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ACOUSTIC TRAUMA*

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General Remarks

IT has long been known that even brief exposures to high-intensity noise will produce a temporary hearing loss.

Similarly, cross-sectional studies on men working in noisy industries have indicated that these men usually exhibit hearing losses that are greater than in the average population.

It has never been clear however whether hearing loss in the individual case appears suddenly as a result of a particular acoustic insult or may in some cases develop gradually with repeated and prolonged exposure.

Only a study in which the same men are tested repeatedly (including an initial pre-exposure test) could possibly answer this question.

The term acoustic trauma is applied to all sound-produced injury of the auditory apparatus. Generally speaking, as the intensity of noise increases there are five important thresholds affecting human function that should be considered.

(a) The threshold of hearing which is equivalent to zero-hearing level.
(b) The noise level which may interfere with rest and sleep.
(c) The noise level which interferes with speech communication.
(d) The threshold of hearing damage risk. This is the noise level which after prolonged exposure of an individual to certain sound-pressure levels and frequency characteristics can produce noise-induced hearing loss.

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(e) The threshold of pain. Since pain is considered a sign of physiological damage, the noise level in the ear canal should never exceed the level of pain no matter how short the exposure period.

My remarks will be confined essentially to this topic but I think it will not be out of place if I referred to the nuisance value of noise which causes concern to the Noise Abatement Society. I suppose noise such as one encounters around airfields and which has given rise to numerous outbursts in the daily press could be regarded as a form of acoustic "trauma" in its broadest concept. A psychological trauma if you wish. At the same time there appears to be an unwillingness to accept certain inescapable facts of the situation. Aviation is here to stay and faster, heavier aircraft with a bigger pay-load entail engines with a bigger thrust and therefore much more noise. Until there is a major break-through in engine or airframe design, aircraft using, for example, London Airport will be extremely noisy and will require a long take-off run.

A considerable amount is being done to minimize annoyance to local residents by adhering to procedures which have been regarded by pilots as at times hazardous. I have no doubt that a pilot would welcome a noiseless aircraft which could take off and land slowly in a short distance. In fact, I should imagine he would welcome such an aircraft more enthusiastically than anyone else.

I am afraid however that for many more years anyone living near a major airport will have to tolerate a great deal of noise.

Now I will return to the main topic.

Injuries resulting from acoustic trauma are caused by industrial noise, gun fire, all detonations and explosions and, in some cases, by head injuries without fracture. There is a close similarity of the audiometric curves produced by noise or gun fire traumata, likewise those caused by closed head injuries. Explosions give rise to a functional loss of a different character. Noise and gun fire traumata cause little or no middle-ear damage, on the other hand explosive traumata often show extensive disruption of the tympanic membrane followed by middle-ear deafness without always producing labyrinthine damage.

Noise and gun fire traumata, and certain cases of closed head injuries, are characterized by early and constant appearance of a localized hearing loss in the region of 4000 c/s—the so-called C_5 dip—best seen on an audiogram. In advanced cases the dip broadens in the higher frequencies and to some extent in the lower frequencies and as a result of this the whispered voice becomes impaired first. The final effect of severe noise and gun fire injuries is a picture of perceptive deafness with a definite loss of high frequencies coupled with a raised bone conduction threshold.

Explosive trauma shows labyrinthine damage in addition to any middle-ear deafness caused by tympanic membrane rupture, but the typical C_5 dip is not present. The audiogram tends to drop steeply in this

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region and deafness is present in the upper 2-3 octaves. Explosive traumata may be accompanied by giddiness, a symptom which only very exceptionally follows noise or gun fire.

The most important characteristic of any kind of sound is the sound pressure wave forms which can be demonstrated on an oscilloscope.

In order to understand the effects of noise upon the middle ear it is necessary and important to know the distribution of the intensity throughout the sound spectrum and this can be shown by frequency analysis.

Sounds as they occur in nature can be classified as of steady characteristics such as tones which produce line spectra or transient characteristics which consist of broad band spectra.

The report from a gun which we classify as noise shows certain characteristics on the oscilloscope which qualitatively are similar to those of an explosion. The noise is of very short duration and of very high intensity. The noise starts with a steep pressure wave front followed by a more gradual fall of pressure until it reaches its starting point. This is followed in turn by a longer though less intense period of negative pressure.

