AUGMENTATION BY FOAM MATERIALS OF LUNG INJURY PRODUCED BY BLAST WAVES - THE ROLE OF STRESS WAVES IN THORACIC VISCERAL INJURY AT HIGH RATES OF ENERGY TRANSFER

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ABSTRACT

Materials have been applied to the thoracic wall of anaesthetised experimental animals exposed to blast overpressure to investigate the coupling of direct stress waves into the thorax and the relative contribution of compressive stress waves and gross thoracic compression to lung injury. The ultimate purpose of the work is to develop effective personal protection to servicemen exposed to blast overpressure.

Foam materials acted as acoustic couplers and resulted in a significant augmentation of the visceral injury; decoupling and elimination of injury may be achieved by application of a high acoustic impedance layer on top of the foam. *In vitro* experiments studying stress wave transmission through the various layers showed a significant incease in power transmitted by the foams, the amplification occured principally at high frequencies. Material such as copper placed upon the foam achieved subtantial decoupling at high frequencies - low frequency transmission was largely unaffected.

The studies suggest that direct transmission of stress waves, and not gross thoracic compressions account for lung parenchymal injury with blast loading. Conventional impacts producing high body wall velocities will also lead to stress wave generation and transmission - if the body wall distortion is not severe, stress wave effects may dominate the visceral respose to the impact with direct compression and shear contributing little to the aetiology of the injury.

1. INTRODUCTION

The blast wave propagated in air following the detonation of high explosive may produce serious internal injury, often with no external indications of trauma. It has been recognised since WW1 that the principal sites of injury to viscera are the lungs and bowel; the injury produced within the parenchyma of the lung has attracted more attention because of the acute physiological consequences of injury to this organ.

The pulmonary injury in the acute phase is haemorrhagic contamination of the alveoli and airways (usually without gross parenchymal laceration) which may lead to establishment of a physiological shunt and thus a reduction of the partial pressure of oxygen in systemic arterial blood. The lung may continue to accumulate fluid over the ensuing hours and days leading to a self-reinforcing pulmonary oedema and to pulmonary infection. Arterial air emboli have also been reported in experimental animals

subjected to severe blast loadings, presumably originating from the pulmonary microvascular damage[10]. The pulmonary contusions produced by the impact of the blast wave are generally termed "blast lung" and at the microscopic level are generally indistinguishable from pulmonary contusion resulting from a non-penetrating impact; on a macroscopic level however, there may be differences in the distribution of contusions produced by the two types of loading.

1.1 Principles of injury

Although the features of an incident blast wave that determine the severity of primary lung contusions have been identified by experiment[1], the biophysical factors leading to the transfer of energy internally and the production of the lung injury following the interaction of the blast wave with the thoracic wall have not been as clearly defined. It was determined in WW2 that the pulmonary contusions originate from the impact of the blast wave upon the chest wall and that the injuries did not result from the passage of the blast wave down the trachea. The incidence of primary lung injury can be defined in terms of the peak incident overpressure, the duration of the incident blast wave, and geometric conditions such as orientation to the blast wave and the proximity to reflecting surfaces[1].

Any impact to the body will generate direct stress waves and shear waves[5]. The body is a very complex structure in which to initiate the propagation of waves - there are marked differences in density, elastic modulus and propagation velocities within thoracic tissues with complex geometric configurations; any sort of impact is likely to excite a broad spectrum of frequencies and stress concentration at multiple sites.

High-speed film of the chest under blast or under impact loading shows that the thoracic cage undergoes considerable compressive strain and exhibits a damped, viscoelastic behaviour. The gross distortion is a "low" frequency response of the thoracic wall taking around 2-3 ms to reach peak deformation in the rat with the blast loading used in the experiments described below; this interval under impact loading is largely governed by the mass of the impactor and the stiffness of the chest wall[4]. The compressive strain may achieve 0.4 - 0.5 and it is tempting to ascribe the resultant visceral injury to local shear or compression underneath the advancing body wall or to shear waves producing strain at sites of fixation such as the hilar region of the lung.

The measurement of the thoracic deformations under impact loadings is technically straightforward but displacements under blast loading are more difficult to make, primarily because of technical difficulties encountered in trying to measure small, fast deformations of a non-uniform surface under severe environmental conditions. Jonsson has made progress in this area and he concludes that gross thoracic deformation with corresponding compression of the lungs is the main origin of the pulmonary injuries[6,9]. Direct coupling of direct stress waves into the thoracic cavity is not considered to be a primary cause of injury[9].

1.2 Protective clothing

Of fundamental importance in the construction of protective clothing designed to attenuate primary blast effects, is the relative contributions of these low- and high-frequency components to lung injury. We define "high" frequency phenomena in this context as the direct coupling of the incident shock wave in air into the lung parenchyma, a process independent

of the gross thoracic compressions produced by the shock and dynamic pressure loadings arising from the blast wave. The direct stress transferred from an incident shock wave will be governed by impedance mismatches with the peak velocity achieved by the pleural surface of the body wall determining the magnitude of the peak stress and dP/dt_{max} transferred into the lung. The severity of parenchymal contusion is considered to be directly related to these parameters- finite element models have shown that transmission of the direct stress wave through the parenchyma leads to a pressure differential between the alveolar space and the capillary which may result in failure of the barrier[2].

