Intra-abdominal hypertension associated with acute lung injury: effects on intracranial pressure

Hipertensão intra-abdominal associada à lesão pulmonar aguda: efeitos sobre a pressão intracraniana

Intra-abdominal hypertension (IAH) is defined as intra-abdominal pressure (IAP) above 12 mmHg and may be categorized as Grade I (12-15 mmHg), Grade II (16-20 mmHg), Grade III (21-25 mmHg) or Grade IV (> 25 mmHg). Recurrent or persistent IAP above 20 mmHg, in association with failure of at least one organ, is called Abdominal Compartment Syndrome (ACS). The mortality and morbidity of IAH and ACS are high and may reach 100% for unattended ACS. Deleterious effects of increased intra-abdominal pressure are not limited to the abdomen but finally impact the pressure balances on other organ systems such as the respiratory, cardiovascular and cerebral systems.\(^{(1-2)}\)

On the chest, IAH displaces the diaphragm cranially, thereby reducing the intra-thoracic volume and increasing the intra-thoracic pressure (ITP); this reduces the compliance of the chest wall, lung and heart cavities, resulting in both respiratory and cardiovascular effects.\(^{(3-4)}\)

Considering the head, IAH increases the intracranial pressure (ICP) and reduces the cerebral perfusion pressure (CPP).\(^{(3)}\) Increased ICP is ascribed to the increased central venous pressure (CVP), reduced brain venous flow and reduced lumbar venous plexus flow, with concomitant imbalance of the intracranial contents, as proposed by Monroe-Kellie.\(^{(3-5)}\) The mechanic effects of inferior vena cava compression during IAH reduces lumbar venous plexus flow and may also be responsible for the increased ICP.\(^{(5)}\)

Not only IAH, but also the increase in ITP due to mechanical ventilation, may determine ICP changes. The use of high inspiratory pressure and positive end-expiratory pressure (PEEP), which results in increased airway pressures, can lead to increased ITP and consequently, to increased central venous pressure, resulting in increased ICP. However, the effects of positive pressure ventilation on ICP are apparently influenced by several factors, including pulmonary and cerebral compliance.\(^{(6)}\)

In the study entitled “Modulation of intracranial pressure in an experimental model of abdominal hypertension and acute lung injury,” the authors evaluated the association between IAH and pulmonary injury on the ICP. According to their results, this interaction impacted the ICP more significantly than IAH alone. The applicability of these findings is immediate.\(^{(7)}\)

The authors found that plateau \(P_{\text{plateau}}\), Peak \(P_{\text{peak}}\) and Pleural \(P_{\text{pl}}\) pressures were significantly increased with IAH and acute lung injury (ALI), without significant hemodynamic changes. With respect to the association of IAH, ALI and 27 cmH\(_2\)O PEEP, significant hemodynamic
changes were observed in mean pulmonary artery pressure (mPAP), pulmonary capillary wedge pressure (PCWP) and CVP, with no cardiac index (CI) changes, and with concomitant increases of P_plateau, P_peak and P_pl transmitted to the cranial cavity. This is confirmed by several authors. (3,6,8-10)

In this study, a high PEEP value caused only a slight 2-mmHg ICP increase. As observed, PEEP would be expected to be transmitted into the thoracic cavity, increasing the CVP and transmitting this pressure into the cranial cavity, leading to increased ICP;\(^\text{6,8-10}\) however, an increased carbon dioxide arterial pressure (PaCO\(_2\)) was observed, which contributed to increase the ICP. Although PaCO\(_2\) contribution was not significant during IAH plus ALI, it may have contributed to the ICP increase, demonstrating the multi-factorial character of this medical condition. Of note, PEEP was given only upon pulmonary injury, where the low parenchymal compliance may have mitigated the transmission of pulmonary pressure into the chest wall,\(^\text{8}\) with only a limited change in P\(_\text{pl}\) (only 3 cmH\(_2\)O after PEEP).\(^\text{9,11-13}\) However, in addition to the pleural pressure, the intrapulmonary pressure, as represented by the plateau pressure, would be expected to be transmitted to other intra-thoracic structures, leading to increased CVP. This was only significantly observed with a 27 cmH\(_2\)O PEEP.

In the absence of lung injury, i.e., IAH alone, intrapulmonary pressures contributed to increase CVP and ICP by compressing the thoracic contents and reducing the cerebral venous return.\(^\text{5,14}\)

Could, therefore, the increased plateau pressure be a modulator of the ICP, or is it just a consequence of the reduced chest wall and pulmonary compliance caused by IAH? It should be noted that with ALI alone, no concomitant significant change involving CVP and ICP was found, although significantly increased P_plateau and P_peak pressures and reduced respiratory system compliance were seen, allowing the inference that pleural pressure reduces cerebral venous return.

It should be noted that in this model, the ICP was changed even in non-injured brain, with normal cerebral autoregulation and compliance; therefore, ICP changes could have been even more significant in injured brains, with a more significant effect of PaCO\(_2\) on ICH pathophysiology.

Although ICP is more frequently used as a therapeutic guide, other data such as cerebral tissue oximetry, cerebral oxygen extraction and cerebral microdialysis can show significant results because they are indicators of hemodynamics and cerebral metabolism, especially with concomitant brain injury.\(^\text{35-17}\)

The reported results have wide clinical applicability and stimulate further research on therapeutic approaches to IAH and increased intra-thoracic pressure impacting the intracranial pressure.

REFERENCES


