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REVIEW

Review of the article - What every paediatrician needs to know about mechanical ventilation

Ligia Febraro[1], Vanessa Soares Lanziotti[2,3]

It is indisputable that invasive mechanical ventilation (IMV) is one of the most frequently performed interventions in critically ill patients in pediatric intensive care units (PICUs), particularly in those with acute respiratory failure (ARF). Over recent decades, technical knowledge regarding mechanical ventilators has advanced significantly; however, alongside these advances, awareness has also grown regarding their adverse effects, especially when ventilation is applied inappropriately, without consideration of the individual respiratory mechanics of each patient. This article addresses the lack of pediatricians' knowledge concerning ventilatory mechanics, which may lead to harm from IMV, outweighing its potential benefits. Ventilatory parameters must be individualized for each patient in order to avoid excessive or insufficient ventilatory support, which may result in three main types of injury: ventilator-induced lung injury (VILI), patient self-inflicted lung injury (P-SILI), and ventilator-induced diaphragmatic dysfunction (VIDD).

The article emphasizes the importance of understanding the basic physiology of ventilatory mechanics in order to adequately manage a patient receiving IMV. One of the essential concepts presented is airflow. All air movement depends on the presence of a pressure gradient. Therefore, for airflow to reach the alveoli, the ventilator must generate positive pressure (peak inspiratory pressure) sufficient to overcome both the elastic recoil of the lungs and the resistive pressure of the patient's airways. Pulmonary elastance (the lung's ability to return to its initial volume after distension, that is, how stiff the lung is) can influence this pressure gradient.

Another fundamental concept discussed is positive end-expiratory pressure (PEEP), defined as the pressure maintained at the end of expiration to prevent alveolar collapse. Peak inspiratory pressure refers to the maximum pressure required to overcome airway resistance. However, the pressure that effectively reaches the alveolar level is known as plateau pressure, and the pressure truly responsible for alveolar distension is the transpulmonary pressure at the end of inspiration. This pressure results from the difference between plateau pressure and pleural pressure (which opposes plateau pressure) and reflects the stress generated by alveolar distension. To identify excessive pulmonary stress, driving pressure can be calculated, defined as the difference between peak inspiratory pressure and PEEP.

[1] Instituto de Puericultura e Pediatrica Martagão Gesteira - Universidade Federal do Rio de Janeiro (IPPMG-UFRJ), Residência Médica em Pediatria (em andamento) - Rio de Janeiro - Rio de Janeiro - Brazil.
[2] Instituto de Puericultura e Pediatrica Martagão Gesteira - Universidade Federal do Rio de Janeiro (IPPMG-UFRJ), Unidade de Terapia Intensiva Pediátrica - Rio de Janeiro - Rio de Janeiro - Brazil.
[3] Instituto de Puericultura e Pediatrica Martagão Gesteira - Universidade Federal do Rio de Janeiro (IPPMG-UFRJ), Unidade de Pesquisa Clínica - Rio de Janeiro - Rio de Janeiro - Brazil.

Correspondence to:
Vanessa Soares Lanziotti.
Instituto de Puericultura e Pediatria Martagão Gesteira - IPPMG. Rua Bruno Lobo, 50, Cidade Universitária, Rio de Janeiro, RJ, Brazil. CEP: 21941-912. E-mail: vslanziotti@gmail.com

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The article addresses the most important mechanisms of lung injury induced by inappropriate mechanical ventilation:

1. **Ventilator-induced lung injury (VILI):** This may be caused by what is known as *barotrauma* (when ventilation is delivered with high peak inspiratory pressures), *volutrauma* (caused by excessive tidal volume), *atelectrauma* (atelectasis leading to compensatory overdistension of the remaining non-atelectatic alveoli), and, finally, *biotrauma* (resulting from atelectasis or alveolar overdistension itself, with subsequent release of inflammatory mediators that may reach the systemic circulation).
2. **Patient self-inflicted lung injury (P-SILI):** This is caused by spontaneous breathing. Allowing spontaneous ventilation has its benefits; however, when it is intense and sustained, it can damage the lungs. In this context, it is important to understand the concept of *pendelluft*, which refers to the transient movement of air from less injured lung regions with greater compliance toward more severely injured regions with lower compliance, leading to excessive distension and increased injury. The article mentions a randomized study in which the use of noninvasive ventilation, dependent on spontaneous breathing prior to intubation, resulted in worse outcomes, which may be explained by the occurrence of P-SILI in these patients.
3. **Ventilator-induced diaphragmatic dysfunction (VIDD):** Excessive ventilatory support and prolonged IMV may lead to diaphragmatic atrophy due to muscle disuse, whereas insufficient ventilatory assistance may result in compensatory diaphragmatic overload, inducing inflammation and thickening of the diaphragm. The degree of diaphragmatic thickening may be decisive in the extubation process and in the prolonged need for noninvasive ventilation after extubation. Other mechanisms of diaphragmatic injury include patient–ventilator asynchrony and the maintenance of high levels of PEEP, which may also contribute to diaphragmatic atrophy.

