The Rise of the Machines: Why the future lies with less injurious adaptive ventilation strategies

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It has been 60 years since Bendixen, Hedley-White, and Laver described the progressive atelectasis and resultant hypoxemia that resulted from prolonged mechanical ventilation. [1] A proposed solution was to raise the tidal volume (V_T) from those recommended by Radford's nomogram for "proper ventilation" to 10 -15 ml/ kg. [2] It was less than four years later that Acute Respiratory Distress Syndrome (ARDS) was first reported. [3] Since then, clinicians and researchers have been searching for the ideal ventilation strategy to minimise the harm and optimise the outcomes from ventilatory support in the critically ill. Within a decade of the original description, animal work identified harm caused by excessive pressure, but it was two decades before Hickling's description of a protective ventilation strategy, and another decade before clear benefits of lowered V_T was confirmed. [4-6]Another twenty years on from this, an analysis of the larger ventilation trials suggested that driving pressure (DP), rather than V_T (per kilogram of ideal body weight) per se was most closely associated with mortality. [7]

In the homogenous lung, each alveolus will be stretched equally. However, in many critically ill patients, the lung acquires distinctly heterogenous alveoli, with disparate compliances. The pressure to adequately distend a diseased and non-compliant alveolus causes over-distension of less diseased, more compliant alveoli. Clearly large tidal volume used historically were harmful, but how low should the V_T be? Amato's concept was that the poorly compliant lungs were identified by those requiring large driving pressures. The lower DP are associated with lower mortality. If the pressure is the cause, limiting DP to less than 15 cmH_2O could translate to an overall survival benefit. This outcome remains unproven with lowered pressures comes lower V_T, especially when low compliance is the issue. Clinicians face a choice – accept a lower minute ventilation (Ve) and the resultant hypercarbia or achieve the desired Ve by using higher set respiratory rates. Hypercarbia and its effects on mortality deserves further serious consideration but is beyond the scope of this discussion. [8,9]

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Increasing the set respiratory rate results in increased energy transmitted to the airway and lung with increased "ventilation power" (work overtime). [10] Both DP and respiratory rate are important components of power calculations although DP have four-fold higher impact than the effect of respiratory rate on mortality. [10] "Ventilator power" is complex, and extremely dynamic. Compliance, resistance, or any operator- initiated changes influence the power, but that transmitted to alveoli may differ from total airway power.

New automatic real time measurement of ventilation power as well as computed power is now possible. [8,11]

Schibler's review of adaptive ventilation provides an excellent overview of the damaging, but potentially avoidable ventilator induced lung injury (VILI). [12] The three potential malfeasants - V_T, DP and "power of ventilation" can all potentially be addressed using an adaptive mode. Mechanical power was associated with mortality in some patients' groups with ARDS but the influences of alteration in flow patterns, power spikes and positive end expiratory pressure (PEEP) are less clear. [10,12] Nevertheless, the dynamic nature changes in compliance resistance, and operator-imposed changes are all potential hazards.

Compliance with optimal ventilation is poor, as clinicians often target short term goals, such as oxygenation and normocapnia rather than universally applying ventilation strategies that produce longer-term benefits to patients, such as smaller V_T. [13] Adaptive mechanical ventilation with automated minimization of inspiratory power may lead to more lung-protective ventilator settings when compared with adaptive mechanical ventilation according to Otis' equation. [14] By automating the response to changes, the human factor can be removed from the equation. The problem is that not all respiratory failure, or even ARDS, is the same, and while V_T, DP and the various iterations of the power are all suspects, there is not a constant association between these and mortality. [15-18] Possibly other metrics, such as mean alveoli or mean airway pressure, are important influences on outcome. [19] There is no doubt the formula to provide ideal ventilation will be complex and will certainly be beyond the ability of any clinician to compute on a breath-by-breath basis, even if they had the time. Adaptive ventilation can effortlessly alter the ventilation parameters to target less injurious patterns. As our knowledge of the important components that influence ventilator induced lung injury improves, adaptive ventilation strategy can modify to target the pattern required.

As Schibler states, we have some understanding of the predictors of poor outcome in ARDS but less knowledge about how those parameters optimized. It must be remembered that "association" does not imply "cause", and the manipulation of oxygenation, compliance, driving or mean airway pressure or mechanical power associated with harm are not guaranteed to automatically improve outcome. If we want to adopt a "perfect strategy, the twice daily ward round, or even an hourly bedside assessment, still will not provide sufficient precision and adequate response times to be useful. We will need real-time guidance and adaption from measured ventilatory parameters, to ensure we achieve the best outcomes for patients.

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