Modelling the relationship between gas exchange and lung mechanics

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Introduction
Selecting appropriate ventilator pressures such as positive end-expiratory pressure (PEEP) is difficult as changes in inspiratory pressures affect both mechanical and gas exchange properties of the lung. Currently no measures or physiological models linking gas exchange and lung mechanics are known to the authors. This study presents a compartmental model of lung mechanics that combines the individual contributions of chest wall and lung tissue elasticity, surfactant function and hydrostatic effects. The aim is to create a model with physiologically relevant parameters that simulates the effects of changes in ventilator settings on both lung mechanics and gas exchange.

Methods
The lungs are divided into 100 compartments (Fig. 1) containing an equal number of alveoli, with different pleural pressures ($P_{pl}$) due to hydrostatic effects ($\rho gh$) down the lungs. Alveolar pressure ($P_a$), as a function of lung volume ($V_L$) and individual alveolar volumes ($V_A$), is modelled as the contributions of hydrostatic effects, surfactant ($P_\gamma$), lung tissue elasticity ($P_e$) and chest wall elasticity ($P_{cw}$). Alveoli are collapsed, open or over-distended depending on the pressure across the alveolar wall ($P_{alveol}$).

Results
Simulated static pressure-volume (PV) curves show good correlation with experimental results for excised rabbit lungs [1], and for pigs with healthy and damaged lungs. Figure 2 (top) illustrates a simulation of a PV curve overlaid on a measured PV curve from a pig with oleic acid lung damage [2]. Figure 2 (bottom) plots the corresponding variation in the total number of collapsed, open and over-distended alveoli. These trends correlate well with changes in measured gas exchange parameters between different PEEP levels for the same animals. The surfactant model enables simulation of hysteresis in the static PV curve. Model parameters with direct physiological interpretation and realistic values can be used to simulate PV relationships for a variety of animals and lung disorders.

Conclusions
The model simulates the effect of alveolar pressure changes on the PV relationship in the lungs, and the gas exchange characteristics defined by the number of collapsed, open and over-distended alveoli. However, the model does not yet simulate sufficient hysteresis in the static PV curve. When correct, such a model could assist clinicians in setting PEEP for control-ventilated patients.

References