

Numerical modeling of primary thoracic trauma because of blast

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ABSTRACT

Purpose: Since explosive blasts continue to cause casualties in both civil and military environments, there is a need for an understanding of the mechanisms of blast trauma at the human organ level, plus a more detailed predictive methodology. The primary goal of this research was to develop a finite element model capable of predicting primary blast injury to the lung so as to assist in the development of personal protective equipment.

Materials and Methods: Numerical simulation of thorax blast loading consisted of the following components: 3D thorax modeling reconstruction, meshing and assembly of various thorax parts, blast and boundary loading, numerical solution, result extraction and data analysis.

Results: By comparing the models to published experimental data, local extent of injury in the lung was correlated to the peak pressure measured in each finite element, categorized as no injury (< 60 kPa), trace (60-100 kPa), slight (100-140 kPa), moderate (140-240 kPa) and severe (> 240 kPa). It seemed that orienting the body at an angle of 45 degrees provides the lowest injury.

Conclusion: The level and type of trauma inflicted on a human organ by a blast overpressure is related to many factors including: blast characteristics, body orientation, equipment worn and the number of exposures to blast loading.

Keywords: blast wave; respiratory trauma; chest wall velocity; thorax modeling; ANSYS software.

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INTRODUCTION

In times of both peace and war, there are considerable reports of civilians and military personnel dying of blast-related injuries. An important subset of blast-related trauma, which has been the focus of this research, was primary blast injury, i.e. injuries which occur as a direct result of interaction and transmission of pressure waves through the body, primarily affecting air-filled organs, such as the lungs (**Figure 1**).

The ear is the most frequently damaged organ from blast overpressure because the auditory system has the lowest threshold for injury. Richmond suggested a human threshold range of 20-35 kPa, and that 50% of the population would show significant injury at 100 kPa.¹ At more lethal levels of a blast, pulmonary contusions

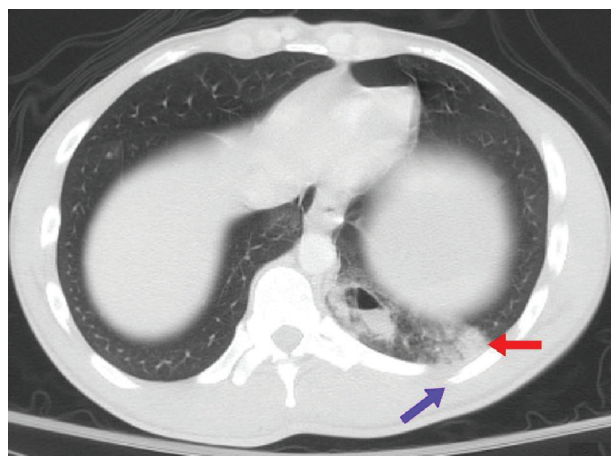


Figure 1. A computed tomographic scan showing a pulmonary contusion, red arrow, accompanied by a rib fracture, blue arrow. (Source: http://en.wikipedia.org/wiki/Pulmonary_contusion)

become evident with large areas of hemorrhaging and interstitial edema. Blast lung injury is the major cause of death in patients who survive initial resuscitation. Early mortality is associated with air emboli and massive pneumothorax and hemothorax, in which both blood (hemo) and air (pneumo) escape into the thoracic cavity. The gastrointestinal tract presents a risk of severe blood loss and infection, which can lead to hemoperitoneum and peritonitis.^{2,3}

Blast lung is a clinical diagnosis and is characterized as respiratory difficulty and hypoxia without obvious external injury to the chest. Patients may have a variety of symptoms, including the shortness of breath and labored breathing (dyspnea), slow heart rate (bradycardia), abnormally low blood pressure, chest pain, cough and hemoptysis. The expectoration of blood or blood-streaked sputum from the larynx, trachea, bronchi or lungs has been seen in more severe cases. A short time after the insult, a bluish discoloration of the skin and mucous membranes can be seen resulting from inadequate oxygenation of the blood. The symptoms listed above are generally the results of tearing or failure of sensitive biological materials, and are sometimes referred to as pulmonary barotraumas.⁴

Several approaches have been used for computational modeling of blast injury and resulting trauma. Earlier models used theoretical and semiempirical correlations to relate the blast shock wave parameters to severity of injury. One of the first fundamental injury biomechanical models was proposed by Stuhmiller and colleagues.^{5,6} In this model, schematically depicted in **Figure 2**, several springs, masses and dampers have been combined to represent thoracic anatomical structures and an impact mass.

