Impairment of Cardiac Performance by Laparoscopy in Patients Receiving Positive End-Expiratory Pressure

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Background: The cardiopulmonary effects of the combination of abdominal and thoracic pressures in humans have not been well delineated.

Objective: To study the cardiopulmonary effects of 15 mm Hg of intra-abdominal pressure in the presence and absence of 10 cm H2O of positive end-expiratory pressure (PEEP).

Design: Prospective.

Setting: University hospital.

Methods: Nine patients undergoing laparoscopic cholecystectomy had pulmonary compliance, cardiac output, exhaled carbon dioxide, and preload (left ventricular end-diastolic volume) determined at 4 points while undergoing ventilation with (1) no PEEP before pneumoperitoneum; (2) 10 cm H2O of PEEP and no pneumoperitoneum; (3) no PEEP and 15 mm Hg of pneumoperitoneum; and (4) 10 cm H2O of PEEP and 15 mm Hg of pneumoperitoneum. Preload and cardiac output were determined by means of transesophageal echocardiography. Pulmonary compliance and exhaled carbon dioxide were determined by an attachment to the endotracheal tube.

Main Outcome Measures: Preload, cardiac output, exhaled carbon dioxide, and pulmonary compliance.

Results: There was no significant change from baseline in preload, cardiac output, or pulmonary compliance when either PEEP or pneumoperitoneum was applied separately. However, there was a significant decrease in preload (P < .01), cardiac output (P = .01), and exhaled carbon dioxide (P = .04) when PEEP and pneumoperitoneum were applied together. Pulmonary compliance was not significantly affected at any of these points.

Conclusions: There was a significant reduction in preload and cardiac output when there was intra-abdominal pressure of 15 mm Hg in the presence of 10 cm H2O of PEEP. This combination of pressures may pose a contraindication to laparoscopic surgery.

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INTRA-ABDOMINAL hypertension can cause both hemodynamic and pulmonary compromise when there is intra-abdominal bleeding, intra-abdominal packing, intestinal obstruction, or pneumoperitoneum. The pulmonary effects of intra-abdominal hypertension tend to culminate in elevated peak airway pressures when intra-abdominal pressure (IAP) reaches 30 to 40 mm Hg; there is also a fall in pulmonary compliance and vital capacity and an increase in alveolar dead space.1 The hemodynamic effects of increasing IAP are marked by decreasing cardiac output (CO) thought to be caused by increasing systemic vascular resistance and decreasing venous return.2,3 In the normovolemic human, an increase in IAP may decrease cardiac output when IAP reaches 40 mm Hg.4,7 At this point, patient survival may depend on lowering the IAP by decompressing the abdomen.8,9

Patients with intra-abdominal hypertension (such as trauma patients with abdominal packing and patients undergoing laparoscopy) frequently receive positive end-expiratory pressure (PEEP) ventilation. An important question to ask is whether the criteria for abdominal decompression in patients receiving elevated PEEP should be the same as for those receiving low PEEP. The answer to this question may clarify the reported discrepancies in the level at which IAP results in hemodynamic consequences. For example, Kashlan et al2 showed no hemodynamic compromise in dogs until the IAP reached 40 mm Hg, whereas Meldrum et al9 demonstrated improved CO when the abdomen was decompressed at a mean IAP of 27 mm Hg. This discrepancy in mean

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PATIENTS AND METHODS

Nine patients at the University of California, Davis, who were slated for laparoscopic cholecystectomy gave consent for this study, which was approved by our Human Subjects Committee. A thorough cardiac and pulmonary history was obtained on all patients. Patients were excluded if they had esophageal obstruction, recent upper gastrointestinal tract bleeding, or previous upper gastrointestinal tract surgery. All patients were given an initial bolus of 2 mL of crystalloid per kilogram per hour of preoperative fast. This was followed intraoperatively by 2 mL of crystalloid drip per kilogram per hour as maintenance fluid. A Foley catheter and peripheral intravenous line were placed in all patients. Six patients had a respiratory mechanics workstation (Ventrak, Novametrics Medical Systems Inc, Wallingford, Conn) attached to the end of their endotracheal tubes. Five of these 6 patients had arterial lines placed because of the anesthesiologists’ need for blood pressure or blood gas monitoring. A Varess needle or Hassan cannula was placed shortly after induction so that the IAP could be measured before insufflation of the abdomen and not assumed to be 0 mm Hg. Pneumoperitoneum was obtained through either Varess needle or Hassan cannula to 15 mm Hg and maintained through the side port of an 11-mm cannula. A carbon dioxide (CO2) insufflator (OpPneu 5050E, Wisap GmbH, Munich, Germany) was used.

