Simulation of Shock Wave Propagation and Attenuation in the Lung: Criteria for Edema Formation

O.V. Manyuhina* and A.I. D'yachenko**

* Institute for Molecules and Materials, Radboud University Nijmegen, Toernooiveld 1, 6525 ED Nijmegen, The Netherlands
** General Physics Institute of the Russian Academy of Sciences, Vavilova 38, 117942 Moscow, Russia

dyachenk@kapella.gpi.ru

Abstract: We study the physical parameters responsible for edema formation in a rabbit lung due to impact load. For simulation we use the model of shock wave propagation developed in [1], where pulmonary parenchyma is presented as continuum homogeneous medium with absorption and distortion. Three types of biomechanically reasonable criteria are introduced to analyze primary blast lung injury. We focuse on calculation of spatial averages over temporal maxima/minima values of the following stress wave parameters: pressures, gradients of pressure, stresses in the structural elements, their gradients — for rigid and free boundary conditions. Average overpressure and underpressure explain edema formation in a rabbit lung in the best way. However, the role of underpressure can be eliminated by increase of parenchyma viscosity which is not well-defined. Experimental data can be explained in terms of different types of criteria. But to make unambiguous choice of physical parameter one needs experiments with variety of shock wave amplitudes, durations and boundary conditions.

Introduction

A few types of lung injury: hemorrhage, rapture of parenchyma and edema occur after blast. Most authors from different groups conlude that stress wave propagation in the lung is the main process involved in the injury. However, to understand the mechanism of lung trauma one needs physical parameter i.e. criterion responsible for its formation due to impact load. Nowadays there is no unique view on this problem and reserchers propose different injury criteria as one or another parameter of the stress wave or movement of the chest wall associated with lung injury. These parameters include:

- the maximal deformation velocity of the chest wall [2],
- the maximum product of local deformation velocity of the chest wall and its relative deformation (the parameter viscous response) [3],
- peak acceleration of the thoracic wall [4],
- tensile stresses or strains in the alveolar walls [5, 6],
- pressure gradient in pulmonary parenchyma [7, 8],
- and others.

Considering proposed mechanisms and criteria we should make a few notes:

(1) In all these suggestions, the basic item is the idea of destructive effect of deformation wave in parenchyma. The specified criteria (i.e. wave parameters responsible) for hemorrhage, edema and rupture are not determined yet. One can suppose that different injuries are caused by different physical wave parameters (deformations, stresses, velocity, released energy or their combination).
(2) Some criteria refer to chest wall deformation rather than to deformation wave in parenchyma. This is because there are techniques to measure chest wall deformation but there is no one to measure the deformation wave in parenchyma.
(3) Until now no paper could demonstrate an injury criterion without ambiguity.
(4) Three space-time types of criteria are reasonable. We consider these types of criteria on an example of one kind of injury — edema, also the same types of criteria are reasonable for other injuries.
   (a) An injury is associated with a maximal value of a parameter reached in any point of the lung parenchyma at any moment of the wave propagation; i.e. a general lung injury is proportional or linearly proportional to a maximal local value of the parameter. This type of criteria can be valid if an injury of the whole lung is initiated by a maximal local damage such as local holes in alveolar walls. In this context term "local" means dimensions much less then the wavelength. As soon as we consider short pulses ~ 0.3 ms, for wave speed ~ 20 m/s a wavelength is about 0.6 cm.
   (b) An injury is associated with an integral of maximal values of a parameter reached in all the points of the lung parenchyma at any moment of the time. It happens if a local edema after blast is produced by a fluid leakage through a local microscopic holes emerged at the moment

ISSN: 1727-1983

of time when maximal value of parameter is reached. The total injury of the lung (for example — a rate of edema formation in lung) should be characterized by an integral of this maximal value of parameter over the volume of pulmonary parenchyma.