The compact models incorporating mass, spring, and damping device elements can be expressed in a general Newton's second law form:

$$M \frac{dv}{dt} + D \cdot v + k(u - u_0) = F_{ext}$$

In this formula M is the tissue mass, v is tissue velocity, u deformation, and F_{ext} external force. In the Newtonian dashpot model, the force F is linearly related to the displacement rate v (or velocity $v = \dot{u} = du/dt$) in the form $F = D\dot{u}$, where D is the damping coefficient equivalent to viscosity η of the dashpot dampening. The model assumes that the lung behaves as compressible gas and uses the Landau-Lifshitz correlation,⁷ which relates the pressure wave in a compressible gas to the piston speed (**Figure 3**):

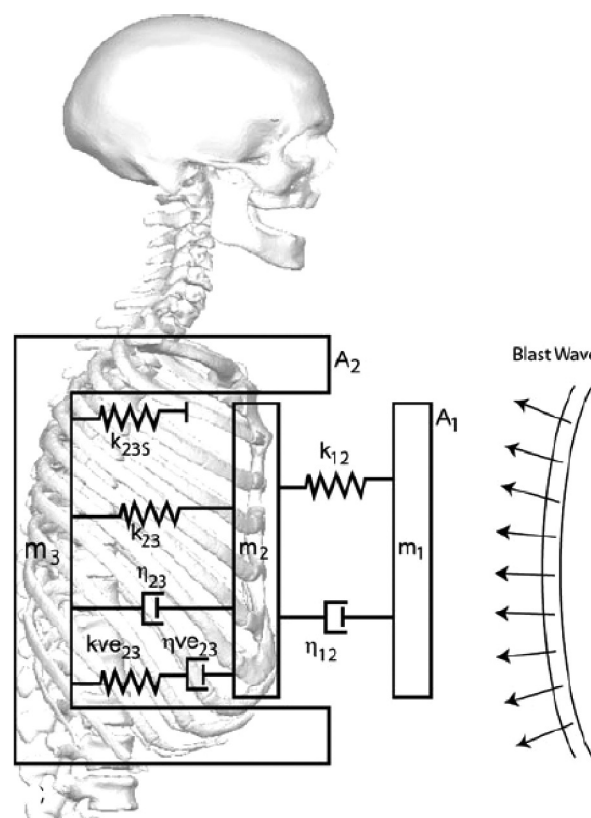


Figure 2. Spring-Mass-Dashpot compact model of thoracic biomechanics and impact injury, where k is the spring constant and η is damping coefficient (Source: Stuhmiller JH, Ho K, Vander Vorst M, et al. A model of blast overpressure injury to the lung. *J Biomech.* 1996;29:227-34.

key: m_1 , impact mass; m_2 , the sternum mass; m_3 , the thoracic mass; Spring k_{12} , the elasticity of the skin and the protective clothing; parallel Voigt spring-mass (k_{23} , η_{23}), the elasticity of the rib cage and attached viscera; series Maxwellian spring-mass (k_{ve23} , η_{ve23}), the viscoelastic thoracic muscle tissue; Spring k_{23s} , the bilinear increase of the thoracic stiffness.

