The Journal of Laryngology and Otology
November 1994, Vol. **108**, pp. 925–929

Review Article

Blast injury of the auditory system: A review of the mechanisms and pathology

R. J. N. GARTH, F.R.C.S.

Abstract
Blast injury of the auditory system is uncommon and our knowledge incomplete. This article reviews the literature to date giving an account of the interactions of blast waves with the ear, the mechanisms of injury, the pathology, the clinical features, and an outline of management principles.

Key words: Blast injuries; Auditory pathways

Introduction

Blast injury to the ear is unusual in peacetime and few otolaryngologists in Great Britain encounter many cases. Sporadic incidents occur as a result of isolated accidents or occasionally there are larger numbers of casualties following a terrorist bombing.

Otological injury is easily overlooked in a patient with multiple injuries and it is interesting that in a review of 1535 victims of terrorist explosions in Belfast, Hadden *et al.* (1978) noted only 15 perforated tympanic membranes. Many of these casualties may have been the victims of unconfined free-field explosions resulting in a low incidence of perforations but it is possible that some injuries to the ear were not recorded. In contrast, Kerr and Byrne (1975) reported on 60 perforated tympanic membranes following a single blast at the Abercorn Restaurant.

Many reports that have followed bombings or military conflicts may be of general interest to the otolaryngologist, but the varied and uncontrolled nature of the explosions means that there is little information of scientific value to add to our understanding of blast injury. Much of our understanding of the biophysical interactions of pressure waves with the ear is a result of investigations using laboratory animals, cadaver ears, or models.

Blast waves and their transmission to the ear

Auditory damage may result from continuous noise, impulse noise or blast trauma and this article focuses on the injuries resulting from blast. Impulse noise and blast can be classified in terms of the source of the pressure or the characteristics of its waveform, but this distinction is arbitrary. Small arms are usually said to produce impulse noise whereas the sound energy from artillery or an explosion is usually described as a blast. The following criteria are useful in differentiating impulse noise from blast:

(a) The peak over pressure of impulse noise is usually less than 2 kPa (160 dB) while the muzzle blast from a large gun may produce tens of kPa.
(b) A blast involves considerable movement of air and combustion products whereas impulse noise does not.
(c) Impulse noise is often associated with low frequency mechanical clatter.

Following a detonation, there is a shock wave of effectively instantaneous over-pressure which travels through air at a velocity greater than the speed of sound. Behind the shock wave is a region of gas flow consisting of combustion products which is known as the dynamic over-pressure. These combine to form a short positive pressure phase which is followed by a longer subatmospheric phase. These are represented by the Friedlander curve (Figure 1).

It is unlikely that many blast victims will be exposed to a simple Friedlander type blast wave as the environment has a significant influence on the waveform. In a confined space such as a building, the initial shock wave will be modified by multiple reflections from surrounding surfaces, and the heating of gases within that space will give rise to a gradual increase in pressure which may last for several hundred milliseconds (Phillips *et al.*, 1989). Figure 2 shows a typical blast wave in such circumstances.

The degree and type of damage are influenced by the peak over-pressure and the duration of the positive pressure phase (James *et al.*, 1982). Other factors such as the orientation of the ear canal to the blast wave will result in further modification as it passes down the ear canal (Figure 3). Stinson (1985) investigated sound pressure distribution at the tympanic membrane using a scaled replica of the human external auditory canal and found areas on the drum with as much as 20 dB of attenuation. Although his investigations used continuous noise and

From the Department of Otolaryngology, Royal Naval Hospital Haslar, Gosport, Hampshire, PO12 2AA.
Accepted for publication: 30 July 1994.

