Biomechanical Model for Pressure Transmission into the Lung, with Application to Blast Injury and Blunt Thoracic Impact

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Abstract

The intrathoracic pressures produced by exposure to air or underwater blast waves of short duration are predicted using a simplified one-dimensional model. The theoretical model is then used to explore the protective effects of various added layers worn as jackets. Predictions are compared with results from a laboratory simulation model based on a water-filled shock tube.

1. Introduction

1.1 Background and motivation

Air blast is a well known hazard to military personnel and bomb disposal crews, and was studied experimentally by Zuckerman during World War II [1]. It can result in extensive lung haemorrhage and the mechanism involves the transmission of pressure waves through the chest wall. Based on the work of Penney et al [2,3] and Jönsson [4], it appears that the detailed damage mechanisms depend on the blast duration. In short-duration blasts (overpressure lasting less than 20 ms), which are the subject of the present paper, the important physical properties of the pressure transmitted into the lung are its peak value P_{max} and its maximum rate of change $(dP/dt)_{max}$. The waveform impulse $\int P\,dt$, which is a measure of momentum transfer, becomes important at longer durations where resonance of the thorax is involved [4].

The same mechanisms of lung damage are expected to operate when divers are exposed to underwater blast waves, caused for example by dredging or demolition work. In either case it would be useful to be able to provide a protective jacket or vest which would minimise the transmitted pressure reaching the lungs. The main aim of this paper is to show how the effectiveness of such a protective layer can be estimated from a simple acoustical model.

1.2 Outline of the present paper

The theoretical model and its predictions are summarised in section 2. It is shown that some types of protective layer actually increase the potential for air blast injury, as measured by P_{max} or $(dP/dt)_{max}$ inside the lung. This is consistent with the experimental findings of Clemedson et al [5], Young et al [6] and Phillips et al [7]. No such detrimental effect is predicted for water blast injury; on the contract, a low-impedance (e.g. air-filled) protective layer is predicted to be highly effective in reducing P_{max} and $(dP/dt)_{max}$ in the lung. Such medical evidence as exists on this point [8,9] supports the prediction.

Section 3 describes a laboratory experiment set up to test the theoretical predictions. A water-filled shock tube was used to send an impulsive blast wave into a simulated lung sample (air filled polyether foam). Transmitted pressure waveforms measured in the foam were compared with the model predictions, but agreement was poor, probably because of the highly

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nonlinear foam compression at incident blast pressures of order 250 bar. The addition of a foam protective layer in the experiment produces no significant change in the transmitted P_{max} value, in contrast to the linear model prediction. This is because the unprotected-chest simulation was already producing as much attenuation, at the measurement point in the "lung", as was subsequently achieved with the additional protective layer in place.

Section 4 discusses the application of the model to other situations involving blunt impact. Finally, conclusions are summarised in section 5.

2. Theoretical Model of Pressure Wave Transmission into the Lung

Figure 1 shows the biomechanical model used. The entire model is one-dimensional; it represents a plane incident wave in the external fluid (air or water), transmitted at normal incidence through a protective layer and the chest wall, and entering the lung.

For greater generality the protective layer is allowed to have a composite structure: an incompressible layer on the outside, bonded to a compressible layer, (thickness L), on the inside. The chest wall is modelled simply as an incompressible layer, with a distributed stiffness (K per unit area) to represent the resilience of the rib cage, and with distributed mass M per unit area.

Typical values of the biomechanical parameters K and M were obtained from the literature [10]. Values of density and sound speed appropriate to lung parenchyma are discussed in Appendix 1. The whole system is modelled here as having a linear response to the blast input.

Although not explored here, the nonlinearity associated with pressure wave propagation in the lung is a potentially important aspect of the lung damage mechanism [2,3]. Waveforms predicted by the present model give the pressure-time history at the point of entry to the lung; their subsequent nonlinear evolution, and the actual damage mechanism, are subjects for further investigation.

2.1 Predicted pressure transmission coefficient

Since the system is treated as linear, it is simplest to calculate its behaviour in the frequency domain. Time-domain results can then be obtained by Fourier transformation.

The pressure transmission coefficient of the protective layer/chest wall combination is defined as

$$T_p(\omega) = \frac{\text{(complex pressure transmitted into lung)}}{\text{(complex pressure incident from outside)}}.$$

Its dependence on angular frequency ω is found by standard methods (Appendix 2) as

$$T_p = \frac{2}{(A \cos k_2 L + iB \sin k_2 L)}, \qquad (1)$$

where A and B are defined in the following section. All remaining symbols are defined in Appendix 3.

