ECRONICON OPEN ACCESS

EC PULMONOLOGY AND RESPIRATORY MEDICINE

Opinion

Lungs-Brain Cross-Talk? or Barrier?

Della Torre V[1]* and Badenes R[2]

[1]*Department of Intensive Care, Imperial College Healthcare Trust, St Mary's Hospital, London, UK*

[2]*Department of Anaesthesia and Surgical Trauma Intensive Care, Hospital Universitari Clinico de Valencia, University of Valencia, Valencia, Spain*

***Corresponding Author:** Della Torre V, Department of Intensive Care, Imperial College Healthcare Trust, St Mary's Hospital, London, UK.

Received: May 08, 2019; **Published:** June 26, 2019

Pulmonary disorders may occur after brain injury, such as Ventilator Associated Pneumonia (VAP), Acute Respiratory Distress Syndrome (ARDS) and Neurogenic Pulmonary Oedema (NPO) [1]. They are key points for the management of brain-injured patients because respiratory failure is an independent risk factor for increased mortality, poor neurological outcome and longer stay in the intensive care unit (ICU) [2]. Brain and lungs strongly interact via complex pathways and recently the hypothesis of a "double hit" model has been described [3]. A consensus doesn't exist on the best ventilator settings for patients with concomitant respiratory failure and brain injury [4,5]. Respiratory failure is the most frequent extra cerebral organ dysfunction in neurocritical care patients [2]. Among lung injuries in neurological patients, VAP presents with a incidence of 21 to 60% [6], ARDS with a incidence of 35% [7] and more specifically ARDS complicates 20 to 25% of Traumatic Brain Injured (TBI) patients [8], 20 to 38% of Subarachnoid Haemorrhage (SAH) patients [9] and 4% of the stroke population [10]; whilst neurogenic pulmonary oedema is present with a incidence of 42% in patients who suffered SAH [9]. *Staphylococcus aureus* (Methicillin-Sensitive) is the most common pathogen causing VAP in patients with TBI, mostly related to decreased level of consciousness and higher risk of aspiration [2].

The pathophysiology of lung injuries after an acute brain injury is still in debate; recently, the "double hit" model has been described [3]. The sympathetic response to increased intra cranial pressure is also important. It has been well demonstrated that direct myocardial injury with Takotsubo's Cardiomyopathy can contribute to NPO [11]. Systemic inflammatory response plays a major role in the development of pulmonary failure after acute brain injury. Cerebral inflammatory response occurs after neurologic disorders, and cytokines (IL-1, IL-6 and IL-8) and tumour necrosis factor (TNF) are produced locally in brain-injured tissue [12]. Alteration of the blood brain barrier permeability allows their discharge into the systemic circulation, following a trans cranial gradient. This systemic production of mediators constitutes an inflammatory environment: the "first hit". Organs are therefore more susceptible to subsequent events, the "second hit", such as mechanical ventilation, infections or surgical procedures. The inflammatory cascade does not occur only in one way: from the brain to the lungs, but also from the lungs to the brain [3].

Recruitment manoeuvres, prone positioning and the use of high PEEP can improve pulmonary gas exchange and respiratory mechanics by reducing ventilation-perfusion mismatches, opening collapsed alveoli, and reducing intrapulmonary shunt [13,14]. However, these techniques may be associated with the development of intracranial hypertension. Moreover, they can decrease mean arterial pressure, resulting in decreased cerebral perfusion pressure [15]. Literature is lacking regarding the management of patients with concomitant TBI and ARDS, there is therefore need for a pragmatic approach into this group of patients [4,5]. Treatment for VAP in patients with cerebral injuries isn't specific, but prevention seems to be a key point. Risk factors for VAP in brain-injured population are numerous and prophylactic measures have to focus on these, including oral care [16]. Surely the high rate of VAP in brain-injured patients is, in part, explained by long duration of mechanical ventilation [16]. Regarding Neurogenic Pulmonary Oedema, few studies have reported specific treatments. Some animal studies have focused on alpha-blocker treatment to limit massive sympathetic discharge after brain injury [17]. The key

point of NPO management is to treat the underlying cerebral injuries to decrease the ICP, reduce the sympathetic discharge and consequently improve the oxygenation [17]. Concerning ARDS, protective ventilation is the best ventilator strategy [18], alongside accurate monitoring of haemodynamic, respiratory and cerebral parameters. We also suggest adding bedside ultrasound, to monitor and evaluate response to treatment. A comprehensive multi-organ ultrasound assessment (including heart, inferior vena cava, lungs and brain) allows evaluation of peripheral and central perfusion, venous congestion, volume status and ICP, and may be a guide for early-stage interventions for ventilated brain injured patients [19].