(c) An injury is associated with a space-time integral of a parameter in all points of the lung parenchyma at all moments of the wave propagation. In fact, this type of criterion was considered by [9], who offered the energy criterion: an injury is associated with mechanical energy dissipation in the lung. The energy is an integral of the local dissipated power over time and lung volume. Other mechanisms can lead to this type of criterion. For example, if local microscopic holes are produced by a prolonged process of stretching of alveolar walls above a threshold, then the total injury of the lung should be characterized by an integral of stress/strain over time above threshold and over the volume of pulmonary parenchyma.

(5) One can consider different types of criteria that are based on the same parameter of deformation wave in pulmonary parenchyma and the same kind of injury. Relationship between criteria of types (a) and (b) depends upon a distribution of the parameter in the lung. If the mean value of parameter in the lung is proportional to its maximal value, then both types of criteria are equivalent for this parameter.

The purpose of our work is: 1) to study correlations between different physical wave parameters and one kind of lung injury — edema in terms of type (b) criteria; 2) to compare results with criteria of type (a) that we analyzed in [1].

Methods

In the present study we use the model developed in [1]. This model is based on the presentation of lung parenchyma as foam-like four-phases continuum and homogeneous medium [10]. The constitutive equations of mass and impulse balance may be linearized for weak blast waves when $P_0 < 50$ kPa, $\tau < 10$ ms, where P_0 is a peak pressure amplitude, 2τ is duration of a triangular pulse [1]. To satisfy the condition of continuity, one should consider pulses with $\tau > 0.1$ ms. At $\tau \sim 0.1$ ms one may regard pulmomonary parenchyma both as continuum and as layered medium.

Assuming one-dimensional model we get equation for time evolution of the displacement u:

$$\rho \frac{\partial^2 u}{\partial t^2} = \left(K + \frac{4\mu}{3} + \frac{1}{\beta}\right)\frac{\partial^2 u}{\partial x^2} + \left(\xi + \frac{4\eta}{3}\right)\frac{\partial^3 u}{\partial x^2 \partial t}. \quad (1)$$

where K, μ are bulk compression and shear modules; η, ξ are shear and bulk coefficients of parenchymal viscosity; β is compressibility of parenchyma. Thus our model takes into consideration absorption and distortion of the wave profile governed by model parameters.

The front surface $x = 0$ is subjected to the triangular pulse with peak pressure amplitude P_0 and duration 2τ.

$$\sigma\big|_{x=0} = -P(t) = -\frac{P_0}{\tau}\begin{cases} t, & 0 \le t \le \tau, \\ 2\tau - t, & \tau < t \le 2\tau, \\ 0, & t > 2\tau. \end{cases} \quad (2)$$

The rear surface of the lung $x = L$ has two types of boundary conditions:

$$u\big|_{x=L} = 0, \quad \text{contact with absolutely rigid body;} \quad (3)$$

$$\sigma\big|_{x=L} = 0, \quad \text{free surface.} \quad (4)$$

The detailed solution of equation (1) with boudary conditions (2) and (3) is presented in [1]. In the next section we use its analytical form to calculate stresses and strains in the lung for two types of boundary conditions.

For our simulations we choose lung length $L = 5$ cm, density $\rho = 0.25$ g/cm^3. The model parameters $K = 74 \cdot 10^3$ g/(cm s^2) and $\mu = 6.2 \cdot 10^3$ g/(cm s^2) are taken from the work [11] with transpulmonary pressure $P_{tp} = 1$ kPa like in considered experiments. The coefficient of viscosity $(\xi + 4\eta/3) \sim 400 - 8000$ g/(cm s) at a frequency ~ 100 Hz and $\sim 40 - 800$ g/(cm s) at a frequency ~ 1000 Hz was assessed in the work [12]. They supposed that viscosity of pulmonary parenchyma times frequency is approximately constant as for some other soft tissues.

Results

Computer simulations were based on the set of experimental data presented by Yen et al. [6], where the rate of edema formation due to impact load was studied. In these experiments the excised rabbit lung was supported on a nylon screen (i. e. soft cloth) or on a rigid plate. The shock wave in the form of a triangular pulse with amplitude 20 kPa and duration 0.34 ms results in increase of lung weight by 40 % in the case of a freely supported lung, but it was more than 120 % in the case of a rigidly supported lung it was more than 120 %. The pressure distribution calculated from analytical equations is presented in figure 1 for two types of boundary conditions.