The primary impetus for the work presented now has been a requirement to develop protective equipment for military personnel exposed to blast. During these investigations defining the severity of injury and its relationship to the magnitude of the stress transferred internally, insight upon the aetiology of non-penetrating injury at high rates of energy transfer resulting in high body wall velocities has been developed which is at present being applied to conventional impact injury produced by solid objects such as projectiles.

2. AIMS

The present study has used anaesthetised experimental animals and physical models to determine:

2.1. The effect of low- and high-acoustic impedance materials covering the thoracic cage either alone or in combination, upon the severity of primary lung injury produced by blast loading.

2.2 Changes in the intrathoracic stress produced by the coverings.

2.3 The transformation of the incident pressure wave both in time and frequency domain by interaction with these materials.

3. METHODS, MATERIALS AND BLAST PARAMETERS

3.1 Materials

The materials used as thoracic coverings and in experiments studying the transmission properties of these materials performed in vitro are shown in TABLE 1; the materials were used either singly or in combination. TABLE 1 also presents the areal densities of the materials; the mean (±SD) areal density of the rat lateral thoracic wall was 5.5±0.7 $kg.m^{-2}$.

TABLE 1 Materials used as thoracic coverings.

DESIGNATION	MATERIAL	THICKNESS mm	AREAL DENSITY $kg.m^{-2}$
FOAM R	Natural rubber foam	9.3	2.01
FOAM V	Viscoelastic open cell polyurethane silicone foam	10.3	0.84
COPPER	Copper sheet	0.58	5.23
KEVLAR	Kevlar 49 resin bonded composite	4.4	5.10

3.2 Blast loadings

All blast loadings were produced by a blast wave generator designed and constructed at CDE. Compressed air at approximately 11 MPa was directed by solenoid to a 0.55 mm thick aluminium bursting disc mounted in a 20 mm internal diameter nozzle. The severity of the loading was adjusted by positioning the target at various distances from the nozzle outlet. In the majority of exposures, the right lateral thorax of the unprotected rat or the exterior surface of the material placed over the rat thorax was positioned 45 mm from the outlet. At this distance, the mean (±SD) peak reflected overpressure was 584±108 kPa, mean overpressure duration 1.65±0.24 ms and positive phase impulse 183±25 kPa.ms (n=10).

3.3 Studies on experimental animals

Anaesthetised rats were exposed to a blast wave from the blast wave generator. The animals remained anaesthetised for 1h and those rats surviving the period were then killed. The degree of contamination of the lung with blood and oedema fluid was assessed quantitatively by calculation of the lung weight/body weight ratio (LWR). In each experiment group, the mean LWR of the animals exposed to blast (with or without coverings) was expressed as ratio of the LWR of a control, unexposed group of animals within the same experimental series. This ratio was designated the quotient of injury, Qi. Thus

$$Qi = (LWR_{\text{blast exposed animals}}) / (LWR_{\text{control animals}})$$

The severity of the lung injury was expressed as minor or uninjured for Qi<1.2, moderate for Qi = 1.2-1.5, severe for Qi = 1.5-1.9 and very severe for Qi>1.9. The majority of rats were exposed lying with their left flanks on a reflecting surface; the blast wave was directed 90 degrees to the plane of the surface onto their right lateral thoracic wall. Some rats were also exposed in free-field by suspending the anaesthetised animal in a thin flexible nylon net. In all experiments, blast loadings were applied to the right lateral thoracic wall.

Rats are more susceptible to blast overpressure than man; the effective mass and stiffness of the body wall is obviously different and injuries to rat lung described in this paper would be unlikely to occur in man at these overpressures. Rats have been used solely to address the general concepts of stress wave interaction with the body - experimental studies with a large animal model of body weight and thoracic wall stiffness closer to man (anaesthetised pigs, 30-60 kg) are continuing in parallel but are not reported in detail in this paper.

All materials were trimmed to cover the lateral thoracic cage only, ensuring that there were no significant increases in the presented area of the thorax resulting from application of the coverings. Preliminary experiments had shown that total enclosure of the whole rat torso with FOAM R significantly increased the severity of blast contusions from a Qi of 1.42±0.06 (mean±SE) in bare rats to a Qi of 2.59±0.16, with a resultant increase in mortality from zero to 5/11. The enveloping foam had resulted in a substantial increase in presented area of the target; Young et al.[13] have claimed that the augmentation of lung injury by bulky materials probably arises from the increase in the presented area, thus increasing the blast energy received by the target. This explanation is contentious, but in order to ensure that this possible effect did not arise in the current experiments, all foams and other materials covered only the surface of the body facing the blast source and were trimmed to cover the upper aspect of the thoracic cage only - they did not result in an increase in presented area.

