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REVIEW ARTICLE

Patient self-inflicted lung injury and ultraprotective mechanical ventilation: a narrative review

Lesión pulmonar autoinducida por el paciente y ventilación mecánica ultraprotectora: una revisión narrativa

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Abstract

Patient self-inflicted lung injury (P-SILI) describes the contribution of the patient's own respiratory effort to lung damage during mechanical ventilation (MV). Ultraprotective mechanical ventilation (UMV) and physiology-guided invasive monitoring may reduce its occurrence.The objective was to review the pathophysiology of P-SILI, define the concept of UMV, and analyze current evidence regarding its clinical use. P-SILI arises from increased lung stress and strain during vigorous inspiratory effort, aggravated edema due to negative transcapillary pressures, and patient-ventilator asynchrony. UMV aims to reduce these mechanisms using lower tidal volumes and plateau pressures than standard protective ventilation (< 6 mL/kg predicted body weight, < 25 cmH_2O), decreasing stress applied to lung tissue. As conclusion, P-SILI represents a key driver of acute lung injury progression. UMV emerges as a strategy to mitigate this phenomenon, although its optimal implementation and impact on clinical outcomes require further validation in controlled trials.

Keywords: *Patient self-inflicted lung injury. Ultraprotective mechanical ventilation. Lung stress. Lung strain.*

Resumen

La lesión pulmonar autoinducida por el paciente (P-SILI) es un concepto fisiopatológico que reconoce la contribución del esfuerzo respiratorio del paciente al daño pulmonar durante la ventilación mecánica (VM). La ventilación mecánica ultraprotectora (VMUp) así como la monitorización invasiva basada en mecanismos fisiopatológicos implicados se pronostica que puede disminuir su incidencia. Esta revisión revisa la fisiopatología de la P-SILI, busca definir el concepto de VMUp y analizar la evidencia actual sobre su aplicación clínica. La P-SILI se genera por aumento en el estrés y la tensión pulmonar durante el esfuerzo inspiratorio, así como la exacerbación del edema por presiones transcapilares negativas y la asincronía paciente-ventilador. La VMUp busca reducir esto mediante volumen corriente y presión meseta más bajos que los estándares (< 6 mL/kg de peso predicho, < 25 cmH_2O), así reducir el estrés generado en el tejido pulmonar y por lo tanto la lesión generada. La P-SILI representa un componente crucial en la evolución del daño pulmonar agudo. La VMUp emerge como estrategia para mitigarla, aunque su implementación óptima y su impacto en desenlaces clínicos requieren mayor validación en ensayos controlados.

Palabras clave: *Lesión pulmonar autoinducida por el paciente. Ventilación mecánica ultraprotectora. Estrés pulmonar. Tensión pulmonar.*

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Introduction

Nowadays, it is recognized that patients receiving mechanical ventilation (MV) may actively contribute to the progression of lung injury through their own respiratory effort. This phenomenon, termed patient self-inflicted lung injury P-SILI, proposes that strong inspiratory efforts, patient-ventilator asynchronies, and markedly negative intrathoracic pressures can intensify injury in an already vulnerable lung parenchyma[1].

Several studies have shown that neuromuscular blockade in patients with acute respiratory distress syndrome (ARDS) significantly improves oxygenation, likely by reducing spontaneous respiratory effort[2]. Complementary animal experiments have demonstrated that artificially increasing inspiratory effort through pharmacologic stimulation can induce severe lung injury, both clinically and histopathologically[3].

The term "P-SILI" was introduced in 2017 to describe this phenomenon, highlighting the risk that a patient's respiratory drive and effort may worsen lung injury-especially in severe ARDS and hypoxemic respiratory failure[1].

In severe ARDS, these efforts may be difficult to control, and the complex patient-ventilator interaction can lead to regional overdistension, exaggerated alveolar recruitment, and heterogeneous ventilation distribution[4-6].