In figure 1 (a) the maximum underpressure (black color) and significant overpressure (white color) are reached on the surface of contact with a rigid plate $L = 5$ cm. While for free lungs (figure 1 (b)) the maximum and minimum pressures are located inside the lung parenchyma (dark and light ovals). If one accepts a hypothesis of lung trauma due to the tensile wave [5] then this result reveals different injury localizations for rigid and free boundary conditions.

The main goal of this study was to identify the physical wave parameter that is responsible for edema, in terms of criteria of (a) and (b) types (see introduction). It means that there should be the same dependencies between the value of physical criterionparameter and the

ISSN: 1727-1983

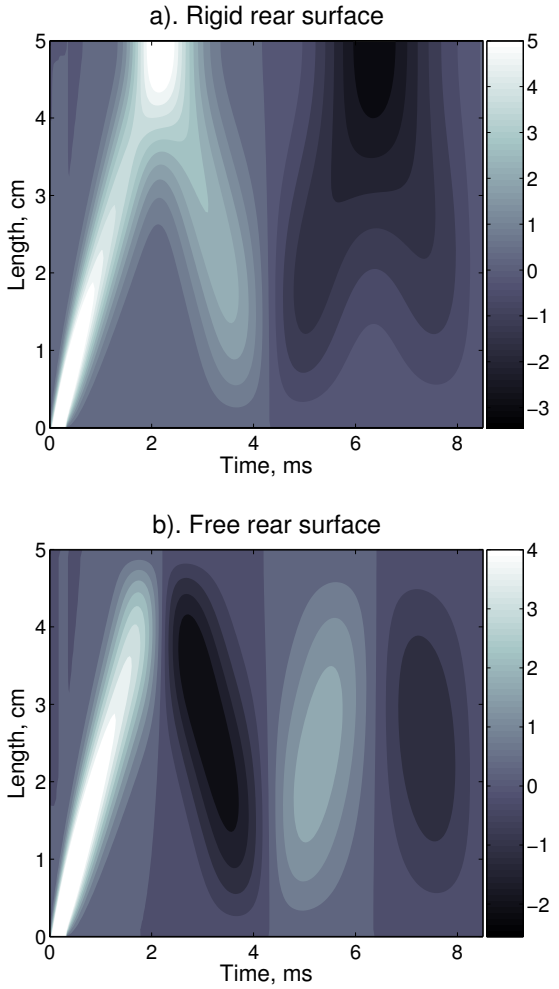

Figure 1: Pressure contour map for free and rigid rear surfaces while impact load with $P_0 = 20$ kPa and duration 0.34 ms. Values of pressure more than (a) 5 kPa and (b) 4 kPa are mapped with white color. The level step in color change is 0.5 kPa.

increase of lung mass for both types of boundary conditions. We have previously [1] considered the following wave parameters in terms of criteria of type (a): pressure in the medium P, strains $\partial u/\partial x$, velocity $\partial u/\partial t$, stresses in structural elements σ_f, energy dissipation, viscous criterion $\partial u/\partial t \cdot \partial u/\partial x$. It was shown that maximum underpressure could be considered as a physical parameter for edema formation in terms of type (a).

This result is closely related to a hypothesis of the mechanism for lung trauma [5]. According to this hypothesis the tensile wave passes after the compression wave. It induces a considerable stretching of alveolar walls, that leads to an increase of permeability of alveolar epithelium and even to its rupture. During compression wave propagation the small airways close and gas is trapped in alveoli. Then tensile wave passes and the airway reopening occurs with greater pressures than airway closing. There are two differences between our results and this hypothesis.