The intrathoracic pressure in anaesthetised rats subjected to blast loading was measured by introducing a Gaeltec Type 12CT pressure transducer mounted upon a 5FG (1.6 mm diameter) catheter into the oesophagus. The output of the transducer was captured upon a Nicolet 4094 digital oscilloscope and transient recorder at a sampling interval of 0.5 μs/point.

3.4 Anechoic chamber studies

The modification of the incident blast wave by interaction with the foams, copper and Kevlar laminate was investigated by placing the materials upon the surface of a 27 m^3 underwater anechoic chamber, exposing the materials to the shock front in air and measuring the wave transmitted into the water through the materials using a hydrophone (FIGURE 1).

The output of the hydrophone was sampled with 9 bit precision at a sampling rate of 200 kHz into a record of 8192 points, giving a resolution in the frequency domain of 24.4 Hz. The waveforms were stored

individually and then averaged in the time domain within each experimental group to suppress noise. All time-averaged signals had a common trigger point.

4. RESULTS

4.1 THE EFFECT OF THORACIC COVERINGS UPON THE SEVERITY OF LUNG INJURY.

4.1.1 Application of foam materials to the thoracic wall

Coverage of the right lateral thoracic wall with FOAM R significantly increased the severity of lung injury; the mean (±SE) Qi was increased from 1.24±0.06 in the group of animals having no thoracic coverings to 1.68±0.16 ($p<0.01$ by Student's t test). Mortality increased from zero to 33%. The intensification of primary blast effects seen with the rats placed upon a reflecting surface was also evident in rats exposed in free-field conditions. The Qi of rats exposed in free field with no covering was 1.18±0.04; coverage of the thoracic wall with FOAM R significantly increased the Qi to 1.77±0.13 ($p<0.001$)

High-speed cine photography of FOAM R exposed to the blast loading upon an unyielding surface and upon the rat thoracic wall showed that the foam underwent considerable compression with subsequent recovery. FOAM V, a viscoelastic foam failed to show any dynamic compression or distortion under identical loading. This foam also produced exacerbation of primary blast lung injury

The severity of lung injury with coverage of the thoracic wall with 10.3 mm FOAM V was compared to that produced in experiments using the natural rubber covering, FOAM R. The Qi with FOAM V was 1.82±0.16 showing considerable enhancement over the Qi of 1.33±0.08 for rats with bare thoraces. The natural rubber foam coverage resulted in a Qi of 1.99±0.14. Although both types of foam resulted in statistically significant augmentation of blast lung injury (at 95% confidence limits), there was no significant difference between the effects produced by the two foams.

Augmentation of lung injury was also evident with a high density cross linked polyethylene foam (areal density 1.07 kg/m^2) and even "bubble pack" packaging material used as thoracic coverings.

4.1.2 The influence of copper and Kevlar upon the severity of lung injury

The application of a 0.58 mm sheet of copper to the thorax failed to reduce significantly the severity of lung injury. Placed alone upon the thoracic wall without foam, the copper reduced but did not eliminate lung injury (Qi=1.11±0.02) when compared to rats with no thoracic coverings (Qi= 1.24±0.05) - this difference was not significant at $p<0.05$.

However, placing the copper <u>on top</u> of both types of foams produced a substantial reduction in the severity of the lung injury - with FOAM R, the injury was eliminated. Thus, rats exposed to blast loading with thoracic covering of 9 mm FOAM R alone had, as expected, an exacerbation of the quotient of lung injury from 1.24±0.05 in the unclothed animals to 1.70±0.17. Facing the rubber foam with the 0.58 mm copper sheet eliminated the primary blast injury to the lungs (Qi = 0.99±0.02).

This elimination of primary blast lung injury was mirrored when Kevlar laminate was used as the hard facing. The appropriate thickness of Kevlar laminate was used to result in the same areal density as the copper sheet

(TABLE 1). Serious primary blast lung injury produced with FOAM R (Qi = 1.61±0.053) was eliminated when the foam was faced with either copper (Qi=1.02±0.014) or the Kevlar laminate (Qi = 1.02±0.016)

Additional experiments have shown that flexible textile Kevlar at the same areal density as the resin-bonded laminate is not effective in reducing the severity of injury when used as a facing on foam materials.

4.2. CHANGES IN INTRATHORACIC OVERPRESSURES

4.2.1 The effect of thoracic coverings on intra-oesophageal pressure

Coverage of the lateral thoracic wall with either type of foam, copper alone or foam faced with copper failed to affect significantly either the peak overpressure, dP/dt_{max}, or rise time. The mean (±SD) peak overpressure measured within the oesophagus in rats exposed to blast with no thoracic coverings was 186±34 kPa (Qi=1.35±0.23); application of FOAM V increased the Qi to 2.05±0.42 and the intrathoracic pressure increased to 256±70 kPa, a value just significant at the 95% level of confidence.

Facing FOAM V with copper significantly reduced the severity of injury but failed to produce statistically significant alteration in the mean peak overpressure (166±77 kPa) when compared to the unprotected animal or to the animal covered by copper alone (162±51 kPa).