These three mechanisms of injury may coexist, and it is necessary to find a balance in titrating ventilatory support to prevent them. This balance varies according to the phase of lung disease in which the patient is, and there are specific strategies to mitigate these injuries (Figure 1).

In order to prevent VILI, especially in patients with reduced lung compliance, such as those with Pediatric Acute Respiratory Distress Syndrome (PARDS), the use of lower tidal volumes is recommended, ideally around 6 mL/kg, avoiding

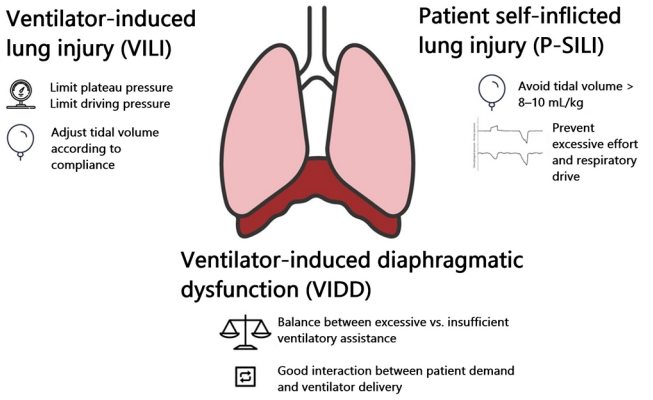


Figure 1. Graphic summary of the main approaches to limit ventilator-induced lung injury (VILI), ventilator-induced diaphragmatic dysfunction (VIDD), and patient self-inflicted lung injury (P-SILI).

excessive ventilation. In addition, patients with lower compliance tend to exhibit higher driving pressure, which increases the risk of VILI. Therefore, it is recommended to maintain driving pressure below 15 cmH_2O by limiting inspiratory pressures (maximum plateau pressure of 28 cmH_2O) and providing higher levels of PEEP. Adequate PEEP helps prevent alveolar collapse, promotes more homogeneous ventilation of the airways, and reduces atelectasis and compensatory hyperdistension of healthier lung regions.

The ideal PEEP level is one that minimizes the number of collapsed and overdistended alveoli, allowing for homogeneous lung redistribution and achieving the highest possible lung compliance, as both collapsed and hyperdistended alveoli reduce compliance. PEEP can initially be titrated using PEEP–FiO_2 combination tables and subsequently individualized by balancing oxygenation and hemodynamic stability. Lung recruitment maneuvers may help identify patients who could benefit from higher PEEP levels. This lung-protective strategy is more easily achieved when ventilation is delivered in pressure-controlled ventilation (PCV) mode, in which inspiratory pressures are set and tidal volume varies according to lung compliance and airway resistance. Plateau pressure can be measured by performing a manual end-inspiratory pause, during which airflow is zero.

Regarding P-SILI, the ideal approach is to objectively quantify the patient's respiratory effort, for example by measuring esophageal pressure during inspiration or by using noninvasive alternatives such as occlusion pressure (Pocc), which is available on some ventilators. Tidal volumes above 8–10 mL/kg during spontaneous breathing should be avoided. Sedation should be optimized, PEEP adjusted to higher levels, and inspiratory effort reduced by allowing the ventilator to fully

control ventilation, using volume-controlled modes when necessary. Daily assessments of readiness for extubation through spontaneous breathing trials are also essential, contributing to a reduction in the risk of VIDD.

The article concludes by emphasizing that mechanical ventilation is a "double-edged sword." Its importance and benefits are undeniable, as it can be life-saving for critically ill children, particularly those with respiratory failure. However, without a basic understanding of respiratory physiology and mechanics, and without appropriate adjustment of ventilatory support to the individual needs of each patient, its adverse effects may outweigh its benefits, contributing to worse clinical outcomes. Current concepts therefore increasingly aim to understand how to achieve an optimal balance between excessive and insufficient ventilatory support.

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