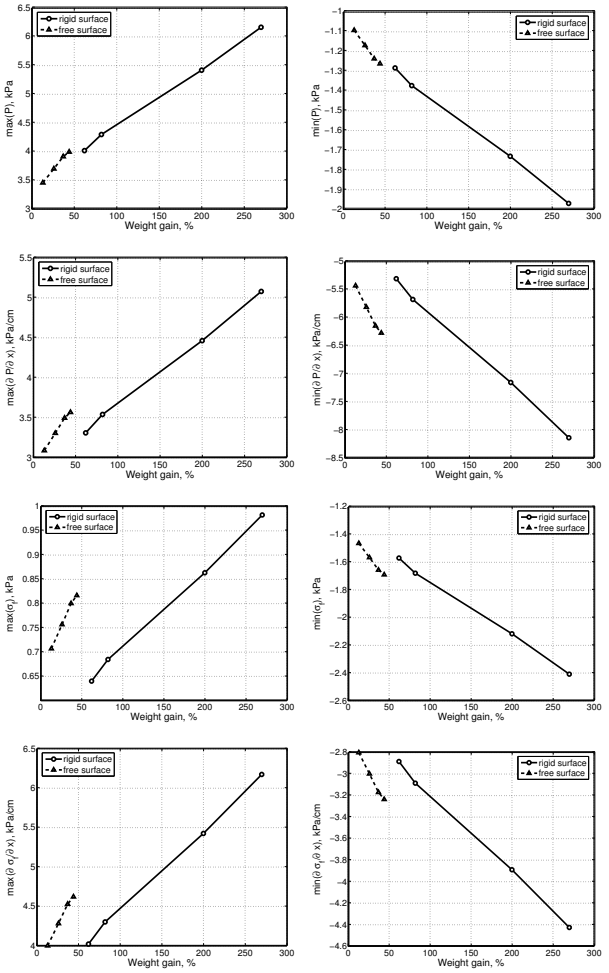

Figure 2: Studied wave parameters

(1) We observed that underpressure in the parenchyma forms a better criterion than tensile stress in structural elements of parenchyma (i.e. alveolar walls). Underpressure includes a drop in gas pressure and tensile stress.
(2) Underpressure forms a criterion even without a greater reopening pressures.

Based on our previous work [1] we suppose that stress wave plays more significant role in edema formation than strain wave. Here we are interested only in stresses and their gradients in pulmonary parenchyma. Eight non-scattered data points with shock wave pressure amplitude $P_0 = 15$–23 kPa for rigidly and freely supported lungs were chosen [6, 1]. Our assessments show that the linear model well describes stress waves of such amplitudes.

The response of lung to each impact load is analyzed in terms of criteria of type (b) for different wave parameters. The results are presented in figure 2. It turns out, that for two upper sketches: spatial averages over temporal maxima values of overpressure and underpressure, the curves for rigidly and freely supported lungs coincide well, i.e. can be presented by one smooth curve. In this case the dependence is linear, however it is not necessary

ISSN: 1727-1983

condition. We can conclude that two wave parameters average overpressure and underpressure can describe edema formation in terms of criteria (b) and our model.

We consider dynamics of rabbit lungs during 8 ms (50τ). For this time pressure and volume relaxation occur and no processes with long time relaxation remain. Therefore, we assume that high frequency harmonics in the spectrum of triangular pulse contribute more to the lung dynamics as well as for edema formation. The main frequency band in experiments [6] and simulation is about 1000–3000 Hz. According to mentioned assessments [12] and assuming the inverse frequency dependence of viscosity, $(\xi + 4\eta/3) \sim$ 20–400 g/(cm s) at 2000 Hz. For our calculations in figure 2 we've chosen the mean value of viscosity $(\xi + 4\eta/3) = 190$ g/(cm s).

According to our simulations the variation of viscosity term $(\xi + 4\eta/3)$ can drastically change the situation in choice of good criteria for edema formation. Shear viscosity η was measured only for low-frequency oscillations, bulk viscosity ξ was not measure at all and this is very sketchy parameter. For $(\xi + 4\eta/3) > 500$ g/(cm s) and $(\xi + 4\eta/3) < 150$ g/(cm s) the curves for underpressure show a big discrepancy for criterion of type (b). The criterion of type (a) is less sensitive to choice of $(\xi + 4\eta/3)$, but for $(\xi + 4\eta/3) > 500$ g/(cm s) there is no underpressure due to the strong attenuation of the wave and one needs another parameter to explain edema formation.

