Noise Pollution
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CHAPTER 7
*Auditory After-effects of Noise**

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7.1 INTRODUCTION

The most obvious effects of intense or prolonged noise were known at least 350 years ago to Francis Lord Bacon (1627), who wrote that 'A very great sound, near hand, hath strucken many deaf'. Furthermore, he had himself experienced a temporary partial loss of hearing coupled with a tinnitus: '... myself, standing near one that lured [whistled loudly to call back a falcon] loud and shrill, had suddenly an offence, as if somewhat had broken or been dislocated in my ear; and immediately after a loud ringing (not an ordinary singing or hissing, but far louder and differing) so as I feared some deafness. But after some half quarter of an hour it vanished'.

Thus, even in Bacon's time the temporary and permanent manifestations of acoustic trauma — effects produced by a single, very intense exposure — were well known. On the other hand, a slow but progressive loss of hearing associated with extended exposures to less intense noises was apparently not as clearly recognized, as suggested by this observation of Bacon's: 'It is an old tradition, that those who dwell near the cataracts of Nilus are strucken deaf, but we find no such effect in cannoniers, nor millers, nor those that dwell upon bridges.' Perhaps cannons and mills were quieter in Bacon's day than they became later. With the Industrial Revolution, at any rate, noise sources became ever louder, so that by 1830 (Fosbroke, 1830), in a review of what was then known about deafness, could refer to damage to hearing 'caused by continued noise, as blacksmith's deafness' as though it were already an established syndrome. However, despite the length of time that noise-induced hearing damage has been recognized, the precise relation between the parameters of the acoustic exposure (intensity, duration, spectral characteristics, and temporal pattern) and the resultant loss of sensory capability is still highly controversial.

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7.2 IMPAIRMENT AND HANDICAP

It is generally accepted that the most serious consequence of noise exposure is indeed partial deafness, particularly insofar as such a loss of hearing produces a social handicap. Unfortunately, there is no widespread agreement on how this social handicap is to be defined and measured. Until the middle of this century, a hearing loss was regarded as handicapping, and therefore compensable as being the result of an occupational hazard, only if it was disabling, in that it led to a loss of earning power of the individual, a condition whose existence could be relatively easily and unambiguously determined. Since then, however, with the gradual acceptance of the principle that a worker is entitled to compensation for any material impairment suffered as a result of employment, handicap has come to be any condition that interferes with everyday living.

Representative of this viewpoint is the definititon of handicap adopted by the American Academy of Otolaryngology: 'an impairment sufficient to affect a person's efficiency in the activities of daily living' (Anon., 1979). Since 'activities of daily living' include so much, yet vary so widely from person to person, recent practice has been to attempt to measure auditory handicap in terms of a reduction in the individual's ability to understand ordinary speech, a simplification that ignores, among other things, the social significance of the perception of warning signals, sounds of nature, and music.

However, even this oversimplification fails to solve the problem, because there is no accepted definition of 'ordinary speech'. Speech consists of messages of various degrees of complexity and redundancy, spoken by talkers differing in age, sex, ethnic background, education and dialect, at a large range of sound levels, in quiet and in the presence of a near-infinite variety of interfering noises. Any test that claims to measure an individual's ability to understand ordinary speech would have to include enough test items to provide a representative range of all these parameters — providing that agreement could be reached on what 'representative' means. No such direct test has yet been developed.

At the present time, therefore, hearing ability is still assessed in terms of the ability to hear pure tones, particularly those at frequencies most important to the understanding of speech, viz., 500 to 4,000 Hz. *Impairment* of hearing is measured by the individual's Hearing Threshold Levels (the number of decibels by which the sound intensity must be increased to be heard, relative to 0 dB Hearing Level, which is the sound level that can be heard by an ear that presumably has never been affected by any deleterious agent) at these frequencies. *Handicap* is then defined in terms of the number of decibels by which the Hearing Threshold Levels (HTLs) exceed the 'low fence', a fixed although somewhat imprecisely-determined empirical line that, in theory, separates individuals who have 'no' difficulty in understanding conversation from those who have 'some' difficulty, by self-report. Just how well this line of

demarcation based on pure-tone thresholds actually divides individuals in terms of speech understanding is a question still being hotly debated; the low fences actually used in different countries and states vary widely. Noble (1979) has recently reviewed the problem of handicap and its measurement in some detail. But because the audiogram is the easiest and best-standardized test of ability to hear that can be administered to large numbers of people, it will doubtless continue to be used to assess the auditory 'after-effects' of noise exposure, no matter what method of relating pure-tone thresholds to handicap is employed.