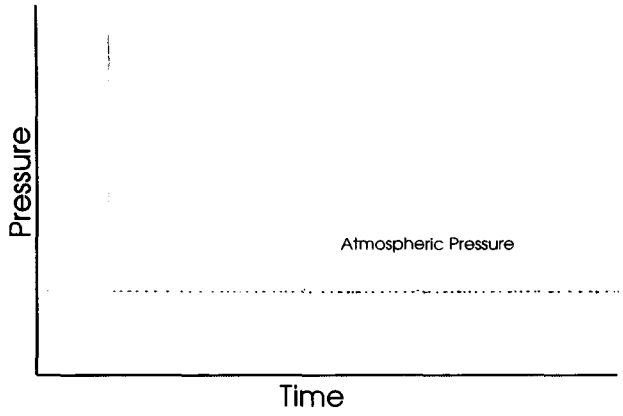

FIG. 1
Friedlander curve. The idealized free-field blast waveform.

cannot be directly extrapolated to a blast, they demonstrate the extent to which the external auditory canal may modify a wave travelling down it. The presence of wax within the ear canal is another factor that may influence the extent of damage. Hirsch (1968) pointed out that wax occluding the outer part of the canal can act as an ear plug and attenuate the blast while wax impacted on the drum may be pushed into the middle ear like a 'ramrod' causing ossicular disruption. With all the variables that may influence a blast wave it is not surprising that apparently similar blasts produce injuries of varying severity.

The effect of blast on the ear

The external ear

Injuries are most likely to result from flying debris rather than as a primary affect of the blast wave. These injuries will be much the same as other penetrating soft tissue injuries from projectiles.

The tympanic membrane and middle ear

The extent of tympanic membrane damage may vary from injection of blood vessels, subepithelial haemorrhages, small split-like perforations (often following the fibres of the lamina propria as shown in Figure 4) to multiple or total perforations. There have been few investigations on the human ear and data on the susceptibility of ears to blast derived from animal models, with differences

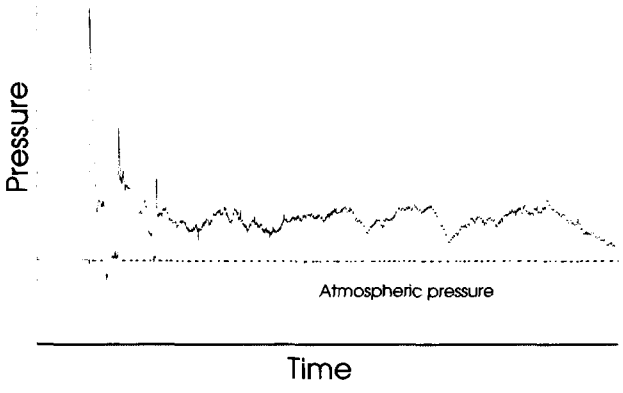

FIG. 2
Complex waveform of a blast in a confined space demonstrating the prolonged positive pressure phase.

from one species to another (Roberto *et al.*,1989), cannot be applied directly to humans. Zalewski (1906) used static over-pressures in one of the earliest experiments on the human ear and found the lowest pressure of tympanic membrane rupture to be 37 kPa (5.4 psi). James *et al.* (1982) collected sufficient data in experiments on human temporal bones to determine the 50 per cent rupture curves as a function of pressure and positive impulse (Figure 5). Obviously there will be considerable variations between individuals in the susceptibility of a tympanic membrane to rupture. Normal biologial variability will result in slightly different physical characteristics of the tympanic membrane and previous injury or aging may also be of significance.

Stuhmiller (1989) used static pressure loading in a model to demonstrate quite marked variations in stress throughout the tympanic membrane with particularly high levels of stress being found around the periphery. If the work of Stinson (1985) can be applied to a blast wave, the uneven pressure distribution over the drum will further complicate stressing patterns within the tympanic membrane. The levels of stress imposed on the drum by a blast are well beyond normal physiological limits and result in alteration of its mechanical characteristics. Stress within the drum may stretch the radial fibres of the lamina propria beyond their elastic limits resulting in rupture of some of these fibres and an increase in tympanic membrane compliance. Eames *et al.* (1975) noted an increase in compliance in chinchillas exposed to repeated 166 dB impulse stimuli which gradually returned to normal over the following two weeks.