2.2 Introduction of dimensionless variables

We define dimensionless variables as follows:

$D_0 = M_A/M$ (dimensionless added mass in front of layer)
$D_1 = \omega M/r_1$ (dimensionless frequency)
$D_2 = r_1 L/Mc_2$ (dimensionless layer thickness)

$D_3 = r_2/r_1$ (dimensionless characteristic impedance of layer)

$D_4 = r_3/r_1$ (dimensionless characteristic impedance of lung parenchyma)

$D_5 = KM/r_1^2$ (dimensionless chest wall stiffness).

Note these are arranged so that the frequency appears only in one term (D_1). Then

$$A = \frac{i}{D_4}(D_0D_1 + D_1 - D_5/D_1) + \frac{1}{D_4} + 1, \tag{2}$$

$$B = \frac{(iD_0D_1 + 1)}{D_3}\left\{1 + \frac{i}{D_4}(D_1 - D_5/D_1)\right\} + \frac{D_3}{D_4}, \tag{3}$$

$$k_2L = D_1D_2 \ . \tag{4}$$

Equations 1 to 4 yield the transmission coefficient $T_p(\omega)$ as a function of dimensionless frequency, $T_p(D_1)$, with 5 (or 4) parameters determining the frequency response. These are: D_0 (if there is an added mass layer in front of the protective material), plus D_2-D_5.

2.3 Results for transmitted-pressure impulse response in air

An equivalent time-domain description of pressure wave transmission into the lung is given by the impulse response function h(t), which represents the transmitted pressure waveform caused by a delta-function pressure waveform incident on the layer. Given $T_p(\omega)$, the impulse response h(t) is obtained by inverse Fourier transformation.

Sample results for h(t) are presented in Figures 2-5. They correspond physically to the transmitted waveform when the incident pulse is of very short duration. The response to an actual incident pulse $P_i(t)$ will differ in shape from h(t), to the extent that the time scale of $P_i(t)$ is not short compared with that of h(t). The parameters D_4 and D_5 are here given values appropriate to the air blast case.

A comparison of Figures 2 and 3 (impulse responses without and with a protective layer) shows that in air, a protective layer can make an incident blast wave *more* hazardous. This has been observed in practice, in terms of increased intrathoracic pressure, for some ballistic jackets as worn by artillery and bomb disposal crews [6,7]. In Figure 3 the parameters D_2 and D_3, representing layer thickness and layer impedance, are varied while the parameters D_4 (= 14.6) and D_5 (= 0.53) are held constant at their values in Figure 2. Systematic variation of D_2 and D_3 shows that a *maximum* value of peak transmitted pressure occurs in the region of

$$D_2D_4 = 1 \ , \ D_3/D_4 = 1 \tag{5}$$

The value of P_{max} in this worst case reaches about 600 units, as compared with 285 units for the unprotected chest. The peak transmitted pressure is quite insensitive to D_2 and D_3 in this region, so that varying these parameters by a factor 3 either side of the worst case has little influence on P_{max}.

Figure 4 shows the first 50 ms of the impulse response; the successive spikes represent multiple reflections in the layer, and are spaced at intervals $2L/c_2$. This is the round-trip transit time of pressure waves within the protective layer.

2.4 Effect of biomechanical parameters on P_{max} for air blast

Varying the chest wall stiffness parameter D_5 has negligible effect on the transmitted peak pressure, as was found by repeating Figure 2 with double the value of D_5. This is to be expected, since at typical D_5 values for air (around 1/2), the natural period of oscillation of the chest wall is far longer than the time scale of the transmitted pressure impulse (around 0.2 ms).

Varying the chest wall mass per unit area M, however, has a large effect on P_{max}. When M is doubled, P_{max} for the unprotected chest is reduced to about half its previous value.

2.5 Effect of an added mass layer on air blast transmission

A composite protective layer, with an incompressible mass layer added in front of the compressible layer (thickness L) discussed in the previous sections, can greatly reduce the transmitted P_{max} and $(dP/dt)_{max}$. This is demonstrated in Figure 5. Here $D_2 = 0.15$ and $D_3 = 14.6$, while D_4 and D_5 retain the values used previously.

Without the added mass layer, Figure 3 shows that P_{max} for this case is close to its maximum value, around 570 units. Adding a mass layer equivalent to the chest wall ($D_0=1$) brings P_{max} below the unprotected-chest value. Further inceases in D_0 cause further reductions, both in P_{max} and $(dP/dt)_{max}$, as can be seen in Figure 5.

