The Quality of Quality Metrics

To the Editor:

Perhaps the most important skills the respiratory care profession offers the medical community are those associated with life support: resuscitation and mechanical ventilation. We might wonder, therefore, why we have such high expectations[1] and yet so few metrics of quality performance in those areas. The study by Walsh et al[2] could be a landmark contribution in that direction. On the one hand, replication of their work will be a challenge for most institutions; what they have done involved special hardware (to port data from ventilators to the electronic health record) and custom web-based analytics software developed by their institution. Having attempted more modest projects that involve custom software and electronic health record integration,[3] I can appreciate the formidable barriers to entry. On the other hand, Walsh et al[2] have provided a detailed set of rules that others can use to assemble quality metrics for the domains of mechanical ventilation, oxygenation, and ventilator-induced lung injury. Their definitions for rules-based algorithms might inform tools already developed in the emerging field of cognitive computing (eg, WatsonPaths).

However, before adopting all of the rules, I would question those related to ventilator-induced lung injury. Specifically, the paper[2] defined barotrauma-free as "peak inspiratory pressure (PIP) $\leq$ 30 cm H_2O" and volutrauma-free as "exhaled tidal volume $>$ 4 mL/kg ideal body weight; $<$ 8 mL/kg ideal body weight." The results in the paper were reported as percentage of time spent in the "free" zones. First of all, these metrics were defined as measures reflecting ventilator settings, not lung trauma classifications as their names imply. Hence, they could be misleading. For example, someone reading only the abstract would probably conclude that freedom from the 2 types of traumas indicated actual patient outcomes. This could be avoided by simply naming these metrics in a way that reflects what they actually measure. For example instead of barotrauma-free, a more precise name might be low-risk PIP, and instead of volutrauma-free a better name would be low-risk tidal volume.

As the authors pointed out in the discussion, the quality of mechanical ventilation based on metrics like ventilator-associated pneumonia or incidence of pneumothoraces is an end point and hence is retrospective in nature. Although not explicitly stated as such, their study seemed to emphasize continuous, near real-time data analysis, rather than intermittent retrospective conclusions. In this context, monitoring PIP and tidal volume (V_T) makes sense. But the names of their associated metrics do not make sense.

As a minor point, the term barotrauma is an anachronism. It was apparently coined back in 1973 to indicate lung damage associated with high PIP. In 1992, Dreyfuss et al[4] showed that it was a high V_T, not a high PIP, that was the major factor responsible for mechanical lung damage. Accordingly, they coined the more accurate term volutrauma.[4] Gattinoni et al[5] provide a very succinct and informative review. Even further, they point out that pressure change (stress) and volume change relative to a baseline (strain) are linearly related by a constant of proportionality, K (in this case defined as specific elastance):

$$\Delta P_{tp} = K \times \Delta V/V_0 \quad (1)$$

where ΔP_{tp} is static transpulmonary pressure (obtained with an inspiratory hold) associated with V_T delivery (P_{tp} = pressure at airway opening minus pleural pressure[6]; ΔP_{tp} = elastance $\times$ V_T = one definition of driving pressure[7]) and ΔV is the V_T relative to baseline or resting lung volume, V_0. Actually the pressure change, ΔP_{tp}, is also measured relative to a baseline (ie, PEEP), corresponding to V_0 (although initially functional residual capacity was used as the reference lung volume in the equation[8]; hence, the reference pressure was ambient barometric pressure). The term K is specific elastance (the reciprocal of static compliance, often used to describe pediatric lung mechanics). It is defined by rearranging Equation 1 as follows.

$$K = \Delta P_{tp}/(\Delta V/V_0) = (\Delta P_{tp} \times V_0)/\Delta V \quad (2)$$

From Equation 2, we see that specific elastance has units of pressure (ie, volumes in the numerator and denominator cancel). Gattinoni et al are fond of saying "It follows that K . . . is the transpulmonary pressure recorded when V_T equals the resting volume, in other words, when the lung doubles its volume,"[5,8] yet I have never seen them explain exactly how it "follows." The proof is that in Equation 2, if ΔV is set equal to V_0, then the term $V_0/\Delta V = 1$, and hence $K = \Delta P_{tp}$. In other words, K is equal to the change in transpulmonary pressure required to make $\Delta V = V_0$ and hence the end-inspiratory volume = $2V_0$, or double the initial lung volume.

The point being, as Gattinoni et al stated,[5] when ΔP is equated to ΔV (Equation 1), "The distinction between volutrauma and barotrauma then vanishes." Hence, they should not be conceived of as 2 different metrics and certainly not defined as ventilator variables instead of patient outcomes, yet it is confusing to use the terms interchangeably when we want to refer to the strain-related cause of ventilator-induced lung injury.