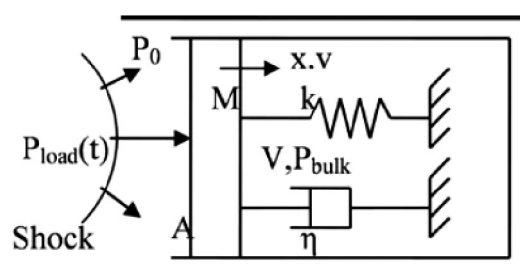


Figure 3. Generalized pleural dynamics compact model coupling external load, thorax wall, and compressible air in the lung.

$$P(t) = P_0 \cdot \left(1 + \frac{1}{2}(\gamma - 1) \frac{v}{c_0}\right)^{\frac{2\gamma}{\gamma-1}}$$

In this formula p_0 , r_0 , and c_0 are the pressure, density, and speed of sound in the undisturbed lung, v is the piston velocity, and γ is the ratio of specific heats.

The primary goal of this research was to develop a

finite element model capable of predicting primary blast injuries in simple and complex environments, while being flexible enough to be used as an engineering tool for the development of protective equipment. This model also provides insight into the mechanisms of trauma, reducing our dependency on animal testing.

MATERIALS AND METHODS

Chest radiography, computerized tomography, high flow oxygen, airway management, tube thoracostomy in the setting of pneumothoraces, mechanical ventilation (when required) with permissive hypercapnia, and judicious fluid administration are essential components in the management and diagnosis of blast lung injury. In this study, a finite element model was created in order to provide insight into the origins of trauma, providing an engineering tool for the development of personal protective equipment. The most recent thoracic injury finite element models have been developed in the context of blast injury by Przekwas and colleagues⁸ and Friend,⁹ ballistic non-penetrating impact and resultant behind armor blunt trauma by Lee and colleagues,¹⁰ Grimal and colleagues,¹¹ Roberts and colleagues,¹² and automotive crash analysis by Forbes.¹³

In this research, raw data for human thorax finite element modeling were obtained from normal adult men (170cm, 70kg). To create the model geometry, computer aided three-dimensional interactive application (CATIA) software was used and then the file was imported in Ansys simulation software. Because various human thoracic components have complex and irregular shapes, it is very difficult to generate hexahedral meshing; therefore, tetrahedral meshing was used. Mesh parameters such as size limits, edge ratios and densities were specified, but due to the complex geometry, actual meshing was performed by the software (**Figure 4**). The chosen material characteristic parameters were all based on relevant blast injury simulation studies (**Table 1**).^{14,15}

The human body is primarily composed of water, with muscles being approximately 73% water in composition. A natural first step in terms of an equation of state would be to use the equation of state for water, with a modified viscosity to more accurately reflect the nature of the muscle tissue. Clemenson has noted that due to the high water content of most tissues, the low modulus of rigidity and the high bulk modulus, a fluid of high viscosity could reasonably approximate stress wave propagation behavior in tissue.¹⁶

