Induced abdominal compartment syndrome increases intracranial pressure in neurotrauma patients: A prospective study

Giuseppe Citerio, MD; Ettore Vascotto, MD; Federico Villa, MD; Simona Celotti, MD; Antonio Pesenti, MD

**Objective:** To evaluate the effect of a stepwise increase in intra-abdominal pressure (IAP) on intracranial pressure (ICP) and to further define the pressure transmission characteristics of different body compartments.

**Design:** A prospective, nonrandomized study.

**Setting:** A multidisciplinary intensive care unit at a university medical center.

**Patients:** Fifteen patients with moderate-to-severe head injury.

**Interventions:** All patients were studied after the initial stabilization and resolution of intracranial hypertension. Measurements were carried out before and 20 mins after IAP was increased by positioning a soft, 15-L water bag on the patient’s abdomen.

**Measurements and Main Results:** Placing weights upon the abdomen generated a significant increase in IAP, which rose from 4.7 ± 2.9 to 15.5 ± 4.1 mm Hg (p < .001). The rise in IAP caused concomitant and rapid increases in central venous pressure (from 6.2 ± 2.4 to 10.4 ± 2.9 mm Hg; p < .001), internal jugular pressure (from 11.9 ± 3.2 to 14.3 ± 2.4 mm Hg; p < .001), and ICP (from 12.0 ± 4.2 to 15.5 ± 4.4 mm Hg; p < .001). Thoracic transmural pressure, calculated as the difference between central venous pressure and esophageal pressure, remained constant because of decreased chest wall compliance. The mean arterial pressure increased from 94 ± 11 to 100 ± 13 mm Hg (p < .001), which allowed the maintenance of a stable cerebral perfusion pressure (82.4 ± 10.3 vs. 84.7 ± 11.5 mm Hg; p = NS) despite the ICP increase.

**Conclusions:** Increased IAP causes a significant rise in ICP in head trauma patients. This effect seems to be the result of an increase in intrathoracic pressure, which causes a functional obstruction to cerebral venous outflow. Routine assessment of IAP may help clinicians to identify remediable causes of increased ICP. Caution should be used when applying laparoscopic techniques in neurotrauma patients. (Crit Care Med 2001; 29:1466–1471)

**Key Words:** intracranial pressure; intracranial physiology; head injuries; respiratory mechanics; abdominal physiology; prospective studies; intensive care; intracranial pressure

Abdominal compartment syndrome (ACS) is a clinical entity comprising a series of pathological changes induced by abdominal pressure elevations that involve both abdominal and extra-abdominal organs and systems. Abdominal hypertension is the common final pathway of various pathologies, either chronic or acute. Severe abdominal trauma and ruptured abdominal aortic aneurysms are the most common causes of abdominal hypertension (1). A comprehensive review of ACS has recently been published (2). The abdominal consequences of an elevated intra-abdominal pressure (IAP) include a reduction in splanchic and hepatic blood flow and an increase in renal venous pressure (3) with renal blood flow redistribution and oliguria. Moreover, elevated IAP produces compression of the inferior cava and dislocation of the diaphragm toward the thoracic cavity.

Cardiovascular consequences (4) include a reduction in cardiac index and stroke volume, resulting from both a decreased venous return and an increase in systemic vascular resistance. Central venous pressure and pulmonary artery occlusion pressure seem to increase mainly as a result of raised intrathoracic pressure. Elevated IAP influences respiratory system mechanics through the elevation of the diaphragm, which causes a reduction in respiratory compliance, increased airway pressure, and hypoventilation (1).

