ISSN: 2582-6751
Journal of Clinical Medical Research

ATHENAEUM SCIENTIFIC PUBLISHERS

Review Article

Revisiting Atelectrauma: (De-)Recruitment Under Compression as a Distinct Mechanical Stressor

Jan van Egmond[1,2*], Jan Paul Mulier[3,4]

[1]Department Anesthesiology, Radboud University Medical Centre, Nijmegen, The Netherlands
[2]Donders Institute for Brain, Cognition and Behaviour, Donders Centre for Cognition, Radboud University, Nijmegen, The Netherlands
[3]Faculty of Medicine and Health Sciences, Basic and applied medical sciences, Ghent University, Ghent, Belgium
[4]Department of Anesthesiology and Pain clinic, Ghent University Hospital, Ghent, Belgium

***Correspondence author:** Jan van Egmond, Department Anesthesiology, Radboud University Medical Centre, Nijmegen, The Netherlands and Donders Institute for Brain, Cognition and Behaviour, Donders Centre for Cognition, Radboud University, Nijmegen, The Netherlands;
Email: Jan.vanEgmond.kf@gmail.com

Citation: Van Egmond J, et al. Revisiting Atelectrauma: (De-)Recruitment Under Compression as a Distinct Mechanical Stressor. Jour Clin Med Res. 2026;7(2):1-5.
https://doi.org/10.46889/JCMR.2026.7205

Received Date: 14-05-2026
Accepted Date: 31-05-2026
Published Date: 07-06-2026

Abstract

Background: Atelectrauma is generally attributed to repetitive opening and closing of unstable lung units during mechanical ventilation. However, physiological airway closure and reopening occur normally during spontaneous breathing without apparent injury, suggesting that cyclicity alone may not fully explain ventilator-induced lung injury. We therefore reconsider atelectrauma from the perspective of the surrounding intrathoracic pressure environment in which recruitment occurs.

Discussion: During spontaneous breathing, inspiration is generated by increasingly negative pleural pressure, resulting in lung inflation within a decompressive pressure environment. In contrast, during positive pressure ventilation, both airway pressure and pleural pressure increase, such that reopening of peripheral lung units occurs under elevated surrounding intrathoracic pressure. We propose that transpulmonary pressure and driving pressure describe lung distension, but do not fully characterize the absolute pressure environment surrounding lung tissue and peripheral airways. As a conceptual approximation, this surrounding pressure environment may be represented by the mean of airway pressure and pleural pressure. Numerical examples demonstrate that comparable transpulmonary pressures may occur under profoundly different surrounding pressure conditions. In acute respiratory distress syndrome, elevated intrathoracic pressure may contribute to airway closure, altered peripheral airway patency, edema formation and loss of alveolar-airway coupling. Experimental observations comparing positive and negative pressure ventilation, as well as a clinical case of occult pneumothorax during positive pressure ventilation becoming apparent during spontaneous breathing, support the physiological relevance of this concept. We suggest that the injurious component traditionally attributed to atelectrauma may arise less from cyclic opening and closing per se than from repeated recruitment occurring within a mechanically constrained and compressed lung environment.

Conclusion: Atelectrauma may be better understood as a context-dependent phenomenon in which elevated surrounding intrathoracic pressure contributes importantly to injury generation. While transpulmonary pressure remains fundamental for describing lung distension, the surrounding pressure environment may represent an additional mechanical dimension relevant to airway closure, recruitment, edema formation and ventilator-induced lung injury. This perspective may have implications for ventilatory strategies aimed at reducing excessive intrathoracic pressure loading.

Keywords: Atelectrauma; Intrathoracic Pressure; Transpulmonary Pressures; Lungs; Positive Pressure Ventilation

Introduction

Recently Marini and Slutsky published an editorial discussing atelectrauma as a central mechanism of Ventilator-Induced Lung Injury (VILI) in Intensive Care Medicine [1]. While the concept that cyclic opening and closing of unstable lung units may contribute to injury is physiologically plausible, we believe that an essential distinction remains insufficiently emphasized: the difference between physiological airway closure and reopening under conditions of elevated intrathoracic pressure.

Physiological Airway Closure

Airway closure is a normal physiological phenomenon [2,3]. In healthy individuals, particularly in dependent lung regions, small airways may close near end-expiration and reopen during inspiration, especially around closing volume. This process occurs repeatedly during normal breathing, including in the seated posture, where gravitational gradients in Pleural Pressure (P_{pl}), combined with abdominal pressure acting on the diaphragm, increase P_{pl} in dependent regions and promote regional airway closure. Importantly, this ubiquitous cyclic behavior is not associated with detectable injury, suggesting that airway closure and reopening per se are not intrinsically harmful.

