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Stress concentration around an atelectatic region: A finite element model

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ABSTRACT

Lung parenchyma surrounding an atelectatic region is thought to be subjected to increased stress compared with the rest of the lung. Using 37 hexagonal cells made of linear springs, Mead et al. (1970) measured a stress concentration greater than 30% in the springs surrounding a stiffer central cell. We re-examine the problem using a 2D finite element model of 500 cells made of thin filaments with a non-linear stress–strain relationship. We study the consequences of increasing the central stiff region from one to nine contiguous cells in regular hexagonal honeycombs and random Voronoi honeycombs. The honeycomb structures were uniformly expanded with strains of 15%, 30%, 45% and 55% above their resting, non-deformed geometry. The curve of biaxial stress vs. fractional area change has a similar shape to that of the pressure–volume curve of the lung, showing an initial regime with relatively flat slope and a final regime with decreasing slope, tending toward an asymptote. Regular honeycombs had little variability in the maximum stress in radially oriented filaments adjacent to the central stiff region. In contrast, some filaments in random Voronoi honeycombs were subjected to stress concentration approximately 16 times the average stress concentration in the radially oriented filaments adjacent to the stiff region. These results may have implications in selecting the appropriate strategy for mechanical ventilation in ARDS and defining a "safe" level of alveolar pressure for ventilating atelectatic lungs.

1. Introduction

Atelectasis, or alveolar collapse, causes a reduction of lung functional residual capacity and is a common clinical condition that occurs during general anesthesia for surgery (Brismar et al., 1985) and in disease states such as the acute respiratory distress syndrome (ARDS) (Gattinoni et al., 1986a,b; Pelosi et al., 1994). ARDS is a syndrome characterized by localized regions of air-less lung tissue adjacent to expanding lung units (Gattinoni et al., 1986a,b; Pelosi et al., 1994). Ironically, the mainstay of treatment for ARDS, namely positive pressure ventilation via a mechanical ventilator, has been found to cause or exacerbate lung injury. Although the exact mechanism for the injury still remains speculative, it has been hypothesized that high inflation pressures causing alveolar overdistension (Webb and Tierney, 1974) and low end-expiratory pressures allowing airway closure, alveolar collapse and cyclic reopening (Muscedere et al., 1994) may be the cause of what is termed ventilator-associated lung injury (VALI). Airway closure and alveolar collapse are also thought to potentiate VALI by reducing the number of alveoli available to distribute the tidal volume, thus increasing the likelihood of alveolar overdistension. Additionally, it has been postulated that atelectasis may cause stress concentrations in the tissues near the interface between the region of collapsed or non-inflating alveoli and the surrounding ventiled alveoli (Denny and Schroter, 2006; Mead et al., 1970).

2. Previous work

The stress concentration around a non-inflating region was first estimated by Mead et al. (1970) using a mechanical model consisting of a two-dimensional hexagonal array of coil springs, which was subjected to uniform tension at its outer boundary. They found the stress in the spring elements increased by about 50% with complete collapse of a central cell, giving rise to a condition of stress concentration in those members. Afterwards they compared predictions of stresses during lung expansion based on

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the mathematical model to changes seen with goat lung lobule inflation with a glued pleural disk and concluded that the mathematical model underestimated the stress (Takishima and Mead, 1972). Other investigators (Menkes et al., 1972a,b) have found similar underestimation of stress concentration in the mathematical model and more sophisticated models of lung micro-mechanics have been published (Fung, 1988; Kimmel et al., 1987; Wilson and Bachofen, 1982). However, these models or continuum mechanics analyses give only estimates of average behavior and none have quantitatively examined the concentration of stress around a region of atelectasis in heterogeneous structures.

The aim of this research is to re-examine Mead's estimate of stress concentration around a non-inflating region of the lung using finite element analysis, with an asymmetric structure more like that of the lung. We model inflation of the lung around an existing atelectatic region, which does not inflate, while the rest of the lung around it does. The stresses within the atelectasis are not supported by the alveolar walls, but by a reduction in pressure of the intra-alveolar fluid, which, depending on surface tension and viscous forces, may not change in volume as the lung is dynamically inflated in mechanical ventilation. When the collapsed region expands little, it may be approximated as being rigid relative to the surrounding tissue.

In this paper we provide quantitative information on the stress concentration of stress bearing tissue elements in the parenchyma around such a region. We compare concentrations seen in a homogeneous hexagonal structure, similar to that of Mead's model, with those taking place in random structures, closer to the structure of the lung. We also analyze the impact of using linear and non-linear tissue elasticity in the calculations. The model is not intended to explain the process of atelectasis formation, parenchymal instability or its associated phenomena. However, our analysis is unique in that it demonstrates that individual stress bearing elements within a random structure can be exposed to much higher stresses than the average stress around a non-expanding region and may also be much greater than those estimated for a uniform structure.