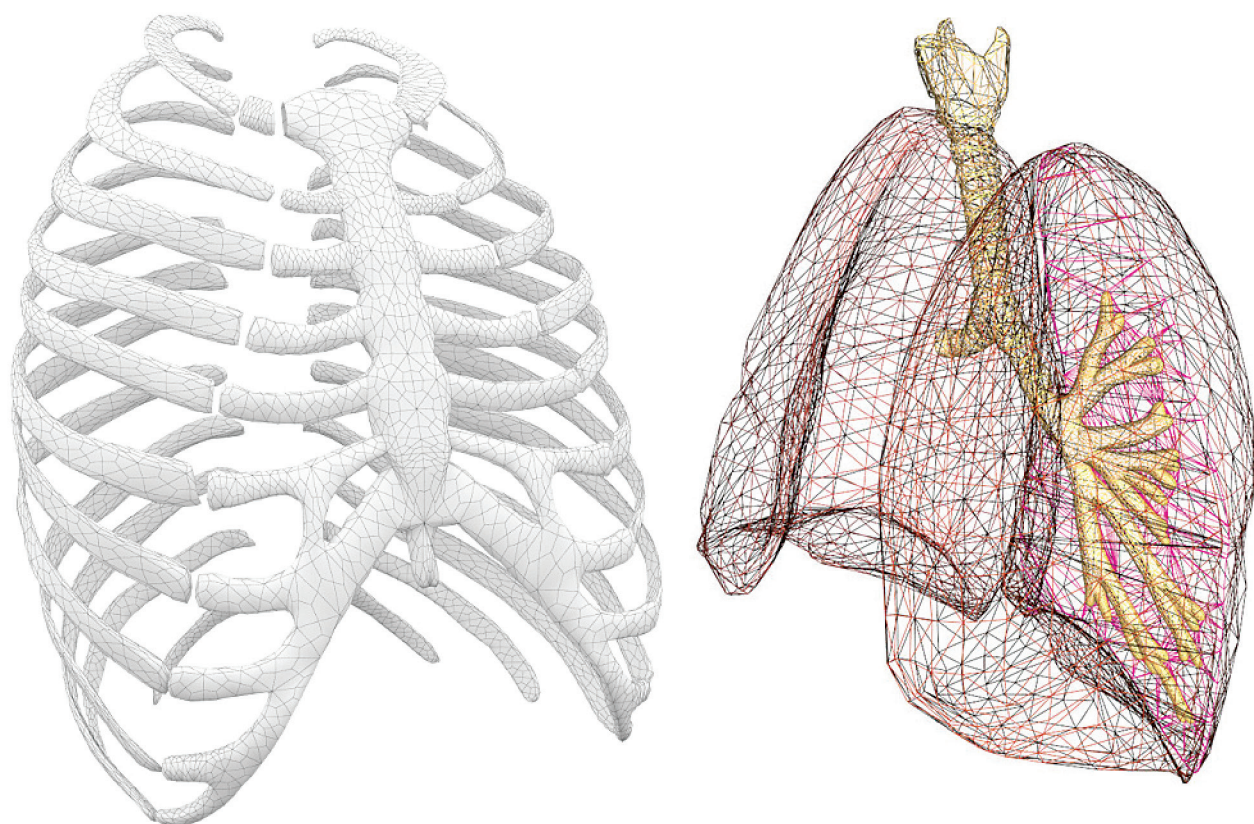


Figure 4. 3D human thorax finite element model.

Table 1. Thorax finite element model material characteristic parameters.

Material	Density (kg/m ³)	Young's Modulus (GPa)	Poisson's Ratio	Sound Speed (m/s)
Thorax bone	1561	7.92	0.379	3073
Soft tissue (fat and muscle)	1000	1.28	0.4	1500
Lung	2000	0.007	0.3	30

Key: GPa, The gigapascal is the unit of pressure (GPa=10⁹Pa=1GN/m²).

Air was modeled as a null material because this material model allows equations of state to be considered without computing deviatoric stresses, thereby providing a good representation of the behavior of a gas (Table 2).

Table 2. Material values for the material model and equation of state for air and ambient elements.

Material	Air
Density	1.293 kg/m ³
Pressure Cutoff,	-0.100 kPa
Dynamic Viscosity Coefficient,	0.0
Specific Heat Capacity at Constant Pressure	1005
Specific Heat Capacity at Constant Volume	718
Initial Temperature	270 K
Initial Relative Volume	1

Key: kPa, The kilopascal is the unit of pressure (kPa=1000Pa=1kN/m²).

For this experiment, computational fluid dynamics were performed assuming a rigid body in the flow field and the pressure was calculated on the entire body surface.

RESULTS

For this modeling, the initial blast overpressure was set as 50Mpa (created by a 0.5 kg Trinitrotoluene-equivalent explosive charge) and detonated at a distance of 1.8 meters of the target. The front surface of the thorax faced toward the blast emission source. Figure 5 presents the pressure contours on the surface of the rigid body at t = 0.002s from the detonation instant.

