Mr Chatburn has disclosed relationships with IngMar Medical and DeVilbiss/Drive Medical.

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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]

Yet there have been some conflicting reports of the higher V_T values positively influencing ventilator-free days[8] or mortality.[4]

Using the term low-risk PIP or low-risk V_T would indicate an understanding of all PIPs or V_T ranges. Could we conclude a PIP of <30 cm H_2O to be low risk? Possibly, a more palatable solution would be the use of a low-risk (PIP <15), medium-risk (PIP 15–25), or high-risk (PIP >25) PIP or low-risk (V_T 5–7 mL/kg), medium-risk (V_T 3 to <5 or 8–10 mL/kg), or high-risk (V_T <3 or >10 mL/kg) V_T. Some may choose to swap ΔP_{tp}, ΔP, or a different V_T range for the parameters we have chosen based on an individual patient disease or condition to determine the risk of VILI. This should be encouraged based on current and ever evolving evidence or clinical expertise. This will create natural experiments that will lead to future discoveries without costly randomized controlled trials.

Our goal was to develop and test a method that identifies conditions and reports variance from clinical practice guidelines or established standards of care. Whether our computer-aided mechanical ventilation program reduces variance and therefore incidences of mortality or morbidity is yet to be determined, but this is an active pursuit of our team. How we choose to define the VILI parameters is not nearly as important as measuring and reporting quality indicators of mechanical ventilation that are not end results of poor quality mechanical ventilation. This will hopefully allow us to see trends in our practice before a negative patient outcome. We will no longer report barotrauma or volutrauma without coupling parameters associated with the actual VILI (eg, large V_T and pneumothorax). We will incorporate Mr Chatburn's suggestions of scaling risk associated with the chosen parameter of VILI.

Thank you for your valuable assessment of and contribution to our work.

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Promoting the Inclusion of Lung Volumes in the Reversibility Evaluation

To the Editor:

I read with great interest the recent paper of McCartney et al[1] highlighting the importance of lung volumes in the investigation of reversibility of air-flow obstruction. Because lung hyperinflation has become a major concern in the management of COPD, such papers should be encouraged. However, 2 points should be highlighted.

First, in the above retrospective study,[1] change in residual volume (RV) was expressed as a percentage from the initial value ([post-bronchodilator minus pre-bronchodilator]/pre-bronchodilator), and 5 thresholds (8, 10, 12, 15, and 20%) were tested. However, in the literature, other interesting ways to express reversibility with different thresholds have been evaluated. Among them, an absolute value decrease of −300 mL (post-bronchodilator minus pre-bronchodilator)[2,3] or a −10% decrease from predicted value ([post-bronchodilator minus pre-bronchodilator]/predicted value)[2,4] were considered as clinically important[2-4]. In a Tunisian study[2] including 366 heavy smokers divided into 2 groups (hyperinflated [n = 314] and free from lung hyperinflation [n = 52]), it was found that in the hyperinflated group, and compared with changes in FEV_1 and FVC (a 12% and 0.2-L increase), the above RV changes detected more respondents (54% for FEV_1 and FVC vs 65% for RV). This was not the case for the group free from lung hyperinflation (23% for FEV_1 and FVC vs 35% for RV). Moreover, in the hyperinflated group free from air flow obstruction (n = 58) and compared with changes in FEV_1 and FVC, the above changes in RV detected more respondents (24% for FEV_1 and FVC vs 71% for RV). According to the authors, it seems essential to include RV as a criterion of reversibility evaluation.[2]

Second, I agree with McCartney et al[1] that "there is no clear consensus on what constitutes reversibility in subjects with air flow obstruction."[5,6] This could be a source of confusion and/or misdiagnosis for clinicians and respiratory researchers. However, to better understand how subjects with COPD respond to bronchodilators, it will be more helpful to derive post-bronchodilator spirometric norms from healthy subjects.[5] However, to date, only 2 post-bronchodilator spirometric reference values in adults have been published.[7,8]

In conclusion, in daily practice, reversibility should be identified in all subjects with COPD using the changes not only in FEV_1 and FVC as primary outcomes, but also RV. Sufficient evidence is now available to justify promoting this message, particularly through the consensus statements of highly influential organizations like the Global Initiative for Chronic Obstructive Lung Disease[9] and the American Thoracic and European Respiratory Societies.[10] It is time for professional societies to standard-