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Stress amplification effect of lung

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Abstract

Under a blast or impact load, rapid movement of the thoracic wall generates stress in lung, a foam-like structure of high compressibility, which is different from general solids. Due to this unique characteristic, it is hypothesized that when lung is subjected to a blast or impact load, there will be an initial low stress progressively developed to a high stress in a short duration in a thin layer of parenchyma near the lung surface. Compared to the incident stress, the actual stress value experienced by lung is amplified, which may cause alveolar-capillary walls to burst, subsequently results in edema or hemorrhage. This hypothesis can explain one significant phenomenon observed in animal tests that the gross thoracic compression do not cause major lung injury and there is a close relationship between thoracic wall velocity and the lung injury degree. According to the hypothesis, under a blast or impact load, there should be a significant injury degree discrepancy between a thin layer of parenchyma near the lung surface and the rest of the lung. Serious injuries should be mainly found in this thin layer, which can be employed to test whether this amplified effect exists or not. The hypothesis may shed some light on the mechanism of blast lung injury.

Introduction

Blast lung injury (BLI) refers to lung injuries such as contusion, edema, tearing and hemorrhage when torso is subjected to blast load. In wartime years, it was often observed in battle fields and weapon-launching sites. Recently, there is an increase in the number of BLI in industrial accidental explosions and terrorist attacks such as bus bombing [1]. In addition, high velocity blunt impact to the chest also causes similar lung injuries. One may suspect that the injury mechanism is similar or even the same [2]. Minimization of injury and more effective design of protective methods require a quantitative model of lung response to such load.

To make the injury mechanism clear, a variety of research methods, e.g. animal tests, theoretical analyses and numerical simulations have been conducted [e.g. 3-5]. It was assumed that the BLI is caused by tension in the alveolar walls during the re-expansion phase after the passage of the pressure wave [6]. Recently, it was hypothesized that this kind of injuries are induced by both the pressure wave and the re-expansion: first, spalling occurs in the alveolar-capillary walls under strong pressure wave; then, the walls are further damaged by the violent re-expansion of the collapsed tissue under the negative pressure wave [7]. However, the injury mechanism has still not been fully understood.

The Hypothesis

In mechanical point of view, the lung is a foam-like structure consisting of air and parenchyma. When establishing the lung model, some researchers considered it as an

elastic and homogeneous multi-phase medium [e.g. 4]. Although it is reasonable in some aspects, this kind of homogeneous model may neglect the compressibility difference between general solids and foam structures: the foam structures are highly compressible while general solids are relatively difficult to be compressed.

Hypothesis: due to the high compressibility of the foam-like lung structure, when subjected to a blast or impact load, the stress in a thin layer of parenchyma near the lung surface is amplified by local violent compaction in a short duration. Specifically, under such load, the rapid movement of thoracic wall impacts lung surface within a short time period, typically several milliseconds, results in an impulse imposing on it. For illustrative purpose, the impulse is assumed to have a square shape, indicated in dashed line in Fig. 1. For a solid material, the impulse will keep its shape and propagate into the inner part. Since the lung has a foam-like structure and the impulse duration is too short to disturb the entire lung, only a thin layer of parenchyma at lung surface is compacted. In a real situation, under such a square impulse, the prescribed stress level at the beginning cannot be achieved due to the high compressibility of lung; instead, an initial low stress is retained, corresponding to the start of the thin layer compaction. With the increase of compaction degree, the stress in this thin layer increases progressively until the layer is highly compacted, which leads to a sharp stress increase, shown in Fig. 1. Different from compaction of open-cell metal foams, “highly compacted” means high compaction degree rather than being compacted into a solid state without air. In fact, the impulse duration is so short that air does not have

sufficient time to escape, subsequently trapped in lung. According to impact dynamics, the area under the square impulse must be the same as that under the solid curve-the stress time history of the thin layer. Therefore the initial low stress in the beginning of the impulse inevitably leads to high stress level afterward, which can be extremely high and may cause the alveolar-capillary walls to burst. Physically, the incident stress is amplified to an extremely high value, suggesting a possible mechanism for BLI.

Interpret of experimental results

Animal tests indicate that the gross thoracic compression is not the major injury mechanism; in addition, there is a close relationship between thoracic wall velocity and the injury degree of BLI [8]. It can be readily explained by this hypothesis. It is obvious that the thoracic wall velocity is directly related to the impact time duration: for the thoracic wall, generally, under the same impulse, the shorter the impact duration, the higher the velocity. Imagine what will happen when the load time duration is very long, even quasi-static: the lung will deform uniformly in a global manner, thus the stress has sufficient time to distribute uniformly throughout the entire lung and the damage level is low. However, under a blast or impact load, the thoracic wall undergoes a rapid movement within a short duration. The stress does not have sufficient time to distribute uniformly, subsequently, compacts a thin layer near lung surface and results in highly concentrated stress in this layer. As described in the previous section, the stress is amplified to a high value, which may cause BLI such as edema and hemorrhage.

Implications

If the hypothesis of stress amplification effect is correct, some implications can be made. (1) Under a blast or impact load, the most seriously injured part of lung should be the surface, while the inner part experiences minor or even no injuries. In the aspect of injury degree, there should be an obvious discrepancy between the thin parenchyma layer near the lung surface and the rest of the lung. In other words, the injury pattern of BLI should be highly local in surface rather than disperse throughout the entire lung. (2) When establishing the BLI criteria for human, direct extrapolation from animal tests using the relationship between impulse, pressure and time duration may lead to inaccurate prediction. In certain cases, it may overestimate the injury threshold. In order to accurately predict the human BLI criteria, the stress amplification effect of lung must be taken into consideration. In fact, the first inference can be employed to experimentally test whether this amplification effect exists or not.

Conflict of interest statement

None declared.

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Fig. 1 Stress time history in a thin layer of parenchyma near lung surface when subjected to a blast or impact load. The dashed line is the incident impulse time history. The photograph in the upper right is synthesized according to the hypothesis. This amplification effect should be experimentally justified.