Primary blast injuries were characterized by anatomical and physiological changes from the force generated by the blast wave impacting the body's surface, and

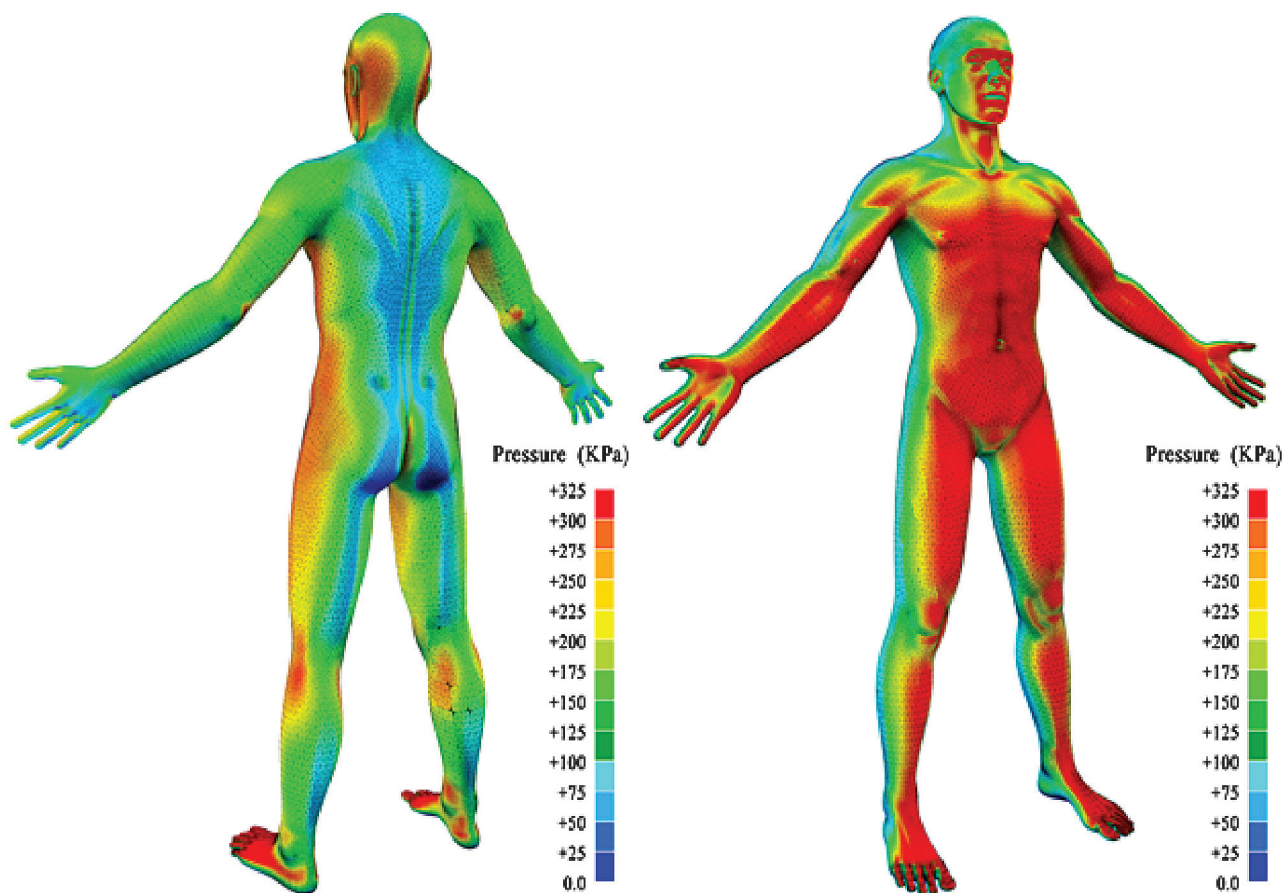


Figure 5. The pressure contours on the surface of the rigid body at t = 0.002s from the detonation instant.

affecting primarily gas-containing structures (lungs, gastrointestinal tract and ears). Exposure to significant blast loading can cause pleural rupture and tearing of the lung that creates a passageway between the lung and pleural space. Because the lung is an elastic material under tension, the vacuum in the pleural space is required to keep it inflated. Major rupture of the pleura can decrease the intra-thoracic pressure and lead to lung collapse.^{17,18} **Figure 6** presents the von Mises equivalent stress distribution within the thorax.

The calculation of the mean value of the peak lung pressures allowed for an overall estimate of the injury level, with 35 kPa predicting threshold damage, 129 kPa for 1% lethality and 186 kPa for 50% lethality.