2.6 Pressure transmission into the lung from underwater blast waves

Because water has a much higher characteristic impedance than air, quite different transmitted pressures in the lung are predicted for *underwater* blast waves compared with air, even when the incident pressure waveform is the same. For the unprotected chest, using a unit-impulse incident pressure as previously gives $P_{max} = 31.5$ units underwater, compared with $P_{max} = 285$ units in air. The lower peak transmitted pressure is due to the low chest-wall and lung impedances compared with water.

Note that underwater blast pressures can be much higher than in air, however, so the factor of 9 advantage in transmitted-pressure reduction underwater does not mean that underwater blast exposure is less dangerous.*

2.7 Protective effect of a compressible layer in underwater blast exposure

A similar parametric study to that described in section 2.3 was carried out for the underwater case, by varying D_2 and D_3 while holding D_4 ($= 4.10^{-3}$) and D_5 ($= 0.04.10^{-6}$) constant. This showed that a simple compressible layer could be very effective in reducing P_{max} from its unprotected-chest value. Generally speaking, the greatest reductions occurred with low impedance layers ($D_3 << 1$) of moderately large thickness (D_2D_4 of order 1). The best result obtained was

$P_{max} = 0.18$ units (factor 0.006 lower than with no protection).

It corresponds to

$$D_2D_4 = 2.2, \quad D_3/D_4 = 0.1 .$$

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* In fact for the same energy release, pressures at any given distance scale approximately as ρc^2, which is about 16000 times higher for water than air.

This D_3/D_4 value is the lowest for which results are available, and may not represent an optimum. It implies that the layer has a characteristic impedance comparable with that of air at atmospheric pressure.

3. Shock Tube Experiment

3.1 Experimental arrangement

A water-filled shock tube was constructed to provide an experimental test of the one-dimensional theory at realistic blast pressures. Figure 6 shows the general arrangement of the shock tube, which was mounted vertically with the incident blast wave travelling upwards towards the test section. The lung and chest wall were simulated by a cylindrical plug of polyether foam faced with a 4 mm thick aluminium disk. A second foam plug was used to represent a protective compressible layer in front of the chest wall.

The foams available had characteristic impedances of 545-620 kg m^{-2} s^{-1} and internal sound speeds of 282-340 m s^{-1}. They were used dry, and for this purpose each sample was sealed in a polythene skin before installation. The tube was then filled with water to the base of the sample.

Pressure waveforms were measured with hydrophones, one in the protective foam layer (where present) and one in the "lung" (50 mm behind the "chest wall"). To generate the shock wave a 9 mm nitrocellulose blank cartridge was detonated at the base of the shock tube. The incident shock was typically of 250 bar peak pressure, and had an exponential tail with time constant 50-60 μs. A transient recorder was used to capture the pressure waveforms in the foam and in the water. The data was then transferred to an Amstrad PC for plotting and processing.

3.2 Results and comparison with theoretical model

Figure 7 shows pressure waveforms measured in the model lung and in the protective foam layer, for the following situations** :

(Lung)	Foam F4	$r_3 = 576$ kg m^{-2} s^{-1}	$c_3 = 300$ m s^{-1}
(Protective Layer)	Foam F6	$r_2 = 595$ kg m^{-2} s^{-1}	$c_2 = 282$ m s^{-1}

The incident shock had a peak pressure 255 bar; this is reduced to 0.62 bar at the measurement point in the protective layer, and to 0.14 bar in the model lung. A theoretical prediction, based on the model of section 2 and using the incident shock waveform as measured, gives P_{max} = 0.065 bar in the model lung. Figure 8 presents the predicted pressure waveform in the lung for comparison.

The lack of accurate agreement between measured and predicted waveform shapes in foam F4 (Figures 7 and 8) is probably due to the nonlinear behaviour of the foams under large impulsive pressure loading. Severe distortion and movement of the foam plugs were observed during the experiment. However, the prediction of P_{max} within a factor of 2 lends some support to the present simple model.

** The corresponding dimensionless parameters are: $D_2 \doteq 100$, $D_3 \doteq 4.10^{-4}$, $D_4 \doteq D_3$. Note that the characteristic time constant M/r_3 was 19 ms in the experimental simulation, compared with around 0.15 ms for the human thorax.

4. Blunt Impact Trauma

The theoretical model proposed here for blast-induced lung pressure waveforms also has applications in other areas. Falls into water represent a dynamically equivalent situation to the underwater blast case, and are known to cause lung haemorrhage at impact velocities of order 20 - 30 m s^{-1} [3,11]. Here the "incident" pressure waveform in the one-dimensional model would be replaced by an imposed external-pressure waveform, corresponding to the time-history of the impact pressure during water entry. At 20 m s^{-1} impact velocity, the pressure is expected to peak around 10 bar, with a rise time of 1 ms [12]. The necessary modification to the model is straightforward, but will not be discussed here.