There are however clinical distinctions between the traumatic effects inflicted by a gun report and those inflicted by an explosion. The peak pressure in an explosion is considerably longer than that due to gun fire. The acoustic energy at the drum is also correspondingly greater and the amplitude of the tympanic membrane displacement is considerably greater than that of a report with an equal sound pressure peak.

For example, small calibre fire-arms, such as pistols and rifles, produce a pure report trauma whilst in the case of guns of heavy calibre the trauma is also of the explosive nature.

Owing to post-mortem cell changes it is difficult to make comparative studies, consequently we have little knowledge about the histological changes which may occur in the human ear following noise or gun and rifle trauma. On the other hand some information is available of the effects of explosive trauma when the subject has been killed. Here we find the tympanic membrane destroyed and the ossicles fractured. The stapes shows multiple fractures and the footplate is driven into the oval window. Hæmorrhages have been observed in the vestibular connective tissue as well as rupture of Reisner's membrane.

To understand the traumatic effects of noise on auditory function we must measure, as stated, certain physical characteristics of sound. Sound itself consists of propagated fluctuations in barometric pressure. These variations in atmospheric pressure may be periodic or random and are called "sound pressures". Measurements of sound pressure may be used to indicate the amount of noise but we must remember that the amount is not the same as the energy in the noise. Temperature, for example, depends on heat but cannot be equated to the heat energy present in a body: likewise the energy in noise is related to but is not the same as

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sound pressure. The ear responds not to the energy of sound but to sound pressure, consequently noise measurement scales are based on sound pressure.

The smallest fluctuation of barometric pressure to which the young human ear will respond is about 0·0002 of a pressure unit, the microbar. Rüedi subjected guinea-pigs with a normal pinna reflex to a mixture of tones comprising all frequencies between 1,750 and 10,000 c/s—that is synthetic noise at a sound pressure level (S.P.L.) of 100 db. for periods of 1,600 to 2,200 hours. Immediately after the experiments the ear reflex was absent to any of the tested frequencies, but after a period of rest the reflex returned except for a persistent localized hearing loss between 3,000 and 8,000 c/s. The histological findings revealed a localized loss of outer hair cells from the end of the first turn till about the middle of the second turn of the cochlea. Subsequent identical histological findings in ears traumatized by noise strongly suggest that in a human ear a localized histological defect at one special point of the cochlea occurs and this is reminiscent of the C_5 dip.

Rüedi also found that pure tones produced much more widespread and rapid hearing loss than noise. Comparing the respective histological changes it was noted that the changes produced by a mixture of tones was always circumscribed and localized whereas pure tones, such as 800 c/s or 3,000 c/s gave rise to irregular, widespread areas of degeneration. Human ears exposed to pure tones at a S.P.L. of 130-140 db. for a period of 3-4 minutes showed at first a widespread hearing loss reaching a maximum of 1-2 octaves above the stimulating frequency. At the same time there occur alterations in pitch which are attributed to a displacement of the basilar membrane at the point of physical stimulation. Bekesy, who studied events within the cochlea from a hydrodynamic point of view, established the presence of eddies whose point of origin is dependent on the frequency and which probably stimulate Corti's organ.

Perlman and Kimura studied the cochlea bloodflow in acoustic trauma which they reported in the Acta of February, 1962. It is generally held that a rise in activity of functionally discrete areas in the nervous system as well as in other organs is accompanied by an increase in regional blood flow. Recent experiments in the cochlea using direct methods (oxygen and hydrogen electrodes) have been concerned with homeostatic mechanisms relating acoustic stimulation to permeability and blood flow in the stria vascularis. The oxygen tension in the cochlea during stimulation has been reported to be reduced (Misrahy *et al.* 1958). The authors observed increase blood flow only during very strong acoustic stimulation.

Their conclusion based on direct examination of the vessels of the stria vascularis during strong acoustic stimulation (120 db.) does not reveal either vasoconstriction or dilatation, while at the same time a marked restriction in microphonic response is recorded. An increase in

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blood flow rate in these vessels can be measured after very strong acoustic stimulation (135-150 db) when the microphonic response is further impaired or permanently abolished.

Some Clinical Observations on the Effects of Noise

We must differentiate from the start between acoustic trauma as an acute process and the hearing loss which is induced as a chronic process and which Eldrege thinks is not strictly speaking traumatic.

