Resistance to alveolar shape change limits range of force propagation in lung parenchyma

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ABSTRACT

We have recently shown that if the lung parenchyma is modeled in 2 dimensions as a network of springs arranged in a pattern of repeating hexagonal cells, the distortional forces around a contracting airway propagate much further from the airway wall than classic continuum theory predicts. In the present study we tested the hypothesis that this occurs because of the negligible shear modulus of a hexagonal spring network. We simulated the narrowing of an airway embedded in a hexagonal network of elastic alveolar walls when the hexagonal cells of the network offered some resistance to a change in shape. We found that as the forces resisting shape change approach about 10% of the forces resisting length change of an individual spring the range of distortional force propagation in the spring network fell of rapidly as in an elastic continuum. We repeated these investigations in a 3-dimensional spring network composed of space-filling polyhedral cells and found similar results. This suggests that force propagation away from a point of local parenchymal distortion also falls off rapidly in real lung tissue.

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1. Introduction

The intra-pulmonary airways are intimately embedded within the lung parenchyma such that the outer aspect of the airway wall is connected to multiple alveolar walls. This produces an important mechanical interdependence between the airways and the parenchyma that affects both; when the lung inflates the parenchyma pulls outward on airway walls tending to increase lumen radii, and conversely when the airway smooth muscle contracts to narrow the lumen it causes local distortion in the surrounding parenchyma. In particular, the capacity of the parenchyma to mechanically oppose airway smooth muscle shortening, and hence luminal narrowing, has major implications for airway responsiveness and has been a subject of much study.

The conventional wisdom about the mechanics of airway-parenchymal interdependence dates back to the classic work of Lai-Fook (Lai-Fook, 1979; Lai-Fook and Hyatt, 1979; Lai-Fook et al., 1978) who assumed the parenchyma to behave as an elastic continuum. The predictions of the continuum model were also shown theoretically by Wilson (Wilson, 1972) to be similar to those of a network of elastic elements arranged in a hexagonal pattern representing a two-dimensional slice of alveolar parenchyma. Nevertheless, these early studies are based on mathematical idealizations and also cannot easily incorporate the effects of regional parenchymal heterogeneities, something that clearly pertains in reality. Accordingly, we recently developed a numerical model of a contracting airway embedded in elastic parenchyma. We represented the alveolar walls in the parenchyma as pin-joined springs arranged in a repeating hexagonal pattern, as this represents the conventional idealization of the appearance of a thin slice of lung tissue (Mead et al., 1970; Wilson, 1972). We found, somewhat unexpectedly, that such a network produces long-range transmission of forces in response to the distortion produced by the narrowing of the embedded airway (Ma and Bates, 2012; Ma et al., 2013a). By contrast, when the springs are arranged in a repeating triangular pattern, or the parenchyma is represented as an elastic continuum, the elastic forces fall off rapidly with distance from the airway wall (Ma and Bates, 2012), in apparent accord with existing theory about airway-parenchymal interdependence (Lai-Fook, 1979; Wilson, 1972).

We hypothesize that the reason for the differences in behavior of the hexagonal spring network versus the triangular network relates to their different shear moduli. Unlike the triangular network the hexagonal network has limited capacity to resist shear, although this depends on the pre-stress in the network elements (Stamenovic, 1990; Wilson, 1972). Exactly how real lung parenchyma behaves mechanically thus remains an open question,

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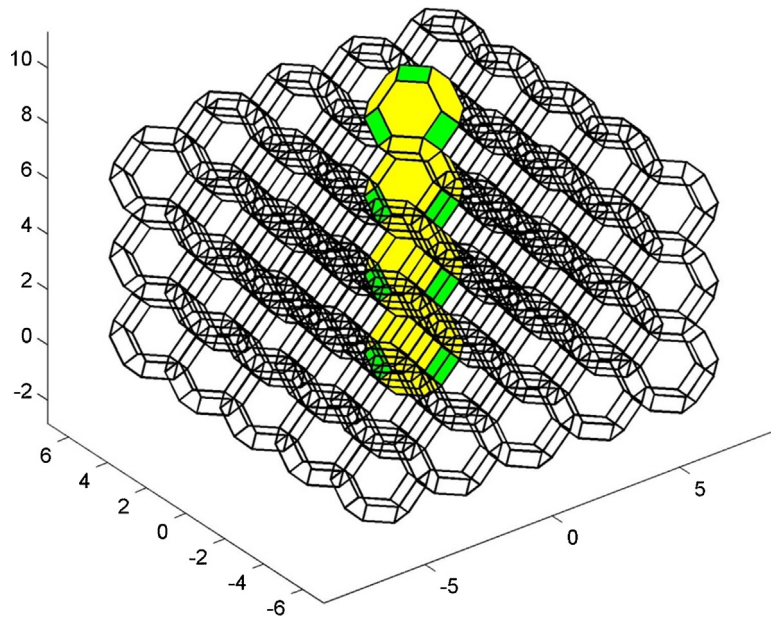*E-mail address:* jason.h.bates@med.uvm.edu (J.H.T. Bates).