After induction, a transesophageal echocardiography (TEE) probe (Sonos 2500, Hewlett Packard, Andover, Mass) was placed into the esophagus and advanced to a position 30 to 35 cm from the incisors to obtain a 4-chamber view of the heart. The left ventricular end-diastolic volume (LVEDV), the left ventricular end-systolic volume (LVESV), and the ejection fraction were measured with the probe in the horizontal plane by the method of acoustic quantification.13 The respiratory mechanics workstation was attached to the end of the endotracheal tube by an in-line adapter in 6 patients. This machine and arterial blood gases were used to determine the ratio of dead space to tidal volume, pulmonary compliance, autoPEEP (see below), and total exhaled CO2 per minute (VCO2). These values were obtained with the respiratory mechanics workstation by the method of Arnold et al.13 Data were analyzed off-line with Analysis + respiratory mechanics analysis software (Version 2.0; Novametrics Medical Systems Inc). A CO2 expirator was obtained by plotting CO2 in exhaled breath against exhaled breath volume. The CO2 was determined with a mainstream capnometer; flow and pressure were determined from a fixed-orifice differential pressure pneumotachograph. Expired CO2 per breath was calculated as the area under the CO2 expirprogram curve. The CO2 production was determined as the VCO2. The ratio of physiological dead space to tidal volume was from the CO2 expirprogram and the arterial CO2 level by the technique of Fowler13 and Fletcher et al.13 Dynamic pulmonary compliance was assessed by the following equation: compliance = tidal volume/(peak inspiratory pressure − PEEP). Total PEEP was determined as the sum of set PEEP and autoPEEP, where set PEEP is the PEEP set on the ventilator. AutoPEEP was measured as the airway pressure in excess of baseline PEEP required to initiate ventilator flow.

The previously mentioned data collected by TEE, the respiratory mechanics workstation, and arterial blood gases were obtained while the patient was placed under 4 conditions within 15 minutes after induction: (1) 0 cm H2O of PEEP and before pneumoperitoneum, (2) 10 cm H2O of PEEP and before pneumoperitoneum, (3) 0 cm H2O of PEEP after pneumoperitoneum at a pressure of 15 mm Hg, and (4) 10 cm H2O of PEEP after pneumoperitoneum. Data acquisition with each manipulation required approximately 1 to 2 minutes. We waited approximately 1 to 2 minutes between each manipulation and its respective data acquisition. No ventilator changes were made other than PEEP during the data acquisition period.

Stroke volume was determined by the equation LVEDV − LVESV. The CO was determined by the equation heart rate × stroke volume. The ventricular end-systolic elastance (Ees) was used as a measure of myocardial contractility.16 The equation for Ees is Ees = LVESP/(LVESV − 0.1 × weight [kilograms]). The LVESP was estimated by the equations LVESP = (0.9 × SBP) − 10 mm Hg for patients 50 years or older and LVESP = (0.9 × SBP) − 20 mm Hg for patients younger than 50 years, where SBP is systolic blood pressure.16

Data were statistically analyzed by means of a factorial repeated-measures analysis of variance with 2 within factors, IAP and PEEP. The 4 treatment combinations were compared by a repeated-measures model with 1 within factor (treatment) and by pairwise post hoc comparisons among the treatments with the use of the Tukey method.17 Relationships were considered statistically significant whenever P < .05. Variables were transformed logarithmically as necessary to satisfy the assumptions of normality and homoscedasticity. As ventilatory volumes and COs were compared within each patient under 4 experimental conditions, the volumes were left unindexed for simplicity.

IAP may have occurred because the patients studied by Meldrum et al had an average of 10 cm H2O of PEEP, whereas Kashtan et al applied no PEEP. In the context of laparoscopy, 15 mm Hg of IAP may not cause hemodynamic compromise unless the patient is also exposed to 10 cm H2O of PEEP.

Although both PEEP and IAP are thought to decrease venous return, there is evidence that both 10 cm H2O of PEEP10 and mild increases in IAP,11 when applied separately, are tolerated in the normovolemic state with no decrease in CO. An animal study showed that CO is reduced when intra-abdominal pressures less than 40 mm Hg are applied in conjunction with PEEP.18 Because elevated intra-abdominal pressures frequently occur in conditions in which PEEP is required in humans, we prospectively studied the cardiopulmonary effects of intra-abdominal pressure in conjunction with PEEP.

Demographic data are given in Table 1. One of the patients had acute cholecystitis and severe coronary artery disease with a fractional shortening of 10% and previous congestive heart failure. Another patient had had a myocardial infarction 12 years before our study and had subsequently sustained a cardiac arrest with a previous
surgery. The other 7 patients had no major cardiac symptoms. One patient had chronic obstructive pulmonary disease but no symptomatic heart disease. The other patients had no pulmonary symptoms.