The first reports (5–8) on this syndrome focused mainly on its surgical implications. Over the last decade, with more widespread use of laparoscopic surgical techniques, there has been a renewed interest in ACS (9–11). The proposed use of laparoscopy for emergency abdominal evaluations in traumatized patients, associated with the knowledge that abdominal trauma frequently coexists with head injury, led Josephs et al. (12) to test the effects of increased IAP on intracranial pressure (ICP) in animals. Under experimental conditions, a pneumoperitoneum caused a rise in ICP, which was greater in animals with preexisting intracranial hypertension. Elevated IAP may also detrimentally affect ICP in humans, as described in two case reports (13, 14) in trauma patients and during laparoscopy. Animal studies (15, 16) subsequently confirmed these first observations and reported not only an increase in ICP but also reductions in cerebral perfusion pressure (CPP). Moreover, these studies determined that the ICP increase could be avoided by performing a thoracotomy or a pericardiopleurotomy. Therefore, with elevated IAP, an intact thorax seems to be essential to cause an increased ICP. These data suggest that an intrathoracic pressure increase could lead to cerebral venous outflow impairment, which in turn increases cerebral blood volume and, subsequently, the observed rise in ICP. Severe abdominal trauma may be associated with head injury in >40% of patients with abdominal trauma (17). In a recent 3-month national survey (18) on head trauma victims in Italy, the presence of associated severe abdominal complications was reported in 6.7% of the patients admitted to the in-
tensive care unit (ICU). We performed this study to evaluate the effects of raised IAP on ICP in neurotrauma victims.

**MATERIALS AND METHODS**

A prospective, nonrandomized study was designed to systematically measure the effects of artificially increased IAP in head trauma patients and to further define the pressure transmission characteristics between different body compartments.

The study protocol was reviewed and approved by our hospital ethics committee. Selected head trauma victims admitted to our multidisciplinary ICU between January and December 1998 were included in the study. Inclusion criteria were as follows:

- **Age:** >16 yrs
- **Moderate or severe head trauma (postresuscitation Glasgow Coma Scale <13 (19))
- **Absence of intracranial hemorrhage lesions:** >20 mL on the most recent computed tomography scan
- **ICP monitoring,** invasive arterial blood pressure measurement, and jugular bulb saturation in use for clinical management
- **ICP stability and adequate CPP,** respectively defined as ICP <20 mm Hg and a CPP >70 mm Hg. Both these criteria had to be fulfilled in the 24 hrs preceding the enrollment
- **Absence of major extracranial lesions and of increased IAP (>10 mm Hg) at baseline
- **Endotracheal intubation and mechanical ventilation
- Hemodynamic stability, i.e., mean arterial pressure (MAP) >75 mmHg without inotropic drug infusion and normovolemia, defined as a normal central venous pressure and a urinary output >1 mL/kg/hr.
- **Patients** were eligible for the study at the end of the acute phase of management, after the evacuation of surgical masses, and when no intracranial hypertension was recorded.

Patients were routinely sedated with propofol (3–6 mg/kg/hr) and fentanyl (1.5 μg/kg/hr). A bolus of pancuronium bromide (0.8 mg/kg) was administered before the test period to guarantee muscle relaxation during the procedure. Propofol (3–6 mg/kg/hr) and fentanyl (1.5 μg/kg/hr) were continuously recorded. Arterial and jugular bulb blood gas samples were drawn and immediately analyzed for PaO₂, PaCO₂, and pH and immediately analyzed for PaO₂, PaCO₂, and pH. 

**Study Protocol.** After baseline data collection with continuous computerized pressure wave recording, a soft 15-L water bag was positioned on the anterior abdominal wall. The weight was uniformly distributed, avoiding contact with the hips and ribs.

A second set of data (which was referred to as the High-IAP step) was collected 20 mins after the weight was positioned. No ICP-influencing drugs or management maneuvers were allowed during the study period.

During both steps of the protocol, ICP, internal jugular pressure (IPP), central venous pressure, IAP, arterial blood pressure, ventilator pressures (positive end-expiratory pressure and peak inspiratory and plateau pressures) were continuously recorded. Arterial and jugular bulb blood gas samples were drawn and immediately analyzed for PaO₂, PaCO₂, and pH immediately analyzed for PaO₂, PaCO₂, and pH immediately analyzed for PaO₂, PaCO₂, and pH and immediately analyzed for PaO₂, PaCO₂, and pH during each protocol step, both before weight positioning and at the end of the 20-min High-IAP step. Cerebral oxygen extraction (CEO₂), i.e., the difference between arterial and jugular saturation (SjO₂), was calculated.

