Model for Pulmonary Response Resulting from High Deformation Rate Loading

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Abstract abstract

Pulmonary contusion or Primary Blast Injury can result from exposure to blast overpressure. The development of improved protection requires the ability to evaluate pulmonary response at the tissue level. A detailed numerical torso model was developed and used to predict response and the potential for injury resulting from short duration loadings. Long duration blast loading (duration > 10ms) is not common; however, the associated injury data necessary for model calibration is the most consistent from various sources in the literature. The first version of the torso model was not able to consider long duration loading due to the large tissue deformations. To address this issue, a hyperelastic tissue model was integrated with a bulk model to address long duration loading. The model was evaluated for long duration overpressures to provide an injury threshold. This model was subsequently investigated for varying overpressures, demonstrating increased predicted injury with increased overpressure. Injury was predicted to decrease for decreasing duration below 10ms. Above 10ms, the injury prediction was relatively constant in agreement with experimental data, and explained by the wave transit time in the lungs. This study expands the range of applicability of a detailed thorax model to predict the potential for primary blast injury, and an enhanced constitutive model for lung tissue to include bulk effects has been proposed.

Keywords lung contusion, Primary Blast Injury, bulk response, numerical blast model

I. INTRODUCTION

Pulmonary contusion injuries most commonly occur from blunt thoracic impacts, for example in vehicle collisions, and range from Moderate (AIS 2) to Critical (AIS 5) [1]. While less common, exposure to blast overpressure can cause contusion of lung tissue, commonly described as Primary Blast Injury (PBI) [2]. These contusion injuries can range from Serious (AIS 3) to Critical (AIS 5) [1]. Although the mode of loading varies between blunt impact and blast, the resulting pulmonary injury is generally accepted to be contusion, described as parenchymal damage occurring at the tissue level resulting in interstitial edema and capillary hemorrhage. PBI has been known for many years [2] and several methods currently exist to predict the potential for injury including correlations based on the blast wave transient pressure [2], analytical models based on experimentally measured data [3, 4], and numerical approaches [5]. All of these models have been validated or calibrated using animal test data, required due to the physiological nature of contusion response. Although primary blast injury to the lungs is often considered to be well-understood with respect to injury tolerance, there are differences in predicted tolerance level for short duration blasts (on the order of 1 to 10 ms in duration, Fig. 1, Fig. 2). Some of this may arise from the experimental tests. For example, very short duration blasts often require very close proximity to the explosive and was achieved in earlier tests using relatively small charges with small animals in close proximity [2]. Proximity and animal size are only two of many parameters that could lead to nonlinear results including the interaction and wrap-around of the pressure wave. Importantly, there is relatively good agreement between many data sources regarding long duration blast loading (Fig. 2), and this data was used for the current study.

The first and most widely referenced body of data and corresponding injury curves are attributed to Bowen and Richmond [2, 7] who related overpressure, the pressure exceeding atmospheric pressure, to primary blast injury. They undertook tests on a variety of animals in the late 60’s and early 70’s using controlled blast loading
from high explosives and shock tubes to demonstrate several important concepts:

- Significant injury or fatality could occur due to lung contusion resulting from exposure to blast.
- The extent and severity of injury depended on the magnitude and duration of the blast overpressure.
- At long durations, the probability of injury was related to the magnitude of the overpressure.
- Injury was only observed if the pressure wave was a ‘fast-rising’ wave.

Based on this experimental data a series of injury curves were proposed using the static or side-on pressure (Fig. 1) and duration of the blast wave to predict injury (Fig. 2). It should be noted that the 50% probability of survival curve (LD50) was based on the experimental data (fatality within 24 hours of exposure), while the threshold, 1% and 99% lethality curves were inferred from the data. The LD50 long duration overpressure was 248 kPa.

