

Complex ventilation problems with no simple solution

Ross Freebairn

DOI: https://doi.org/10.53097/JMV.10067

Cite: Freebairn R. Complex ventilation problems with no simple solution. J Mech Vent 2022; 3(4):196-198.

"For every complex problem there is an answer that is clear, simple, and wrong". H L Menken

The healthy lung contains approximately 300 million alveoli, although the critically ill the number of functional units would be considerably less. ¹ Each alveolus has its unique compliance and resistance, and with that each has a unique time constant and hysteresis. These affect the filling and emptying of the individual alveolar sac, and the energy required to distend the lung.

In contrast when determining a ventilation strategy for the critically ill patients at any time we can only usually determine a single parameter such as pressure, volume or flow for the entire respiratory system, with the others become dependent variables and we can measure only the "average" compliance and resistance. The inevitable result is that some alveoli will have sub optimal pressure and volume applied to them while some being over distended, others being underinflated. With many diseases processes heterogeneity of the lung increase, with a much wider range of ideal settings being required. Clinicians want a simple parameter to which to titrate our ventilation strategy. Unfortunately, previous targets have in several circumstance resulted in harm rather than benefit.

Sixty years ago, clinicians strove for metabolic and respiratory normality, aimed at normalizing carbon dioxide and oxygen levels, no matter what the physiological cost.²

Thirty years ago, recognizing that aspects of mechanical ventilation were causing harm, the focus shifted to simple respiratory mechanics with limitation of plateau pressure and tidal volumes. ^{3,4}

Subsequently this evolved to the concern about the driving pressure, mean airway pressure and most recently to the "power" of ventilation. ^{5,6,7} Changes in driving pressure, mean airway pressure and power is, in some circumstances associated with a change in outcome, but not always. ^{8,9}

The initial power calculation measures the entire energy expended over time in distending the lung and moving gas in and out of the alveoli and large airways.

Authors

1. BHB, MBChB, Dip Obs, FRCPE, FANZCA, FCICM, Intensive Care Consultant at Hawke's Bay Hospital, in Hastings, New Zealand Corresponding author: ross.freebairn@xtra.co.nz

Conflict of interest/Disclosure: None Funding: None

Journal of Mechanical Ventilation 2022 Volume 3, Issue 4

This open-access article is distributed under the terms of the Creative Commons Attribution Non-Commercial License (CC BY-NC) (http://creativecommons.org/licenses/by-nc/4.0/), which permits reuse, distribution and reproduction of the article, provided that the original work is properly cited, and the reuse is restricted to noncommercial purposes. For commercial reuse, contact: editor@journalmechanicalventilation.com

However, while the total energy transmitted to the respiratory system is easy to measure, much of this energy is dissipated and absorbed into the large and medium airways. ARDS in particular is not associated with pathology of the transmitting airways but of the alveolar sacs and alveoli. For mechanical damage to occur there by power, energy must be transmitted to this area.

Gas flow rates, and thus energy in the alveoli sac are very low and the gas exchange at the alveoli sac level occurs by diffusion rather than bulk flow. Having said this, the pressure, flows and thus energy, while much smaller by in comparison to the outer airway, are much larger than would occur during spontaneous breathing or gentle positive pressure ventilation. Working out where the energy is absorbed would appear to be important.

Calculating the determinants of the pressure resistance compliance and flows, and thus the power absorbed in the functional alveoli, and understanding the interaction between these potential determinants of harm is therefore a worthwhile exercise. If we begin to understand the effects manipulating ventilation at the alveolar level, we may be in a position to reliably reduce the damage done.

Daoud and Franck in this edition of the journal proffer an eloquent disguisition on alveolar compliance and resistance and describe ways in which we could make estimates of the effect of ventilation changes, using esophageal balloon manometry measure the trans alveolar pressure, and estimating the alveolar tidal volume using volumetric capnometry. ¹⁰ The article like the subject it addresses is complex and requires an active rather than passive read. It outlines the concepts clearly and highlights the need for accurate and exacting measurement. Complicating this is the need to provide simultaneous diaphragmatic and alveolar protective ventilation, which further complicates modelling of controlled ventilation strategies. ¹¹ It remains something to be addressed in the future.

Our previous understanding, which was informed only by the gross airway measurements, is insufficient as a guide to address the problem of protective ventilation and while it may be simple and clear is probably the wrong approach. Daoud and Franck' s dissertation is worthy of a close read, as it opens many areas of discussion and provides an insight into the difficulties we will face. As the authors themselves note, the subject will require further thought, and the theory needs to be supported by clinical investigation.

References

1. Lumb A, Thomas C.Functional anatomy of the respiratory tract. Nunn's Applied Respiratory Physiology. 8th ed2010 1:1-13e2.

2. Bendixen HH, Hedley-Whyte J, Laver MB. Impaired oxygenation in surgical patients during general anesthesia with controlled ventilation. A concept of atelectasis. N Engl J Med 1963; 269:991-996.

3. Hickling KG. Low volume ventilation with permissive hypercapnia in the Adult Respiratory Distress Syndrome. Clin Intensive Care 1992;3(2):67-78.

4. Slutsky AS. Consensus conference on mechanical ventilation-January 28-30, 1993 at Northbrook, Illinois, USA. Intensive Care Med 1994; 20(2):150-162.

5. Bellani G, Grassi A, Sosio S, et al. Driving pressure is associated with outcome during assisted ventilation in Acute Respiratory Distress Syndrome. Anesthesiology 2019; 131(3):594-604.

6. Freebairn RC. What do mean airway pressures mean?. Crit Care Med 2020:767-769.

7. Zhang Z, Zheng B, Liu N, et al. Mechanical power normalized to predicted body weight as a predictor of mortality in patients with acute respiratory distress syndrome. Intensive Care Med 2019; 45(6):856-864.

8. Lanspa MJ, Peltan ID, Jacobs JR, et al. Driving pressure is not associated with mortality in mechanically ventilated patients without ARDS. Crit Care 2019;23(1):424.

9. Fuller BM, Page D, Stephens RJ, et al. Pulmonary mechanics and mortality in mechanically ventilated patients without acute respiratory distress syndrome: A cohort study. Shock 2018; 49(3):311-316. 10. Daoud EG, Franck C. Alveolar mechanics: A new concept in respiratory monitoring. J Mech Vent 2022; 3(4):178-.188. 11. Goligher EC, Dres M, Patel BK, et al. Lung-And diaphragm-protective ventilation. Am J Respir Crit Care Med. 2020;202(7):950-961.



Submit a manuscript

https://www.journalmechanicalventilation .com/submit-a-manuscript/



Free membership

https://societymechanicalventilation.org /membership/