In some road traffic collisions the thorax strikes a flat, non-penetrating object, and pressure waves will be transmitted into the lung in this case also. The same one-dimensional model can be applied in the early stages of impact to estimate intrathoracic pressure. The shape and amplitude of the resulting pressure waveform will depend on the mechanical impedance of the impacting object, as well as on the impact velocity.

5. Conclusions

The main conclusions from the study are summarised below. Further details may be found in reference [13].

(1) A simple one-dimensional prediction model has been devised for pressure wave transmission into the lung under blast exposure, both in air and underwater. The model allows the effects of various protective layers surrounding the thorax to be predicted.

(2) Based on the model, it is found that air blast penetration into the lung can actually be increased by an inappropriate design of protective layer. In particular, a compressible layer whose properties are close to the "worst case" defined by

r_2 = 6000 kg m^{-2} s^{-1} (impedance similar to lung parenchyma)
L/c_2 = 0.15 ms (acoustic transit time through layer)

produces 2 × the peak transmitted pressure of the unprotected case, when the incident pressure wave consists of a sharp impulse.

(3) A good protective layer designed for air blast attenuation consists of an incompressible mass layer on the outside, followed by a compressible low-impedance layer. The mass layer should have a mass per unit area several times that of the chest wall, i.e. at least 4 kg m^{-2}, for significant P_{max} reductions.

(4) A large reduction in underwater blast transmission is achievable by using a low-impedance compressible protective layer, with no added mass. A factor of 0.006 on P_{max} was predicted for the case

r_2 = 600 kg m^{-2} s^{-1} (impedance about twice that of air at atmospheric pressure)
L/c_2 = 0.33 ms (acoustic transit time through layer)

when the incident pressure wave consists of a sharp impulse.

(5) The same theoretical model can be used to predict intrathoracic pressure waveforms, and hence possible lung damage, during blunt thoracic impact.

(6) Limited experimental support for the predictions of the one-dimensional model has been obtained from a laboratory model based on a water-filled shock tube, using incident shock pressures of around 250 bar. The fact that severe distortion was observed in the plastic foam samples (used to simulate lung parenchyma and protective layers) suggests that a nonlinear model would have been better able to explain the transmitted waveform shape.

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APPENDIX 1

Effective density and sound speed in lung parenchyma

The lung normally consists of about 80% air and 20% tissue by volume. The tissue has approximately the same density and characteristics as water and can be regarded as incompressible. Hence the lung has an average density of

$$\rho = 200 \text{ kg m}^{-3} \qquad \text{(at 1 bar, 15}^{\circ}\text{C)}$$

which is due almost entirely to the tissue component.

The effective sound speed is estimated by treating the air in the lung as adiabatic, under the rapid compressions involved in blast or impact injury. The pressure-volume relationship for lung parenchyma is then

$$P(V - V_t)^{\gamma} = \text{constant},$$

where V is the total volume of unit mass of lung, and V_t is the volume of the tissue component. The index γ equals 1.40 for air.

Differentiating the adiabatic relationship to find $c^2 = -V^2(\partial P/\partial V)_{ad}$ gives the sound speed c as

$$c = V\left(\frac{\gamma P}{V-V_t}\right)^{1/2}, = 29.6 \text{ ms}^{-1} \text{ (at 1 bar, 15}^{\circ}\text{C)}. \qquad \text{(A1)}$$

APPENDIX 2

Calculation of pressure transmission coefficient from model

The model and notation are defined in Figure 1. Pressure waves are incident from the left onto mass layer M_A, with pressure P_i. A reflected wave P_r is sent back to the left, while on the other side of the mass layer, standing waves P_a and P_b are set up in the protective compressible layer (medium 2). Finally, a transmitted wave P_T emerges to the right, travelling into the lung parenchyma.

The pressure transmission coefficient, $T_p = P_T/P_i$, is then calculated using the following information:

- Each wave system travels at speed c appropriate to the medium, in the +x or -x direction.
- The characteristic impedance is $r = \rho c$, for each wave system.
- The velocity normal to the layer (v_A) is the same on both sides of mass layer M_A, and likewise for the chest wall layer, since both are regarded as incompressible.
- Transmission impedance of M_A is $i\omega M_A = Z_A$
- Transmission impedance of chest wall is $i(\omega M - K/\omega) = Z$.