Fig. 1: Bowen injury curves (adapted from [6])

Subsequent studies by Stuhmiller et al [3, 8] utilized additional test data, primarily on goats, to develop an analytical model based on normalized work [3], which used the pressure-time measurements from an experimental device known as the Blast Test Device (BTD) for input. The BTD is a right circular cylinder with four evenly spaced flush-mounted pressure transducers around the circumference. Stuhmiller developed a mathematical model using the proposed normalized work to predict injury for unprotected simple and complex loading cases, correlated to blast field measurements using pressure histories from a BTD. The normalized work value was correlated to lung injury by first performing tests on animals (sheep in this case) and repeating the test with the BTD. The level of injury was correlated to the normalized work values determined from the pressure gauges. This model is used in a propriety computer program (Injury 8.2) to predict injury.

Axelsson has also proposed a mathematical model for blast injury using a non-linear differential equation to represent deformation of the torso based on the applied pressure (measured using the BTD) and validated using blast tests with sheep [4]. Axelsson correlated peak inward chest wall velocity ranges with injury: 3 – 4.5 m/s for threshold lung damage, 8 – 12 m/s for LD1 and 12 – 17 m/s for LD50, with a chest wall velocity of 12.8 m/s corresponding to 50% lethality.

More recently, Bass et al [9] and Rafaelis et al [10] have reviewed a larger set of data and proposed revised pressure-duration injury tolerance curves. In particular, one study focused on a re-analysis of existing data supplemented with new data to identify the injury risk associated with exposure to long duration blast waves, defined as blast waves exceeding 10ms in duration. The corresponding LD50 value was approximately 200 kPa overpressure [10].

The prediction of response to blast loading is essential for the development of improved blast protection [6, 11]. Blast injuries to the human body include fragmentation, global motion of the body, burn, inhalation and other effects. PBI refers to injuries caused by interaction of the blast wave with the human body, and affects gas-containing organs such as the lungs, tympanic membrane and gastro-intestinal tract [2]. This injury is a consequence of impedance coupling between the stress wave and tissue, and is of significant importance in cases where the human body is unprotected. A shock or blast wave is generated by the detonation of a high
explosive which can be considered as a nearly instantaneous transformation of the explosive to a highly compressed gas. Expansion of these high pressure gases into a lower pressure medium such as air creates a shock wave that propagates through the quiescent air represented by a discontinuous jump in pressure, density, velocity and internal energy. A discontinuity in density, known as the contact surface, follows the shock wave and is the boundary between the explosive detonation products and air [12]. Although this can be an important consideration for very close proximity blast loading, the current study is focused on interaction of the effects of the blast wave at greater standoff distances. Future studies will investigate the effects of close proximity blast. The impact interaction of a blast wave on with a deformable body may lead to deformation which in turn affects the blast wave (fluid) flow. This coupled effect may be significant in terms of the overall response of the structure, particularly when considering complex blast environments where the loading duration may be relatively long.

Several numerical approaches have been considered to investigate the effect of blast waves on animals or humans [5, 13, 14]. The primary challenges for these models include appropriate application of the blast load, and incorporation of the mechanical properties required to predict response of the tissues and the potential for injury. Detailed thorax models have been developed, primarily in the automotive community and some in the blast community to predict thoracic response, and a limited number have investigated pulmonary response and the potential for injury [15]. However, full three-dimensional models of the torso are computationally prohibitive to implement due to the required mesh refinement required to accurately resolve shock propagation through the torso. Some challenges for existing models include the ability to account for body orientation with respect to the blast wave [16], the incorporation of protection [6], and the ability to consider complex-shaped blast waves or blasts occurring within enclosures. Notwithstanding these important issues, all detailed finite element models must include representative constitutive description of the constituent materials. Although the existing models have been calibrated using experimental measurements of the blast field with injury to animal subjects, they are somewhat limited in that pressure measurements behind personal protection are inherently challenging to interpret, and the methods have not been verified for this situation. In order to predict the effectiveness of blast protection, an appropriate predictive model is necessary. This led to the development of a human torso numerical model and prediction of injury at the organ level for the evaluation of protection concepts.

The model used in this study has been applied successfully to investigate primary blast injury resulting from applied blast loading [5], the effects of body orientation on predicted injury [16] and the effect of protection on primary blast injury [6,11]. An explicit finite element code (LS-Dyna, LSTC) with a coupled Eulerian model representing the blast flow was incorporated with a Lagrangian model of the torso.