For our simulation we've chosen non-scattered part of experimental data. In order to explain data scattering of the experiment [6] in terms of considered criteria, we supposed that such irregularity is induced by physiological variety of rabbit lungs. We analyze the optional influence of variation in lung length and lung density on the values of considered parameters.

The 20 % increase in lung length leads to 10 % reduction in spatial average over temporal maxima underpressure and overpressure. The 20 % density increase leads to 8 % decrease in edema formation if spatial average over temporal minima of pressure in structural element is chosen as a criterion responsible for edema.

Discussion

There is a principle opportunity to differ strain and stress in a criterion of lung trauma because a lung does not obey Hook's law. Stress and strain are not related in a unique way. Due to viscosity of the lung parenchyma an increase of the pressure P_0 and a decrease of τ in a triangular pressure pulse will increase stress amplitude, but may keep strain amplitude unchanged. If an experimental score of injury is increasing in such circumstances one may consider that stress is a causal factor of lung injury. In this way the differential roles in lung injury of mechanical factors such as pressure, stress, deformation can be identified as soon as experimental study includes a wide variety of pressure pulses. Different boundary conditions give the same opportunity for the identification of a causal factor as a wide variety of pressure pulses. Stress is zero at free boundary and strain is zero at rigid boundary. In an experimental set of both boundary conditions stress and strain are not related in a unique way.

Dependence of injury score upon a causal factor should be the same in all sets of available experimental data. The corresponding curves should fit a smooth line, i.e. coincide well. We consider this as athe strong requirement main obligatory condition for a choice of a "good" criterion (figure 2 two upper sketches). Significant discrepancy of two branches of dependence for free and rigid conditions demonstrate "bad" criteria (figure 2 all other sketches). Linearity of dependence is an additional but non-obligatory condition requirement for a good criterion. Some injury scores like percentage of ruptures may have a nonlinear dependence upon a causal factor.

Unlike previously developed models [13, 14, 7] used for the study of lung injury, our model takes into account the attenuation of the wave amplitude due to the dissipation of energy in pulmonary parenchyma. We believe, that it can be more reliable for identification of a causal factor of blast lung injury.

In considered experiments with impact duration 0.34 ms the simulated attenuation length is about 3 cm (figure 1). Thus before reflection at the rear surface the wave amplitude is significantly damped. Average overpressure is three times higher than the absolute value of average underpressure (figure 2). In spite of this fact, the simulation reveals that both values can form criteria for edema formation. Similar results were received in experiments [15], where critical values for overpressure 240 kPa and underpressure -76.1 kPa correspond to LD50. They demonstrated that underpressure can be used to indicate the severity of lung injury, and this relationship can be expressed with received linear regression equation.

On one hand, it seems that underpressure should be the best criterion to explain edema formation, because it's role was justified in terms of criteria (a) and (b). On the other hand, unlike overpressure in criteria of type (b), it appeared to be sensible to variation in a poorly defined viscosity term. For $(\xi + 4\eta/3) > 500$ g/(cm s) the damping of compression wave is strong and there is no underpessure. Therefore, integral of maximum overpressure is the only apparent parameter to describe edema formation. Other candidates associated with criteria of type (b) could be wave parameters like minimum stresses in structural elements and maximum pressure gradients. They show small discrepancy for two types of boundary conditions. For further insight into the criteria of lung injury and the responsible physical parameters of stress wave one needs to compare simulation results and experimental data on lung trauma for shock waves with different amplitudes, durations and boundary conditions.