In summary, I am suggesting that we use the term terms low-risk PIP instead of barotrauma-free and low-risk tidal volume instead of volutrauma-free in defining quality metrics of mechanical ventilation. Furthermore, I suggest that we use the term volutrauma (as a patient outcome quality descriptor) instead of barotrauma because volutrauma is more obviously associated with the measurement of V_T, in contrast to barotrauma, which is easily mistaken to be associated with measurement of PIP instead of ΔP_{tp} (which is also less convenient to measure than V_T).

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The Quality of Quality Metrics—Reply

In reply:

Mr Chatburn's letter to the Editor highlights some important points about our paper.[1] Let's first start with barotrauma. Barotrauma is used to describe the manifestations of extra-alveolar air during mechanical ventilation often associated with high airway pressures.[2] Mr Chatburn is correct, our term barotrauma-free or volutrauma-free categorization may be misleading because the system does not presently incorporate radiology reports, which would include extra-alveolar air. However, the system does assess conditions known to be associated with barotrauma and lung injury. It may be pertinent to use the term "barotrauma risk."

Volutrauma is as an ultrastructural lung injury due to overdistention occurring during mechanical ventilation.[2] Barotrauma and volutrauma reflect 2 sides of the same phenomenon: lung injury due to excessive distending volumes and/or excessive airway pressure. Alveolar pressure can be easily estimated by measuring plateau pressure. We chose a peak inspiratory pressure (PIP) threshold of 30 cm H_2O because is it equal to or higher than plateau pressures and is a generally accepted threshold beyond which most clinicians are concerned that lung injury exacerbation may occur. Mr Chatburn suggests the measurement of transpulmonary pressure (P_{tp}) gradient as a method of quantifying lung injury risk. Although we do not disagree that ΔP_{tp} may be an important measure for severely lung injured patients, it is not the standard of care for all, certainly not for pediatrics, and is somewhat cumbersome to perform and yields only approximate results[3] because it cannot possibly determine whether regional gas exchange units are experiencing hyperinflation or atelectasis. Pressure limitation has been a strategy for decades used to limit overdistention of the lung that would be potentially detected by ΔP_{tp}.[3] In our ICU, we utilized almost exclusively pressure-targeted modes of ventilation, and when volume-targeted modes of ventilation are chosen, we often do not set a pause pressure; therefore, plateau pressure is not continuously available for assessment by our computer-aided mechanical ventilation system. Second, we chose to use PIP because it is continuously measured and often the result of high Δ or driving pressure levels, which Mr Chatburn points out has evolved to be an important parameter to monitor and potentially even control. The association of high airway pressures with barotrauma and the availability of measure led us to choose a PIP. It was not our intent to insinuate that PIP was the only determinate of risk of ventilator-induced lung injury (VILI). This is clearly a limitation of our method because we chose to monitor for a PIP of >30 cm H_2O regardless of lung disease. This may prevent the computer-aided mechanical ventilation system from early identification and subsequent intervention, which is the primary objective. Our system is designed to be completely customizable based on the patient condition and in the future will provide different thresholds for lung injury risk. Importantly, even lower PIP ranges should be goal of therapy when a patient is spontaneously breathing because they are probably contributing to their ΔP needed to ventilate. In our study, we rarely identified subjects with PIPs of >30 cm H_2O because our practice is to pressure-limit. In pediatric ARDS, there is an association of mortality with high PIPs regardless of tidal volume (V_T).[4] We have plans to continue to evolve our methods to identify expected PIPs by degrees of hypoxemia, patient effort, and total respiratory system compliance to improve our VILI risk categorization. In addition, PEEP levels have been correlated to incidences of barotrauma,[5] indicating that PEEP also may play a role in total or regional lung volumes, but there is insufficient and conflicting evidence for us to monitor for this potential mechanism of VILI.

VILI can occur because of high and low lung volumes, which, as Mr Chatburn points out, makes the term barotrauma itself a little misleading. Dreyfus et al[6] coined the term volutrauma, demonstrating that lung stretching, not airway pressure, was an important factor in lung injury and that PEEP could be used to reduce the associated edema and end-expiratory lung collapse. We chose to use V_T because, like PIP, it is a variable that we can measure 12 times/min. Intermittent assessments of PIP and V_T by respiratory therapists can underestimate the duration and extent of delivered V_T, especially in modes of ventilation that do not control or target V_T. Duration of insult and its association with VILI are not well described in the literature. Therefore, we developed a system that can determine the dose and therefore risk of VILI through a nearly continuous assessment by reporting the percentage of time spent within the risk area. We targeted a V_T range of 4–8 mL/kg/ideal body weight but often fell short in practice of staying within range, primarily exceeding the high limit of 8 mL/kg. This is an important institutional observation and may enable targeted clinical interventions to adhere to clinical practice guidelines.

On the other hand, the literature is not clear in pediatrics that tidal volumes should be tightly regulated in *all* patients. There have been no randomized controlled trials assessing normal versus high V_T values in pediatric subjects with ARDS; however, there has been an independent association of normal V_T values and lower mortality.[7]