The torso model included the thoracic cage, lungs and mediastinum with tissue material properties from the literature and rate dependent tissue models [5,17]. The geometry of the model (Fig. 3) was based on a mid-sternum section (transverse plane between the 5th and 6th thoracic vertebrae) of an approximate 50th percentile male [5]. The torso depth (230mm) was in good agreement with the literature for a 50th male subject (298.5mm). The model included layers of elements to simulate the ribs, and layers to simulate the intercostal tissues, with the simplification that the ribs occurred in the transverse plane. However, static compression simulations of the individual rib showed that this effective rib provided the correct force-displacement response [18]. Previous studies have investigated the required mesh density for a consistent prediction was approximately 2.5 mm [17]. The current model included a mesh size of approximately 2mm with a total of 33,312 solid elements.

The blast flow was coupled to the torso using a penalty coupling algorithm. A study of the ALE approach to model shock and expansion waves showed that this method produced excellent results in terms of the magnitude and transients when the mesh size was 0.5 mm [19]. For the current study, it was found that a mesh size of 2mm in the area of interaction with the torso was sufficient to produce consistent results. The element size was graded away from this zone for computational efficiency. It should also be noted that the coupling algorithm required the mesh density of the torso and air to be comparable to avoid leakage of the air through the torso model. For the current study, the air model was increased in size (Fig. 4, total of 177944 solid elements) to avoid interaction with the model boundaries. This allowed for a maximum simulation time of approximately 16 ms before reflected waves from the blast wave source reached the torso model.
There is a great deal of literature on the subject of the mechanical properties of lung tissue, and the evaluation of lung injury [20, 21, 22]. For the current study, the deformational or deviatoric properties were based on those proposed by Yuen [21] as implemented in the lung tissue model proposed by Vawter (Eqn. 1). In the case of high rates of loading, the bulk response of the tissues, lung tissue in particular, is important in the predicted response while at lower rates and larger deformations the deviatoric and viscous properties are more important. The torso model, previously used for short duration loading, did not include deviatoric response for the lung and this was found to be important for long duration loading where larger deformations could occur. Similarly, the hyper-viscoelastic constitutive model from Vawter (Eqn. 1) included a linear bulk modulus and was not suitable for larger overpressures.

$$W(I_1, I_2) = \frac{C}{2\Delta} e^{(\alpha I_1 + \beta I_2)} + \frac{12C_1}{\Delta(1+C_2)} [A^{(1+C_1)} - 1], \quad A^2 = \frac{4}{3}(I_1 + I_2) - 1$$

**Where**

- \( W \) = Strain energy per unit volume
- \( I_1, I_2 \) = Strain invariants
- \( C, \alpha, \beta \) = Material constants; \( C=1.15\times10^{-3} \) MPa, \( \alpha=0.213 \), \( \beta=-.343 \)
- \( C_1, C_2 \) = Surface tension constants; \( C_1=1.002\times10^{-3} \) MPa, \( C_2=2.040 \)
- \( \Delta \) = Typical unstressed alveolar diameter; \( \Delta=1.00\times10^{-1} \) mm
This material model incorporated bulk effects using a constant bulk modulus, a common assumption for many materials. However, in the case of lung tissue the measured wave speed and corresponding bulk modulus is relatively low. For example, the acoustic wave speed in air is approximately 343 m/s at 20°C and 1 atmosphere of pressure. The wave speed in soft tissues is approximately 1500 m/s. However, the experimentally measured wave speed in lung tissue varies from 20 to 60 m/s depending on the species and conditions such as transpulmonary pressure.