From this information equations (A2) to (A5) are obtained:

$$P_i + P_r - P_{ao} - P_{bo} = Z_A v_A , \quad (A2)$$
$$v_A = (P_i - P_r)/r_1 = (P_{ao} - P_{bo})/r_2 \quad (A3)$$
$$P_{aL} + P_{bL} - P_T = vZ , \quad (A4)$$
$$v = (P_{aL} - P_{bL})/r_2 = P_T/r_3 . \quad (A5)$$

Here subscripts (0,L) on P_a and P_b indicate the values at x=0 and x=L (Fig. 1). These are related by

$$P_{aL}/P_{a0} = e^{-ik_2L} , \quad P_{bL}/P_{b0} = e^{ik_2L}$$

where $k_2 = \omega/c_2$.

APPENDIX 3

List of symbols

c_i (i=1 to 3)	sound speed in layer i
D_j (j=0 to 5)	dimensionless variables, sec. 2.2
h(t)	impulse response function, sec. 2.3
k_2	ω/c_2
K	effective stiffness per unit area of chest wall (taken as 10^5 Pa m^{-1})
L	thickness of protective layer
M	effective mass per unit area of chest wall (taken as 0.9 kg m^{-2})
M_A	added mass per unit area in front of protective layer
P_{max}	peak pressure in lung
r_i (i=1 to 3)	characteristic impedance (ρc) of layer i
T_p	pressure transmission coefficient, sec. 2.1
ρ_i (i=1 to 3)	density of layer i
ω	angular frequency

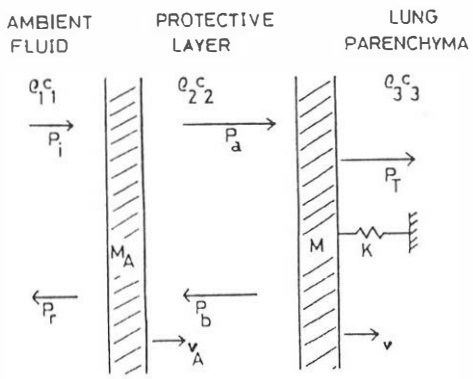


FIG 1 FLUID DYNAMIC MODEL OF PROTECTIVE LAYER CHEST WALL LUNG WITH ADDED MASS LAYER

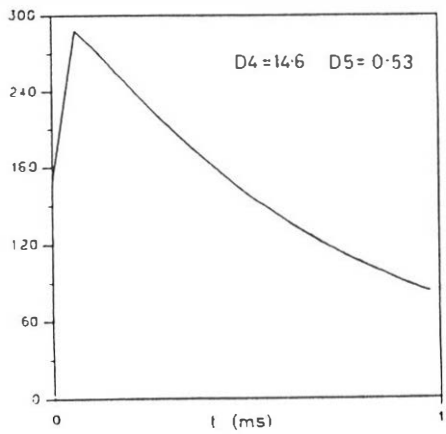
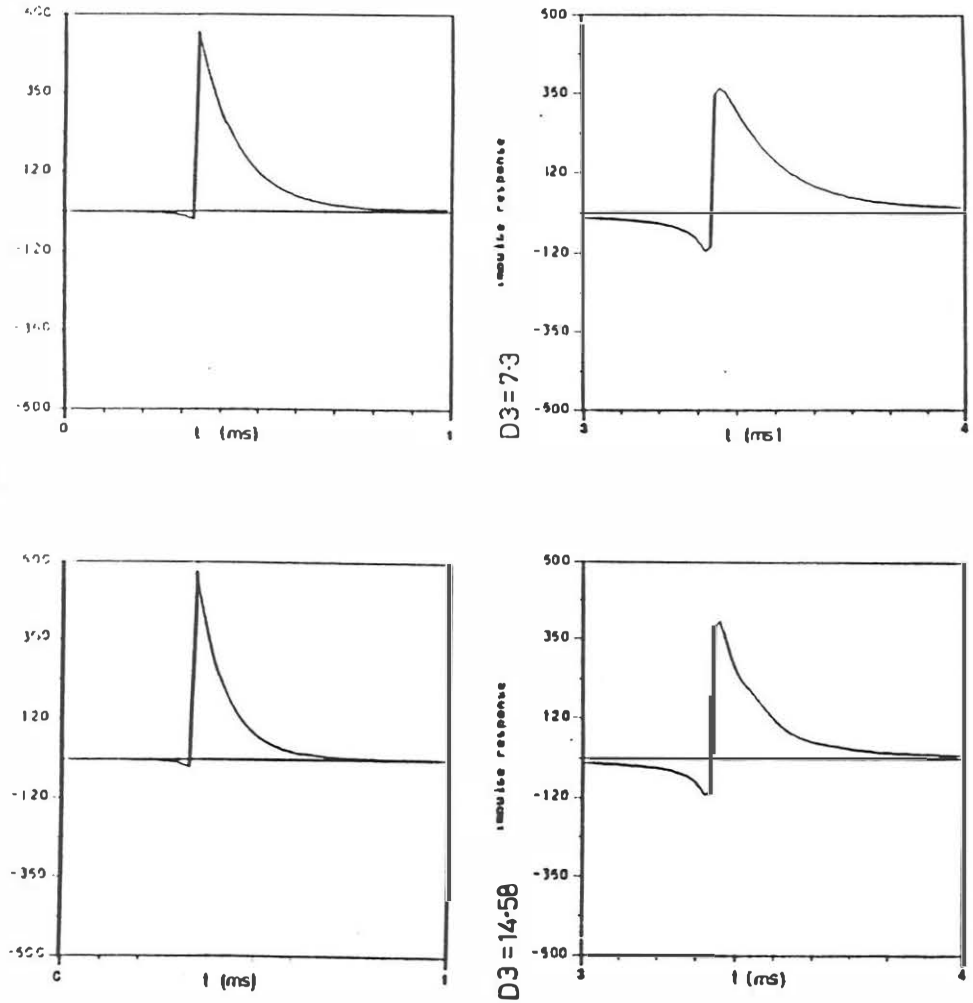