ICP values were obtained in 4 patients by a ventricular catheter (Codman & Shurtleff, Randolph, MA) and in 11 patients by an intraparenchymal monitoring device (Codman & Shurtleff). If a ventricular catheter was placed, cerebral compliance was measured using the pressure-volume index (PVI), according to the method described by Marmarou et al (20). PVI, calculated as PVI = ΔV/log10 Po/Pm, where ΔV is the injected volume, Po is the initial pressure, and Pm is the peak pressure, describes the slope of the line produced by the logarithmic transformation of the cerebrospinal fluid pressure/volume curve and expresses the amount of volume necessary to raise the pressure by a factor of 10. The mean of three pairs of tests (cerebrospinal fluid "withdrawal" and "addition") was recorded as the PVI result.

IPP, central venous pressure, and arterial pressure were monitored by standard indwelling catheters. IPP and central venous pressure catheter positions were previously confirmed radiologically. IAP was measured with the technique described by Iberti et al (21). The patient’s bladder was filled with 100 mL of saline solution and the urinary catheter was connected to a pressure transducer. The zero-reference point was set at the mid-axillary line for all pressure transducers, except for the ventricular ICP, which was set at the mid-ear level. All the pressure lines were connected to a continuous data collection system (MacLab, AD Instruments, Mountain View, CA), which was in turn connected to a Macintosh computer. The system stored in digital format all the pressure traces, sampled at a rate of 20 samples/sec. CPP was calculated as the difference between MAP and mean ICP. Ventilator variables were recorded from a SERVO 900C ventilator (Siemens-Elema, Solna, Sweden). In eight patients, an esophageal balloon was also placed and connected to a pulmonary monitor (Bicore CP100, Bicore Monitoring Systems, Irvine, CA). This measurement allowed the partitioning of respiratory system compliance into its pulmonary and chest wall components (22). Thoracic transmural pressure was calculated as the difference between mean central venous pressure and mean esophageal pressure. Because of the arbitrary setting of the esophageal pressure reference level, the absolute transmural pressure values are not precise. For this reason, the basal transmural pressure was normalized to 0 mm Hg and the High-IAP transmural pressure was computed as the difference from this baseline level.

Safety criteria for the patients were rigidly implemented: a CPP decrease <60 mm Hg and an ICP >25 mm Hg for >5 min required termination of the protocol. All patients met these safety criteria during the entire study and no study complications were recorded.

All collected data were stored and analyzed by means of the Data Desk 6 statistical analysis program (Data Description, Ithaca, NY). All summary data are expressed as mean ± SD. Differences between groups were analyzed using Student’s paired t-test. Differences in proportions were analyzed by a chi-square test. Linear regressions using ICP as the dependent variable and IAP, central venous pressure, and IJP as independent variables were performed to quantify the relationship between these variables. Statistical significance was set at p < .05.

**RESULTS**

Fifteen consecutive male patients, out of a total of 72 head-injured patients admitted to our ICU, were enrolled in the study. The mean age was 42.3 ± 19.2 yrs (range, 18–62 yrs). The median postresuscitation Glasgow Coma Scale score was 7. The mean interval between the traumatic event and protocol enrollment was 5.3 ± 2.9 days. The patients’ demographic data are listed in Table 1.

The main results are shown in Table 2 and are summarized in Figure 1. Positioning a weight on the abdomen generated a significant increase in IAP, from 4.7 ± 2.9 to 15.5 ± 4.1 mm Hg (p < .001). In all patients, the IAP rise was associated with a significant, rapid increase in central venous pressure (from 6.2 ± 2.4 to 10.4 ± 2.9 mm Hg; p < .001), IJP (11.9 ± 3.2 to 14.3 ± 2.4 mm Hg; p < .001), and ICP (from 12.0 ± 4.2 to 15.5 ± 4.4 mm Hg; p < .001). Central venous pressure seemed to be linearly related to IAP (R = .604, p < .05). A weak but significant correlation (R = .57, p < .05) was found between IPP and central venous pressure. No significant correlation was found between IAP and ICP. Over the range of basal ICP recorded, the baseline ICP values were correlated linearly with the ICP levels during weight positioning (R = .791, p < .001) (Fig. 2). The ICP rise was independent of baseline ICP (chi-square; p = NS). PVI values did not show any significant change (27.3 ± 19 vs. 26.6 ± 13; p = NS). Analysis of the continuous digital recording showed that the rise in central venous pressure, IJP,
and ICP occurred rapidly and immediately after weight positioning. All the described pressure increases took two to three cardiac cycles to reach their peak and occurred simultaneously (Fig. 3). During the entire High-IAP period (about 20 mins), all the pressures reached stable levels.