The Surrounding Pressure Environment, From Forced Expiration to Positive Pressure Ventilation

It is well established that during forced expiration, an increase in P_{pl} leads to dynamic compression of the intrathoracic airways, resulting in flow limitation and airway closure. This phenomenon, described in terms of the equal pressure point and downstream airway collapse, occurs even in healthy lungs and represents a fundamental mechanical property of the respiratory system. The observation that elevated surrounding pressure may narrow or close compliant airways therefore extends beyond pathological lungs and reflects a general mechanical principle [4]. By analogy, any increase in intrathoracic pressure irrespective of its origin may exert a similar compressive influence on peripheral airways. During Positive Pressure Ventilation (PPV), elevation of airway pressure (P_{aw}) is accompanied by a rise in P_{pl}, thereby altering the pressure environment surrounding the lung parenchyma. Although the pressure distribution differs from that during forced expiration, the resulting increase in absolute intrathoracic pressure may nevertheless promote airway narrowing or closure, particularly in dependent lung regions where P_{pl} is highest (Fig. 1). Transpulmonary pressure (P_{tp}), $P_{tp} = P_{aw} - P_{pl}$ describes the distending pressure of the lung and remains fundamental for understanding lung inflation. However, transpulmonary pressure and driving pressure do not fully characterize the absolute pressure environment surrounding lung tissue and peripheral airways. Because the lung parenchyma is mechanically interposed between main airways and pleural cavity (Fig. 1).

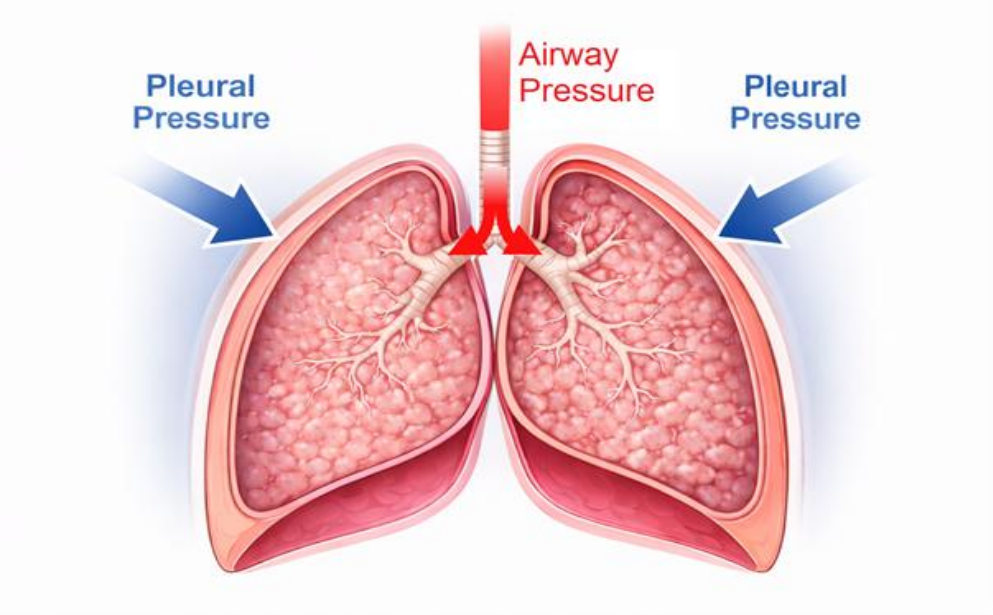

Figure 1: Pressures acting on the lung parenchyma during positive and negative pressure ventilation. The lung parenchyma is sandwiched between the pleural space (exposing P_{pl}) and the larger airways (exposing P_{aw}). Airway pressure is transmitted throughout the bronchial tree into the alveolar space and therefore contributes directly to the mechanical compression experienced by the lung tissue.

From a mechanical perspective, parenchyma is simultaneously exposed to P_{aw} from the luminal side and P_{pl} from the thoracic compartment. As a first-order conceptual approximation, the surrounding pressure environment (inthrathoracic pressure P_{it}) acting on the parenchyma may therefore be represented by the mean of P_{aw} and P_{pl}: $P_{it} \approx (P_{aw} + P_{pl})/ 2$.

We do not propose P_{it} as a replacement for P_{tp}, but rather as an additional descriptor of the absolute pressure environment surrounding the lung. This simplified representation is intended as a conceptual framework and does not account for regional heterogeneity, gravity-dependent gradients or local stress concentrations.