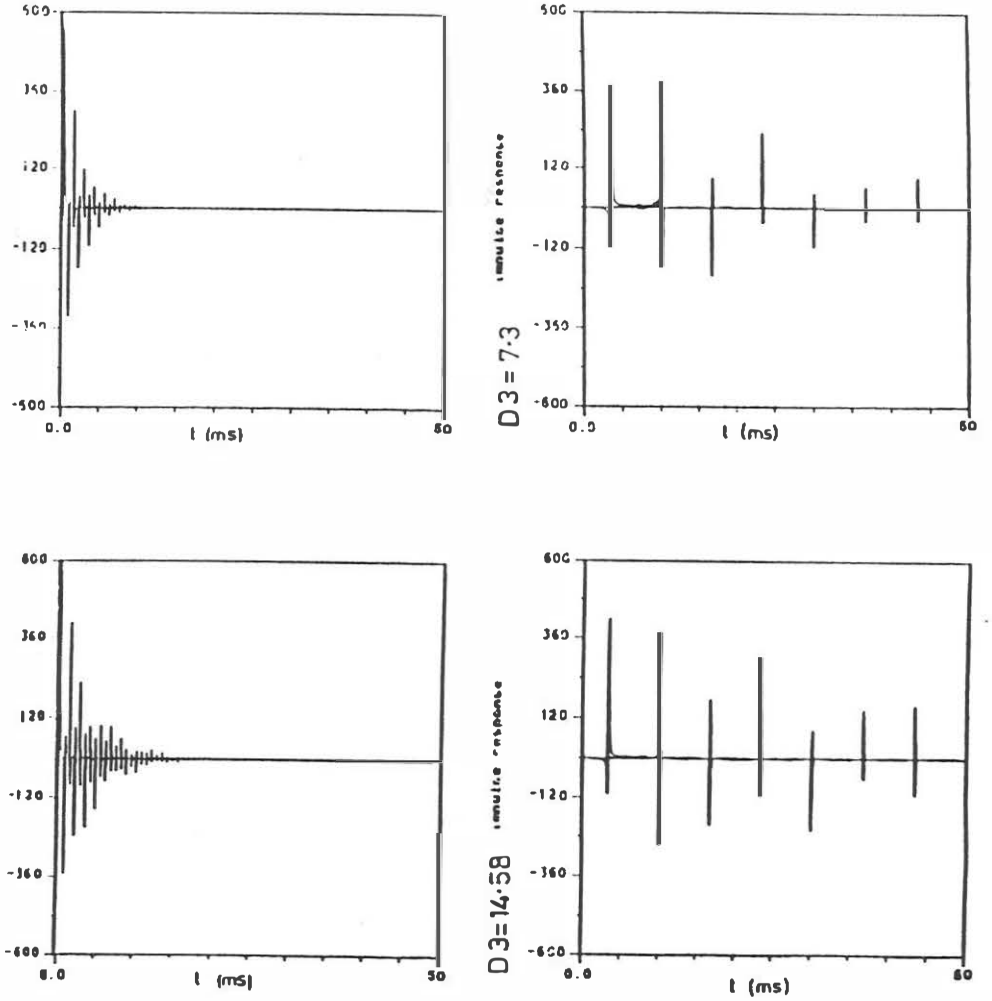
FIG 2 IMPULSE RESPONSE IN AIR, NO LAYER

FIG 3



Width of time base 1ms

FIG 4

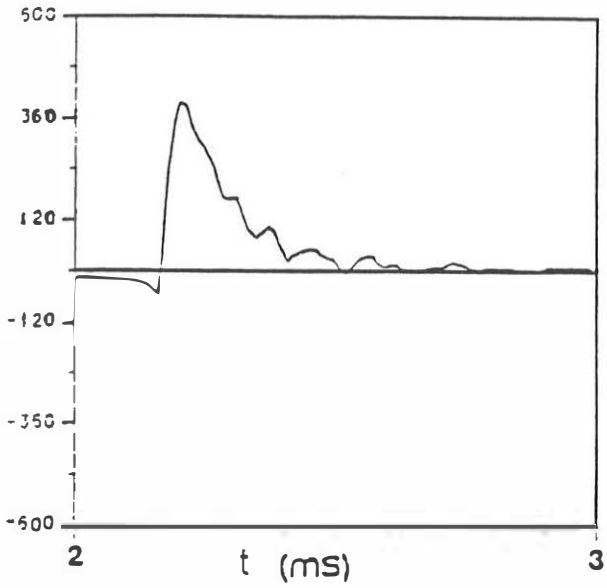


Width of time base 50ms

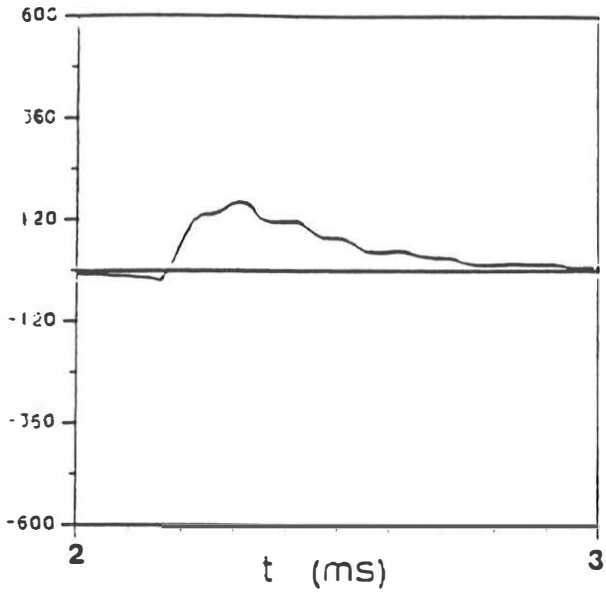
FIG 5

EFFECT OF ADDED MASS, M_A, IN FRONT OF FOAM LAYER (M = chest wall mass)