Oesophageal pressure has been used in the literature as an index of the stress transferred into the thoracic cage[9,13]. Its principal advantage is that it is a convenient site to introduce a gauge particularly in experimental models such as rats or even humans where emplacement within the parenchyma of the lung is not feasible. For stress waves generated at the lateral thoracic wall, the oesophageal pressure represents the stress after interaction with parenchyma and other structures and is not the input to the thoracic system. Its use as an indicator of the potential for thoracic injury in impact and blast loadings is debatable.

We are at present measuring overpressures at several sites within the lung parenchyma of anaesthetised pigs subjected to air-blast loadings from a shock tube to compare with intra-oesophageal pressure measurements. For the purposes of the current studies with rats however, the input to the thoracic system and its modification by materials was modelled *in vitro*.

4.3 TRANSMISSION OF THE INCIDENT STRESS WAVE BY THE MATERIALS

4.3.1 Coupling in the time domain

The time averaged waveforms for copper and FOAM R coverings are presented in FIGURE 2. At the hydrophone position within the anechoic chamber, (480 mm below the surface), the mean (±SD) peak transmitted overpressure was 140±37 kPa (n=10) with direct coupling of the airblast into the water with no interposing materials. FOAM R upon the surface of the water increased the mean peak overpressure to 174±53 kPa, however FOAM V resulted in a reduction to 84±16 kPa, even though both materials increase significantly the severity of injury in the rat model.

Estimates of dP/dt_{max} on the individual waveforms were unacceptable due to noise; the rise time of the leading edge (defined as the interval between 10% and 90% of the peak overpressure) was significantly modified by the foams. From a mean rise time of 11.8±6.8μs in the directly coupled

waveform, FOAM R and FOAM V resulted in an increase of the rise time to 76.7±7.7 and 35.3±9.7μs respectively.

Placing copper upon the surface of the water resulted in insignificant changes in the rise time (7.6±1.4μs) and a reduction in the peak transmitted pressure (76.6±18.5 kPa) - broadly similar results were experienced with Kevlar. The most notable and unequivocal changes in the transmitted waveform were achieved when the foam materials were faced with either copper or Kevlar. (FIGURE 2d). A copper facing upon FOAM R produced a mean transmitted overpressure of 22.7±2.8 kPa, a reduction to 16% of the level measured with no foam or other coverings. The rise time was extended from 11.1±6.8μs to 160.9±37.8μs.

In the time domain therefore, the foams produced equivocal changes in peak transmitted pressure but extended the rise time of the leading edge; facing the foam with copper significantly reduced the peak transmitted overpressure and lead to a very substantial increase in rise time.

4.3.2 Coupling in the frequency domain

Logarithmic spectra were produced in the frequency domain and the frequency content of the transmitted pressure profiles compared to that of the waveforms transmitted without materials by subtracting logarithmic spectra, thus giving the ratio of coated to uncoated transmission in dB.

Both FOAM R and FOAM V significantly increased the power transmitted into the anechoic water chamber when placed upon the water surface. FOAM V produced approximately 5 dB amplification over a relatively confined bandwidth of about 0.5-2 kHz. The coupling produced by FOAM R was more substantial with a 5-10 dB enhancement over 0.5-3.5 kHz (FIGURE 3a). Copper alone or Kevlar alone do not significantly modify the severity of injury in the rat model; this was mirrored in the comparative logarithmic spectra with minor changes in the transmitted frequencies over the bandwidth 0.5-5 kHz (FIGURE 3b).

Placing the Kevlar or copper as facings upon both types of foams achieved substantial decoupling over a wide frequency range. Copper upon FOAM V resulted in a 10-20 dB reduction over a bandwidth of 2-6 kHz. Frequencies of less than 2 kHz were largely unaffected. A very similar pattern was also seen with Kevlar as a facing upon FOAM V.

It was observed in the experimental studies upon rats that copper and Kevlar facings were more effective at reducing the severity of lung contusions produced by blast loading with FOAM R as the backing material - the comparative log spectra supported this empirical observation. Both copper and Kevlar upon FOAM R were extremely efficient at decoupling the incident power and resulted in up to 20-30 dB reductions over a 3.5-6 kHz bandwidth (FIGURE 3c). At 2-3.5 kHz, a 10-20 dB loss of transmission was evident but little reduction was seen at <2 kHz.

5. DISCUSSION

The augmentation of primary blast lung injury by foam materials can be explained in terms of impedance matching. The foams appear to be acting as acoustic couplers/transformers resulting in an augmented transfer of direct stress waves from the air into the tissues of the body. The absence of injury when copper or Kevlar facings upon foams are used may be explained in terms of the decoupling effects of these high impedance layers.

