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Coupled 3D impedance model of the Human Lungs

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ABSTRACT

Mechanical ventilation is an indispensable tool in the treatment of critical care patients suffering from Acute Respiratory Distress syndrome (ARDs) or Acute Lung Injury (ALI). However the mortality rates of these diseases remains high, 50 and 60% respectively. In addition, it is widely known that ventilation is the cause of a number of further associated injuries. This is despite the adoption of modern (protective) ventilation strategies, where lower tidal volumes are administered while retaining a positive pressure at the end of expiration, in order to avoid collapse of the lung. The primary reason for this strategy is widely accepted to be protective against valotrauma. However, controversy also still surrounds PEEP ventilation techniques. Understanding the reason why the lungs still become damaged or inflamed is a key question sought by the medical community. Computational modelling represents an approach that can provide more in depth knowledge into how the human lung functions mechanically, both during normal breathing and with the assistance of mechanical ventilation. Of particular interest is the consideration of what conditions within the lung mechanically change during ventilation. Currently a number of limitations exist for modelling the lower airways. Firstly the resolution of CT imaging is currently restricted, and hence smaller vessels are not visible during segmentation of the 3D models. Secondly, it is in any case not computationally feasible to simulate the full 23 generations. Because of these aforementioned points alternative methods for modelling the lung must be sought. Here we employ a geometric multiscale approach to modelling the entire human lung.

The numerical method for modelling the lung fluid and structure mechanics utilises a validated in house ALE fluid structure interaction (FSI) finite element code. This has been previously used to simulate air flow in the lower airways (Wall and Rabczuk [3]). The 3D lung lower airway geometry used for simulations is a 6 generation patient specific model derived from medical CT images. The simulations consider conditions of normal and ventilator assisted breathing, which is of utmost importance when elucidating the different dynamics that occur in the lung under natural and mechanical ventilation. A further novel aspect of this simulation is the inclusion of the cartilage rings along the length of the trachea, which are well known to be of high stiffness and provide structural support for this region of the airway. A common issue with numerical modelling of a portion of a larger system is the outflow boundary conditions on the artificially created surfaces, where the vessels terminate (in this case due to the limits of CT resolution). For this reason, it is important to consider a reduced dimensional model

that truncates the airway region from the alveoli level back to the terminus of the 3D geometry, hence providing a complete simulation of the human lungs. This was achieved using a 1D transmission line impedance model for flow in bifurcating pipes, as outlined by Olufsen *et al* [1]. The geometry of the 1D bronchiole tree is derived from published measurements in literature. The coupling of the 3D model to the 1D impedance based model is achieved using a Dirichlet to Neumann approach previously outlined for blood vessel simulations by Vignon-Clemental *et al* [2]. This allows for the distribution of airflow through the upper part of the lower lungs to be governed by the peripheral resistance rather than the more commonly simulated traction free outflow conditions, which represents an unphysiological state. In addition simulation of diseased state lungs is very interesting within this framework, as if a region of the lung is diseased or collapsed the distribution of air may lead to over distension in the healthy regions, due to over delivery of air to this region. Overall the model sets the framework for information to be obtained regarding the effects of ventilation on lung wall, in particular lung wall stresses, which clinical trials have suggested are a critical factor in determining the survival of mechanically ventilated patients.

The intended future direction of this research is to couple the 1D-3D FSI lower airway simulations with an alveolus model, as described in Wiechert *et al* [4]. This will allow for a complete model that includes the important lung parenchyma, which has the potential to provide interesting and insightful information about how the lung functions mechanically overall. In addition applying the previous mentioned methods to the transport of small particulates, for example aerosol from inhalers, would provide an understanding of how these particles distribute in the lung.

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