The simulation showed that the peak front thoracic surface overpressure approximately doubled the incident overpressure. The peak overpressures on the left- and right-side thoracic surfaces were similar to the incident overpressure, and the peak overpressure on the rear surface of the thorax was approximately half of the incident overpressure. Inside of the thorax, the lower lobe of the lung experienced the longest stress duration. It is generally recognized that blast injuries that result in rupture of the lung and high risk of lung emboli follow sudden pressure increases up to at 15 psi (100 kPa).

DISCUSSION

After blast loading, a forward-propagated plane shock wave was generated. When the blast reached the surface of the thorax, because of impedance differences between the

body and air, a portion of the blast wave passed into the body, while the other portion was reflected and combined with the following blasts, leading to a stronger blast loading. When the incident pressure (P_i) wave impinges on a target that is not parallel to the direction of the wave's travel, it is reflected and reinforced, producing what is known as reflected pressure. The reflected pressure is always greater than the incident pressure at the same distance from the explosion. The reflected pressure varies with the angle and peak pressure of incidence of the shock wave (**Figure 7**).¹⁹

Blasts in enclosed areas (bunker, building, large vehicles) are associated with greater morbidity and mortality due to reflections and superposition of waves. When the blast wave impacts a barrier, the gas is further compressed and ultimately begins to travel back toward the incident wave, creating a region of intensely pressurized air. In addition to the enclosure, proximity to walls and corners will increase the risk of primary blast injury. Although it appears that orienting the body at an angle of 45 degrees provides the lowest injury. The drop in injury noted at 45 degrees as compared to the 0 and 90 degrees should be further investigated.

Comparing the required blast for threshold injury in different body orientations gives an idea of the effect that reflective pressure can have on injury. When lying end-on to the blast, a static pressure of 12 psi (83 kPa) is required for threshold injury, while side-on subjects would need 10 psi (69 kPa) and a subject against a reflector would only need 5 psi (34 kPa), which reflects to 12 psi. This simple