Acoustic trauma is characterized by a permanent threshold shift resulting from an injury either from a single exposure to an explosive episode or a brief exposure to a high-level noise. A feature of the environment is peak sound pressures in excess of 135 db. Examples of acoustic trauma are gun fire and jet engines. Dieroff studied the sonotraumatic effects of chiselling on the ear. The noise thus produced reaches intensities up to 130 db. and shows up in the impulse record as an explosion with a very high but short-lived energy peak so that in chiselling true acoustic trauma may develop.

On the other hand noise-induced hearing loss results from exposure over a period of months or years to a relatively steady machinery noise at over-all levels of about 105-110 db. in the frequency bands from 300 to 2,400 c/s.

When considering these points we are faced with some questions which must be answered.

(1) Is there really a risk of cumulative auditory effects on subjects exposed to high-intensity noise?
(2) How may these possible effects be detected in their early stages, both now and in the future?
(3) If cumulative ill-effects do exist, at what levels and with what duration of exposure do they become significant risks to subjects?

It is unsafe to extrapolate from present noise exposures to the more severe exposures that must be anticipated in the future. Brief exposures to most industrial noise will produce little or no damage to the ear. The otologist, when called upon to evaluate the causal relation of hearing loss to noise, must bear in mind certain factors. These are:

(1) The overall noise level to which the man was exposed.
(2) The frequency composition or spectrum of the noise, probably as expressed in octave band sound pressure levels.
(3) The duration and time distribution of noise during a typical work-day.
(4) The total duration of noise exposure expected during the work life.

It is still difficult to set a safe level of noise exposure because our knowledge about the relations of hearing loss to noise exposure is not

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sufficient. What we can state however is that whenever employees are exposed to continuous noise for at least 5-6 hours a day for 5 days a week, 50 weeks in the year for several years, then if the sound pressure level in any of the four octaves 300-600 c/s, 600-1,200 c/s, 1,200-2,400 c/s and 2,400-4,000 c/s exceed 85 db. a programme of hearing conservation must be instituted and put into effect. I need hardly add that this is primarily a medical responsibility.

The impairment to hearing caused by exposure to noise, whether it be in industry, in military service or in sport is a problem which is of increasing importance to the otologist. The loss is sensory-neural and loudness recruitment is present. Glorig and Davis have remarked that in spite of extensive high tone hearing loss present in cases they reviewed discrimination of speech was good. This, they say, is in contrast to what one finds in Ménière's Disease where recruitment is associated with poor discrimination, tinnitus and loss of tone quality. My experience over a considerable period with cases of hearing loss arising from exposure to noise does not accord with this view. I found discrimination of speech was poor and tinnitus was present.

I think we can state that the ageing process (presbyacusis) is independent of acoustic trauma. The age effect continues during the development of the noise effect and after the noise effect is complete. Once the permanent threshold shift is established in a worker he may as well continue to work indefinitely in that or any lesser noise but if he moves into a more intense noise he must expect another increase in his permanent threshold shift.

The 4,000 c/s dip is the most characteristic pattern of noise-induced hearing loss whereas the advancing age audiogram shows a gradual slope.

Studies under varying conditions and intensities of noise have been made. Van der Waal observed engine-room personnel and found that 91 per cent. had a threshold shift of 25 db. or more. When compared with threshold audiograms of a comparable group of deck personnel who are exposed to noise well below the harmful level there is a significant difference in threshold shift. He further observed that the threshold shifts do not increase proportionally to the exposure duration of the noise. A particular threshold is reached which hardly shows any further increase during the next years.

Kylin investigated the temporary threshold shift (T.T.S.) and auditory trauma following exposure to steady-state noise. Observations were made on 220 young individuals with normal hearing who were exposed to octave bands of noise during a two-hour period, ranging from 75-150 c/s to 4,800-10,000 c/s and sound pressures ranging from 70-115 db. Ten subjects were exposed to each octave band pressure level. An audiogram was taken immediately before and another 15 minutes after exposure. It was shown that the 75-150 c/s band needed about a 20 db. higher octave band

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pressure level than the 2,400-4,800 c/s or the 4,800-10,000 c/s bands to produce a significant temporary threshold shift. It was further shown that a close relation exists between the rate of recovery and the initially measured threshold shift and that the capacity for recovery appears to differ in different individuals.