The sites of perforations were recorded by Pahor (1981) who found 60 per cent to be central, 25 per cent to be anterior and 15 per cent to be postero-superior. As the tympanic membrane is displaced medially by the positive phase of the blast, the volume of the middle ear will decrease, and there will be some increase in middle ear pressure. The size of this pressure increase will be determined by the dimensions of the middle ear and how quickly air can be displaced into the mastoid system or down the eustachian tube. In theory this increase in

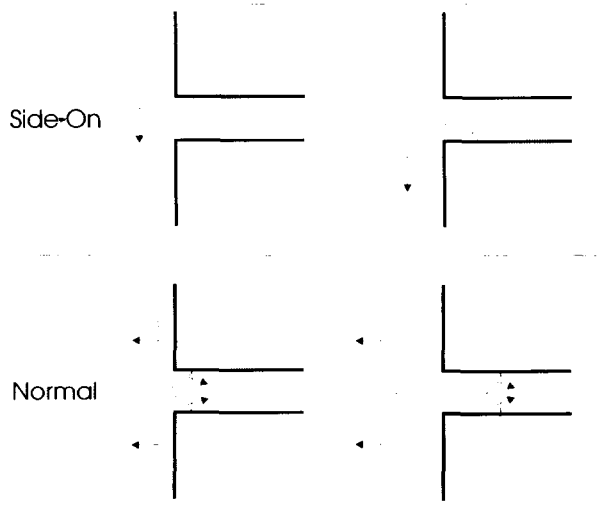

FIG. 3
Diagrammatic representation of a blast wave passing down the external auditory canal showing the reflections in the canal resulting from shock waves normal and side-on to the canal. (From James *et al.*, 1982).

https://doi.org/10.1017/S0022215100128555 Published online by Cambridge University Press

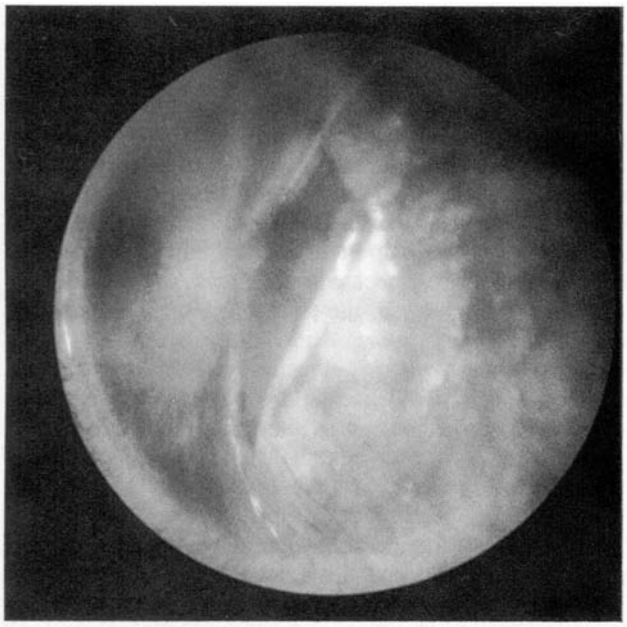

FIG. 4
Perforation in line with the radial fibres of the tympanic membrane.

middle ear pressure will cushion the tympanic membrane giving some degree of protection from rupture, so an ear with a large mastoid system or patulous eustachian tube may be more vulnerable to blast injury. The extent to which this mechanism protects the tympanic membrane is uncertain. White *et al.* (1970) suggested that the tympanic membrane may be displaced medially to the extent that it 'bottoms out' on the promontory and the ossicles. If this is so, the drum may gain some protection in these areas making perforation less likely. Further evidence for this theory is provided by the fact that the drum is more easily perforated by static negative pressures (where the tympanic membrane would gain no support from the middle ear structures).

