ISSN: 2582-6751
Journal of Clinical Medical Research

ATHENAEUM SCIENTIFIC PUBLISHERS

Short Communication

The Stiff Lung Cries Out for Negative Pressure Ventilation

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Citation: Van Egmond J. The Stiff Lung Cries Out for Negative Pressure Ventilation. Jour Clin Med Res. 2025;6(3):1-3.

https://doi.org/10.46889/JCMR.2025.6307

Received Date: 23-09-2025

Accepted Date: 13-10-2025

Published Date: 20-10-2025

Introduction

Before the worldwide adoption of positive pressure ventilation (following the polio pandemic, Copenhagen, 1952), patients with atelectasis were curatively treated in the iron lung. After the transition to positive pressure, however, atelectasis became the main complication [1]. An early animal study drawing attention to this problem was countered with the argument that there can be no difference between positive and negative pressure ventilation (Loring) [2-4]. The claim was that transpulmonary pressure alone determines lung volume. As long as the transpulmonary pressure is the same, there is no difference. This message was repeated again in 2023 by Butler [5].

But Is There Really No Difference?

There is a fundamental difference and it is easy to demonstrate with a thought experiment. Place a weight on the abdomen of a supine person. Lung volume decreases and pleural pressure rises. Assume equal compliances of lung and chest wall (they are close in reality), each X ml/cmH_2O. Let the weight reduce lung volume by 2·X. This means pleural pressure rises by 2 cmH_2O and trans-thoracic pressure (pleural minus extrathoracic) falls accordingly. To restore the original lung volume, we must return trans-thoracic pressure to its baseline value. This can be done in two ways:

- By reducing ambient pressure, as in the iron lung (Negative Pressure Ventilation, NPV)
- By raising airway pressure, as in Positive Pressure Ventilation (PPV)

In both cases, the transpulmonary pressure and therefore the volume, is restored. But here lies the key difference:

With PPV, pleural pressure rises 4 cm H_2O above its baseline.

With NPV, pleural pressure is restored back to its baseline negative value.

Both achieve the same lung volume, but only NPV restores the pleural environment to its original physiological state.

Our Thought Experiment in Real Life

A compelling real-world equivalent of this thought experiment is the obese patient. Excess abdominal and thoracic tissue applies continuous external pressure on the lung, raising pleural pressure and, in the absence of extra effort of the patient, reducing end-expiratory volume. In such patients, the airways are not simply narrowed but often completely compressed. Behazin and colleagues have shown that there exists a measurable Airway Opening Pressure (AOP): below this threshold, no air enters the lung because conducting airways to the alveoli remain sealed shut [6]. Only when airway pressure exceeds the AOP does ventilation become possible. This phenomenon is the clinical manifestation of the lung being physically compressed between airway and pleural pressures-a striking confirmation of the difference between positive and negative pressure ventilation.

Why Pleural Pressure Matters

At relaxed end-expiration, pleural pressure is normally subatmospheric (≈ -5 cm H_2O, on average). The lung is "pulled away" from the chest wall. When airway pressure is raised to 10 cm H_2O in PPV, pleural pressure rises toward 0 cm H_2O. In this state, the lower lung regions are already pressed against the chest wall. Any further volume expansion means the lung is squeezed between airway pressure and pleural pressure, creating parenchymal stress and airway closure. These phenomena occur early

in PPV but are absent in NPV, where pleural pressure is simply restored to baseline.
The lung tissue is caught between pleural pressure and airway pressure. Normally, at FRC, these pressures are -5 and 0 cm H_2O, respectively. But with 10 cm H_2O airway pressure, the tissue is compressed between 0 and 10 cmH_2O. Averaged, that is an increase from -2.5 to +5 cm H_2O. Spread over an area of ~1000 cm^2, this corresponds to a 5 kg weight pressing directly on the lung tissue over that surface.

Several authors have measured pleural pressures in ARDS patients to optimize PEEP [7-10]. They consistently report values ≥ 15 cm H_2O, while PEEP itself is 10-15 cm H_2O. The lung is thus forcefully compressed and small airways are squeezed shut, leading inevitably to airway closure and atelectasis. Hedenstierna reports AOPs of 15 cm H_2O [11].

Lung-Protective Strategies: A proper Answer?
Protective strategies demand the lowest possible plateau and end-expiratory pressures, with the smallest feasible tidal volume. This makes sense:

- Low tidal volume minimizes the amplitude of sliding and shear of pleural surfaces
- Low pressures minimize the force pressing the lungs against the chest wall

Such strategies are increasingly applied not only in the ICU but also in the operating room and yet there is a paradox: during ventilation we restrict tidal volume to avoid "overstretch," but once extubated we encourage the patient to take the deepest possible breaths. Why? Increasing lung volume while pressing it against the thorax wall is harmful. Deep breaths evoked by strong negative pleural pressures are curative, resolving the atelectasis created by preceding PPV [12,13].

Conclusion
The danger of PPV is not transpulmonary pressure nor driving pressure. It is pleural pressure.

- PPV elevates pleural pressure above baseline, causing compression and atelectasis
- NPV restores pleural pressure to its physiological baseline, preventing airway closure and stress, resolving atelectasis

It is not only the stiff lung, but every lung requiring ventilatory support that cries out for negative pressure. Are we willing to listen?

Conflict of Interest
The author declares no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

Funding
No external funding was received for this case report.

Acknowledgment
None

Author's Contribution
Author read and approved the final manuscript.

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