Relationships Between Continuum Lung Tissue Strains, Alveolar Wall Strains and the Potential for Injury

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I. INTRODUCTION

Lung injury is a major risk in many impact scenarios, including automotive crash, sports, ballistic impact, and blast. Finite element (FE) models of the human body, including the lungs, are often used to investigate the potential for lung injury in these scenarios [1-3]. In such models, the lungs are modelled at the organ level using continuum elements, which do not capture the alveolar microstructure of lung tissue. It has been postulated that even during compressive loadings to the thorax, the maximum principal strains (tensile) in the alveolar walls may exceed critical values for increased permeability of the lung epithelium resulting in injury [4]. However, the link between continuum level deformation of lung tissue and the corresponding deformations to the alveolar wall where injuries occur has not been sufficiently characterized. This study uses a FE model of an alveolar cluster to determine the relationships between continuum level deformations at the lung organ scale, alveolar wall strains and the potential for injury.

II. METHODS

A FE model of a representative alveolar cluster was created and solved using a commercial explicit FE code (LS-DYNA R11, LSTC). The 1.3 mm³ cube comprised tetrakaidecahedral structures meshed with single integration point hexahedral elements (Fig. 1(a)) representing alveolar wall tissue. The tetrakaidecahedron has been identified in previous investigations as a suitable approximation for lung parenchyma [4-5]. The average diameter of a tetrakaidecahedral cell in the model was 200 μ m, with wall thickness of 12 μ m, to match human alveoli. The characteristic element size used in the model was 10 μ m.

A hyperelastic constitutive model was used for the alveolar tissue, based on experimental stress-strain data [6-7]. The alveolar cluster model was simulated in various modes of deformation (uniaxial tension/ compression, biaxial tension/compression, volumetric tension/compression, and simple shear) using prescribed displacements (Fig. 1(b)). For the biaxial modes, the model was stretched equally in two orthogonal directions, with no constraints in the third direction. The deformation of the entire cluster model in these simulations was analogous to the deformation of a continuum scale lung tissue element in larger human body models, where a single element would correspond to an alveolar cluster (as shown in Fig. 1(a)). In this study, the strains in the alveolar wall tissue are denoted ε_{AW} , and the strain of the entire cluster is denoted ε_{Cont} (labelled in Fig. 1(b)).



Fig. 1. (a) Alveolar cluster model shown with continuum scale human model (WALT v5.3 [3]) and (b) deformation cases with contours of first principal strain (not to scale).

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III. INITIAL FINDINGS

The maximum first principal strain (MPS, corresponding to peak tensile strain) and minimum third principal strain (3PS, corresponding to peak compressive strain) histories of the alveolar wall elements for each simulation were extracted, and the 95th percentile values were determined at each time step. Next, the relationships between the global strain of the whole alveolar cluster (ϵ_{Cont}), and the 95th percentile strains in the alveolar wall tissue (ϵ_{AW}) were determined (Fig. 2). The results demonstrate approximately linear relations for uniaxial tension/compression and simple shear, but with smaller magnitudes of ϵ_{AW} compared to ϵ_{Cont} . In the compressive loading cases, buckling of the alveolar walls was observed at approximately $\epsilon_{Cont} = -0.45$ (uniaxial) and $\epsilon_{Cont} = -0.2$ (biaxial and volumetric), as indicated by the non-linearity in the curve shape in Fig. 2(a)–(c), and can be seen in Fig. 1(b).



Fig. 2. Relationships between the 95th percentile alveolar wall strains (ϵ_{AW}) and continuum level strains (ϵ_{Cont}) for (a) uniaxial tension/compression, (b) biaxial tension/compression, (c) volumetric tension/compression, and (d) simple shear.

IV. DISCUSSION

The relationship between alveolar strains and resulting injury is not well characterized in the literature, however an initial postulation can be made by comparing against the physiological range of strains in the alveolar wall. A review article [8] reported that the alveolar strains incurred during quiet breathing can be up to 5% ($\epsilon_{AW} = 0.05$), and up to 40% ($\epsilon_{AW} = 0.40$) during maximal inspiration. Assuming that tensile strains in the alveolar wall beyond this range ($\epsilon_{AW} > 0.40$) represent an increased potential for injury, the results of this study provide threshold values of continuum level strain that correspond to this maximum alveolar strain (Table I). Due to the buckling behaviour of the model, the uniaxial and biaxial compression cases did not reach this value.

Values of continuous level strain (ϵ_{CONT}) corresponding to an alveolar wall strain (ϵ_{AW}) of 0.40						
Uniaxial	Uniaxial	Biaxial	Biaxial	Volumetric	Volumetric	Simple Shear
Tensile	Compressive	Tensile	Compressive	Tensile	Compressive	
ε _{Cont} = 0.480	N/A	$\epsilon_{Cont} = 0.447$	N/A	$\epsilon_{Cont} = 2.02$	ε _{Cont} = -0.645	ε _{Cont} = 0.459

Table I Values of continuum level strain (ϵ_{cont}) corresponding to an alveolar wall strain (ϵ_{aw}) of 0.40

The results presented in Fig. 2 and Table I provide a link between continuum scale elements used to represent lung tissue, as is common in human body models for simulating impacts to the thorax, and the strains that may occur at the scale of the alveolar walls where actual injuries occur. Consequently, these relations can inform predictions of lung injury from the results of continuum lung models. Initial investigations using a human body thorax model [3] (shown in Fig. 1(a)) in a pendulum impact condition known to cause lung injury produce continuum levels strains in the lungs that exceed some of these thresholds.

The alveolar cluster model used in this study did not include surface tension effects, which are known to be important to alveolar mechanics. However, since the simulations used prescribed displacements to simulate the various deformation modes, the predicted alveolar strains should generally be consistent with models that include surface tension effects, although the predicted stresses would likely be different. Future work will further investigate the stresses and deformations of the alveolar wall in impact conditions, the effects of air and surface tension, and the suitability of various injury metrics at the alveolar and continuum scales.

V. REFERENCES

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