vasoconstriction may have mediated the pulmonary vessel changes observed here (1, 7).

Long-term anti–IL-5Rα therapy may affect pulmonary vascular abnormalities in patients with eosinophilic asthma. ■

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The Proper Assessment of Pendelluft and Absolute End-Expiratory Lung Volume

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To the Editor:

We read with great interest the article by Menga and colleagues that was published in a recent issue of the *Journal* (1). They compared the respective effects of helmet pressure support (noninvasive ventilation [NIV]) and continuous positive-airway pressure (CPAP) to high-flow nasal oxygen in hypoxemic respiratory failure. This study demonstrated that helmet NIV reduced inspiratory effort and mitigated the pendelluft phenomenon. However, several factors might influence the reported findings.

First, we identified the discrepancy between the method description and the results for the pendelluft calculation. We believe that, according to their illustration in Figure 1 and the equation in the online supplement, the method proposed by the authors is very similar to one used in a previous study (2, 3). If that's the case, the pendelluft amplitude should always be positive, since the pixel-per-pixel-TidalΔZ is always larger than breath-cycle-TidalΔZ by definition. However, in Figure E7 (the correlation between pixel-per-pixel-TidalΔZ and breath-cycle-TidalΔZ), the pixel-per-pixel-TidalΔZ was smaller. This discrepancy is confusing, and we are not sure how to interpret the results. In the main text, the authors claimed to use the LuMon system, but in the supplement, they used an electrical impedance tomography (EIT) device from MBMED. Would this be the cause, as different manufacturers might have different methods of parameter calculation?

Second, the local negative pleural pressure generated by diaphragmatic contraction is not evenly distributed but rather concentrated in the dependent lung regions because of a vertical pressure gradient from nondependent to dependent areas, resulting in an increased dorsal ventilation (2). Because of the heterogeneity, it is not justified to utilize TIV(Tidal Impedance

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Variation)/ΔP_L(transpulmonary pressure) for calculating pixel compliance in patients with spontaneous breathing, regardless of the type of tidal impedance (global or the sum of all pixel TIV calculations) used.

Finally, for a given stress, the lung strain may be quite different, depending on the size of the lung. At the same time, the strain on the lungs is changing because of changes in end-expiratory lung capacity during the treatment with helmet CPAP or helmet NIV, and the opening of the dorsal alveoli. The lungs exhibit viscoelastic behavior, whereby their stress response is dependent on both the amplitude and rate of strain (4). Therefore, it is debatable to generalize the end-expiratory lung impedance (EELI) derived from $k = 13.7$. In addition, it should be noted that the ratio between the two derivatives cannot be assumed to be identical to that of their absolute values, as shown in Equation 2b: $\frac{EELI_{pixel,abs}}{EELI_{lung,abs}} = \frac{EELI_{pixel,derived}}{EELI_{lung,derived}}$.

The calculation of EELI depends on the selected reference point for image reconstruction, and the absolute value is not unique. The authors correctly pointed out this issue in chapter 5 of the supplement, so we are unable to follow why they would still calculate the $EELI_{lung}$ using Equation 1b.

This is a fascinating study, as both EIT and esophageal pressure are potent tools. The combination of these two instruments yields additional indicators that warrant further exploration. The critical issues in the EIT data analysis posed questions on the present findings, which require further clarification. ■

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Reply to Wang *et al.*

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From the Authors:

We sincerely thank Wang and coworkers for their thoughtful comments on our study (1). These are greatly valued, as they help in refining our understanding of this complex topic.

We acknowledge the discrepancy identified concerning the pendelluft calculation. The pixel-per-pixel Tidal impedance variation (TidalΔZ) is indeed, by definition, always larger than the breath-cycle TidalΔZ, as evidenced when comparing the values for TidalΔZ in Table 2 and Table E1 in our study (1). The confusion arose as a result of a labeling error in Figure E7 in the online supplement (1), in which the *x*- and *y*-axes were inadvertently swapped. We are grateful to Wang and colleagues for spotting this oversight, allowing us to rectify the error promptly. A corrected version of the figure has been inserted in the online supplement (1); the *Journal* is also publishing an erratum to notify its readership of the change.

In their letter, Wang and colleagues provide several interesting physiological and methodological observations, sparking an engaging discussion on the complex physiology of transpulmonary pressure distribution and the limitations of electrical impedance tomography. It is indeed true that the propagation of transpulmonary pressure in the lungs is not homogeneous, with regional inhomogeneities potentially amplifying transpulmonary pressure in nearby areas. This theory, proposed more than 50 years ago, has been substantiated by Cressoni and coworkers using computed tomography scans, showcasing certain regions acting as "stress raisers" for the surrounding areas (2, 3). This might be particularly prominent during spontaneous breathing, in which the intense inspiratory effort coupled with local inhomogeneities may result in increased force delivery to the dorsobasal regions of the lungs (4).

In this sense, because positive end-expiratory pressure (PEEP) improves the aeration of dorsal areas and reduces the magnitude of pendelluft (5), it may also decrease the inflation inhomogeneities. This may explain why, in our study, the magnitude of pendelluft was lower with helmet continuous positive airway pressure (CPAP)

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