Commentaries on Viewpoint: Could lobar flow sequencing account for convection-dependent ventilation heterogeneity in normal humans?

COMMENTS ON VIEWPOINT: COULD LOBAR FLOW SEQUENCING ACCOUNT FOR CONVECTION-DEPENDENT VENTILATION HETEROGENEITY IN NORMAL HUMANS?

To the Editor: Based on 4DCT imaging (3), this Viewpoint (5) investigates how lobar flow sequencing can contribute to generate a positive phase III slope. Results suggest that interlobar differences in expansion and deflation during the respiratory cycle contribute to Scond. We agree that further imaging measurements, such as measurement of 1D flow profiles in the main bronchi with hyperpolarized gas MRI (1) can provide added regional insight into lobar flow asynchrony. From our experience, measurement of such flow profiles at the entrance of each lobe is feasible but technically challenging in airways below the main bronchi because of the background signal from gas in the lungs outside of the airway of interest.

We also believe that the regional detail of the CT images could be further put to use by looking at the sublobar nonlinearity and hysteresis of lung deformation. After all, much of the heterogeneity in specific ventilation occurs at smaller length scales than the lobes. For instance it would be interesting to see if normal Curv and Scond values could be simulated by feeding an idealized airways tree (such as Ref. 4) with localized ventilation imaging data, e.g., from dynamic CT or HP gas MRI (2).

Ultimately lung ventilation cannot be fully modeled from elementary consideration of diffusion-convection equations of mass transfer in a simple branching model of the lungs alone and anatomically specific information from functional imaging like this can help inform these models and add to our understanding of global lung function tests in this way.

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AN ATTEMPT TO EXPLORE VENTILATION HETEROGENEITY IN THE LUNGS

To the Editor: Ventilation heterogeneity, which refers to uneven gas distribution during respiration, is present in normal lungs (2). Because of its clinical implication in respiratory diseases, multiple mechanisms including gravity distribution and regional pleural pressure difference have been proposed to justify this phenomenon (1–3); however, the origin of ventilation heterogeneity remains controversial. The slope of N2 phase III in breath wash-out test has been considered as a critical indicator of nonuniform ventilation (3, 4). Verbanck et al. (3) explored the potential mechanism underlying the nonzero phase III slope in normal human, suggesting lobar expansion could function as a potential contributor.

Intriguing data were provided in this article showing that lobar flow sequencing demonstrates a compatible pattern with the nonzero N2 phase III slope. Therefore, the flow dynamics within the five lung lobes may partially account for this positive phase III slope (3). However, both expiratory sequencing and ventilation heterogeneity can generate a nonzero phase III slope (3). Whether and how this lobular flow sequencing is associated with ventilation heterogeneity remains unclear.

Ventilation heterogeneity is also an important contributor for airway hyperresponsiveness manifested in patients with asthma (2). At cellular levels, reactive oxygen species (ROS) plays a critical role in hyperresponsiveness induction (5). In our view, an attempt to explore the potential correlation between heterogeneous ventilation and ROS overproduction may provide insights into the progression of airway inflammation in respiratory diseases.

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