5.1 Simple transmission model

The principles of transmission of acoustic waves through interfaces between materials of dissimilar properties are well established for acoustic waves[8]. If a compressive stress wave in a material encounters an interface, the magnitude of the reflected and transmitted waves is dependent upon the relative characteristic acoustic impedances of the materials across the interface. The characteristic acoustic impedance (Z) is the product of the speed of sound in the material (c) and its density (p). For a one-dimensional acoustic wave of pressure amplitude P_i travelling through a medium, a, incident upon a boundary with a material, b, a reflected wave (P_r) and a transmitted wave (P_t) will be generated. The magnitudes of the incident and transmitted waves are related by the expression

$$P_t = P_i * ([2*Z_a]/[Z_a + Z_b]) \quad \ldots.1$$

and

$$P_r = P_t - P_i$$

For conventional engineering materials, the characteristic acoustic impedance, Z, may also be expressed in terms of the Young's modulus, E. As $c = (E/p)^{0.5}$, Z in equation 1 may be substituted by $(E*p)^{0.5}$.

As an illustration of the principles of acoustic coupling and decoupling, equation 1 may be used to calculate the direct stress transferred into the lung after propagation of a compressive stress in the air through the multilayer interfaces (air/single and multilayer materials/soft tissue/lung). The calculation of these stresses can only be considered to be semi-quantitative - equation 1 is not strictly valid for waves of large amplitude and additionally, the acoustic wave velocity in foam materials is determined principally by the effective shear modulus, not by E. Other complicating factors are that p and c are not constant for air and (by assumption) for lung but are a function of the pressure, additionally, the effects of oblique incidence and multiple internal reflections within each material are also not considered. Nevertheless, this simple one dimensional model can provide an approximation of the stress or pressure transmitted to the thorax of the animal that may be compared to the actual severity of lung injury resulting from the interaction of the incident blast wave upon the various materials covering the thorax.

The relative acoustic impedances of air/FOAM V/body wall/lung is assumed to be 1/4.7/3700/23; the acoustic impedance of air at atmospheric pressure is 415 $Pa.s.m^{-1}$ and copper has a relative impedance of 7.6×10^4 compared to air.

Using the simple model above, the peak stress transmitted through the bare, uncovered chest wall into lung for an incident stress in air of 100 units is estimated to be 2.5 units. Interposition of FOAM V increases this stress to 4.2 units but facing FOAM V with copper significantly reduces the transmitted stress to 0.0006 units. Thus, the peak stress transmitted into the simple lung material are ranked in the same order as the severity of lung injury seen in the rat model. More rigorous descriptions of the transmission of energy through multilayer materials of different characteristic impedances are available elsewhere[8].

5.2 High-frequency or low-frequency injury mechanism?

The phenomenon of augmentation of lung injuries by foam materials was first described by Clemedson et al.[6]. They had presumed that the effect occurred only with combinations of foams and blast loadings that were compressed

during exposure. It was suggested that the increased severity of injury arose from potential energy stored in the compressed foam being released during a critical period within the chest wall deflection, resulting in a greater impulse transferred to the chest. The demonstration in the current experiments that augmentation of blast injuries will occur with highly viscoelastic foams which, on the evidence of high-speed cine photography, do not compress under the blast loading, makes the partial impulse explanation suggested by Clemedson unlikely.

A notable limitation of the current studies is that we have been unable to measure the displacement of the rat thoracic wall when covered by materials, and therefore, we have not been able to study the influence of the coverings upon the gross displacement and peak velocity achieved by the wall. Experiments are currently underway using anaesthetised pigs instrumented with accelerometers mounted upon rib to study the motion of the thoracic wall under blast loading. The demonstration that application of copper or Kevlar alone to the thoracic wall failed to reduce significantly the severity of lung injury is an interesting observation; these materials doubled the effective mass of the body wall and would be expected to reduce the peak velocity and gross deflection of the body wall under the same blast loading. Their failure to diminish significantly the injury is indirect evidence that the gross deflection is not a major determinant of the severity of injury; FFT analysis of the transmitted waveforms demonstrated little loss in transmission over a wide frequency band (FIGURE 3b) and thus corresponded to the lack of effect demonstrated *in vivo*.

FOAM R resulted in an increased transmission of power within the bandwidth 0.5-3.5 kHz (FIGURE 3a). The resonant frequency of the rat thoracic wall is around 0.35-0.8 kHz[7] and it is conceivable that the foam was increasing the deflection of the chest due to resonance at this frequency. The elimination of the thoracic injury with the high acoustic impedance materials placed upon the foams resulted from significant decoupling at frequencies much higher than these resonant frequencies (FIGURE 3c) and would suggest that the primary lung injury was a high-frequency damage mechanism. Preliminary results with the pig model have also shown exacerbation of lung injury with FOAM V; the thoracic wall resonances in this model are about 70-150 Hz[7] and in the anechoic chamber studies, FOAM V did not result in significant increases in transmission at these very low frequencies.

The pattern of injury in blast injured lungs would also suggest a high-frequency direct stress wave damage mechanism. Contusions are generally most severe close to the inner surfaces of the lung adjacent to the mediastinum and liver and in acute angles within the parenchyma such as the antero-medial border where stress concentration will occur[5]. The peripheral pleural surface and parenchyma directly compressed by the gross deflections of the thoracic wall are usually less severely injured, suggesting that direct shear or compression are not the principal injury mechanisms.