This paradigm has transformed the understanding of ventilator-induced lung injury (VILI). It is now interpreted as the result of the interaction between mechanical support and spontaneous respiratory activity. Under this perspective, conventional protective ventilation may be insufficient to prevent injury progression. This has led to the exploration of ultraprotective MV (UMV) strategies, which further reduce ventilatory parameters (tidal volume < 6 mL/kg, plateau pressure < 25 cmH_2O), often requiring extracorporeal CO_2 removal (ECCO2R) to maintain adequate gas exchange[7].

This narrative review aims to analyze the pathophysiology of P-SILI and evaluate the emerging role of UMV as a potential strategy to mitigate its deleterious effects.

P-SILI: epidemiology, pathophysiology, and identification

P-SILI results from excessive mechanical forces applied to a heterogeneous, vulnerable lung. Its epidemiology is not well established, as it represents a pathophysiological mechanism rather than an independent diagnosis. Most available studies are narrative reviews, animal or computational models, and clinical observations in contexts such as ARDS or COVID-19[8].

No global incidence rates have been identified, as P-SILI overlaps with other forms of lung injury and is not routinely diagnosed[9]. ARDS affects approximately 10% of critically ill patients in ICUs, with mortality of 30-50% pre-COVID-19, and P-SILI is considered a contributing factor in patients who generate vigorous respiratory efforts, though without specific quantification[10].

Its central mechanisms can be divided into four interrelated components:

Increased lung stress and strain

During spontaneous inspiration, diaphragmatic contraction generates negative pleural pressure. This increases transpulmonary pressure (PL), the true determinant of stress. In ARDS lungs with reduced functional residual capacity, even normal or high effort can produce disproportionately high stress and strain in opened regions, leading to overdistension[11]. This parallels ventilator-induced volutrauma but is driven by the patient.

Worsening of pulmonary edema

Markedly negative pleural pressures significantly increase the transmural gradient across pulmonary capillaries, promoting edema formation. Increased venous return and right ventricular preload may further elevate capillary pressures, especially in patients with left-sided dysfunction[12].

Pendelluft effect and atelectrauma

In heterogeneous lungs, rapid inspiratory effort may redistribute gas from long time-constant regions to short time-constant regions without net airflow at the airway opening. This causes overdistension of healthier units while collapsed units remain derecruited or worsen, perpetuating atelectrauma[6].

Patient-ventilator asynchronies

Asynchronies such as ineffective effort, double triggering, and excessive effort worsen P-SILI. Double triggering results in two consecutive breaths, generating high effective tidal volume and peak pressure. Ineffective efforts increase the work of breathing without ventilation, contributing to fatigue and injury[13].

Identification of P-SILI

Patients at greatest risk are those with acute respiratory failure and vigorous inspiratory efforts. Risk is amplified by lung heterogeneity, underlying inflammation, and non-invasive or assisted ventilation. Other risk factors include pulmonary hypertension, intense diaphragmatic effort, and pre-existing lung disease[14].

Early detection depends on monitoring excessive respiratory effort. Esophageal pressure (Pes) and P0.1 are validated tools[15].

Excessive negative pleural pressures increase PL, producing gas redistribution (Pendelluft), regional overdistension, endothelial/epithelial injury, and worsening lung damage[16].

Pes permits estimation of PL and evaluation of compartmental mechanics. Proposed safety limits for dynamic PL are < 15-20 cmH_2O[9].

P0.1 correlates with respiratory drive and excessive inspiratory effort when > 3.5-4.0 cmH_2O and identifies low effort when < 1.0 cmH_2O, making it a practical tool to guide ventilation and prevent P-SILI[15].

Ultraprotective invasive MV: a response to P-SILI?

Protective ventilation (tidal volume 6-8 mL/kg, plateau pressure < 30 cmH_2O) became standard after the ARDSNet trial reduced mortality from 40% to 31%. However, in severe ARDS, mechanical power remained high (> 15 J/min), contributing to persistent VILI[17].

If lung injury is driven by both ventilator and patient effort, a logical strategy is minimizing all mechanical forces-leading to the concept of UMV.