In order to examine the relation between T.T.S. and permanent noise deafness, Oppliger et al. examined personnel between the age of 16 and 20 during their apprenticeship in the metal industry. The T.T.S. was measured after exposure to white noise of different intensities. The hearing loss was tested annually. No correlation was found between T.T.S. of a patient and his permanent hearing loss at 4,000 c/s.

Gravendeel investigated the influence on the human ear of noise produced by fire-arms and concluded that there was no simple relation between the hearing loss shortly after firing (mainly temporary) and the permanent hearing loss ultimately remaining. This view they based mainly on the difference of average location of the permanent and temporary dips in the continuous audiogram.

Two possible assumptions were made: *firstly*, temporary and permanent dips each had their own genesis, and *secondly*, the permanent dips arose from the temporary dips by incomplete and asymmetrical recovery. In the case of continuous noise, e.g. Diesel engines, it seems the reversible and irreversible dips were possibly brought about by the same mechanism and that the permanent dips derived from the temporary dips by incomplete but symmetrical recovery.

Nixon and Glorig studied the noise-induced permanent threshold shifts (P.T.S.) at 2,000 and 4,000 c/s. Samples of industrial workers were drawn from environments in which continuous steady-state noise had an average S.P.L. in the octave bands 150-4,800 c/s that ranged from 77-96 db. Length of time on a job and age effect were taken into consideration. Median hearing levels were calculated for 2,000 and 4,000 c/s.

It was shown that:

(a) There is a maximum noise-induced P.T.S. at 4,000 c/s which occurs within the initial ten years of exposure to eight-hour day exposure to continuous steady noise of any intensity greater than 70-75 db. in the 1,200-2,400 or 2,400-4,800 c/s octave band.
(b) There is no apparent maximum noise-induced P.T.S. occurring at 2,000 c/s within the exposure ranges studied.

Boenninghans and Roser (1962) investigated the sensitivity to noise in ground staff of big Airports from Boeings 707 (Turbojet) and Vickers Viscounts (Turboprop). The sound intensity was measured at a distance of 10 m. (32 ft.) when starting up and in idling aircraft. Levels up to 120 db. were obtained. Frequency analysis showed the greatest sound intensity at 3,000 c.p.s. Observation showed a T.T.S. of 20-30 db. at C_5

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after a single exposure for 10 minutes at a distance of 32 ft. from the jet of the Boeing. 382 men of the ground staff were examined having been employed at the Airport (Frankfurt) for varying periods and giving no previous history of hearing loss. Pure tone audiometry observed increasing hearing defects in proportion to the length of exposure to noise and varying from several weeks to 10 years.

The downward threshold shift was more marked in men who had been employed for long periods than in those who had only worked in that environment a few months. There was a markedly larger number of employees who became deafened within the first few months; in the long service personnel the incidence of deafness was less.

King recently reviewed the incidence of occupational deafness in the Royal Air Force for the year 1954. The figures he obtained relate to cases referred for specialist opinion either at hospital clinics or at the Central Medical Establishment. In all, 385 case histories of patients with hearing loss were reviewed. These yielded 170 cases of conductive deafness and 215 cases of perceptive deafness of all types. The 215 cases of perceptive deafness were broken down and those cases listed as of occupational origin were recorded as such by the examining otologist at the time of the examination.

Thus are found:	Occupational deafness	136 cases
	Perceptive deafness of known ætiology other than occupational	53 ,,
	Perceptive deafness of unknown origin	26 ,,

In 1954 occupational deafness constituted 35·3 per cent. of all forms of deafness and 63·2 per cent. of all cases of perceptive deafness. It is the greatest single cause of deafness and the precipitating cause was found to be as follows:

Small arms, blast, etc.	59 (43 per cent. of occupational deafness)
Other noises, aircraft engines, etc.	77 (57 per cent. ,, ,,

Men of many branches were involved in this Survey and the branches with the greatest number of men involved were:

Engineering, R.A.F. Regiment, Armaments and Aircrew.

And now I want to draw attention to the further destruction of partially-deafened children's hearing by the use of powerful hearing aids, which was reported by Kinney recently. In 1953 he reported 16 cases of progressive sensori-neural hearing loss and stated that the progress was not noted until a wearable hearing aid had been put on these children and in every instance the increased loss was more marked in the ear in

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which the hearing aid was used. The question of hearing aid trauma was raised on several occasions but the possibility was invariably deprecated. In 1957, however, Silverman stated that the evidence from the relation of hearing loss to noise exposure suggests the possibility of damage to an ear exposed to the level of sound pressure generated by a hearing aid.

