Β. ΣΤΡΟΓΓΥΛΑ ΤΡΑΠΕΖΙΑ-ROUND TABLES

Β1. ΛΟΓΟΣ ΑΝΤΙΛΟΓΟΣ: ΗΡΘΕ ΤΟ ΤΕΛΟΣ ΓΙΑ ΤΗ ΣΤΑΡΤΗΓΙΚΗ ΜΗΧΑΝΙΚΟΥ ΑΕΡΙΣΜΟΥ ΑΝΟΙΚΤΟΥ ΠΝΕΥΜΟΝΑ;

Β1.Α. INTRAOPERATIVE PROTECTIVE MECHANICAL VENTILATION: THE 'OPEN LUNG' STRATEGY

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Introduction

Since its initial application, mechanical ventilation remains an effective tool for respiratory support in the perioperative setting either in patients with respiratory failure or in the intraoperative setting for the substitution of respiratory function in patients under neuromuscular blockade. Although its usefulness is undeniable, mechanical ventilation itself can cause or exacerbate lung injury. In 1967, the term "respirator lung" was used to describe the diffuse alveolar infiltrates and hyaline membranes found on postmortem examination of patients who had undergone mechanical ventilation. (1) Given the dire consequences of this injury, for the last 50 years research has been focused on detecting potential mechanisms that lead to ventilator-induced lung injury (VILI) and on establishing ventilatory strategies to prevent it.

Mechanisms of lung injury

- **DREYFUSS MODEL**

In 1985, Dreyfuss showed that only few minutes of ventilation with high peak pressure is sufficient to cause lung interstitial edema in rats (2). This type of lung damage attributable to the application of high airway pressure was termed **barotrauma** and its cause was not questioned until Dreyfuss, some years later, performed the same experiments having rats' chest walls strapped. In this case, even though airway pressures were extremely high, tidal volumes (TV) were low and lung lesions were absent (3) .Based on these findings, it was assumed that high tidal volume and not high airway pressure (Paw) was responsible for VILI and the term **volutrauma** was introduced to describe this type of damage. The pathophysiological mechanism underlying this concept become clearer, if we realize that the force acting on the lung structures is not the Paw but the transpulmonary pressure (Prs) which is equal with the difference between Paw and pleural pressure (Ppl). In support of this, in Dreyfuss experimental model, rats with a strapped thorax, although ventilated with high Paw, maintained low Prs and thus were protected from developing VILI.

- **GATTINONI MODEL**

Later on, in 2003, Gattinoni attempted to describe the mechanisms of VILI in terms borrowed from bioengineering (4). In this respect, he defined as "Stress" the forces developing into the lung structure due to the application of Prs and as "Strain" the lung deformation (relative to its resting position, V_0) attributable to TV. Within physiological limits, stress and strain are almost linearly related and that relationship

seems to be preserved both to healthy lungs and lungs with VILI (5).This means that volutrauma (strain) and barotrauma (stress) are one and the same, representing just the two sides of the same coin. Moreover, mechanical ventilation below the lower inflection point causes lung damage due to cyclic opening and closing of alveoli (6). In addition, the forces acting on the lung parenchyma can be multiplied when applied at the interface between closed and open lung units (7). Thus, atelectatic alveoli can act like "stress risers" amplifying a harmless amount of stress to a dangerous one, generating atelectotrauma (8). The biological response triggered by barotrauma, volutrauma and atelectotrauma is known as bio-trauma and it is responsible for development of multiorgan dysfunction in patients with lung injury. (9)

Protective lung ventilation: Open Lung Strategy

Taking into account the pathophysiological mechanisms of lung damage described above, the "Open lung Ventilation" protective strategy aims to minimize stress and stain accumulated in the lungs during mechanical ventilation targeting to a homogenous lung without stress risers (10). Specifically, this protective strategy is based on three pillars. Firstly, it prioritizes ventilation with low TV (≤ 6 ml /Kg IBW) so that global lung strain remains in safe limits. Secondly, it recommends maintaining low transpulmonary pressure to reduce global stress exerted in the lung. Even when not possible to determine transpulmonary pressure, it proposes using the **driving pressure** of the respiratory system as a surrogate, targeting at values less than 12-15mmHg. Finally, this strategy endorses the use of optimal end expiratory pressure (PEEP) to eliminate stress risers and achieve lung homogeneity. In this way the reduction of both local and global stress is promoted.

Conclusion

Overall, the open lung strategy aims to protect the lung from the injurious effect of mechanical ventilation, focusing on restoring normal physiology and anatomy in ventilated lungs.

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