Conclusion

We've studied different criteria of edema formation phenomenon on the basis of recently developed model [1]

ISSN: 1727-1983

and experimental data of [6]. We've introduced three types of criteria where the same physical parameters compose damaging factors in three different ways. In this paper the spatial averages over temporal maxima/minima values of the following wave parameters were analyzed: total overpressure and underpressure, their gradients, pressure in the structural elements, and their gradients. The best criteria are based on the average values of overpressure and underpressure. The result depends on the choice of viscosity of lung parenchyma $(\xi + 4\eta/3)$. We've chosen the mean value of viscosity $(\xi + 4\eta/3) =$ 190 g/(cm s) at 2000 Hz, as frequency band about 1000–3000 Hz is the main in experiments [6]. We demonstrated that there is no unique criterion to describe available experimental data. Simulation revealed what characteristics of the lung should be measured and what types of experiments with shock waves should be performed to make an unambiguous choice of lung injury criterion.

Acknowledgements

A.I.D. gratefully acknowledges support from the Russian State Program of Support for Leading Scientific Schools project NSh1996.2003.1. O.V.M. would like to thank K.V. Zakharchenko for a critical remarks.

References

[1] D'YACHENKO, A.I. and MANYUHINA, O.V. Modeling of weak blast wave propagation in the lung. *J. Biomech.*, in press.

[2] JONSSON, A., CLEMEDSON, C.J., SUNDQVIST, A.B., and ARVEBO, E. Dynamic factors influencing the production of lung injury in rabbits subjected to blunt chest wall impact. *Aviat. Space Environ. Med.*, 50:325–337, 1979.

[3] HAFFE, T. The viscous criterion. *Bioscience*, 39:248–253, 1989.

[4] COOPER, G.J., PEARCE, B.P., SEDMAN, A.J., BUSH, I.S., and OAKLEY, C.W. Experimental evaluation of a rig to simulate the response of the thorax to blast loading. *J. Trauma*, 40:38–41, 1996.

[5] FUNG, Y.C., YEN, R.T., TAO, Z.L., and LIU, S.Q. Hypothesis on the mechanism of trauma of lung tissue subjected to impact load. *J. Biomech. Eng*, 110:50–56, 1988.

[6] YEN, R.T., FUNG, Y.C., and LIU, S.Q. Trauma of lung due to impact load. *J. Biomech.*, 21:745–753, 1988.

[7] GRIMAL, Q., WATZKY, A., and NAILI, S. A one-dimensional model for the propagation of transient pressure waves through the lung. *J. Biomech.*, 35:1081–1089, 2002.

[8] GRIMAL, Q., NAILI, S., and WATZKY, A. A high-frequency lung injury mechanism in blunt thoracic impact. *J. Biomech.*, 38:1247–1254, 2005.

[9] STUHMILLER, J.H. Biological response to blast overpressure: a summary of modeling. *Toxicology*, 121:91–103, 1997.

[10] D'YACHENKO, A.I. and LYUBIMOV, G.A. Equations of pulmonary parenchyma dynamics, studying as a multiphase continuum. In *Fluid dynamics 2*, pages 241–258. Hemisphere Publishing Corporation, New-York, Washington, 1991.

[11] TEPPER, R.S., WIGGS, B., GUNST, S.J., and PARE, D.P. Comparison of the shear modulus of mature and immature rabbit lungs. *J. Appl. Physiol.*, 87:711–714, 1999.

[12] D'YACHENKO, A.I. and LYUBIMOV, G.A. Propagation of sound in pulmonary parenchyma. In *Fluid dynamics*, pages 641–652. Plenum Publishing Corporation, New-York, Washington, 1988.

[13] JAHED, M., LAI-FOOK, S.J., PRAMODE, K.B., and STEVE, S.K. Propagation of stress waves in inflated sheep lungs. *J. Appl. Physiol*, 66:2675–2680, 1989.

[14] STUHMILLER, J.H., HO, K.H., VANDER, VORST, M.J, DODD, K.T., FITZPATRICK, T., and MAYORGA, M. A model of blast overpressure injury to the lung. *J. Biomech.*, 29:227–234, 1996.

[15] ZHANG, J., WANG, Z., LENG, H., and YANG, Z. Studies on lung injuries caused by blast underpressure. *J. Trauma*, 40(3S):77S–80S, 1996.