An increase in peak inspiratory pressure from 22.3 ± 7.5 to 28.2 ± 4.5 cm H$_2$O (p < .001) and in plateau pressure from 18.0 ± 3.3 to 21.9 ± 3.5 cm H$_2$O (p < .001) was recorded. The respiratory system compliance significantly decreased from 58.9 ± 9.75 to 44.9 ± 9.42 mL/cm H$_2$O (p < .001) in all patients. Mean esophageal pressure (Pes) recordings in eight patients demonstrated that this reduction was exclusively the result of the chest wall component of respiratory system compliance. The chest wall compliance significantly decreased from 204.7 ± 37.1 to 123.6 ± 38.0 mL/cm H$_2$O (p < .001). The pulmonary compliance was unchanged (108.9 ± 40.1 vs. 101.8 ± 29.2 mL/cm H$_2$O; p = NS).

Pes increase from 9.3 ± 1.9 to 13.1 ± 3.4 cm H$_2$O (p < .01). This increase correlated linearly with IAP elevations (R = .77, p < .05). Transmural pressure, i.e., the difference between central venous pressure and mean esophageal pressure, also remained constant during the two protocol steps (transmural pressure at High-IAP – basal transmural pressure = −0.2 ± 1.9 mm Hg; p = NS). The respiratory mechanics data for eight patients are shown in Table 3.

Gas exchange during High-IAP showed a slight reduction in PaO$_2$ (from 137.9 ± 37.7 to 126.0 ± 37.2 mm Hg; p < 0.05). The PaCO$_2$ (37.1 ± 2.7 vs. 36.5 ± 2.9 mm Hg) and pH values (7.48 ± 0.04 vs. 7.48 ± 0.05) remained unchanged.

Systolic arterial pressure did not change significantly (148 ± 20 vs. 154 ± 23 mm Hg; p = NS). Diastolic pressure increased from 66 ± 8 to 73 ± 7 mm Hg (p = NS) and MAP increased from 94 ± 11 to 100 ± 13 mm Hg, (p < .05). Cerebral perfusion pressure remained stable (82 ± 10 vs. 85 ± 12 mm Hg, p = NS).

No change was detected in SjO$_2$. (66.1 ± 7.9% vs. 64.2 ± 7.8%; p = NS) or CEO$_2$. (31.1 ± 7.7% vs. 32.8 ± 7.8%; p = NS).

**DISCUSSION**

The most important finding of this study is that an elevation of pressure in the abdomen produces a significant increase in ICP, demonstrating in a cohort of patients with head injury the transmissions of pressure between distant compartments. Animal studies (15, 16) have established that an acute increase in IAP can cause an increase in ICP, mediated by a rise in the intrathoracic pressure, causing an obstruction to venous cerebral outflow. The ICP rise must be considered a possible component of the abdominal compartment syndrome. Two case reports (13, 14), one in a trauma patient and the other during a laparoscopy, have reported the same effect in humans.

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**Table 1. General demographic data from the study population (n = 15)**

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<tr>
<th>Patient</th>
<th>Age (yrs)</th>
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<th>Days from Injury</th>
<th>P VO$_2$ (mL)</th>
<th>RR (bpm)</th>
<th>PEEP (cm H$_2$O)</th>
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<td>SDH + ICH</td>
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GCS, Glasgow Coma Scale (19) score; TV, tidal volume; RR, respiratory rate; PEEP, positive end-expiratory pressure; GOS, Glasgow Outcome Scale score (1 = death, 2 = vegetative state, 3 = severe disability, 4 = moderate disability, 5 = good outcome); ICH, intraparenchymal hematoma; SDH, subdural hematoma; EDH, extradural hematoma; DAI, diffuse axonal injury.

