ATS 2017 Highlights

Respiratory Structure and Function Early Career Professionals

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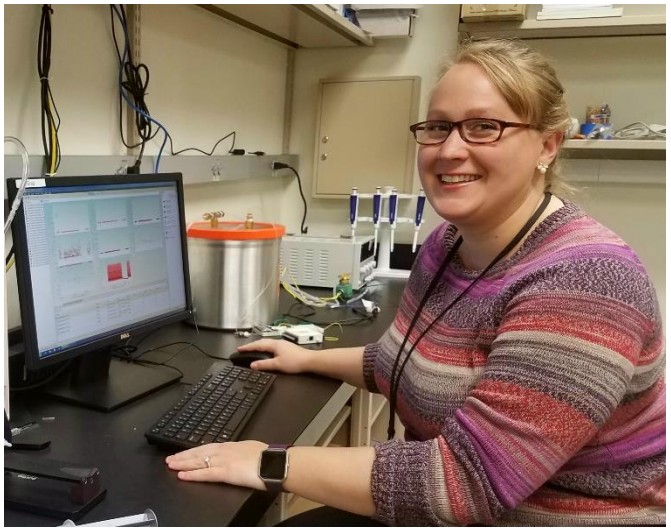

Michelle Mellenthin, PhD

Postdoctoral Fellow
Univ. of Colorado, Denver USA

More about Michelle

Is your research clinical, basic science or translational?

Basic and translational science.

Tell us about your research?

My research aims to understand the physical mechanisms of ventilator induced lung injury pathogenesis. To predict the microscale effects of ventilation, I fit numerical models to measured lung function in mechanically ventilated rodents. In future studies, these simulations will be coupled with electrical impedance tomography to develop optimized ventilation strategies.

Where do you see yourself in 5 years?

One of my short-term career goals is to do exciting research, and while I enjoy teaching, I am open to working in either industry or academia.

What do you find is the major benefit of RSF Assembly Membership?

Becoming a part of their extensive network of professionals. I am encouraged by the interactions that I have had thus far and the interest of well-established ATS members in the development of postdoctoral fellows and other early career professionals.

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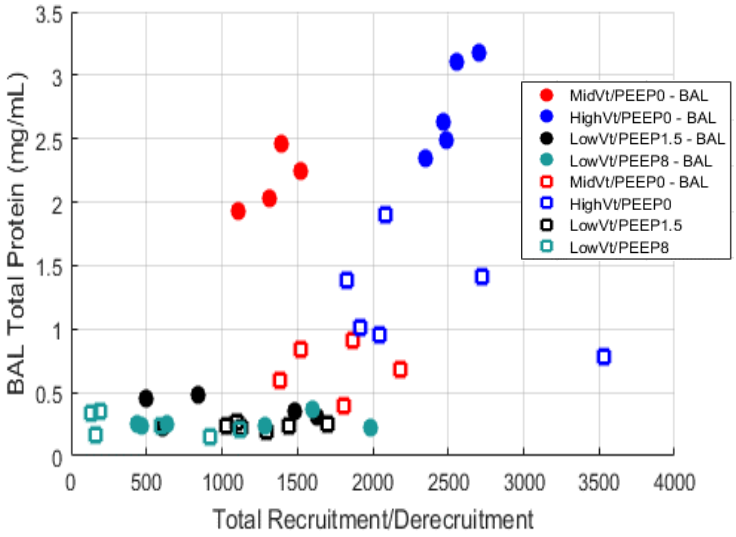

Contributions of Atelectrauma in Ventilator Induced Lung Injury to the Blood-Gas Barrier

Introduction: Damage to mechanically ventilated lungs is caused by overstretch (overdistension) and the repetitive opening/collapse (recruitment/derecruitment or R/D) of alveoli. Pulmonary blood-gas barrier injury leads to increased permeability, which can be quantified by measuring the concentration of protein in the airspace. Blood-gas barrier damage is related to the underlying mechanisms of injury, so a numerical model that predicts distension and R/D was developed to study this relationship.

Methods: A single compartment viscoelastic model was fit to lung mechanics measurements on 45 mechanically ventilated BALB/c mice. Mice in four experimental groups were ventilated at different tidal volumes and positive end expiratory pressures (PEEP) for about 1.75 hours. Half of the mice also received an injurious bronchoalveolar lavage (BAL). Volume and pressure were measured during dynamic pressure-volume (PV) loops and recorded at 5-min intervals. The model used measured data to predicted R/D for each PV loop. These measures were compared to BAL total protein concentration measured in the airspace at the end of the experiment.

Results: The figure compares the concentration of BAL total protein to total R/D, which is defined here as the product of the respiratory rate with the sum of the increase in R/D during inspiration for each measured PV loop. The HighVt/PEEP0 group sustained the most injury to the pulmonary blood-gas barrier, as indicated by the higher concentrations of BAL protein.

Conclusions: This data suggests that injury to the blood-gas barrier in the lung, as reflected by increases in total BAL protein, relates to increased total R/D.

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