Β1.Γ. COMMENTARY-CONCLUSIONS ON THE DEBATE: IS IT THE END OF OPEN LUNG VENTILATION STRATEGY?

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Introduction

Mechanical ventilation supports gas exchange and alleviates the work of breathing when the respiratory muscles are overwhelmed by an acute pulmonary or systemic insult or when paralyzed, as during surgery when neuromuscular blockade is used to facilitate the work of surgeons.

As every powerful treatment, mechanical ventilation has its adverse side effects, which are classically referred to as ventilator-induced lung injury (VILI).[1] In this perspective, ventilator management warrants close attention because inappropriate ventilation can result in injury to the lungs or respiratory muscles and worsen morbidity and mortality.[1]

During the last decades research emphasis has shifted from treating to preventing ventilator induced lung injury (VILI) using preemptive ventilator strategies applied to the normal lung in patients at high-risk.[2]

Ventilation Strategies

- **Open Lung Approach (OLA)**

Protective mechanical ventilation during anesthesia aims at minimizing lung injury and its inflammatory response, and has been associated to a decrease in postoperative pulmonary complications (PPCs).[5, 8]

The Open Lung Approach recommends a protective ventilation strategy with the use of a low, more physiologic tidal volume ($V_T \leq$ 6 ml /Kg IBW).[4] A potential side effect of low-V_T ventilation is the reduction of the functional volume of the lung manifested as lung collapse.This increases lung heterogeneity and thus the driving pressure (DP). Driving pressure is the pressure gradient needed to generate a given V_T. It is calculated as plateau pressure minus PEEP and scales V_T to the size of the functional lung volume.[5] Another potential consequence of lung collapse is the impairment in ventilatory efficiency. For this reason, this approach combines low V_T with application of lung recruitment maneuvers (RMs) and an individualized positive end-expiratory pressure (PEEP) level that prevents lung collapse. A main challenge is the selection of optimal end expiratory pressure (PEEP) in order to eliminate stress risers and achieve homogeneity in the lung.

Preemptive OLA ventilator strategies have been shown to reduce the complications of mechanically ventilated patients with the believed mechanism to be maintaining an open, homogeneously ventilated lung and minimizing repetitive alveolar collapse and expansion (RACE) with each breath.[4-6] However, existing preemptive strategies use the same "one-size-fits-all" approach that is currently used to treat established ARDS and have not yet shown a clear reduction in ARDS incidence.[7]

- **Closed Lung Ventilation**

A strategy of mechanical ventilation that is protective to lungs may also cause harm to other organ systems. The potential for harm caused by protective ventilation was reported in PROVHILO trial, in which patients receiving higher PEEP and lung recruitment maneuvers (RMs) developed intraoperative hypotension more frequently and needed more vasoactive drugs.[8] These findings were in part in line with the finding that protective ventilation was associated with a higher incidence of intraoperative hypotension in a previous French trial.[7]

Advocates of the "Rest Lung Model" consider that the use of low tidal volumes, rather than PEEP, recruitment maneuvers, or a combination of these two, is the most important determinant of protection in intraoperative mechanical ventilation. In this model protective ventilation includes low tidal volumes (approximately 6 to 8 ml/kg IBW) combined with low PEEP because the use of higher PEEP and recruitment maneuvers would not confer further protection against PPCs and could deteriorate the hemodynamics.

The conception of "Let the Lung Rest" during mechanical ventilation is supported by the findings of a recent large multicenter international trial (ART) that demonstrated that an Open Lung strategy improved arterial oxygenation and driving pressure compared with the control group, but appeared to worsen patient outcomes, including mortality. ART investigators commented that high PEEP can reduce VILI from injurious tidal opening and closing but also raises intracardiac pressures, including right atrial pressure, which impedes venous return and cardiac output. Moreover, especially in the absence of significant lung recruitment, PEEP increases right ventricular afterload by compressing alveolar septal capillaries, increasing pulmonary vascular resistance. Shock from acute right ventricular failure, and high levels of PEEP may have contributed to mortality.