Finite element modelling of the transmission of stress waves in pulmonary parenchyma have supported these empirical observations and demonstrated that, due to reflection and reinforcement of the stress waves within the thoracic cage, the stress at internal sites may exceed the peak stresses peripherally, close to the body wall. The modelling has also demonstrated that direct stress waves may also steepen up during transmission through the parenchyma because of increased wave velocities of the high pressure components[3] and consequently result in greater pressure differentials across the alveolar/capillary interface. These pressure differentials may

lead to failure of the barrier to produce haemorrhagic contamination of alveoli.

5.3 Role of direct stress transmission in non-penetrating impacts

Non-penetrating injuries, whether they are produced by blast exposure or by impact, may be categorized as direct or indirect[5]. Direct injuries are those occurring adjacent to the displaced body wall and generally result from local shear or compression of underlying soft tissue or by direct laceration by, for example, displaced, fractured ribs. Indirect injuries are classified as lesions evident at sites not directly involved in the compressive strain and can arise from both transmitted shear waves, (typified by aortic rupture and some splenic and hepatic lacerations) and from direct stress waves transmitted into the body.

Injuries resulting from either local or indirect shear tend to be associated with gross compression of the body wall; stress wave injuries are associated with high body wall velocities and are largely independent of gross thoracic or abdominal distortions. Pulmonary contusions and bowel contusions without gross laceration are typical stress wave related injuries.

Shear-type and compression injuries tend to predominate in road traffic injuries as a result of relatively large body wall deflections occuring over long periods of time with quite low body wall velocities. Non-penetrating impacts in the defence field, such as the retardation of bullets by body armour, tend to result in significantly higher body wall velocities. As the effective mass of an impactor decreases and impact velocity increases, the lower momentum of these types of impact results in small body wall deflections associated with the high body wall velocities. The pattern of injury transposes to a direct stress wave dominated aetiology[11] in which the severity of injury is not governed primarily by the gross deflections, but by the peak body wall velocity.

It is notable that over the last few years, emphasis has shifted away from thoracic injury thresholds being described simply in terms of gross thoracic deflection. The evolution of the Viscous Criterion is a recognition that the rate of energy transfer is an important determinant of the severity of injury[12]. Body wall velocities in motor vehicle impacts rarely exceed 20-25 $m.s^{-1}$ but in the defence field, peak body wall velocities approaching 80-90 $m.s^{-1}$ may occur. Stress wave effects may dominate the response to the impact and any model or criteria derived to predict biological effects or to assess materials developed to offer protection to security forces must take adequate account of this phenomenon and not rely solely on gross displacements predicted by mathematical models or simulants.

The demonstration in this paper of increased severity of injury and increased rates of mortality following "protection" of the thoracic cage with foam materials, serves to highlight the role of direct stress waves in impact injury at high rates of energy transfer.

6. CONCLUSIONS

6.1. *In vivo* and *in vitro* experimental studies suggest that lung injuries arising from exposure to blast overpressure are produced principally by direct coupling of the incident stress wave into the thorax and not from gross distortion of the thoracic wall and viscera.

6.2. Foam materials applied to the thoracic wall increase the stress transferred internally to result in more severe injuries; acoustic coupling is considered to be the biophysical basis of this phenomenon. The foam materials increase the transmission of the high-frequency components of the stress wave.

6.3. Decoupling, and thus elimination of injury may be achieved by facing foam materials with a rigid, high acoustic impedance layer. The high acoustic impedance layer applied in isolation to the thorax is ineffective.

6.4. Low momentum, high velocity impacts resulting in high initial body wall velocities but small gross body wall distortions, will induce direct stress waves in the body. Injuries from this mechanism in susceptible viscera such as lung and bowel may dominate those resulting from gross compression and shear. Injury criteria based upon gross compression will be inappropriate under these circumstances.

7. REFERENCES

1) BOWEN I G, FLETCHER E R, RICHMOND D R, (1968). Estimate of man's tolerance to the direct effects of air blast. Technical Progress Report DASA-2113, Defence Atomic Support Agency, Department of Defense, US Government.

2) BUSH I S, CHALLENER S A, (1988). Finite element modelling of non-penetrating thoracic impact. Proc IRCOBI, Bergisch-Gladbach, FRG.

3) BUSH I S, CHALLENER S A (1989). Internal report to MOD.

4) COOPER G J, MAYNARD R L, (1986) An experimental investigation of the biokinetic principles governing non-penetrating impact to the chest and the influence of the rate of body wall distortion upon the severity of lung injury. Proc IRCOBI, Zurich, Switzerland.

5) COOPER G J, TAYLOR D E M (1989). Biophysics of impact injury to the chest and abdomen. J RAMC, 135,58-67.

6) CLEMEDSON C-J, FRANKENBURG L, JONSSON A et al. (1971). Effects on extra- and intrathoracic pressure pattern and lung injuries of rigid and soft protection of thorax in blast exposed rabbits. Forsvarmedicin 7, 172-190.