Equations of state provide relationships between pressure, volume and internal energy and are necessary to predict material response if the material exhibits a non-linear bulk response or undergoes significant volumetric compression. Several common equations of state (EOS) exist including the Gruneisen EOS (Equation 2), which was used to represent the lung material properties in the original torso model. In this equation, the pressure is a function of the non-linear material bulk modulus (first term in equation 2) and the volumetric work (second term in equation 2). Parameters for this equation are available for a wide range of materials [23], and the rule of mixtures can be used to determine the constants for combinations of materials [24]. An equation of state may also be incorporated using a non-linear bulk modulus, which is a function of volumetric compression.

\[
p = \frac{\rho_0 C^2 \mu [1 + (1 - \frac{\gamma_0}{2}) \mu - \frac{a}{2} \mu^2]}{1 - (S_1 - 1) \mu - S_2 \frac{\mu^2}{(\mu + 1)^2}} + (\gamma_0 + a\mu)E
\]

Where
- \( p \) = Pressure (Pa)
- \( \rho_0 \) = Density (kg/m³); \( \rho_0=288 \) kg/m³
- \( C \) = Acoustic wave speed (m/s); \( C=26.1 \) m/s
- \( \mu \) = \( p / \rho_0 \) - 1
- \( \gamma_0 \) = Gruneisen Gamma; \( \gamma_0=1.618 \)
- \( E \) = Specific internal energy (J/m³)
- \( a \) = First order correction (not used in the current model)
- \( S_1, S_2, S_3 \) = Slope coefficients for the particle-shock velocity curve; \( S_2=1.309 \)

The goal of the current study was to use a detailed coupled thorax-blast model to investigate long duration loading for the purpose of further validating the model, since there is good agreement in the research community regarding human tolerance to long duration (> 10 ms) blast loading. Initial investigations with the original model demonstrated that it was not stable beyond 8-10 ms due to large deformations in the elements. In a similar fashion, when the Vawter equation with a constant bulk modulus was considered, the model was unstable at higher incident pressures due to the lack of an equation of state, and required an artificially high linear bulk modulus. To address these issues, an equation of state was integrated with the Vawter equation to extend the current model to long duration blast loading.

II. METHODS

The available experimental data for lung tissue properties were reviewed and the deviatoric properties were incorporated in the Vawter model (Eqn. 1) following Yuen [21]. The bulk properties of lung tissue play an important role in the overall response of lung tissue to insult. In particular, the low acoustic wave speed resulting from the low volumetric stiffness of the gaseous constituent and high density of the tissue constituent creates nonlinear bulk behavior at the pressures experienced under typical blast loading conditions.

Lung tissue presents an interesting challenge in terms of bulk properties since it consists of tissue, fluid and air. Several studies have been undertaken on two-phase flow (bubbly water) [24] and this provided a basis for
the estimation of bulk material properties from the constituent materials. From a bulk perspective, the tissue and fluid can be considered to have bulk properties comparable to those of water, with the second constituent being air. The relevant properties for each material are listed in Table 1. When combined using a rule of mixtures [2] and the conservation equations [23], the resulting macroscopic bulk properties for lung tissue can be determined. The results of this analysis were used to include nonlinear bulk effects through the incorporation of an equation of state for the lung tissue.

The resulting equations were incorporated into a special version of LS-Dyna version 971 R4.2.1 (LSTC) using a commercial Fortran compiler (Intel Fortran Version 11). The constitutive model implementation was verified using single element test cases compared to the experimental test data.

Table 1: Properties of air and tissue (water) for rule of mixtures analysis

<table>
<thead>
<tr>
<th></th>
<th>Co</th>
<th>S1</th>
<th>Density (kg/m³)</th>
<th>v0</th>
<th>gamma</th>
<th>T</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air</td>
<td>343 m/s</td>
<td>1.03 (-)</td>
<td>1.204</td>
<td>1 (-)</td>
<td>1.400 Cp/Cv</td>
<td>20 C</td>
</tr>
<tr>
<td>Water</td>
<td>1485 m/s</td>
<td>2.00 (-)</td>
<td>101325 Pa</td>
<td></td>
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</tr>
</tbody>
</table>

The thorax model, including the revised material model, was subjected to different blast load scenarios to evaluate lung response. The model response was compared to the previous model and found to be in agreement for short duration loads when no deviatoric properties were included in the Vawter model. Initial analyses were undertaken with long duration (greater than 10ms) blast loads to verify the model injury predictions and evaluate the injury metrics.