It is known that hearing aid manufacturers vie with one another to produce hearing aids which are smaller, more disguisable, more powerful and with greater fidelity. Claims are made that these aids have a maximum acoustic gain of 80 db. and a power output of up to 140 db. The idea of fitting children between the ages of 2 and 12 with powerful binaural hearing aids received and still receives some attention in certain quarters. It is claimed that speech intelligibility is considerably increased. In the education of the totally or sub-totally deafened child high intensity binaural amplification may prove useful. It is a matter of nothing to lose and with the possibility of some gain.

Kinney carried out hearing tests on partially deafened children who had been wearing powerful hearing aids both of the monaural and binaural types. He laid down certain criteria:

(a) The loss is of the sensori-neural type.
(b) The aid was worn for a year.
(c) Threshold hearing tests were carried out prior to the use of the aid.
(d) Two similar tests were carried out after 6 months use of the aid.

Otological screening was carried out and each child had normal or nearly normal speech. Observation was carried out in 198 selected cases, of which 146 were in a control group and had been using a monaural hearing aid which was not particularly powerful. The remaining 52 had used a powerful one and in certain instances a binaural hearing aid.

Of the 146 cases 10·3 per cent. showed a further loss of hearing in the ear in which the aid had been used.

Of the 52 cases 39 used their aid in one ear only. Of these 39 (48·8 per cent.) showed an increased loss of an average of 20 db. in the used ear. Of the 52, 13 had been using a binaural hearing aid, and of these 69 per cent. showed an average increased loss of 25 db. in each ear. One could logically ask in how many of these cases would there have been an increased loss if the hearing aid had not been used. This is not an easy question to answer.

As otologists, we must consider the ætiology of the hearing loss. It is probable that the hereditary case of hearing loss is the least likely to suffer hearing aid trauma.

Kinney sums up his conclusions as follows:

(1) In sensori-neural partially deafened children no hearing aid of more than 40 db. gain should be used in spite of the fact that a more powerful aid may possibly give better speech hearing im-

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provement at the first fitting. (I think that perhaps it would have been better if he had placed a restriction on the total output of the aid (say 120 db.) rather than on the gain.)

(2) The use of binaural amplification in such children should be condemned.

(3) Children between the ages of 5 and 16 who wear a hearing aid should have their hearing evaluated by an otologist at least every 6 months.

I would also bring to your notice the risk of exposing ears which have been subjected to tympanoplasty or stapedectomy to sound pressure levels which, though regarded as safe for normal ears, could be dangerous to operated ones. Whatever may be the advantages bestowed by these operations, alterations of the mechanics in sound transmission carry this risk.

The field of acoustics is not confined to the classical auditory frequency spectrum but has taken into its realm the ultrasonic range and at the other extreme the infrasonic bands of mechanical vibration. We are developing more and more powerful sources of energy so that in some parts of the spectrum we are reaching the limit at which it becomes physically impossible for the medium in which we wish to work to transmit higher levels of energy. At these high intensity levels new phenomena emerge which may dominate the picture.

Acoustic energy has its biological effects and the possibility of serious effects on man has been forced on our attention by the development of the jet, turbo-jet, ram-jet propulsion plants for aircraft and by the attainment of near-sonic and in the near future to supersonic velocities by aircraft so propelled.

Aerodynamic noise may be a powerful source of ultrasonic as well as sonic energy. In our consideration of biological effects of noise we must consider discomfort thresholds in addition to danger zones. To be useful any such observations must be accompanied by approximate intensity measurements and spectral analysis. The concept of a discomfort threshold implies the stimulation of sense organs, and each sense organ will have at least one frequency band to which it is most sensitive. For example, somewhere near the lower end of the auditory spectrum is the resonant frequency of the eyeball. Blurring of vision is likely to be greatest at this frequency.