**Table 2. Experimental data from 15 patients**

<table>
<thead>
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<th>Variable</th>
<th>Basal Values</th>
<th>High-IAP</th>
<th>p Value</th>
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<td>IAP, mm Hg</td>
<td>4.7 ± 2.9</td>
<td>15.5 ± 4.1</td>
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<tr>
<td>CVP, mm Hg</td>
<td>6.2 ± 2.4</td>
<td>10.4 ± 2.9</td>
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<tr>
<td>DP, mm Hg</td>
<td>11.9 ± 3.2</td>
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<tr>
<td>ICP, mm Hg</td>
<td>12.0 ± 4.2</td>
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<tr>
<td>MAP, mm Hg</td>
<td>94 ± 11</td>
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<td>CPP, mm Hg</td>
<td>82 ± 10.3</td>
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<td>SjO$_2$, %</td>
<td>66.1 ± 7.9</td>
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<td>NS</td>
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<tr>
<td>CEO$_2$, %</td>
<td>31.1 ± 7.7</td>
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<td>PVI, mL</td>
<td>26.6 ± 13.0</td>
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<tr>
<td>PaO$_2$, mm Hg</td>
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<td>PaCO$_2$, mm Hg</td>
<td>37.1 ± 2.7</td>
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<td>pH</td>
<td>7.48 ± 0.04</td>
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</table>

IAP, intra-abdominal pressure; CVP, central venous pressure; IP, internal jugular pressure; ICP, intracranial pressure; MAP, mean arterial pressure; CPP, cerebral perfusion pressure; SjO$_2$, jugular bulb oxygen saturation; CEO$_2$, cerebral oxygen extraction; PVI, pressure-volume index; NS, not significant.
In our study, the rise in IAP occurred immediately after positioning of the weight and was transmitted to the respiratory system, as demonstrated by the esophageal pressure increase. The IAP increase, displacing the diaphragm upward, reduced the respiratory system compliance. Partitioning of the respiratory compliance into its components demonstrated that this reduction was exclusively attributable to the decrease in chest wall compliance. The increase found in the recorded intrathoracic pressures was immediately transmitted to intrathoracic veins, and the central venous pressure rose from 6.2 to 10.4 mm Hg. In fact, the transmural pressure, i.e., the difference between Pes and central venous pressure, did not change. The Pes zero reference point influences the absolute (negative) value of transmural pressure. For this reason, we only analyzed changes in transmural pressure from a normalized basal value (chi-square, \( p = \text{NS} \)).

Figure 1. Effect of the application of the weight on intra-abdominal pressure (IAP), central venous pressure (CVP), internal jugular pressure (IJP), and intracranial pressure (ICP). See text for details. Mean value (white line), 75th percentile (upper box), 25th percentile (lower box), highest value (upper error bar), and lowest value (lower error bar) are represented. *Paired t-test, \( p < .05 \).

Figure 2. Over the range of basal intracranial pressure (ICP) values examined in this study, the baseline ICP values were found to be correlated with the ICP levels reached during weight positioning (High-IAP ICP). This relationship was best fitted by a linear regression model: \( \text{ICPhigh} = 0.9762 \times \text{ICPbasal} + 3.7524 \) (linear regression, \( R = .791, p < .001 \)). The ICP rise was independent of baseline (chi-square, \( p = \text{NS} \)).

Figure 3. Example of a digital recording before and during positioning of the weight on the abdomen. We observed, through analysis of the continuous digital recording, that the rise in central venous pressure (CVP), internal jugular pressure (IJP), and intracranial pressure (ICP) occurred rapidly and immediately after weight positioning. All of the described pressure increases took two to three cardiac cycles to reach their peak and occurred simultaneously in all the monitored channels. ABP, arterial blood pressure; IAP, intra-abdominal pressure.
reflected only an extrinsic venous compression. Moreover, it was linearly correlated with IAP increases.

Mechanical displacement of the diaphragm upward, with the resulting reduction in its range of excursion, produces atelectasis of basal lung segments, as reported in animal studies (23, 24). These alterations could explain the statistically but not clinically significant reduction in arterial $P_{a\text{CO}_2}$. Stable maintenance of muscle relaxation, sedation, and respiratory settings enabled us to maintain $P_{a\text{CO}_2}$ and $pH$ constant, thus avoiding secondary changes in the patients’ ventilatory drive. These alterations could explain the statistically but not clinically significant reduction in arterial $P_{a\text{CO}_2}$ and $pH$ constant, thus avoiding secondary changes in the patients’ ventilatory drive.

