Lung Shock Injury Behind Body Armor

D. Pang, M. Panzer, K. Sirhan, J. Op 't Eynde, R. Salzar, J. McEntire, and C. Bass

I. INTRODUCTION

Modern body armor protects against ballistic threats by transferring incoming projectile momentum into the body armor materials at the cost of deformation of the body armor. Though this approach has been successful in reducing injuries and fatalities of personnel wearing body armor [1], the armor backface deformation into the torso may induce impact-related injuries to the chest wall and lung [2–4] termed behind-armor blunt trauma (BABT). In body armor design, the risk assessment of the potential for BABT is a critical consideration in balancing protection and personnel borne mass [5–7]. Typical current injury assessments assume deformation depth is well correlated with thoracic behind armor injuries [8–9]. In contrast, our group recently discovered the potential for strong compressive shocks damaging lung tissue in high-rate blunt impacts typical of those that generate BABT [10–11]. Since the speed of sound in physiological lungs is relatively much lower than in air or in all other tissues at ~20 m/s over most of the lung air volume range [12], in BABT thoracic backface chest wall velocities may substantially exceed the local material speed of sound. Injury to the lung via compressive shock is a competing mechanism with other potential BABT sources such as rib fractures.

A theoretical shock model is employed to provide a framework for injury risk assessments of BABT from high-rate armor backface impact. This shock model was previously used to assess the risk of pulmonary injuries from blast exposure [11]. Porcine experiments were performed to assess the competing risks for BABT injury source and to confirm predictions of the injury model.

II. METHODS

Theoretical Modeling

Formation of injurious compressive lung shocks requires local acceleration of the chest wall, distance from the wall, and time [13]. The distance from the material wall (x) for shock formation can be calculated as

$$\mathrm{x} = \mathrm{x_v} + \mathrm{c_0}\left\{\left(\mathrm{t}-\mathrm{t_\gamma}\right) + \frac{3(\gamma+1)}{32\gamma}\,\frac{a_{CW}\left(\mathrm{t}-\mathrm{t_\gamma}\right)^2}{\mathrm{c_0}} - \frac{45(\gamma+1)^2}{2048\gamma^2}\,\frac{\mathrm{a_{CW}^2}\left(\mathrm{t}-\mathrm{t_\gamma}\right)^3}{\mathrm{c_0}^2} + \cdots\right\} \qquad (1)$$

where c_0 is the material speed of sound, γ is the ratio of specific heats, a_{cw} is the chest wall acceleration, and x_γ and t_γ are the shock initiation distance and time. Given the material speed of sound in physiological lungs under normal respiratory conditions is much lower than in air or tissue ($c_0 = 20$ m/s, $\gamma = 1.4$) [14-16], the available distance is conducive to shock formation under realistic impact chest wall deformation rates. Since the lung compressive speed of sound is insensitive to lung inflation, compressive shock injuries are implied to occur further from the pleural interface but within the typical lung depths of ~180 mm or more in human or porcine lungs.

Experimental Testing

Fifteen live porcine impacts on the mid-thoracic ribcage were conducted with three controls using an indenter representative of a deforming armor backface [4][10] at velocities of 20-70 m/s and impact energies of up to 1217 J with one hour survival period. The experimental protocol was approved by the local Institutional Animal Care and Use Committee (IACUC). Injuries to the lung were assessed with post-test necropsy and local histology. Photographs of the lungs were digitised using ImageJ such that the surface area of present contusions was traced relative to the total visible surface area of the lung. The extent of surface contusion was calculated as a percentage of total surface area and was modelled by a second-degree polynomial regression.

III. INITIAL FINDINGS

Shock formation distance in the lung is shown in Fig. 1A. The shock formation distance weakly decreases with increases in chest wall acceleration. Histological findings from the experimental testing using porcine specimens

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(Fig. 1B) shows alveolar damage away from the impacted wall. The appearance of this damage is consistent with the shock predictions in Fig. 1A.

