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Kinematics of human thorax facing a blast: one possible explanation of lung's degradation

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1. Introduction

Lung injuries are the primary cause of death due to an explosion induced shock wave. It is therefore essential to understand the mechanisms able to cause those injuries for protection purposes. For the moment, blast's effects on the lung are not well described. Global injury criteria used in the literature, such as intra-pulmonary pressure or thoracic cage velocity thresholds (Axelsson and al. 1996, Boutillier and al. 2017), are not directly related to physical mechanisms explaining the degradation of the lung. Moreover, for the moment those degradation's mechanisms are not well understood.

As a consequence of blast loading, hemorrhages appear in the injured lung. Some authors made the hypothesis that they can be induced by an overstretching of the alveolar wall or by an important blood pressure in the capillaries (Fung and al. 1988; Wang and al. 1983). Nevertheless, there is currently no explanation of the appearance of one or more of those mechanisms as a consequence of blast loading.

The displacement of the thoracic cage induces a complex global movement of the internal organs which, to the authors' knowledge, has not been studied. The main goal of this study is to demonstrate the role of this complex kinematics in inducing the overstretching of the alveolar walls, which can result in the lung's injuries. Therefore, we will rely on macroscopic and microscopic observations of injuries in order to sort out the possible mechanisms at the microscopic scale and to understand the localization of those injuries. Next, we will simulate the effect of the blast on the thorax in order to understand its kinematics and to visualize the level of possible injury through a maximal principal strain criteria.

2. Methods

2.1 Injuries description

Biological models exposed sideway to a blast have been used in the literature to evaluate the biological

response of a personal frontally exposed (Boutillier and al. 2017). Additional autopsies revealed that hemorrhages appear in the lung's pinched part between the rib cage and the diaphragm. Those injuries appear in correspondence of the flexible part of the rib cage.

Microscopic observations described in the literature showed that alveolar walls can be more stretched after the blast exposure (Arora and al. 2017). This can be caused by an overdistention of the alveolar walls during the blast exposure. This observation reinforce our hypothesis on the role of overstretching of the alveolar wall in the lung's degradation. As the degradation is localized in the pinched part of the lung, we make the hypothesis that the mechanism of degradation is a structural effect due to the crushing of the lung by the thoracic cage.

2.2 Simulation

In order to understand the macroscopic effects of the blast on the thorax, we used the THUMS model (Toyota 2021). This finite element model represents the geometry and material properties of the organs of the human body of a 50th percentile man. We simulated a blast from a 5 kg of TNT explosion at 3 m away arriving frontally on the model.

We modified the lung's mechanical behaviour implemented in THUMS model in order to be closer to the properties of the real organ. The lung were modelled with a time-independent foam behaviour with a bulk modulus equal to 0.14 MPa, corresponding to the compressibility of the air contained in the lungs (Fung and al. 1985), which increases with compression to simulate densification of the organ. The bulk modulus in the THUMS model was equal to 1.4 MPa, which was too large regarding the modulus of the lung.

3. Results and discussion

Simulations on the macroscopic scale show the presence of significant stretching of the right lung. This stretch is localized in the part of the lung between the thoracic cage and the diaphragm. Therefore, the stretching is found in the pinched part of the lung (Figure 1), the same localization was found for the degradations in the biological model. The localization of the maximum of principal strain are thus coherent with the localization of the degradations.

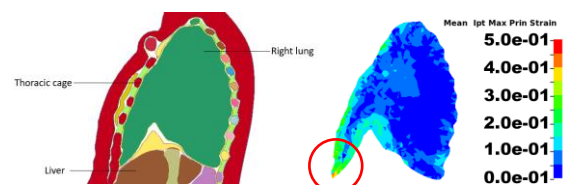


Figure 1 Localization of the parenchyma stretching (logarithmic scale)

This elongation can be generated with the kinematics of the thorax submitted to a blast, with the thorax's geometry and the relative movement and deformation of the thoracic cage, the mediastinum and the lung. The thoracic cage, put into motion by the blast load, will crush the lung against the liver, which results in its pinching. At the same time, the mediastinum will push the lung upwards. Those two action results in a stretching of the parenchyma.

The study of the alveolar wall in traction shows that a rupture occurs for a deformation of 130% of the length of the alveolar walls in the collapsed lung (Fukaya and al. 1968). For comparison, a total lung's capacity corresponds to a deformation of approximately 80% with respect to the residual volume. As the deformations in our simulation are calculated with respect to THUMS initial lung's volume, it is important to change the reference volume to define the failure deformation. Supposing that Fukaya's lung's volume equals to its residual volume, that corresponds to deformation of the order of 40 - 45% for a lung at rest within the thoracic cage (2 L volume). The simulation of a blast effect on the thorax shows a significant portion of the lung with stretching above 42% (Figure 2). Therefore, the stretch of the lung seems important enough to induce degradation of the alveolar walls.

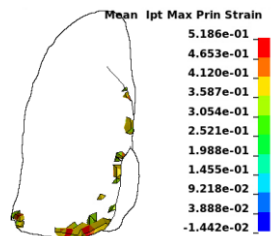


Figure 2 Stretching of the right lung above 42% (logarithmic scale)

4. Conclusions

The kinematics of the thorax induces stretching of the parenchyma in the part of the lung pinched between the thoracic cage and the diaphragm. Therefore the localization of this stretched zone is coherent with the area where hemorrhages are observed in the degraded lung. This stretching is of the same order as the strain at rupture of the alveolar walls. This strengthens our hypothesis that the lung's degradation is due to an overstretching of the structural part of the alveolar wall caused by the kinematics of the thorax facing a blast. Therefore the maximum principal strain of the lung could be used as a pertinent criterion of degradation.

In the future, it would be interesting to study the strain at rupture of the alveolar wall at higher strain rates, for a more precise value of the failure criterion. As information such as the value of stretching at rupture

come from the literature, and several hypothesis were made in the simulation, it would be interesting to have more precise information or even to make some simulation at the alveolar scale. Studies on the change of permeability of the alveolar wall during its stretching may provide a more precise value of the maximum possible strain before bleeding.

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