There has been debate about the relative contribution of the positive and negative phases of the blast wave to middle ear injury. As perforations of the drum may have an inverted flap, and cholesteatoma may develop within the middle ear, it would suggest that many of these injuries result from the positive phase of the blast. The negative phase is of less importance and it is likely that, where an everted flap of drum is found, the negative phase has merely everted this segment after a positive phase perforation.

There is a considerable difference in the reported incidence of ossicular damage, ranging from no cases among a total of 89 perforated tympanic membranes reported by Pahor (1981) and Kerr and Byrne (1975) to 33 per cent of the ears undergoing tympanoplasty reported by Sudderth (1974). The difference may well lie in the selection criteria in the latter series. The variety of ossicular injuries in this series are shown in Table I and in addition to these, fracture/dislocation of the stapes footplate has also been recorded (Singh and Ahluwalia, 1968).

As a tympanic membrane is ruptured by a blast wave, small fragments of squamous keratinizing epithelium may be distributed throughout the middle ear cleft and, if they are still viable, result in cholesteatoma formation. Kronenberg *et al.* (1988), in a series of 210 ears found cholesteatoma in 7.6 per cent. Six of these were cholesteatoma pearls and 10 were extensive cholesteatomas of the middle ear and the mastoid. It is not clear whether the latter cases resulted from fragments of epithelium implanted throughout the middle ear and mastoid system or whether they started within the middle ear and spread rapidly through a well pneumatized mastoid.

The inner ear

Victims of blast injury often have a profound sensorineural hearing loss and tinnitus immediately after the explosion. This temporary threshold shift and tinnitus are often fairly short lived and hearing largely returns within a few hours. Some patients find that their hearing improvement is gradual, but some will be left with a permanent