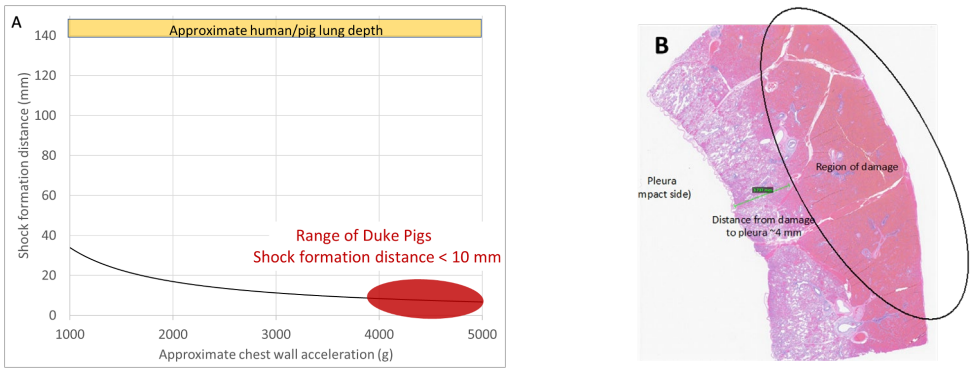

Fig. 1.: (A) Predicted lung shock distance from the chest wall using equation 1. For increasing chest wall acceleration, shocks are produced closer to the chest wall. (B) Shock formation in experimental animals at moderate severity shows damage away from the pleura consistent with the predictions from equation 1.

The extent of contused lung area is found to increase with a corresponding increase in impact energy that is well characterised by polynomial regression (R^2=0.67) (Fig. 2A). The nonlinear dependence of whole lung contusions with impact energy is consistent with the prediction that shock damage is the source of alveolar disruption, and there is an apparent asymptotic effect for higher impact. With increasing energy, damage was found to be more extensive, spread into the contralateral lung from the impact side, and initiated closer to the lung surface. Since these lung injuries are consistent with previous injury criteria developed for similar chest wall velocity ranges in blast exposure, injury assessments for BABT were developed as an extension of these blast injury assessments as shown in Fig. 2B.

A. B.

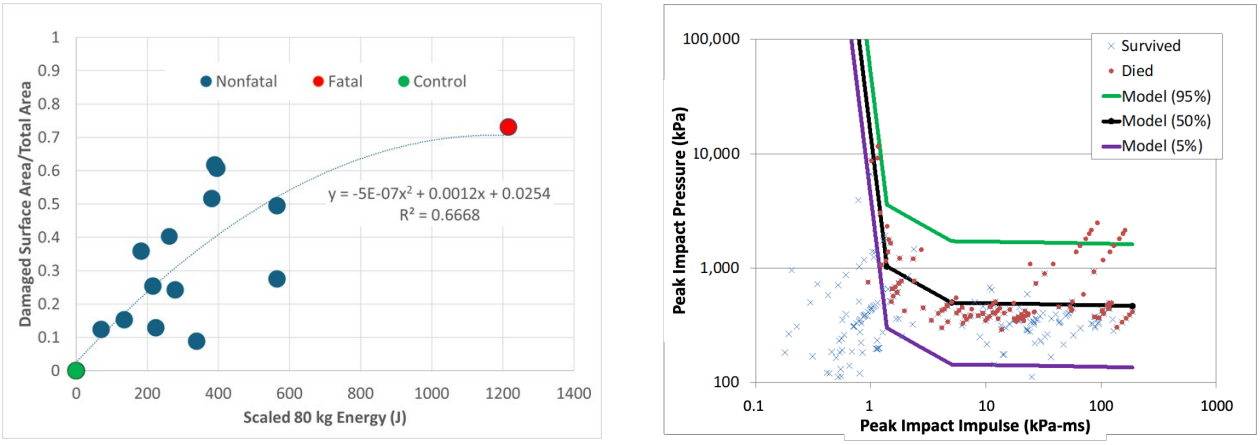

Fig. 2.: (A) Experimental ratio of contused surface area to whole lung demonstrates saturation effect of lung compression damage from a localised backface deformation with increasing impact energy. The red point constitutes a fatality. The green point is a control animal. (B) Proposed BABT injury risk assessment extended from proposed blast injury risk assessment [11].

IV. DISCUSSION

This study provides preliminary injury risk estimates for our recent discovery of a novel, but ubiquitous, injury risk for behind armor blunt trauma. A novel compressive lung shock injury mechanism is proposed as the source of parenchymal lung injuries for backface armor deformations producing thoracic motions greater than ~20 m/s. Injury risk modeling has been extended to incorporate observed injuries in the contralateral lung and has been verified by lung contusion results in the experimental model. This novel risk assessment is essential for assessing competing risks in thoracic injuries from rib fractures and other potential injuries.

V. REFERENCES

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