Antonio Luis Eiras Falcão[1], Douglas Guimarães de Oliveira[2]

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

1. Discipline of Surgical Physiology and Metabolism Studies, Faculdade de Ciências Médicas, Universidade Estadual de Campinas – UNICAMP - Campinas (SP), Brazil.
2. Surgery Post-Graduation Program (MSc Degree), Faculdade de Ciências Médicas, Universidade Estadual de Campinas – UNICAMP – Campinas (SP), Brazil.

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 ($P_{plateau}$), Peak (P_{peak}) and Pleural (P_{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_2O 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;[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,[8] with only a limited change in P_{pl} (only 3 cmH_2O after PEEP).[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_2O 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.[3,14]

Could, therefore, the increased plateau pressure be a modulator of the ICP, or it is 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.[15-17]

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

1. Malbrain ML, Cheatham ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. Intensive Care Med. 2006;32(11):1722-32.
2. Rizoli S, Mamtani A, Scarpelini S, Kirkpatrick AW. Abdominal compartment syndrome in trauma resuscitation. Curr Opin Anaesthesiol. 2010;23(2):251-7.
3. Bloomfield GL, Ridings PC, Blocher CR, Marmarou A, Sugerman HJ. A proposed relationship between increased intra-abdominal, intrathoracic, and intracranial pressure. Crit Care Med. 1997;25(3):496-503.
4. Pelosi P, Luecke T, Rocco PR. Chest wall mechanics and abdominal pressure during general anaesthesia in normal and obese individuals and in acute lung injury. Curr Opin Crit Care. 2011;17(1):72-9.
5. Rosenthal RJ, Friedman RL, Kahn AM, Martz J, Thiagarajah S, Cohen D, et al. Reasons for intracranial hypertension and hemodynamic instability during acute elevations of intra-abdominal pressure: observations in a large animal model. J Gastrointest Surg. 1998;2(5):415-25.
6. Lowe GJ, Ferguson ND. Lung-protective ventilation in neurosurgical patients. Curr Opin Crit Care. 2006;12(1):3-7.
7. Zampieri FG, Almeida JR, Schettino GPP, Park M, Machado FS, Azevedo LCP. Modulação da pressão intracraniana em um modelo experimental de hipertensão abdominal e lesão pulmonar aguda. Rev Bras Ter Intensiva. 2011;23(2):164-9.
8. Bein T, Kuhr LP, Bele S, Ploner F, Keyl C, Taeger K. Lung recruitment maneuver in patients with cerebral injury: effects on intracranial pressure and cerebral metabolism. Intensive Care Med. 2002;28(5):554-8.
9. Zhang XY, Wang QX, Fan HR. Impact of positive end-expiratory pressure on cerebral injury patients with hypoxemia. Am J Emerg Med. 2010 Apr 30. [Epub ahead of print]

10. Caricato A, Conti G, Della Corte F, Mancino A, Santilli F, Sandroni C, et al. Effects of PEEP on the intracranial system of patients with head injury and subarachnoid hemorrhage: the role of respiratory system compliance. J Trauma. 2005;58(3):571-6.
11. Plataki M, Hubmayr RD. Should mechanical ventilation be guided by esophageal pressure measurements? Curr Opin Crit Care. 2011;17(3):275-80.
12. Talmor D, Sarge T, O'Donnell CR, Ritz R, Malhotra A, Lisbon A, Loring SH. Esophageal and transpulmonary pressures in acute respiratory failure. Crit Care Med. 2006;34(5):1389-94.
13. Talmor D, Sarge T, Malhotra A, O'Donnell CR, Ritz R, Lisbon A, et al. Mechanical ventilation guided by esophageal pressure in acute lung injury. N Engl J Med. 2008;359(20):2095-104.
14. Mascia L, Grasso S, Fiore T, Bruno F, Beraldino M, Ducati A. Cerebro-pulmonary interactions during the application of low levels of positive end-expiratory pressure. Intensive Care Med. 2005;31(3):373-9.
15. Hlatky R, Robertsonb CS. Multimodality monitoring in severe head injury. Curr Opin Anaesthesiol. 2002;15(5):489-93.
16. Cecil S, Chen PM, Callaway SE, Rowland SM, Adler DE, Chen JW. Traumatic brain injury: advanced multimodal neuromonitoring from theory to clinical practice. Crit Care Nurse. 2011;31(2):25-36; quiz 37.
17. Vidgeon SD, Strong AJ. Multimodal cerebral monitoring in traumatic brain injury. J Intensive Care Soc. 2011;12(2):126-33.