

Yet there have been some conflicting reports of the higher V_T values positively influencing ventilator-free days⁸ 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 H₂O 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_{IP} , 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 al¹ 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 important²⁻⁴. In a Tunisian study² 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 FEV₁ and FVC (a 12% and 0.2-L increase), the above RV changes detected more respondents (54% for FEV₁ and FVC vs 65% for RV). This was not the case for the group free from lung hyperinflation (23% for FEV₁ and FVC vs 35% for RV). Moreover, in the hyperinflated group free from air flow obstruction ($n = 58$) and compared with changes in FEV₁ and FVC, the above changes in RV detected more respondents (24% for FEV₁ 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 al¹ 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 FEV₁ 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 Disease⁹ and the American Thoracic and European Respiratory Societies.¹⁰ It is time for professional societies to standard-

ize the spirometric criteria of airway reversibility in COPD.

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Promoting the Inclusion of Lung Volumes in the Reversibility Evaluation—Reply

In reply:

We greatly appreciate the thoughts Dr Ben Saad has shared with us about hyperinflation and our research. We think his research on lung volume reversibility¹ complements ours.² The question of whether residual volume change was more sensitive than FEV₁ or FVC change is a problematic one because all thresholds for responsiveness are arbitrary and can be manipulated by setting them higher or lower. What we found was that residual volume change correlated poorly with FEV₁ and FVC, which led us to conclude that we were describing a novel group of responders.

The research of Dr Ben Saad¹ shows that there were more residual volume responders in the hyperinflated group but not the control, which suggests that there are probably differences between hyperinflated people and those that are not hyperinflated. This makes us wonder whether we should use obstructed patients to help guide us to what expected change is and not use healthy subjects.³

Ultimately, we think the more we expand our knowledge about lung volumes the better off we will be understanding obstructive lung diseases. We think residual volume changes add important information about obstructive lung diseases.

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A Shout Instead of a Whisper: Let's Get the Graphics Right

To the Editor:

In July of 2013, Mark Siobal and colleagues wrote a paper on volumetric capnography in *RESPIRATORY CARE*.¹ In a subsequent letter to the Editor,² I pointed out an error in their Figure 8. In that figure, the quantity $V_{D_{alv}}$ is represented as the shaded areas between the volume curve and the CO₂ axis. Although the vertical axis is labeled simply CO₂, there are horizontal lines labeled P_{aCO_2} , P_{ACO_2} , etc, implying that the vertical axis represents pressure. The problem is that if the vertical axis is pressure, then the areas on the graph have the wrong dimensions; they are not volumes. Unfortunately, Mr Siobal failed to heed my advice and, in his most recent paper on the same subject,³ has made the same error 7 more times (Figs. 4, 5, 6, 14, 15, 18, and 19; most of which explicitly label the vertical axis as P_{CO_2} in mm Hg). Figure 6 is particularly egregious because its legend says "From reference 106, with permission." Checking that reference,⁴ we see it repeatedly shows the vertical axis labeled as *Exhaled FCO₂* (carbon dioxide fraction), not P_{CO_2} , for volumetric CO₂ curves.

Here is the significance of the error. The dimensions of any area on an x-y plot are the dimensions of the vertical axis times the dimensions of the horizontal axis. For example, if the y axis has the dimension of length (L) and the x axis also has the dimension of length, then any area on the graph has the dimensions