3. Cellular materials modeling

Many materials, both natural and synthetic, have a cellular structure made up of an interconnected network of filaments, struts or plates. Natural cellular materials include cork, wood and trabecular bone, as well as lung alveoli, of interest here. Engineering cellular materials include structural honeycombs and foams. The mechanical behavior of both two-dimensional, honeycomb-like materials with prismatic cells and three-dimensional, foam-like materials with polyhedral cells has been successfully described by modeling their mechanisms of deformation and failure (Gibson and Ashby, 1997). Random Voronoi cellular structures are typically used to model materials made of heterogeneously shaped cells. Although 2D models do not give identical results as 3D models Ma, Breen and Bates (Ma et al., 2013) utilized 2D non-linear springs to provide insights about network behavior a hexagonal-like array of the lung parenchyma, Ito et al. (2006) used a similar 2D network of hexagonal arrays and analyzed how the distribution of forces changes and the maximum force increases by adding non-linearity. Suki and Bates (2008) used a 2D Voronoi-like array of non-linear springs to model the lung parenchyma. They applied both springs running along the elements between the nodes and rotational springs at the nodes between adjacent cells. For the mechanical behavior of the lung tissue they considered a stress–strain curve with exponential stiffening. Here, we model two-dimensional cellular structures made of hexagonal and Voronoi cells, using an approach similar to Ma et al. (2013), Ito et al. (2006) and Suki and Bates (2008) respectively, where the filaments also represent the stress bearing elements in the parenchyma.

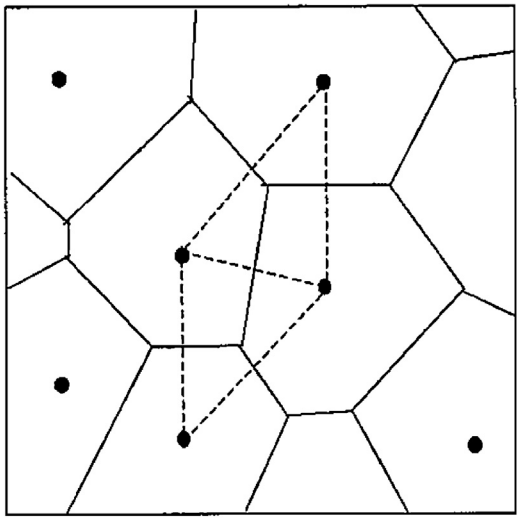

Fig. 1. Construction of a two-dimensional Voronoi structure. The solid lines are the perpendicular bisectors. The dashed lines are the lines connecting the seed points.

3.1. Generation of Voronoi and hexagonal models

Voronoi structures were initially developed to represent crystallization from a set of random nucleation points from which all the crystals begin to grow at the same time and continue to grow at the same rate. Two-dimensional Voronoi structures are created by drawing the perpendicular bisectors of lines connecting seed points generated with a uniform random distribution, as illustrated in Fig. 1. The Voronoi cell corresponding to an individual seed point is formed by the polygon of perpendicular bisectors that are closer to that seed point than to any other. In this study, we used hexagonal arrays and Voronoi honeycombs with approximately 500 cells within a square with sides of approximately 22 cells. Voronoi honeycombs were generated using software developed by Silva and co-workers (Silva and Gibson, 1997; Silva et al., 1995). To produce cells with approximately uniform size, nucleation points closer than a critical distance, equal to 0.7 of the average edge length, were eliminated.

For both the hexagonal and Voronoi honeycombs the value of the filament thickness was set to give a constant thickness, normalized by the unit length, equal to 0.01. It is worth noting that the filament thickness is only used for the purpose of calculating the stress in the filament. For a 2D honeycomb, each filament is a two-dimensional object, with a length and a thickness. Given the normalization of the results by the stress in the filament for a honeycomb without atelectasis, the actual thickness of the filament becomes unimportant. Indeed, it is the relative differences in stress and not the actual value of stress within each filament that is relevant. To approximate the geometry of the stress bearing filaments in the lungs, we made the filaments slim by setting filament thickness to unit length ratio constant and equal to 0.01. In our case, for two-dimensional filaments, one can think of the stress as the axial tensile force in each member divided by the product of the filament thickness times a unit depth out of the plane of the honeycomb. Non-expanding regions were defined by assigning exaggerated high stiffness to the filaments within a small central region of the honeycomb. The high stiffness of the central region prevents it from deforming, simulating the lack of inflation of an atelectatic region that does not re-expand with lung inflation. These regions consisted of either a single central cell or that cell plus all