In summary, a "wiser use" of ventilatory strategies could have a beneficial effect on brain oxygenation, even if high PEEP, recruitment manoeuvres and proving position are applied. Further studies are warranted to explore pathophysiological processes and evaluate optimal ventilator settings in brain and lung injured patients. Strict monitoring, including non-invasive, bedside US of cardio-vascular, respiratory and cerebral systems, is required to optimise the management of these challenging neuro-critical care patients.

Key Messages:

1. Pulmonary disorders are often associated with Brain Injuries.
2. Brain and lungs interacts, via complex pathways, explained by the "double hit model" theory.
3. Mechanical ventilation can cause increased intra cranial pressure and decrease of cerebral perfusion pressure.
4. Brain-lungs focused ventilator settings and bed side ultrasound are required to optimise management of neuro-critical care patients.

Bibliography

1. Lee K and Rincon F. "Pulmonary complications in patients with severe brain injury". *Critical Care Research and Practice* (2012): 207247.
2. Pelosi P., *et al*. "Management and outcome of mechanically ventilated neurologic patients". *Critical Care Medicine* 39.6 (2011): 1482-1492.
3. Mrozek S., *et al*. "Brain-lung crosstalk: Implications for neurocritical care patients". *World Journal of Critical Care Medicine* 4.3 (2015): 163-178.
4. Della Torre V., *et al*. "Acute respiratory distress syndrome in traumatic brain injury: how do we manage it?" *Journal of Thoracic Disease* 9.12 (2017): 5368-5381.
5. Frisvold SK., *et al*. "What respiratory targets should be recommended in patients with brain injury and respiratory failure?" *Intensive Care Medicine* 45.5 (2019): 683-686.
6. Lepelletier D., *et al*. "Retrospective analysis of the risk factors and pathogens associated with early-onset ventilator-associated pneumonia in surgical-ICU head-trauma patients". *Journal of Neurosurgical Anesthesiology* 22.1 (2010): 32-37.
7. Hoesch RE., *et al*. "Acute lung injury in critical neurological illness". *Critical Care Medicine* 40.2 (2012): 587-593.
8. Rincon F., *et al*. "Impact of acute lung injury and acute respiratory distress syndrome after traumatic brain injury in the United States". *Neurosurgery* 71.4 (2012): 795-803.
9. Veeravagu A., *et al*. "Acute lung injury in patients with subarachnoid hemorrhage: a nationwide inpatient simple study". *World Neurosurgery* 82.1-2 (2014): e235-e241.
10. Rincon F., *et al*. "The prevalence and impact of mortality of the acute respiratory distress syndrome on admissions of patients with ischemic stroke in the United States". *Journal of Intensive Care Medicine* 29.6 (2014): 357-364.

11. Davison DL., *et al*. "Neurogenic pulmonary edema". *Critical Care* 16 (2012): 212.

12. Habgood MD., *et al*. "Changes in blood brain barrier permeability to large and small molecules following traumatic brain injury in mice". *European Journal of Neuroscience* 25.1 (2007): 231-238.

13. Heuer JF., *et al*. "Acute effects of intracrainal hypertension and ARDS on pulmonary and neuronal damage: a randomized experimental study in pigs". *Intensive Care Medicine* 37.7 (2007): 1182-1191.

14. Gattinoni L., *et al*. "Effects of positive end-expiratory pressure on regional distribution of tidal volume and recruitment in adult respiratory distress syndrome". *American Journal of Respiratory and Critical Care Medicine* 151.6 (1995): 1807-1814.

15. Mascia L., *et al*. "Cerebro-pulmonary interactions during the application of low levels of positive end expiratory pressure". *Intensive Care Medicine* 31.3 (2005): 373-379.

16. O'Grady NP., *et al*. "Preventing ventilator associated pneumonia: does the evidence support the practice?" *Journal of the American Medical Association* 307.23 (2012): 2534-2539.

17. Lu WH., *et al*. "Different impacts of α- and β-blockers in neurogenic hypertension produced by brainstem lesions in rat". *Anesthesiology* 120.5 (2014): 1192-1204.

18. ARDSnet ventilation protocol summary (2008).

19. Corradi F., *et al*. "Combined lung and brain ultrasonography for an individualised brain-protective ventilation strategy in neurocritical care patients with challenging ventilation needs". *Critical Ultrasound Journal* 10.1 (2018): 24.

Volume 8 Issue 7 July 2019