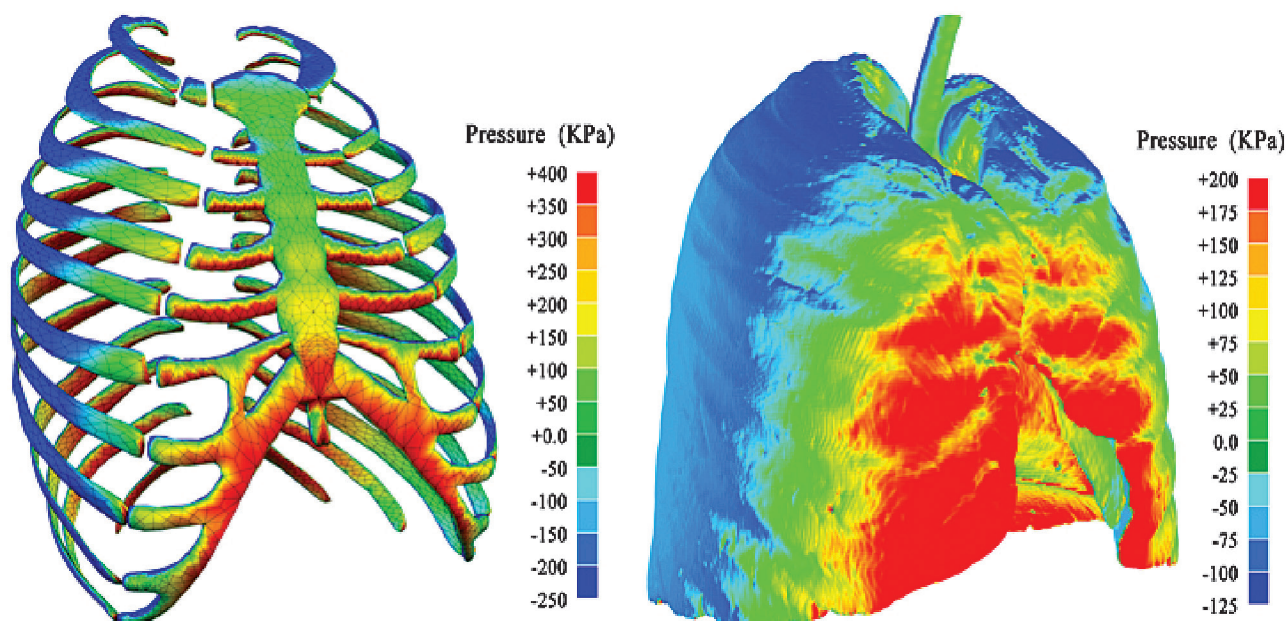


Figure 6. The von Mises equivalent stress distribution within the thorax.

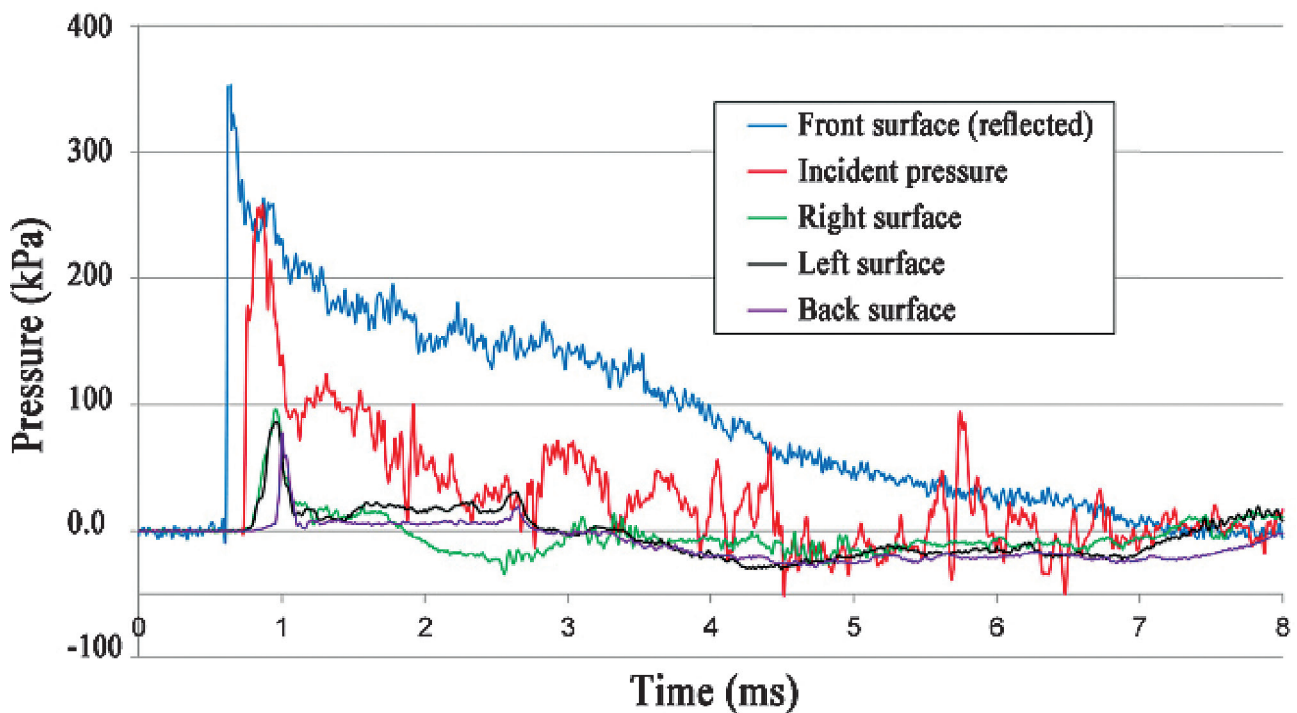


Figure 7. The incident and reflected pressure around the rigid body area following blast loading.

comparison shows that for different incident blast waves, the effective blast loading can be the same, depending on orientation and environment.²⁰ The simulations show when the body is oriented at an angle of 45 degrees the predicted blast lung injury is at a minimum.