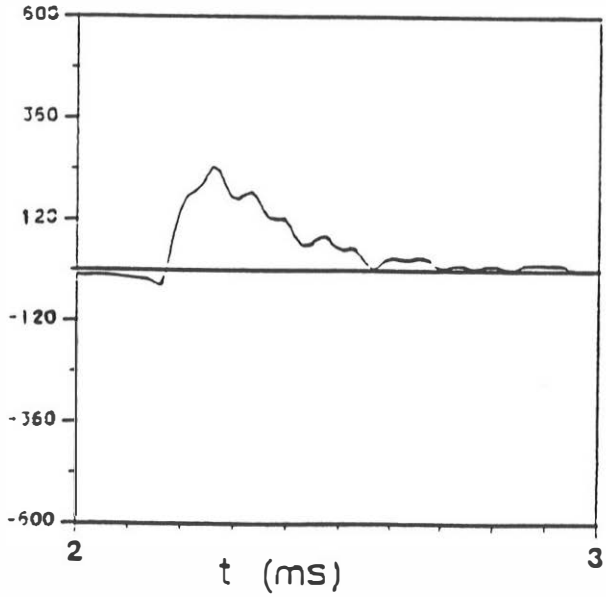
$M_A/M = 0{\cdot}5$



$M_A/M = 2{\cdot}0$



$M_A/M = 1{\cdot}0$



$M_A/M = 4{\cdot}0$

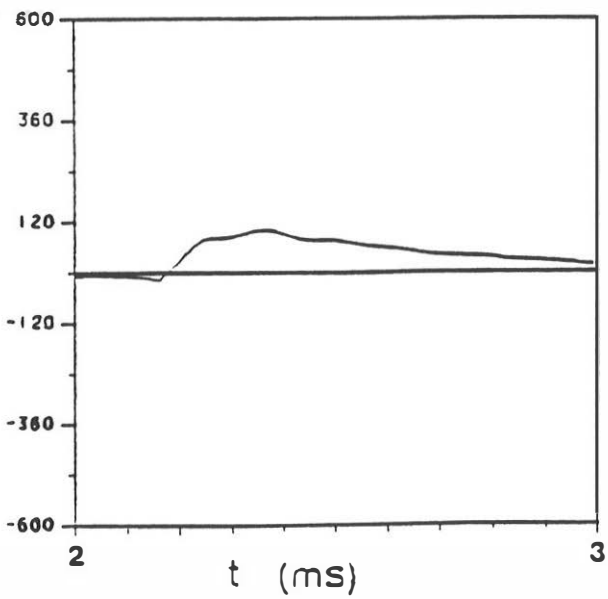


