ± 0.5	3.2 ± 0.9
P bladder	11.9 ± 2.6	11.8 ± 2.6	16.7 ± 2.3	16.7 ± 1.9	22.5 ± 2.4	21.9 ± 2.6

Increasing IAP did not influence measured variables of cardiovascular status (HR, MAP, Sat O₂, and CO) at any level of IAP generated in our study. *IAP* (intra-abdominal pressure), *PEEP* (positive end-expiratory pressure, cmH₂O), *HR* (heart rate, bpm), *SAP* (systolic arterial pressure, mmHg), *DAP* (diastolic arterial pressure, mmHg), *MAP* (mean arterial pressure, mmHg) *CO* (cardiac output, L/min), *P bladder* (bladder pressure, mmHg).