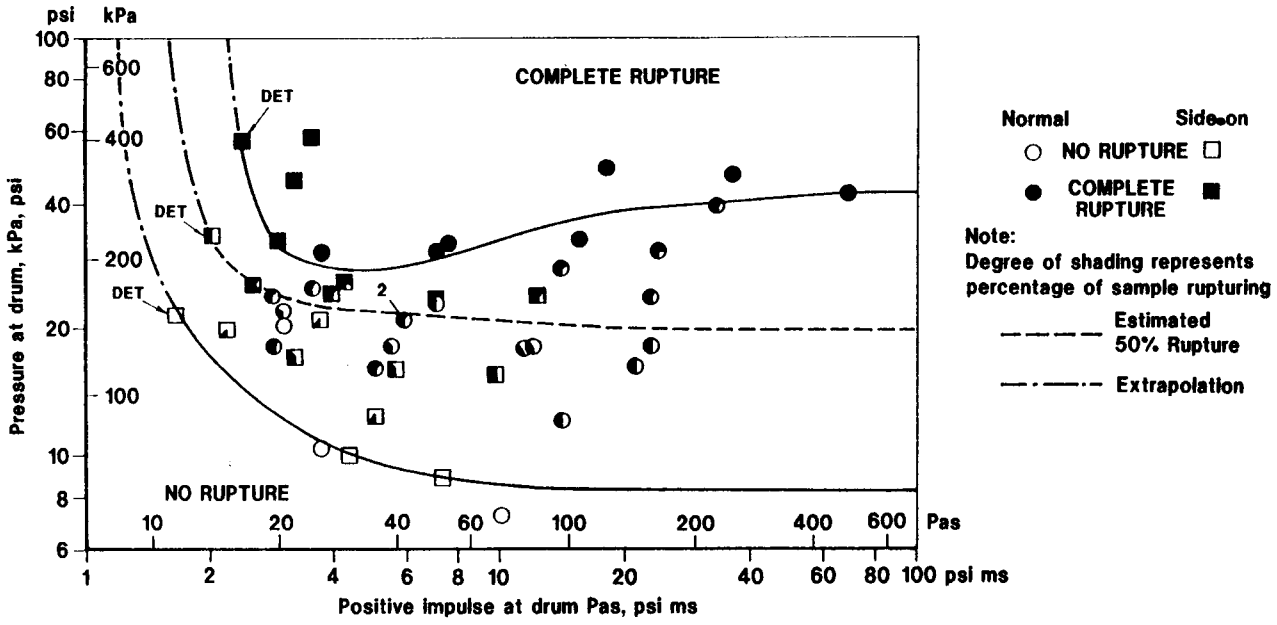

FIG. 5
Pressure-impulse rupture criteria for human cadaveric temporal bones. (From James *et al.*, 1982).

https://doi.org/10.1017/S0022215100128555 Published online by Cambridge University Press

TABLE I
OSSICULAR INJURIES*

Incudo-malleolar joint disruption	25%
Incudo-stapedial joint disruption	4%
Incudo-stapedial disruption + stapes superstructure fracture	2%
Incus dislocation	2%
Fractured stapes superstructure	1%

*From data of Sudderth (1974).

hearing loss. Teter *et al.* (1970) studied the audiograms of blast victims and described four different audiometric configurations, though the commonest audiometric picture in blast victims seems to be a high tone sensorineural loss. The 4 kHz dip associated with noise induced hearing loss is not usually a feature in blast injury which suggests a different mechanism of injury. Many patients may be unaware of their hearing loss as it is often confined to the high tones and good speech discrimination is retained.

Surprisingly, vertigo is an uncommon problem following blast injury. Pahor (1981) found only two cases amongst the 110 pub bombing victims, one of whom had suffered previous vertiginous episodes and in the other it was felt that litigation and compensation were of significance. Kerr and Byrne (1975) felt that the vertiginous cases in their series were associated with a post-concussional state. Perilymph fistulae may occur but these are rare: one case was reported following a stapes footplate disruption (Singh and Ahluwalia, 1968) and they have been also demonstrated by Yokoi and Yanagita (1984) who found a 40 per cent incidence in animal studies. This does not fit with the clinical findings in most series and may be a feature of the unusually long (200 ms) overpressure in their blast experiments.

Electron microscopy has given considerable insight into the damage occurring within the cochlea. It is postulated that the temporary threshold shift results from changes in the integrity of the tight cell junctions of the reticular lamina, changes in the membrane permeability, or holes in the reticular lamina. These breaches allow mixing of the perilymph and endolymph which alters the ionic environment and interferes with the physiological events within the cochlea. As the breaches in the reticular lamina are repaired the temporary threshold shift resolves. There have been a number of changes shown to account for the permanent threshold shift. Hamernik *et al.* (1984b) described changes in the chinchilla exposed to repeated 160 dB impulses. Damage included loss of hair cells in some ears and in more severely damaged ears long segments of the organ of Corti became detached to float in the scala media alongside cellular debris. Cilia of the outer hair cells were found to be bent, fused or broken and some giant cilia were found. The inner hair cells were damaged to a lesser extent. Holes were found in the reticular lamina with cytoplasmic extrusions at the site of maximal cochlear disruption and Claudius cells appeared to be able to spread their membranes to cover a considerable area of damage. Macrophages within the cochlea increased, reaching a peak at 12–16 days, though phagocytic activity was still evident at 30 days. Yokoi and Yanagita (1984) found similar changes concentrated at the basal turn of the cochlea. Kumagami (1992) noted degenerative changes in the endolymphatic sac and some degree of endolymphatic hydrops in guinea pigs exposed to blast, though there are not usually any features of hydrops in human blast victims.

There is some evidence to suggest that tympanic membrane perforation and ossicular disruption may give some protection to the cochlea. Akiyoshi *et al.* (1966) found less cochlear damage in guinea pigs exposed to a larger blast (resulting in disruption of the middle ear) than in those exposed to smaller blasts. Hamernik *et al.* (1984a) and Eames *et al.* (1975) reported similar findings in animals exposed to multiple shock waves. This is not surprising as the initial disruption of the conducting mechanism will reduce energy transmission to the cochlea during subsequent shock waves. Interestingly, Kerr and Byrne (1975) found no evidence of this phenomenon in their series of blast victims, though none had ossicular damage.