Nine patients had ventricular volumes determined by TEE under each of the 4 conditions. Pulmonary compliance, autoPEEP, airway pressures, and \( VCO_2 \) could be determined in the 6 patients who had respiratory mechanics workstation analysis. The ratio of dead space to tidal volume could be described in only the 5 patients who had arterial lines for an arterial blood gas measurement in each of the 4 conditions. All patients had urine outputs of greater than 50 mL/h during surgery.

Thoracic and abdominal pressures are shown in Table 2. The mean autoPEEP was 2.80, 3.40, 0.90, and 3.25 cm H\(_2\)O for conditions 1, 2, 3, and 4, respectively. AutoPEEPs were statistically similar in all 4 groups. Total PEEP were 2.80, 13.40, 0.90, and 13.25 cm H\(_2\)O. The average IAP before insufflation was 0.8 mm Hg. After insufflation of the abdomen with CO\(_2\), the average IAP was 15 mm Hg as it was set by the insufflating machine for conditions 3 and 4.

The cardiopulmonary variables evaluated under the 4 conditions are shown in Table 3, and the statistical analyses of these variables are shown in Table 4. The end-diastolic volume had a significant decrease from baseline only when both PEEP and 15 mm Hg of IAP were applied in condition 4. The end-diastolic volume in condition 4 was also lower than that in conditions 2 and 3. Neither 10 cm H\(_2\)O of PEEP (condition 2) nor 15 mm Hg alone (condition 3) had a statistically significant effect on preload because conditions 1, 2, and 3 were statistically similar by multiple comparisons.

The end-systolic volume was significantly higher (\( P < .01 \)) in condition 3 than in all other conditions. The end-systolic volume in conditions 1, 2, and 4 were statistically similar. When compared with baseline (condition 1), there was a decrease in CO and stroke volume only when PEEP and pneumoperitoneum were applied simultaneously (condition 4). There was a significant reduction in \( VCO_2 \) only when PEEP and IAP were applied together (\( P = .04 \)).

Previous studies have shown that humans require IAPs of greater than 25 mm Hg during laparoscopy to suffer hemodynamic consequences.6,7 The present study demonstrates that 15 mm Hg of IAP can cause hemodynamic consequences if 10 cm H\(_2\)O of PEEP is applied simultaneously. Previous studies of the hemodynamic effects of intra-abdominal hypertension have provided reasons why this may be the case. Toomasian et al18 demonstrated that increased IAP causes a decrease in vena cava flow and an increase in intracavitary right and left atrial pressures. Positive end-expiratory pressure also decreases vena cava flow.19,20 It is possible that PEEP and IAP are additive in their effects on preload.

Hypovolemia also decreases venous return. Only 20 to 40 mm Hg of IAP is required to reduce the CO in hypovolemic dogs.2,18 We maintained normovolemia in our patients so that we could specifically look at the effects of PEEP and IAP in the normovolemic human. Because data were obtained after the patients had been given their initial crystalloid bolus and all data acquisition occurred during the first 15 minutes after induction, all patients were considered normovolemic in condition 1. The small increase in either PEEP (10 cm H\(_2\)O) or IAP (15 mm Hg) alone was well tolerated in these normovolemic patients. The end-diastolic volume decreased in condition 4 most likely because of decreased venous return resulting from the combination of elevated PEEP and IAP.

We used TEE instead of Swan-Ganz catheterization to estimate preload in this study for several reasons. When PEEP is elevated, wedge pressure may correlate poorly with CO.21,22 The LVEDV is accurately measured by TEE23 and may correlate better with CO than does right ventricular end-diastolic volume measured by volumetric Swan-Ganz catheters.21 Estimating preload by means of wedge pressure in the experiment by Burchard et al10 may have led to an incorrect conclusion. In their study, hemodynamics were monitored in dogs while the dogs received various PEEPs and IAPs. The wedge pressure increased with increasing IAP and PEEP, leading to the conclusion that the diminished CO in animals receiving PEEP and elevated IAP resulted from depressed cardiac function. In our study, the decrease in LVEDV in those with elevated PEEP and IAP suggests that CO decreased because of an additive effect of PEEP and IAP in decreasing venous return. The ejection fraction remained the same in all 4 conditions, and the Ees actually increased in condition 4, indicating that depressed myocardial contractility is not the reason for the low CO. The increase in Ees in condition 4 suggests that these patients increased their myocardial contractility to compensate for their low preload and CO.