7) GIERKE H E von (1968). Response of the body to mechanical forces- an overview. Ann. N. Y. Acad. Sci. 152, 172-186

8) KINSLER L E, FREY A R, COPPENS A B, SANDERS J V, (1980). Fundamentals of acoustics. 3rd Edition, J Wiley and Sons.

9) JONSSON A., (1979). Experimental investigations on the mechanism of lung injury in blast and impact exposure. Linkoping University Medical Dissertation No 80, ISBN 91-7372-307-X.

10) MAYNARD R L, COOPER G J, SCOTT R (1989). Mechanisms of injury: Bomb blast injuries and explosions. In "TRAUMA" by S Westaby (Ed), Heinemann, London.

11) TAYLOR D E M, WHAMMOND J S, COOPER G J et al. (1989). Stress/strain characteristics of flat bones in relation to nonpenetrating injuries of the chest and head. In "Material Properties and Stress Analysis in Biomechanics" by A L Yettram (Ed), Manchester University Press.

12) VIANO D C, LAU V K (1988). A viscous tolerance criterion for soft tissue injury assessment. J. Biomechanics, 21, 387-399.

13) YOUNG A J, JAEGER J J, PHILLIPS Y Y et al. (1985). The influence of clothing on human intrathoracic pressure during airblast. Aviation, Space and Environmental Medicine 56, 49-53.

FIGURE 1: Experimental set-up for the determination of stress wave transmission by the materials *in vitro*

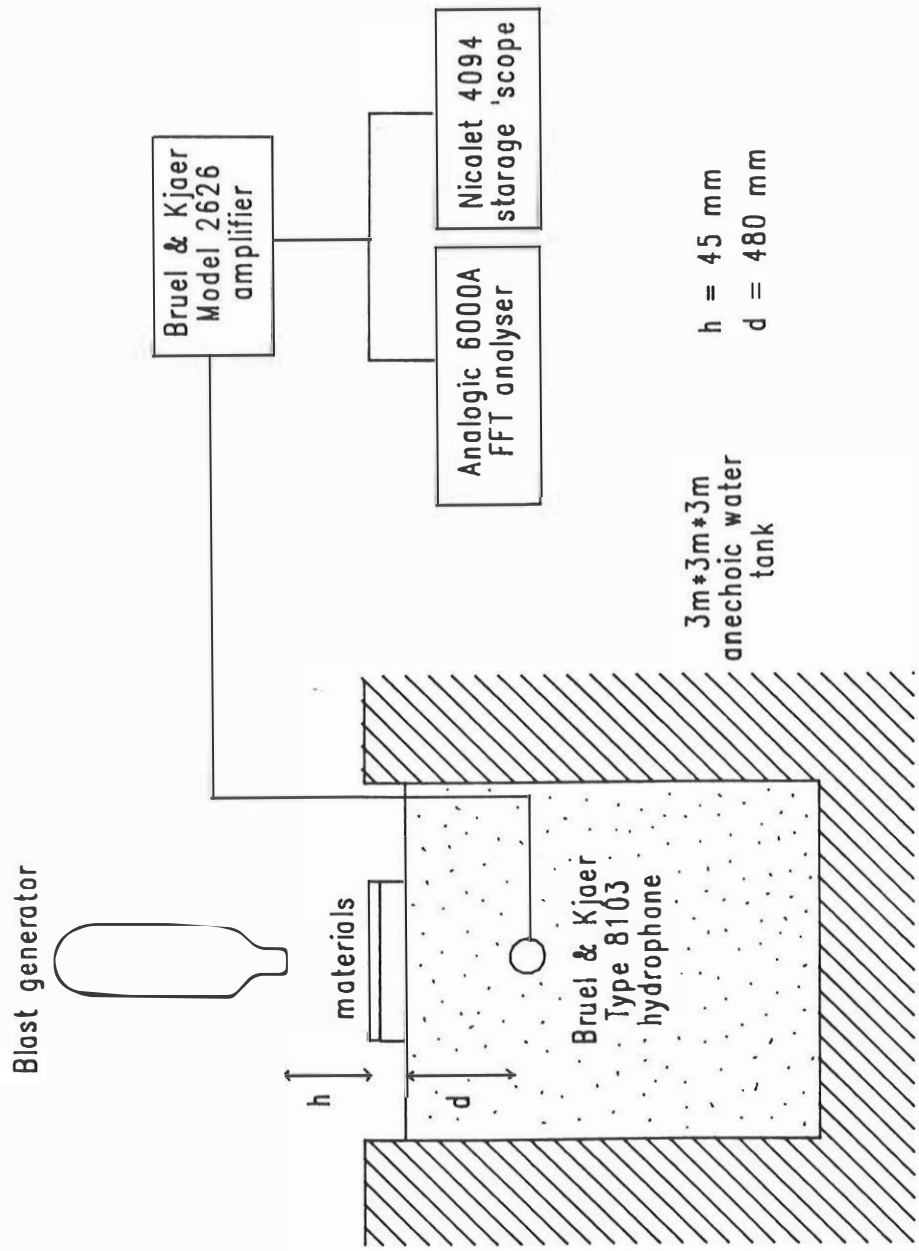

FIGURE 2: Overpressures coupled from air into the anechoic water chamber following interaction with materials upon the water surface. The initial loading was a shock front in air. Waveforms shown are the average of between 5 and 10 individual responses within each group.

a) Direct coupling of the air blast with no intervening materials.
b) 9.3 mm natural rubber foam, FOAM R
c) 0.58 mm copper sheet
d) 0.58 mm copper facing upon 9.3 mm FOAM R.