A series of blast load scenarios were then evaluated using the torso model including the deviatoric and bulk properties for the lungs. The specific effects investigated using the model included:

- Predicted response for long duration (>10 ms) fast-rising incident pressure waves with 248 kPa and 200 kPa overpressures.
- The effect of blast load duration for fast-rising incident pressure waves with 248 kPa and 200 kPa overpressures.

In all cases, the blast loading was constant overpressure for the prescribed duration, typical of long duration pulses.

The response was evaluated by tracking the peak dynamic pressure in the lungs, based on previous studies of lung response [5,15,17].

III. RESULTS

Application of the rule of mixtures and conservation equations using the data in Table 1 provided estimates of the bulk properties for lung tissue assuming a density of 288 kg/m³. One interesting aspect of this analysis is that the predicted wave speed in lung is quite low relative to other materials, and in particular with respect to the constituent materials. The predicted wave speed for a density of 288 kg/m³ was 26.3 m/s (Fig. 5) and was relatively constant across a range of volume ratios except at the extremes corresponding to a single constituent.
Fig. 5: Predicted wave speed as a function of porosity (volume ratio) for lung tissue (vertical line=288kg/m³)

The resulting bulk properties were integrated with the Vawter constitutive model using a non-linear bulk modulus. It should be noted that, in this first implementation the nonlinear bulk effects were introduced through the use of a nonlinear bulk modulus which effectively captured both the material and volumetric work portions of the bulk response. Future efforts will involve integration of the Gruneisen equation of state with the Vawter model. The resulting model was integrated and compiled into an explicit finite element code (LS-Dyna Version 971V4.2.1, LSTC 2010). Single element models were used to evaluate the material model response in tension, compression and confined compression (pressure versus relative volume, Fig. 6) and were in good agreement between the original Gruneisen model and the new model.

Fig. 6: Pressure versus relative volume for the lung model, demonstrating nonlinear bulk response

The thorax model was investigated using long duration loading, with the baseline case defined as a 248 kPa overpressure with 15 ms duration, corresponding to the LD50 value presented by Bowen (Fig. 2). Propagation and interaction of the blast wave with the torso (Fig. 7) demonstrates the reflected pressure at the front of the torso at 2.5-3.0ms, followed by development of the flow around the torso up to 5.0 ms. An image showing the overpressure predicted within the lung (Fig. 8) identifies most of the injury to occur in the anterior portion of the lungs, similar to that predicted for short duration loading [5], and also demonstrates the potential for injury behind the intercostal tissue (Fig. 8, right side, circled) which is unique to blast injury. The amount of injury was defined as the lung overpressure corresponding to 18% volume of the lung model. This has been identified as a threshold based on lung weight corresponding to LD50 in animal studies.
Following initial verification of the model and calibration of the injury threshold, analysis was undertaken to investigate response at other levels of blast loading (Fig. 9) based on the predicted volume of injured tissue. This included data normalized to the long duration injury criterion proposed by Bowen (248 kPa overpressure) and by Rafaels (200 kPa overpressure). Fig. 10 shows the predicted injury as a function of blast load duration.

Unlike short duration blast loading, where the thorax compression is relatively small, long duration loads can lead to significant compression and significant global displacement of the torso later in time (Fig. 11). The thorax velocity was also compared to the Axelsson criterion (LD50 for chest velocity greater than 12.8 m/s) (Fig. 12).
Fig. 9: Predicted injury versus blast overpressure for long duration (15 ms) blast loads

Fig. 10: Predicted injury for varying blast durations (248 kPa overpressure)

Fig. 11: Anterior and posterior compression velocity of the thorax (248 kPa overpressure)
IV. DISCUSSION

A detailed coupled torso and blast model was enhanced to evaluate long duration blast loads, typically defined as having durations longer than 10 ms. Enhancements included a larger surrounding air model to avoid wave reflections between the blast wave source and torso, refinement of the air material model, verification of the representative rib force versus deflection, and implementation of an enhanced lung tissue model. The lung constitutive model implementation was verified using single element test cases compared to the experimental test data. The expected hyper-viscoelastic response and the nonlinear bulk response were demonstrated. At short durations, the response of the torso model with the new constitutive model was similar to the original model, which incorporated only bulk material properties. However, at longer durations the original model was not numerically stable due to large element deformations so that long duration loading could not be evaluated. It was noted that there was some leakage of the air material into the lung material at higher overpressures and this will be evaluated for the next version of this model.