UMV uses very low tidal volumes (< 4-6 mL/kg predicted body weight) and plateau pressure < 25 cmH_2O, aiming to reduce cyclic and static stress and limit volutrauma and P-SILI. By lowering mechanical power and energy applied to the lung, it seeks to interrupt lung injury progression[7].

A 2007 Italian pilot study explored low tidal volume ventilation in ARDS and showed reduced inflammatory markers and improved compliance without significant hypercapnia. This highlighted the insufficiency of 6-8 mL/kg in heterogeneous lungs and encouraged further investigation of ultralow tidal volumes[18].

Current clinical evidence

The systematic review and meta-analysis by Stommel et al. (2024) found that $ECCO_2R$ facilitates UMV by lowering $PaCO_2$ and increasing pH. Although mechanical load decreases, no mortality or length-of-stay benefit was demonstrated, indicating a need for stronger evidence[19].

In severe ARDS on extracorporeal membrane oxygenation (ECMO), the ECMOVENT study compared quasi-apneic ventilation (1 mL/kg) versus. pressure-controlled ventilation with ΔP 8 cmH_2O and positive end-expiratory pressure 14 cmH_2O. Both strategies significantly reduced mechanical load without differences in survival or ECMO duration[20].

Similarly, Costa et al. (2024) found that extended lung protection with controlled ΔP (≤ 15 cmH_2O) and respiratory drive modulation improved lung recovery in COVID-19 ARDS[21].

Clinical implications and future perspectives

Identifying patients at risk of P-SILI is essential. Persistent asynchrony elevated respiratory drive, vigorous effort, and inadequate gas exchange despite protective MV may prompt the use of more aggressive strategies.

Recent literature supports the idea that UMV reduces mechanical injury and may improve recovery, but evidence on mortality remains inconclusive. Advanced monitoring-Pes, inspiratory effort, P0.1-may reduce P-SILI by tailoring interventions to underlying physiology[22,23].

Conclusion

P-SILI represents a central component in acute lung injury pathophysiology, particularly in patients with preserved spontaneous breathing. Excessive effort may worsen injury through stress amplification, gas redistribution, and asynchrony. UMV facilitated by extracorporeal support represents a logical evolution of protective ventilation aimed at minimizing all mechanical aggression. Although high-level evidence on mortality impact is still emerging, its physiological basis is solid. A personalized approach guided by patient-ventilator interaction, P0.1, and Pes monitoring may improve outcomes, especially in severe ARDS.

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Conflicts of interest

The authors declare no conflicts of interest.

Ethical considerations

Protection of human subjects and animals. The authors declare that no experiments on humans or animals were performed for this research.

Confidentiality, informed consent, and ethical approval. This study does not involve personal patient data, medical records, or biological samples, and does not require ethical approval. SAGER guidelines do not apply.

Declaration on the use of artificial intelligence. The authors declare that no generative artificial intelligence was used in the writing or creation of the content of this manuscript.