The decrease in \( VCO_2 \) in condition 4 was most likely caused by reduction in CO. Without PEEP the \( VCO_2 \) is expected to increase by as much as 75% because of CO\(_2\) absorption of the pneumoperitoneum when the abdomen is insufflated to 15 mm Hg of pressure. This increase in \( VCO_2 \) starts after 30 minutes of insufflating the abdomen.24 However, the \( VCO_2 \) did not
change in any condition other than condition 4, which was the last manipulation. In this condition the \( \text{VCO}_2 \) decreased, most likely because of the fall in CO noted by echocardiography in condition 4. If CO remained constant, \( \text{VCO}_2 \) would probably have remained similar in all 4 conditions until 30 minutes after pneumoperitoneum, at which time \( \text{VCO}_2 \) would rise if there were no ventilator changes. Therefore, it is unlikely that acidemia and hypercapnea caused by CO\(_2\) absorption were responsible for hemodynamic changes, because data were acquired early after insufflation, the respiratory rate and tidal volume were not changed, and the \( \text{VCO}_2 \) did not rise.

There is a potential problem with interpreting the data of these 4 conditions in which there were sequential manipulations. The most obvious problem is that we intended that there be 0 cm H\(_2\)O of PEEP in condition 3 within minutes of decreasing the PEEP from 10 cm H\(_2\)O in condition 2. Therefore, it is important to measure the total PEEP (set PEEP + autoPEEP). In condition 3 the average total PEEP was only 0.9 cm H\(_2\)O when the set PEEP was 0 cm H\(_2\)O. Because the auto-PEEP was similar in all 4 conditions, sequential manipulations did not confound this aspect of the data. Sequential manipulations are not as much a problem with respect to IAP because no IAP was applied in the first 2 conditions and because, as IAP was applied, it was continuously measured to ensure an accurate esti-

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<th>Table 2. Abdominal and Thoracic Pressures of 9 Patients at 4 Conditions of PEEP and IAP Obtained by Pneumoperitoneum*</th>
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<td>Pressures†</td>
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<td>Set PEEP</td>
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<td>AutoPEEP</td>
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<td>Total PEEP</td>
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<td>IAP</td>
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* PEEP indicates positive end-expiratory pressure; IAP, intra-abdominal pressure. Condition 1 was PEEP of 0 cm H\(_2\)O and IAP of 0 mm Hg; condition 2, 10 cm H\(_2\)O and 0 mm Hg; condition 3, 0 cm H\(_2\)O and 15 mm Hg; and condition 4, 10 cm H\(_2\)O and 15 mm Hg.
† See “Patients and Methods” section for an explanation of set PEEP and autoPEEP.
‡ Intra-abdominal pressure was not measured in condition 2.

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<th>Table 3. Cardiopulmonary Variables of 9 Patients at 4 Conditions of PEEP and IAP Obtained by Pneumoperitoneum*</th>
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<td>Variables</td>
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<td>End-diastolic volume, mL</td>
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<td>End-systolic volume, mL</td>
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<tr>
<td>Ejection fraction, %</td>
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<td>Stroke volume, mL</td>
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<td>Cardiac output, L/min</td>
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<td>Systolic blood pressure, mm Hg</td>
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<td>Heart rate, beats/min</td>
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<td>Ees, mm Hg/mL</td>
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<tr>
<td>Exhaled CO(_2), mL/min</td>
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<td>Dead space to tidal volume ratio</td>
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<td>Pulmonary compliance, mL/cm</td>
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<td>Airway pressure, cm H(_2)O</td>
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<td>Mean airway pressure, cm H(_2)O</td>
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* PEEP indicates positive end-expiratory pressure; IAP, intra-abdominal pressure; Ees, ventricular end-systolic elastance (a measure of myocardial contractility); CO\(_2\), carbon dioxide. See the first footnote to Table 2 for definitions of conditions.

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<th>Table 4. Multiple Comparisons of the 4 Conditions of PEEP and IAP for the Variables in Table 3*</th>
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<td>Variable</td>
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<td>Exhaled CO(_2) per minute</td>
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<td>Pulmonary compliance</td>
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<td>Peak airway pressure</td>
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<td>Mean airway pressure</td>
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<td>AutoPEEP‡</td>
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<td>Myocardial contractility (Ees)</td>
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* PEEP indicates positive end-expiratory pressure; IAP, intra-abdominal pressure; and CO\(_2\), carbon dioxide. See the first footnote to Table 2 for definitions of conditions.
† According to the Tukey method.
‡ See the “Patients and Methods” section for an explanation.
CONCLUSIONS

An IAP of 15 mm Hg in the normovolemic human can result in a significant reduction of preload and CO in the presence of 10 cm H₂O of PEEP. The cardiac and pulmonary effects of 15 mm Hg of IAP without PEEP are well tolerated. Consideration should be given to avoiding the combination of these 2 pressures in clinical practice. In situations where this combination cannot be avoided, care should be taken to closely monitor the heart and to optimize preload.

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REFERENCES