Central nervous system

Morest (1982) demonstrated degenerative changes in the ascending pathways of cats and chinchillas exposed to acoustic trauma. However, Pratt *et al.* (1985) did not detect any central effects of blast using brain stem audiometry to assess 37 human blast survivors. It is unlikely that such changes occur to any extent in humans as speech audiometry usually shows good speech discrimination suggesting insignificant retro-cochlear degeneration.

Treatment

Most clinicians would agree that ears without middle ear damage do not require active treatment. There is little evidence that treating the sensorineural loss with steroids, vasodilators or vitamin supplements makes any difference to the recovery, though there is often little to lose by trying these, particularly if the hearing loss is profound or in an only hearing ear.

Eighty-three per cent of perforations heal spontaneously (Kerr and Byrne, 1975) which makes it difficult to justify immediate grafting. Ears with perforations should be kept dry and ear drops or antibiotics reserved for cases with infection or significant debris in the canal. Perforations that do not heal spontaneously may be grafted at a later date and any ossicular damage reconstructed.

In patients with persistent vertigo or a fluctuating hearing loss, a perilymph fistula must be considered. Although this complication is rare it is important, so a tympanotomy should be performed if symptoms persist and a fistula is suspected. If a fistula is found at tympanotomy it should be closed with a graft.

With cholesteatoma occurring in 7.6 per cent of ears (Kronenberg *et al.*, 1988), it is prudent to review cases where the tympanic membrane has been perforated and if there is any suspicion of cholesteatoma the middle ear and mastoid should be explored. Cholesteatoma following blast injury may be extensive.

Prevention

In a military setting the design of armoured vehicles may give the occupants considerable protection from an external blast. For those not lucky enough to have this protection, ear muffs or ear plugs may be used. While ear muffs are generally regarded as giving the best protection

to people exposed to continuous or impulse noise, Jonsson (1990) has shown that ear plugs give more effective protection from blast waves. Ear plugs gave sufficient protection in models exposed to over-pressures of 190 kPa to make perforation unlikely, with only 14 kPA recorded in the ear canal. With ear muffs, pressures of 100 kPa were recorded in the canal. The nature of ear muffs means that they are easily displaced by the blast and therefore of limited value.

It is likely that the design of ear plugs is of importance, as some smaller plugs could be pushed down the canal by the blast resulting in direct injury to the tympanic membrane and middle ear. With a larger plug fitting into the concha in much the same way as a hearing aid mould, there would be no chance of this occurring. Ear plugs containing a valve or perforated metal disk are available and provide good protection against impulse noise while allowing the user to hear speech. It is likely that these would also provide good protection against blast waves, though they have not been evaluated in this setting. The design of ear plugs for blast protection is an area where further research is needed.

References

Akiyoshi, M., Amemiya, A., Sato, K., Takeda, T., Shoji, T. (1966) On the pathogenesis of acoustic trauma in rabbits and guinea pigs due to explosion. *International Audiology* **5:** 270–271.

Eames, B. L., Hamernik, R. P., Henderson, D., Feldman, A. S. (1975) The role of the middle ear in acoustic trauma from impulses. *Laryngoscope* **85:** 1582–1592.

Hadden, W. A., Rutherford, W. H., Merrett, J. D. (1978) The injuries of terrorist bombing: a study of 1532 consecutive patients. *British Journal of Surgery* **65:** 525–531.

Hamernik, R. P., Turrentine, G., Roberto, M., Salvi, R., Henderson, D. (1984a) Anatomical correlates of impulse noise-induced mechanical damage in the cochlea. *Hearing Research* **13:** 229–247.