The rise in arterial blood pressure could be explained by the change in intrathoracic pressure. Its elevation, although decreasing the venous return, may facilitate the systolic ejection as demonstrated by physiologic studies (25). Pressure in the abdominal compartment is directly transmitted through the thoracic compartment as demonstrated by the strong correlation between IAP and central venous pressure. Venous pressure rose also in the cerebral compartment, as shown by vein pressure recorded at the jugular bulb, which produced a small but significant increase. However, this rise was only slightly correlated with central venous pressure modifications. A possible explanation for the loss of pressure transmission between the intrathoracic and the cerebral venous systems may be found by considering the ability of the jugular veins to distend. Despite this, ICP increased significantly. This rise, mean value 3.75 mm Hg, was independent of the ICP starting level. All patients were in a stable condition, with a starting ICP <20 mm Hg. The PVI, i.e., the volume needed to produce a ten-fold increase in the ICP, described a normal intracranial compliance. The starting ICP probably lies on the flat portion of the Starling curve, and the application of the weight to the abdomen did not induce a clinically relevant ICP increase. The mean ICP increase is similar to the mean IUP increase. The mechanism producing the rise in ICP seems to be an obstruction of venous drainage, documented by an IJP rise, producing increased pressure in the intracranial compartment. Recently published experimental data (26) in a porcine model of raised ICP, using cerebral blood flow measurement, demonstrated a decrease in the brain venous outflow without alteration of the cerebral microcirculation.

The increase in MAP maintained a stable cerebral perfusion pressure and thus the cerebral blood flow remained stable, as could be extrapolated from jugular saturation data. These results do not agree with animal studies (16) in which ACS negatively influenced cerebral perfusion pressure. In those studies, a fall in MAP was responsible for the fall in CPP. In their model of ACS, which was produced by pneumoperitoneum, Bloomfield et al. (16) did not observe an increase in MAP. Although we used a different technique to increase IAP (i.e., applying an external weight on the abdominal wall), which may explain these differences, we believe that MAP decreased in their animal model because of the hypnotic drug, high dose pentobarbital, administered during their study. The vasoplegic effect of this drug could be responsible for the slight, nonsignificant reduction in MAP found in their model. Consequently, they also observed a reduction in CPP, determined by the concomitant rise in ICP. Also, this occurred only at a very high IAP, >25 mm Hg, which was not reached in our experimental observation.

Although only patients without intracranial hypertension were studied, the effect on ICP was statistically significant, even if it remained in a clinically acceptable range. For safety reasons, we did not study the effects of high IAP in patients with clinically significant intracranial hypertension. It is, however, reasonable to suppose that in the presence of a sustained ICP caused by the different position on the Starling pressure/volume curve and by reduced compensatory capacity, the effect of high IAP could be more harmful.

According to these data, laparoscopic techniques causing IAP pressure levels comparable with the experimentally high IAP reported here should be used, if at all, with caution in patients with concomitant head and abdominal injury. Moreover, routine assessment of IAP or abdominal distention could help clinicians in identifying remediable causes of increased ICP.

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REFERENCES


Table 3. Experimental data from eight patients

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<th>Variable</th>
<th>Basal Value</th>
<th>High-IAP</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
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<td>Respiratory system compliance, mL/cm H2O</td>
<td>64.9 ± 14.0</td>
<td>52.6 ± 9.6</td>
<td>.001</td>
</tr>
<tr>
<td>Chest wall compliance, mL/cm H2O</td>
<td>204.7 ± 37.1</td>
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</tr>
<tr>
<td>Pulmonary compliance, mL/cm H2O</td>
<td>101.8 ± 29.2</td>
<td>108.9 ± 40.1</td>
<td>NS</td>
</tr>
<tr>
<td>Plateau airway pressure, cm H2O</td>
<td>18.1 ± 1.9</td>
<td>20.2 ± 1.8</td>
<td>.001</td>
</tr>
<tr>
<td>Change in transmural pressure, cm H2O</td>
<td>0±</td>
<td>−0.2 ± 1.9</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS, not significant.

*Basal transmural pressure was normalized to 0 mm Hg, see text for details.
vascular, pulmonary and renal effects of massively increased intra-abdominal pressure in critically ill patients. Crit Care Med 1989; 17:118–121

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