Ultimately, allowing part of the lung to stay closed with permissive atelectasis may be more patient-protective than aggressive efforts to keep the lung open.

Protective lung ventilation: Keep the lung open or closed? More questions than answers

In the last years, the effort to reduce mechanical ventilation-related lung damages converted into the widespread acceptance of the open lung strategy (ie, high PEEP associated with a low tidal volume) as the best way for treating not only patients with ARDS but also patients with normal lungs at risk for VILI as during surgery with high inflammatory load. However, several banks of clinical data seem to contrast with this belief.[7-8]

After many years of research several issues on protective ventilation are still presented as open: (1) The definition and the assessment of VILI in the clinical setting seem, per se, to be questionable; (2) What is the mortality attributable to VILI in mechanically ventilated patients?; (3) Which are actual mechanical triggers of VILI?; (4) Which are the lung conditions that favor it? (5) Do all mechanisms have the same contribution to VILI development? (6) Does better physiology translates into improved clinical outcome? (7) Which is the best way to open the lung: is optimal PEEP really optimal? (8) Which is the optimal mechanical breath? (9) Should we leave the patient breathe or not when in severe ARDS? (10) Is the concept of safe mechanical ventilation utopia?

Only when these questions are answered, we will be able to formulate a rational and, most likely, a predictably effective approach to VILI prevention.[1]

THE TRUTH LIES BETWEEN TWO MODELS: GATTINONI'S ENERGY MODEL AND THE DAWN OF PERSONALISED MECHANICAL VENTILATION

The evolution of VILI since its first description is characterized by the use of terminology that reflects the cause of the injury rather than its effects. Initially barotrauma was at the basis of VILI and afterwards volutrauma and atelectrauma were added as possible mechanisms to conclude in our days that Ergotrauma and mechanical power can include all above mentioned mechanisms and explain better the development of VILI through interaction of mechanical energy of the ventilator and the respiratory system of the patient.[4]

Therefore, under the term VILI different pathophysiological mechanisms are included, each one of them with its own different pathways, ultimately leading to possibly different manifestations.

In the proposed energy model by Gattinoni and colleagues, VILI is caused by the delivery of a critical amount of mechanical energy to the lung. The mechanical energy is either so great as to cause stress-at-rupture (eg, pneumothorax) or, when below the stress-at rupture threshold, it is delivered for a sufficient time.

In other words, the greater the mechanical energy applied over time, the greater the amount of lung damage in the extracellular matrix and the cell membrane. Above a certain, unknown threshold of energy/time, the rate of microscopic damage will eventually overcome the repair capability of the lung structures, leading to VILI.

Therefore, energy and time are the 2 essential components of VILI development and, considered together, they define the mechanical power. All mechanical factors implied in ventilation (Tidal Volume, Driving Pressure, Flow, Resistances, Respiratory Rate, and PEEP) are different components of a unique physical variable, which is the energy delivered over time, that is, the mechanical power.

Although the relative weight of each component may vary in the different ventilatory settings, there is no doubt that its dynamic element plays a major role. Another important factor is the distribution pattern of the energy applied to the lung which mainly depends on the homogeneity of the lung.

Therefore, whatever attempt to reduce or abolish the risk of VILI needs a characterization of the patient's lungs (size and homogeneity) and a proper set of all ventilation components. This may be the only way to rationally estimate the risk of VILI for a given ventilatory setting.1

CONCLUSION

Overcoming the clinical challenges and personalized mechanical ventilation

The most important lesson in the 65 years since Bjorn Ibsen's intervention with positive pressure ventilation in the Copenhagen poliomyelitis outbreak, has been that the outcomes of mechanically ventilated patients can best be improved by finding ways to prevent iatrogenic injury from the ventilator itself. Further advances in prevention of injury from mechanical ventilation

might be achieved by tailoring mechanical ventilation to the physiological characteristics of the individual patient.

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