Hamernik, R. P., Turrentine, G., White, C. G. (1984b) Surface morphology of the inner sulcus and related epithelial cells of the cochlea following acoustic trauma. *Hearing Research* **16:** 143–160.

Hirsch, F. G. (1968) Effects of overpressure on the ear – a review. *Annals of the New York Academy of Science* **152:** 147–162.

James, D. J., Pickett, V. C., Burdett, K. J., Cheesman, A. (1982) The response of the human ear to blast. Part 1: The effect on the ear drum of a short duration, fast rising pressure wave. *Army Weapons Research Establishment/Chemical Defense Establishment Report* **No. 04/82.**

Jonsson, A. (1990) Pressure measurement in the external auditory canal of a human head model exposed to air shock waves. *National Defence Research Establishment (Stockholm) FOA Report*, C20802–23.

Kerr, A. G., Byrne, J. E. T. (1975) Concussive effects of bomb blast on the ear. *Journal of Laryngology and Otology* **89:** 131–143.

Kronenberg, J., Ben-Shoshan, J., Madan, M., Leventon, G. (1988) Blast injury and cholesteatoma. *American Journal of Otology* **9:** 127–130.

Kumagami, H. (1992) Endolymphatic hydrops induced by noise exposure. *Auris, Nasus, Larynx (Tokyo)* **19:** 95–104.

Morest, D. K. (1982) Degeneration in the brain following exposure to noise. In *New Perspectives in Noise Induced Hearing Loss* (Hamernik, R. P., Henderson, D., Salvi, R., eds.), Raven Press, New York, pp 87–93.

Pahor, A. L. (1981) The ENT problems following the Birmingham bombings. *Journal of Laryngology and Otology* **95:** 399–406.

Phillips, Y. Y., Mundie, T. G., Hoyt, R., Dodd, K. T. (1989) Middle ear injury in animals exposed to complex blast waves inside an armoured vehicle. *Annals of Otology, Rhinology and Laryngology* **98:** 17–22.

Pratt, H., Goldsher, M., Netzer, A., Shenhav, R. (1985) Auditory brain stem evoked potentials in blast injury. *Audiology* **24:** 297–304.

Roberto, M., Hamernik, R. P., Turrentine, G. A. (1989) Damage of the auditory system associated with acute blast trauma. *Annals of Otology, Rhinology and Laryngology* **98:** 23–34.

Singh, D., Ahluwalia, K. J. S. (1968) Blast injuries of the ear. *Journal of Laryngology and Otology* **82:** 1017–1028.

Stinson, M. R. (1985) The spatial distribution of sound pressure within scaled replicas of the human ear canal. *Journal of the Acoustic Society of America* **78:** 1596–1602.

Stuhmiller, J. H. (1989) Use of modelling in predicting tympanic membrane rupture. *Annals of Otology, Rhinology and Laryngology* **98:** 53–60.

Sudderth, M. E. (1974) Tympanoplasty in blast-induced perforation. *Archives of Otolaryngology* **99:** 157–159.

Teter, D. L., Newell, R. C., Aspinall, K. B. (1970) Audiometric configurations associated with blast trauma. *Laryngoscope* **80:** 1122–1132.

White, C. S., Bowen, I. G., Richmond, D. R. (1970) The relation between eardrum failure and blast induced pressure variations. *Space Life Sciences* **2:** 158–205.

Yokoi, H., Yanagita, N. (1984) Blast injury to sensory hairs: a study in the guinea pig using scanning electron microscopy. *Archives of Otorhinolaryngology* **240:** 263–270.

Zalewski, T. (1906) Experimentelle Untersuchungen uber die Resistenzfahigkeit des Trommelfells. *Zeitschrift für Ohrenheilkunde* **52:** 109–128.

Address for correspondence:
R. J. N. Garth, F.R.C.S.,
ENT Department,
Royal Naval Hospital Haslar,
Gosport,
Hants PO12 2AA.