Fig. 1. Illustration of a $5 \times 5 \times 3$ collection of 14-hedral units each with elastic elements comprising the edges. The central shaded line of polyhedral are designated to represent a small airway to which the adjacent polyhedral, which represent alveoli, are attached.

and one that has some important implications; our studies using 2D models have shown that whether the parenchyma behaves more like a network of hexagonal or triangular cells has a major effect on how nearby cells are able to influence the ability of each other to contract (Ma and Bates, 2014). The purpose of the present study was therefore to determine how important resistance to shear is in settling this question. We address this in both 2 and 3 dimensions using computational models that exhibit a range of abilities to resist shear.

2. Methods

We have postulated previously (Ma and Bates, 2012) that the key to long-range force transmission in a network of elastic elements lies in its shear modulus, the modulus of a hexagonal network of springs being much lower than that of a triangular network. We investigated this concept in the present study by first adapting our previous 2D model of an airway embedded in a hexagonal network of elastic walls (Ma and Bates, 2012, 2014; Ma et al., 2013b) so that it now includes an empirical mechanism for resisting a change in the shape of each hexagonal cell in the network. This mechanism is similar to that which we employed previously for the airway lumen (Ma and Bates, 2014) and is governed by a shape-restoring force applied to each spring junction (node) in addition to the elastic recoil forces exerted by the attaching springs. Each hexagonal cell in the network is bordered by 6 nodes, and the shape-restoring force that is applied to each node and points toward the hexagonal centroid is

$$F_i = S(R_i - R_{i0}) \qquad (1)$$

where $i = 1, \ldots, 6$. S is a constant that determines the force magnitude and R_i is the normalized radial distance of the node from the centroid of the hexagonal cell following airway narrowing given by

$$R_i = \frac{r_i - r_m}{r_m} \qquad (2)$$

where r_i is the radial distance of the ith node from the centroid and r_m is the mean distance for all 6 nodes. R_{i0} is the corresponding normalized radial distance of the node prior to airway narrowing. All nodes, except those at the network border and those around the

airway, thus experienced 3 different F_i corresponding to the 3 cells they bordered.

As in our previous studies (Ma and Bates, 2012, 2014; Ma et al., 2013a, 2013b), the middle section of the network was removed to represent the airway lumen. The springs around the perimeter of the resulting hole represent the airway wall, and each has a stiffness that is ten times that of each of the springs in the parenchymal network, which themselves were assigned a stiffness of unity. The equilibrium configuration of the model was determined as that for which the resultant forces acting on each node were zero.

We then developed a 3D version of the model in which each alveolus is represented as a truncated octahedron (14-hedron). Each 14-hedron has 8 hexagonal faces, 6 square faces, 36 edges of equal length, and 24 nodes. These 14-hedra are space-filling, so they share nodes, edges and surfaces. The edges of the 14-hedron are represented by springs connected at the nodes by pin-joints. In order to imbue the 14-hedra with an adjustable shear modulus, additional cross springs were added to the hexagonal and square surfaces so that they connected between alternate nodes as illustrated in Fig. 2. The stiffness of the cross springs is a fraction, α, of the stiffness of the springs along the edges of 14-hedron. Thus, α provides control over the shear modulus of the 3D model in an analogous fashion to the parameter S in Eq. (1) for the 2D model. A small airway in the 3D model is represented by a column of connected 14-hedra as shown in Fig. 1, similar to other 3D models used by other investigators for different purposes (Denny and Schroter, 1997, 2006). The stiffness of each spring bordering the airway is 10 times the stiffness of each spring along the remaining 14-hedron edges in the network.

The procedure we used for simulating airway contraction in both the 2D and 3D models was similar to that used in our previous studies (Ma and Bates, 2012, 2014; Ma et al., 2013a, 2013b). The nodes around the outer boundary of the network were fixed in place such that the length of each spring in the network was 11% above its zero-load length, so the network began in a pre-stressed state. The equilibrium lengths and the elastic constants of the parenchymal springs were both set to unity. Airway contraction proceeded in small steps through gradual reductions in the zero-load length of all springs comprising the airway wall until airway contraction had reduced lumen area to 40% of its initial value had been achieved. At each step in this process, the elastic equilibrium configuration