The chest wall velocity was found to correlate well with the level of trauma sustained and compared well with the Bowen and colleagues curves²¹ for threshold lung damage. Using the chest velocity-pressure wave relationship, Stuhmiller and colleagues⁵ constructed the formula for the amount of energy delivered to the lung, assuming that the thoracic response to blast is dominated by inertia and external pressure loading, p_{load} . By ignoring the stress in the rib structure and internal wave reflections he could write in general Newton's second law as:

$$m \frac{dv}{dt} = P_{load}(t) - P_0 \cdot \left(1 + \frac{1}{2}(\gamma - 1) \frac{v}{c_0}\right)^{\frac{2\gamma}{\gamma-1}} - \frac{P_0 L}{L - x}$$

Where v is the chest wall velocity, x is the displacement, m is the chest mass per unit wall area, and L is the lung volume per unit of chest wall area. Having p_{load} one can calculate the chest wall speed v .

Associating the adjusted severity of injury index (ASII) with data from Axelsson and Yelverton,²² inward chest wall velocities (V) from the model can be directly used to predict injury levels (Table 3). Using mathematical model along with the experimental testing on sheep

Table 3. Injury as a function of peak inward chest wall velocity.²⁴

Injury Level	ASII	Chest wall Velocity (m/s)
No injury	0.0 – 0.2	0.0 – 3.6
Trace to slight	0.2 – 1.0	3.6 – 7.5
Slight to moderate	0.3 – 1.9	4.3 – 9.8
Moderate to extensive	1.0 – 7.1	7.5 – 16.9
>50% lethality	> 3.6	> 12.8

Key: ASII, Adjusted Severity of Injury Index.

also allowed for a simple mathematical correlation between the ASII and the inward chest wall velocity: $ASII = (0.124 + 0.117 V)^{2.63}$.

An underwater explosion is an explosion beneath the surface of water. The type of explosion may be chemical or nuclear. Water has a much higher density than air, which makes water harder to move and also relatively hard to compress. These two together make water an excellent conductor of shock waves from an explosion. Studies have shown that sections of the body at greater depths show much greater injury, which also explains the predominance of gastrointestinal injuries over lung injuries in victims submerged vertically in water. Underwater blasts are more lethal due to the higher density of water, the higher speed of the wave front and the improved coupling of the stress wave to the body.

It has also been found that repeated blast exposure at relatively low levels of pressure causes injury. This is of particular interest for safe operation of munitions and

workplace safety. Experimentation has shown that there is a marked difference between the overpressure required for severe injury due to one blast, as opposed to twenty blast exposures.⁵ A study was conducted using sheep and swine exposed to blasts at one minute intervals in order to examine the effect of multiple blasts on injury. It was found that a given blast that resulted in 1% mortality when delivered only once, produced 20% mortality when delivered twice and 100% when delivered three times.²⁰

CONCLUSION

This study developed three-dimensional finite element models for the human thorax based on real human body data and carried out numerical simulations on the pressure fields surrounding the chest area and the stress fields inside the thorax under blast loading. Trauma evaluation for the numerical models was focused on pressure within the lung.

Auditory, respiratory and digestive system blast injury correlated to many parameters including: peak overpressure of the incident blast wave (an overpressure of 60-80 psi or 414-552 kPa is considered potentially lethal), positive impulse, distance from the incident blast wave and the frequency content of the input. The orientation of the human body relative to the blast wave and any reflecting surfaces also has a profound effect on the level of injury because geometry of the person and environment strongly influence the blast field and pressure impacting the various areas of the body.

To further improve the performance of the model, it is suggested that deviatoric strength be added to the lung model along with a frictionless contact definition at the lung/rib interface. This change would permit the accurate modeling of long-term deformation in the lung. Since computer performance increases, a finer mesh and a truly three-dimensional model of the torso would allow a more in-depth study of the complex wave interactions that cause injury within the lungs.

CONFLICTS OF INTEREST

None Declared.

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