Type	State	P_{pl}	P_{aw}	P_{tp}	VT	P_{it}	P_{surr}
Normal	SB EE	-5	0	5		-2.5	0
Normal	SB EI	-10	0	10	5 x Compl = 500 ml	-5	0
Normal	NPV EE	-5	0	5		-2.5	0
Normal	NPV EI	-10	0	10	5 x Compl = 500 ml	-5	-10
Normal	PPV EE	-2.5	5	7.5		+1.25	0
Normal	PPV EI	2.5	15	12.5	5 x Compl = 500 ml	+8.75	0
ARDS*	PPV EE	17	14	-3		+15.5	0
ARDS*	PPV EI	21	29	8	484 ml	+25.0	0

End-expiratory and end-inspiratory values for P_{pl}, P_{aw}, P_{tp}, VT, P_{it} and P_{surr} during spontaneous breathing and mechanical ventilation for a normal healthy person and an ARDS patient. VT = Tidal Volume, SB = Spontaneous Breathing, EE = End–Expiratory, EI = End-Inspiratory, P_{surr} = Surrounding Pressure, ARDS = Adult Respiratory Distress Syndrome.
*) Data taken from Talmor, et al., [5]

Table 1: Numerical illustration of the separate role of P_{it}.

Numerical Illustration

A simplified numerical illustration helps clarify this distinction (Table 1). During spontaneous breathing at end-expiration, P_{pl} is typically approximately -5 cmH_2O while P_{aw} equals atmospheric pressure. During inspiration, P_{pl} becomes more negative, thereby generating lung inflation in a decompressive pressure environment (P_{it} negative). In contrast, during PPV both P_{aw} and P_{pl} increase, such that lung inflation occurs under elevated surrounding pressure conditions (P_{it} positive). Importantly, comparable P_{tp} may therefore occur under profoundly different absolute pressure environments. In patients with obesity or ARDS, P_{pl} may remain markedly elevated even at end-expiration. Under such conditions, dependent lung regions are particularly susceptible to airway closure. This may produce apparently paradoxical situations in which measured P_{aw} is lower than P_{pl} (-3 cmH_2O marked red in Table 1). In the presence of airway closure, however, P_{aw}–P_{pl} no longer reflects the true P_{tp} of the closed alveolar compartment. If gas remains trapped distal to a closed airway, alveolar pressure must remain greater than P_{pl} to prevent complete collapse, implying that the closed compartment has become mechanically decoupled from the proximal airway opening.

Airway Closure and Loss of Alveolar-Airway Coupling

Experimental observations may support the physiological relevance of P_{it}. Klassen, et al., demonstrated in *ex-vivo* porcine lungs that NPV achieved similar tidal ventilation with substantially smaller driving pressures than PPV [6]. Moreover, peripheral air leakage through standardized pleural defects was markedly greater during NPV, suggesting more effective transmission of ventilation to peripheral lung regions. Similar observations have been reported by Eckert, et al., supporting the concept that the mode by which pressure is applied to the lung may influence peripheral airway patency and regional ventilation independently of P_{tp} alone [7].

A comparable phenomenon may occur clinically. We recently described a patient with a surgically confirmed pneumothorax that remained radiologically occult during ongoing PPV, despite direct intraoperative aspiration of air by a puncture through the pleura into the peripheral lung by the surgeon [8]. Remarkably, the pneumothorax became immediately apparent upon return to spontaneous breathing. This observation is consistent with the possibility that elevated P_{it} during PPV may reduce peripheral airway patency or functional communication with pleural air collections, whereas spontaneous negative-pressure breathing restores peripheral connectivity.

This perspective suggests that reopening of peripheral lung units during PPV does not occur under physiological decompressive conditions, but rather under externally imposed compressive loading. Consequently, the injurious component traditionally attributed to atelectrauma may not arise from cyclic opening and closing per se, but from repeated recruitment within a mechanically constrained pressure environment. Recruitment against compression may therefore represent the relevant mechanical stressor.

Supportive Clinical Observations

This interpretation is consistent with several clinical observations. Prone positioning, particularly when accompanied by abdominal unloading, reduces regional P_{pl} and redistributes compressive forces, thereby facilitating recruitment and improving compliance [9,10]. Similarly, preservation of spontaneous breathing during assisted ventilation partially offsets ventilator-induced increases in P_{pl}, generating tidal volume through negative P_{pl} swings rather than through further elevation of P_{it} [11]. The beneficial effects of these strategies may therefore reflect, at least in part, reduction of compressive loading rather than solely improved ventilation-perfusion matching.

The concept of Patient Self-Inflicted Lung Injury (P-SILI) may also be reconsidered within this framework [12,13]. Vigorous inspiratory effort in a heterogeneous lung may generate large regional pressure gradients and dynamic flow redistribution. However, in the absence of airway closure or obstruction, cyclic expansion alone is unlikely to be intrinsically injurious. Rather, injury may arise when strong inspiratory effort interacts with regional closure (such as a closed ventilator valve), reopening phenomena or excessive transvascular pressure gradients promoting edema formation.