The thorax model was subjected to different blast load scenarios to evaluate lung response. Initial analyses were undertaken with long duration (greater than 10ms) blast loads to evaluate injury as a function of overpressure. As expected, the predicted injured volume increased with increasing overpressure. The effect of blast duration was investigated (Fig. 10) and demonstrated increasing predicted injured tissue volume with increased duration, up to approximately 10 ms. Beyond this value, the predicted injury was constant, corresponding to the LD50 threshold, in agreement with experimental results [2,10]. For the long duration LD50 threshold defined by Bowen (248 kPa overpressure, Fig. 7), the torso was found to reach maximum compression velocity of 9.4 m/s, defined as the velocity difference between the anterior and posterior of the torso, approximately 1.5 ms after first contact by the blast wave (Fig. 11). For comparison, the absolute values of the anterior and posterior velocities were investigated (Fig. 11, Fig. 12) and the anterior velocity at 248 kPa overpressure (14.11 m/s) was found to be reasonably consistent with the LD50 value proposed by Axelsson (>12.8m/s). This is likely more comparable to the Axelsson model since it did not include global body acceleration, a reasonable assumption for shorter duration loading. The chest wall velocity increased with increasing overpressure, as did the maximum compression of the torso (Fig. 12), occurring around 8 ms when the anterior and posterior velocities were equal. It was also noted, based on the stress wave transmission through the lungs, that the wave traversed the lungs (anterior to posterior) in approximately 7 to 8 milliseconds due to the low wave speed in this tissue. Fig. 10 suggests that the transition to long duration loading could begin around 6 ms; however, this is based on the dynamic lung pressure metric, which still requires further
evaluation. The predicted chest wall kinematics and lung wave propagation times are in agreement with the currently accepted value of 10ms for long duration loading.

V. CONCLUSIONS

The development of enhanced protection requires improved knowledge of human body response to blast. The benefits of a detailed coupled torso model include the ability to evaluate different types of loading, including non-traditional blast loading and protection. One of the primary limitations of the original model was the inability to provide response predictions for long duration loading. Although long duration loading is not common, this was considered important since the most consistent experimental data between different sources is for long duration (>10ms) blast loading. To address this limitation, the detailed thorax and blast model was enhanced to include a larger surrounding air mesh, refinement of the air material model, verification of the representative rib force versus deflection, and implementation of an enhanced lung tissue model. The lung constitutive model implementation was verified using single element test cases.

Long duration LD50 blast loading was applied to the torso model (248 kPa from [2] and 200 kPa [10]) and the response was evaluated by tracking the predicted peak dynamic pressure in the lung tissue. This metric has been used in previous versions of this model and provided good correlation to expected injury and sensitivity to the presence of protection. However, further analysis of the existing experimental data is required to verify this as an injury metric. The proposed dynamic pressure threshold corresponding to injury was comparable to values from previous models. The volume of tissue exceeding the LD50 threshold increased with increasing blast overpressure for long duration blast loading. Importantly, the volume of tissue exceeding the LD50 threshold decreased for short duration blast loading, but was relatively constant for long duration loading. This was explained by the wave speed in the lung tissue and the time to maximum compression of the thorax was of comparable duration.

The proposed enhancements to this model extend the capabilities to long duration loading and provide expected response based on the dynamic overpressure in the lungs. The primary limitation of the current study is the need for further validation across a wider range of loading. Future studies will investigate short duration loading with a corresponding analysis of the existing blast data in the literature.

A gap identified in the literature is the need for additional mechanical test data for lung tissue, particularly at higher rates of strain. For the current study the tissue deformations were generally small for most load cases considered. The prediction of lung contusion based on an injury metric provided a good correlation with the existing data; however, more detailed data regarding the specific location of the injuries within the lungs is needed. Lastly, the current model uses a continuum-level approach to model the lung tissue. Further investigation of the injury mechanism using multi-scale modeling approaches should be undertaken in the future.

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VII. REFERENCES


