On modeling edematous alveolar mechanics

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TO THE EDITOR: Chen et al. (1) recently published in the Journal of Applied Physiology a modeling study of lung inflation effects on stretch and stress of an alveolar septum separating aerated and flooded alveoli (1). Chen et al. base their model on two images from an experimental study by Jahar Bhattacharya, David Lederer, and me (3). Chen et al.’s fundamental analysis is sound, a natural extension of our experimental findings, and I appreciate their interest in our study. However, I have concerns about their approach.

Chen et al. consider cases in which flooded alveolar surface tension (1) exceeds or (2) equals aerated alveolar surface tension, or (3) is zero. Only case 2 is physiological.

Theory and data suggest case 1 to be unlikely. The liquid phases and interfaces of adjacent alveoli are connected. Diffusion and Marangoni flow should ensure equal surface tension between adjacent alveoli, likely even under dynamic conditions given the low respiratory rate. These factors may underlie Schurch et al.’s finding of equal surface tension between nearby, albeit aerated, alveoli of differing size (4). Furthermore, my current group recently demonstrated equal surface tension in adjacent aerated and flooded alveoli (2).

Regarding case 3, only in an excised lung that has been degassed and reinfated with liquid can surface tension be zero. When flooding extends beyond the alveoli into an airway, the interface in the airway will form a meniscus such that distal liquid phase pressure in flooded alveoli will be less than air pressure. Furthermore, Chen et al. mistakenly cite one of our original images as evidence that an alveolus can be completely flooded and immune to surface tension effects (3). Our image depicted an optical section obtained above the height of the meniscus that was present in the alveolus.

Results from Chen et al.’s physiologic case 2 are presented in their Figs. 8 and 9. Unfortunately, three aspects of Chen et al.’s methodology raise questions about these results.

First, Chen et al. use the parameter λ to indicate tissue stretch above functional residual capacity in Eq. 1 but above unstressed length in Eq. 11. This inconsistent use of λ affects results throughout their report.

Second, in solving Eqs. 2 and 14, Chen et al. must know the transpulmonary pressure, $P_{ALV}$, meniscus radius, $R_b$, relation. They presumably generate a linear relation from the two data points in our study (3). Without knowing the contour of the $P_{ALV}$-$R_b$ relation, it is unclear whether the difference in stretch and stress contours of Chen et al.’s Fig. 8 is meaningful.

Third, Chen et al. implicitly assume that, with lung inflation, the lengths of all aerated alveolar walls excepting the bowed septum, and of the straight line $SS'$, increase proportionately. As this assumption has not been validated, Chen et al.’s predictions of inflation effects on aerated alveolar geometry (Fig. 9) may not be valid.

Given these concerns, Chen et al. might consider estimating the propagation of error through their model. Reporting confidence limits for model outputs would enable assessment of whether reported differences are meaningful.

DISCLOSURES
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REFERENCES

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