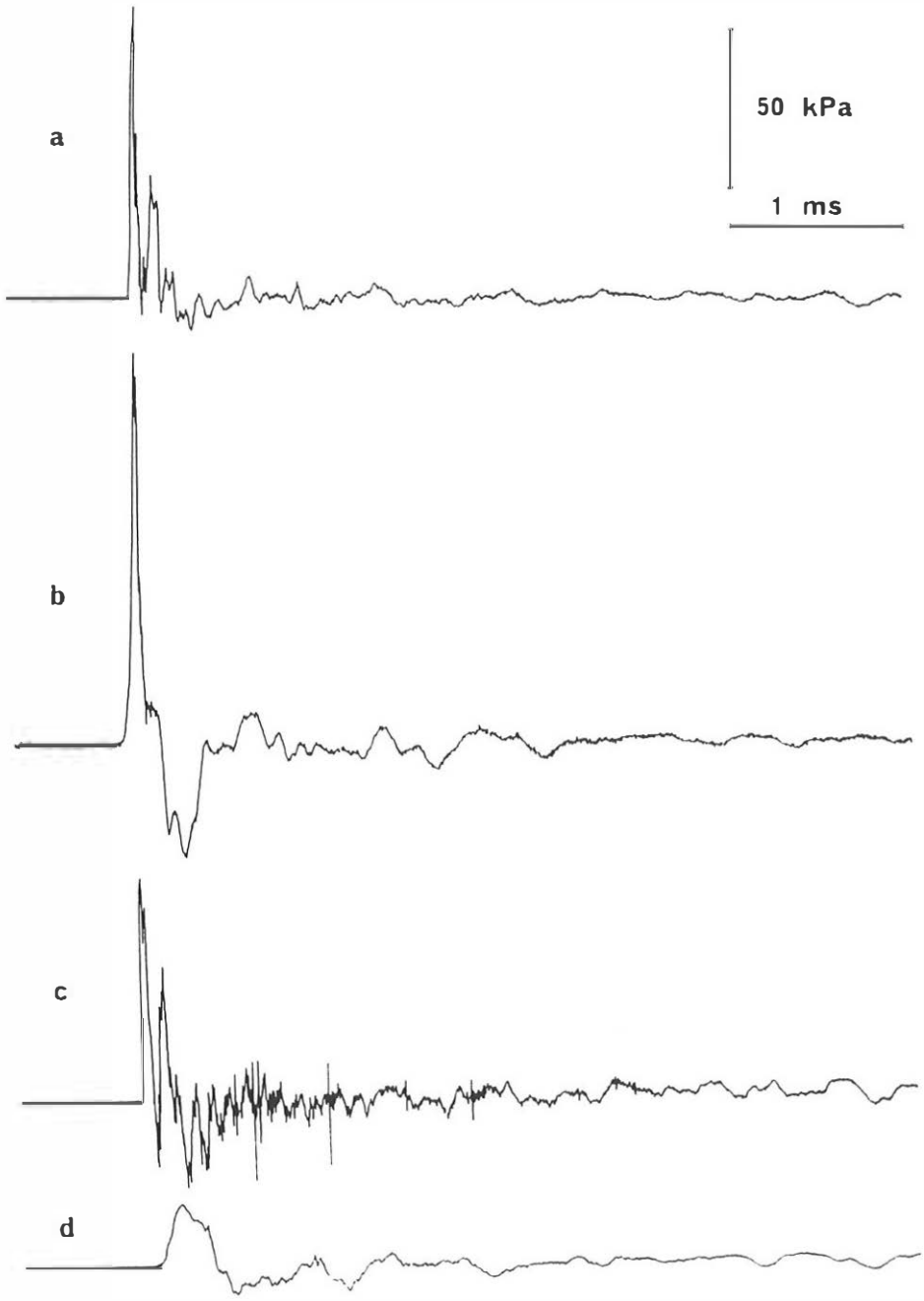

FIGURE 3: Transmission characteristics of FOAM R and copper. The figures show the result of subtraction of the logarithmic spectrum in the frequency domain of the directly coupled waveform (acquired in the absence of any coverings) from the logarithmic spectrum of the waveforms transmitted with each type of covering. Transforms were performed on the time-averaged waveforms of FIGURE 2. The peak evident at just less than 2 kHz is a resonance of unknown origin in the directly coupled waveforms and is not an artefact of signal analysis.

a) 9.3 mm natural rubber foam, FOAM R
b) 0.58 mm copper sheet
c) 0.58 mm copper facing upon 9.3 mm FOAM R.