References

1. Brochard L, Slutsky A, Pesenti A. Mechanical ventilation to minimize progression of lung injury in acute respiratory failure. Am J Respir Crit Care Med. 2017;195:438-42.
2. Coggeshall JW, Marini JJ, Newman JH. Improved oxygenation after muscle relaxation in adult respiratory distress syndrome. Arch Intern Med. 1985;145:1718-20.
3. Mascheroni D, Kolobow T, Fumagalli R, Moretti MP, Chen V, Buckhold D. Acute respiratory failure following pharmacologically induced hyperventilation: an experimental animal study. Intensive Care Med. 1988;15:8-14.
4. Morais CC, Koyama Y, Yoshida T, Plens GM, Gomes S, Lima CA, et al. High positive end-Expiratory pressure renders spontaneous effort noninjurious. Am J Respir Crit Care Med. 2018;197:1285-96.
5. Yoshida T, Roldan R, Beraldo MA, Torsani V, Gomes S, De Santis RR, et al. Spontaneous effort during mechanical ventilation: maximal injury with less positive end-expiratory pressure. Crit Care Med. 2016; 44:e678-88.
6. Yoshida T, Torsani V, Gomes S, Santis RR, Beraldo MA, Costa EL, et al. Spontaneous effort causes occult pendelluft during mechanical ventilation. Am J Respir Crit Care Med. 2013;188:1420-7.
7. Gattinoni L, Marini JJ, Pesenti A, Quintel M, Mancebo J, Brochard L. The "baby lung" became an adult. Intensive Care Med. 2016;42:663-73.
8. Sklienka P, Frelich M, Burša F. Patient self-inflicted lung injury-A narrative review of pathophysiology, early recognition, and management options. J Pers Med. 2023;13:593.
9. Deshwal H, Elkhapery A, Ramanathan R, Nair D, Singh I, Sinha A, et al. Patient-self inflicted lung injury (P-SILI): an insight into the pathophysiology of lung injury and management. J Clin Med. 2025;14:1632.
10. Carteaux G, Parfait M, Combet M, Haudebourg AF, Tuffet S, Mekontso Dessap A. Patient-self inflicted lung injury: a practical review. J Clin Med. 2021;10:2738.
11. Yoshida T, Amato MB, Kavanagh BP, Fujino Y. Impact of spontaneous breathing during mechanical ventilation in acute respiratory distress syndrome. Curr Opin Crit Care. 2019;25:192-8.
12. 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;359:2095-104.
13. Blanch L, Villagra A, Sales B, Montanya J, Lucangelo U, Luján M, et al. Asynchronies during mechanical ventilation are associated with mortality. Intensive Care Med. 2015;41:633-41.
14. Cruces P, Retamal J, Hurtado DE, Erranz B, Iturrieta P, González C, et al. A physiological approach to understand the role of respiratory effort in the progression of lung injury in SARS-CoV-2 infection. Crit Care. 2020;24:494.
15. Simón JM, Montosa CJ, Martínez Carmona JF, Delgado Amaya MJ, Luna Castro J, Rodríguez Carmona A, et al. Effects of three spontaneous ventilation modes on respiratory drive and muscle effort in COVID-19 pneumonia patients. BMC Pulm Med. 2023;23:333.
16. Battaglini D, Robba C, Ball L, Silva PL, Cruz FF, Pelosi P, et al. Noninvasive respiratory support and patient self-inflicted lung injury in COVID-19: a narrative review. Br J Anaesth. 2021;127:353-64.
17. Acute Respiratory Distress Syndrome Network, Brower RG, Matthay MA, Morris A, Schoenfeld D, Taylor Thompson B. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. N Engl J Med. 2000; 342:1301-8.
18. Terragni PP, Rosboch G, Tealdi A, Corno E, Menaldo E, Davini O, et al. Tidal hyperinflation during low tidal volume ventilation in acute respiratory distress syndrome. Am J Respir Crit Care Med. 2007;175:160-6.
19. Stommel AM, Herkner H, Kienbacher CL, Wildner B, Hermann A, Staudinger T. Effects of extracorporeal CO_2 removal on gas exchange and ventilator settings: a systematic review and meta-analysis. Crit Care. 2024;28:146.
20. Rodriguez Y, Thomachot A, Deniel G, Mezidi M, Chauvelot L, Yonis H, et al. Physiological and clinical effects of two ultraprotective ventilation strategies in patients with veno-venous extracorporeal membrane oxygenation: the ECMOVENT study. Ann Intensive Care. 2025;15:111.
21. Costa EL, Alcala GC, Tucci MR, Goligher E, Morais CC, Dianti J, et al. Impact of extended lung protection during mechanical ventilation on lung recovery in patients with COVID-19 ARDS: a phase II randomized controlled trial. Ann Intensive Care. 2024;14:85.
22. Cronin JN, Formenti F. Experimental asynchrony to study self-inflicted lung injury. Br J Anaesth. 2023;130:e44-6.
23. Yoshida T, Amato MB, Kavanagh BP. Understanding spontaneous vs. ventilator breaths: impact and monitoring. Intensive Care Med. 2018; 44:2235-8.