I will only refer very briefly to the subject of hearing loss in relation to compensation. This opens a field for considerable discussion. No unanimity exists, even among those who have legislated for this occupational hazard. A primary assumption is that compensation for impairment of hearing need not be paid because there is a measurable shift in the sensitivity of hearing but only when there is significant handicap in the hearing of every day speech. Said in another way, compensation should

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be paid for practical impairment of hearing, but impairment does not begin at the zero of the audiometer scale. In the U.S.A. there appears to be an increasing agreement that hearing impairment for purposes of workmen's compensation should be based upon the hearing threshold levels of the 3 frequencies 500, 1,000 and 2,000 c.p.s. These frequencies are stated to represent the band most important for the understanding of speech. (I believe that 3,000 c.p.s. should be included. Recent speech tests on persons with a high tone hearing loss strengthen this suggestion.) In spite of many severe 4,000 cycle dips and some threshold shifts at 3,000 c.p.s. good scores were obtained in word discrimination. A Committee on conservation of hearing recommended that hearing impairment should be considered to begin only when the average hearing level for the three frequencies 500, 1,000 and 2,000 c.p.s. exceed 15 db.

I must however make very brief reference to the prevention of the ill-effects of noise. These can be considered under three headings:

(1) Avoidance of noise.
(2) Reduction of noise.
(3) Individual protection.

I will deal with the latter. With increasing S.P.Ls. I strongly favour the ear muff rather than the ear plug or warden. As a rule there is a wide difference of attenuation values in ear plugs. The reason is that some people get a good fit and some do not. In general the goodness of a fit and the amount of attenuation are directly proportional to the amount of discomfort the wearer can tolerate. The ear plug is as a rule inserted at a minimum distance: loosely fitting plugs may be comfortable to wear but they will not attenuate noise.

In recommending ear muffs I must stress that their attenuating properties are related to mass, volume and adequacy of seal. The sealing rings which give good attenuation are those with a free-flowing liquid and which must be airtight. One of the major factors which affects the attenuation of ear muffs, particularly at low frequencies, is the ability of the muff to seal itself to the head in the area surrounding the ear, thereby reducing leakage of sound into the cup. The head-band force should be great enough to maintain the sealing action without causing discomfort.

May I finally summarize some of the conclusions I have reached after being interested for nearly 25 years in the problem of acoustic trauma.

(1) There can be no doubt that an exposure to noise of an intensity in excess of 90 db. will ultimately give rise to some degree of hearing impairment.
(2) I am not convinced that temporary threshold shift is a basis on which to assess or forecast the permanent threshold shift. I think that the temporary threshold shift, if it becomes evident in the

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early stages of exposure to noise and recovery is prolonged, may be an indication of the susceptibility of the individual to noise.

(3) The necessity of the pre-employment evaluation of the hearing status of anyone about to engage in any form of noisy occupation is a *sine qua non*. Thereafter periodical checking of auditory function is essential, especially if a change of occupation becomes necessary. I need hardly stress the necessity of standard conditions of testing, properly calibrated standard equipment conforming to B.S.I. specifications, and reliable technical staff. These should be objectives of medical responsibility in order to identify and treat those cases of hearing loss that are amenable to treatment particularly those hearing losses that are not due to noise exposure, the prevention of significant noise induced hearing loss and the proper ear protection and utilization of those who do incur hearing loss from any cause.

(4) With rising S.P.L. the conventional means of ear protection, viz. ear wardens or ear muffs will soon become obsolete. Some other measures of protection against the effects of intense noise will have to be considered.

(5) In assessing any degree of hearing impairment I believe that the only practicable and realistic method is speech audiometry. Speech audiometry carried out preferably in a free field binaurally is, in my opinion, the only fair method of assessing a hearing disability. The linguistic material is important and should be made up of meaningful words, consisting of a consonant, vowel, consonant and should be based on the frequency occurrence of certain sounds in the English language and the frequency with which certain sounds are likely to be mistaken. Responses at threshold intensities between different tones of different pitch and differing intensity is no real indication of the ability to understand and discriminate speech. There is a relation but no correlation between the two.

(6) And in conclusion may I make a plea for a better understanding by otologists of the problem of noise-induced hearing loss—from all its aspects. This has been left too much in the hands of physicists, acoustic engineers, psychologists and audiologists. All these have a contribution to make but it is the otologist who must ultimately make the final assessment as to cause and effect when any dispute arises involving compensation. In the witness box he must be familiar with all the implications of the problem if he wants to avoid discomfiture during cross-examination.

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