FIG 6 SHOCK TUBE EXPERIMENT

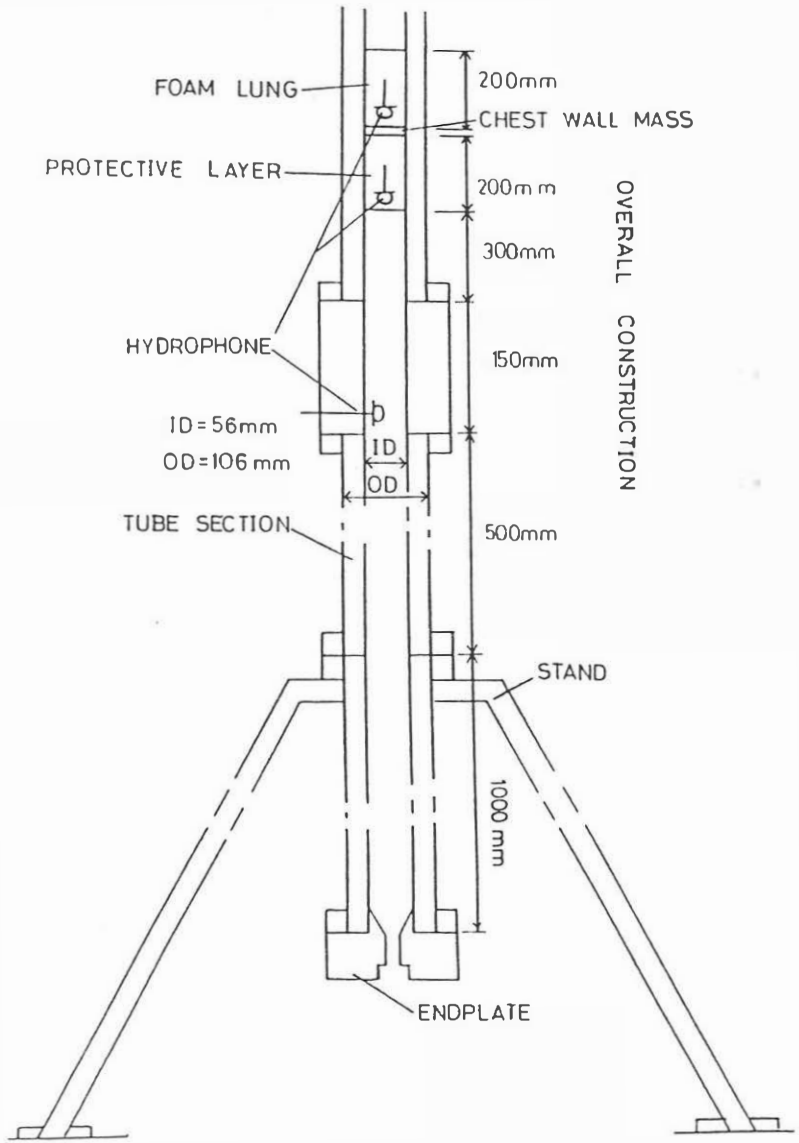


FIG 7 PRESSURE IN F4 AND PROTECTIVE LAYER F6. L = 200MM

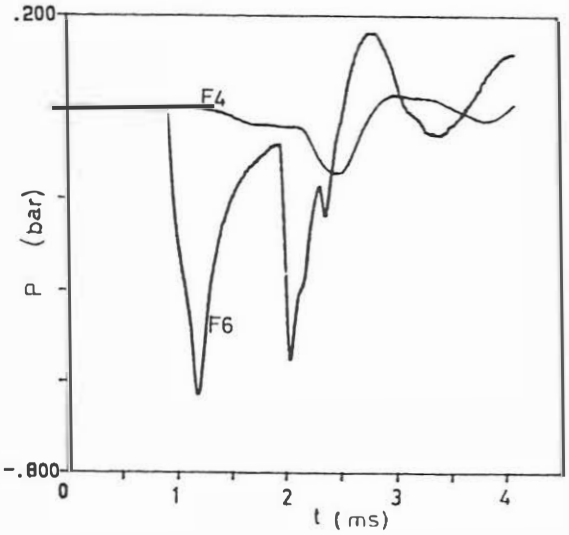


FIG 8 PREDICTED PRESSURE IN F4