Similarly, the concept of volutrauma may warrant partial reinterpretation. Volutrauma takes place when lungs are inflated beyond vital capacity. Large tidal volumes below vital capacity are often tolerated during physiological spontaneous breathing considerably better than during PPV, despite comparable or greater volume excursions. During spontaneous breathing, lung inflation occurs while P_{pl} is strongly negative, whereas during PPV similar tidal volumes require elevated airway and pleural pressures. This suggests that at least part of so-called volutrauma may reflect pressure-related injury occurring within a compressed and mechanically constrained lung environment.

Taken together, these considerations suggest that discussions of VILI should more clearly distinguish between physiological airway closure and reopening and reopening occurring under elevated surrounding P_{it}. The latter, rather than cyclicity itself, may represent the critical injurious mechanism.

Implications for Ventilatory Strategy

This perspective may have implications for ventilatory strategy. In addition to limiting tidal volume and driving pressure, approaches that reduce excessive P_{it} loading including optimization of chest wall mechanics, judicious use of PEEP, preservation of spontaneous breathing (or Neurally Adjusted Ventilatory Assist, NAVA), flow-controlled ventilation strategies or reconsideration of negative pressure ventilation may more directly address the mechanical conditions associated with injury [14].

Conclusion

We propose that atelectrauma should be reconsidered as a context-dependent phenomenon in which the surrounding pressure environment rather than cyclic reopening alone determines whether airway closure and reopening remain physiological or become injurious. Transpulmonary pressure remains essential for describing lung distension but may not fully capture the mechanical environment experienced by peripheral airways and lung parenchyma during mechanical ventilation.

Conflict of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

Funding Statement

This research did not receive any specific grant from funding agencies in the public, commercial or non-profit sectors.

Acknowledgement
The authors have no acknowledgments to declare.

Data Availability Statement
The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Ethical Statement
The project did not meet the definition of human subject research under the purview of the IRB according to federal regulations and therefore was exempt.

Informed Consent Statement
Informed consent was obtained from all participants included in the study.

Authors' Contributions
All authors contributed equally to this paper.

References

1. Marini JJ, Slutsky AS. Atelectrauma: Promotion and prevention. Intensive Care Med. 2026;52:353-5.
2. Dollfuss RE, Milic-Emili J, Bates DV. Regional ventilation of the lung, studied with boluses of 133xenon. Respir Physiol. 1967;2:234-46.
3. Milic-Emili J, Torchio R, D'Angelo E. Closing volume: A reappraisal (1967–2007). Eur J Appl Physiol. 2007;99:567-83.
4. Hunter JG, Prajapati P, Bera K, Gupta A, Gupta A. Imaging depiction of hypoxic pulmonary vasoconstriction using dual-energy CT. Cureus. 2023;15(3):e36551.
5. 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.
6. Klassen C, Eckert CE, Wong J, Guyette JP, Harris JL, Thompson S, et al. *Ex-vivo* modeling of perioperative air leaks in porcine lungs. IEEE Trans Biomed Eng. 2018;65:2827-36.
7. Eckert CE, Harris JL, Wong JB, Thompson S, Kassis ES, Tsuboi M, et al. Preclinical quantification of air leaks in a physiologic lung model: Effects of ventilation modality and staple design. Med Devices (Auckl). 2018;11:433-42.
8. Mulier JP, van Egmond J, Beckers Perletti L, van Peteghem C, Desender L, Debaerdemaeker L. Delayed detection of iatrogenic pneumothorax after port catheter insertion under positive pressure ventilation: A case report. AA Pract. 2026;20:e02186.
9. Guérin C, Reignier J, Richard JC, Beuret P, Gacouin A, Boulain T, et al. Prone positioning in severe acute respiratory distress syndrome. N Engl J Med. 2013;368(23):2159-68.
10. Goins E, Grace A. Proning for ARDS. NUEM Blog. 2025.
11. Mauri T, Cambiaghi B, Spinelli E, Langer T, Grasselli G. Spontaneous breathing: A double-edged sword to handle with care. Ann Transl Med. 2017;5(14):292.
12. Morais CCA, Koyama Y, Yoshida T, Plens GM, Gomes S, Lima CAS, et al. High positive end-expiratory pressure renders spontaneous effort noninjurious. Am J Respir Crit Care Med. 2018;197(10):1285-96.
13. 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(12):2738.
14. Kuo NY, Tu ML, Hung TY, Liu SF, Chung YH, Lin MC, et al. A randomized clinical trial of neurally adjusted ventilatory assist versus conventional weaning mode in patients with COPD and prolonged mechanical ventilation. Int J Chron Obstruct Pulmon Dis. 2016;11:945-51.