7.3 HEARING DAMAGE

7.3.1 Tinnitus

Inability to hear weak sounds is not, of course, the only manifestation of damage from noise. Its opposite, in a sense, is also a frequent result: the hearing of sounds that do not exist. Tinnitus, the ringing in the ears mentioned by Bacon, is a common accompaniment of noise-induced hearing loss. However, although intractable tinnitus is often distressing to the individual concerned, its measurement is difficult. Furthermore, only seldom does noise cause a permanent tinnitus without also causing hearing loss. So although the person with tinnitus should probably be considered more impaired than someone with the same audiogram but without tinnitus, the question of how *much* more impairment a particular tinnitus represents has not yet been solved.*

7.3.2 Paracusis

In addition, some sounds may be heard, but heard incorrectly. Musical paracusis is said to exist when the pitch of tones near a region of impaired sensitivity due to noise is shifted; i.e., a tone is heard, but one having an inappropriate pitch. Unfortunately, direct measurement of paracusis is possible only in highly musical persons, so the phenomenon has received little attention. If the paracusis is greater in one ear of a given individual than in the other, then *binaural diplacusis* will be found: a particular tone will give rise to different pitches in the two ears, and the magnitude of this difference can be inferred by having the listener adjust the frequency of a tone in one ear to match the pitch of a fixed tone in the other. Again, however, noticeable degrees of paracusis or diplacusis that are attributable to noise exposure only occur in conjunction with a considerable loss of sensitivity, so the importance of paracusis *per se* in determining social handicap is still unknown.

* Chapter 6 by Evans, E. F. (in this volume).

7.3.3 Speech Misperception

Noise-induced hearing loss, it will be shown later, is frequency-specific, almost always involving high frequencies, especially around 4 kHz, much more than low frequencies. The noise-impaired auditory system therefore acts, broadly speaking, like a filter, so that the spectrally complex sounds of speech, particularly consonants may be heard, but heard incorrectly. For example, in a study designed to find differences in the associated auditory characteristics of high-frequency losses presumably caused by steady noise *vis-a-vis* similar losses caused by gunfire, individuals with a high-frequency loss that began at 2,000 Hz consistently heard an initial 't' as a 'p'; e.g., when given the word 'tick', with no opportunity to see the lips of the speaker, they almost always responded 'pick' when forced to choose between 'tick' and 'pick' (Ward *et al.*, 1961). In recent years, these misperceptions of speech sounds have received increasingly greater attention, and eventually some standardized consonant-confusion test may be adopted as a part of a battery of speech tests designed to assess the ability to 'understand ordinary speech'.

7.3.4 Physiological Measures of Hearing Damage

All of the preceding indicators of damage to hearing are associated with a measurable loss of threshold sensitivity. However, damage to the auditory mechanism may occur without affecting the threshold. Evidence from animal studies, for example (Henderson *et al.*, 1974), implies that several hundred of the hair cells that have been presumed to be important in the process of hearing may be destroyed before a change in threshold is measurable. This clearly casts doubt on the adequacy of threshold sensitivity as the only behavioural indicator of damage. To date, however, efforts to find a psychophysical indicator that is more sensitive to physiological damage have been unsuccessful, although many have been proposed, such as the ability to (1) identify small changes in intensity or frequency of a signal (difference limens), (2) discriminate signals from interfering sounds (masking), or (3) detect longer signals more easily than shorter ones (temporal integration). The problem is that the existence of diffuse hair-cell destruction can be validated only in experimental animals, while reliable estimates of difference limens, masking, and temporal integration can easily be obtained only in man. Until such time as some method for determining hair-cell loss in the intact organism is developed, therefore, the auditory threshold remains the most dependable indicator of the physiological normalcy of the auditory system, just as it is the most easily measured indicator of its normal functioning.

7.4 THRESHOLD SHIFTS, TEMPORARY AND PERMANENT

If the auditory threshold is measured before and after a noise exposure, giving

values of HTL_0 and HTL_t, respectively, then the difference HTL_t-HTL_0 is by definition the threshold shift (T), which may be temporary (TTS) or permanent (PTS). The term *noise-induced* may legitimately be attached if it can be established that no other reason for the difference exists. Thus a TTS associated with, for example, an eight-hour exposure to noise in an iron-smelting plant can reasonably be termed a noise-induced TTS (NITTS) provided that: (1) the same audiometer was used for both the pre- and post-exposure tests, and its physical characteristics did not change; (2) the testing technique, recording technique, and ambient noise level in the test-room were adequate and invariant; (3) the subject's criterion for deciding when he heard a tone was same (i.e., there was no 'learning'); (4) body noises in the subject's head were not different (e.g., he did not have a pounding heart because of exertion just prior to either test); and (5) the heat and fumes from the molten metal did not influence the threshold directly. While the first two requirements can be met fairly easily, that the others were fulfilled can be verified only by the use of appropriate controls. In this case, such a control group would be a group of workers exposed to all the same conditions except for noise. Complete elimination of the noise being impossible, the best solution would be to require the use of well-fitted ear protectors by half of the workers on one day of testing, by the other half on another, in order that each worker could serve as his own control.

Laboratory tests of TTS are usually conducted with conditions so well regulated that a formal control experiment is usually not necessary except for assessing 'learning effect' when initially naive test subjects are used. Because of the ease of performing such research, there is a considerable body of knowledge concerning the effects of spectrum, level, duration, and temporal pattern of exposure on NITTS in man: the pre-exposure Hearing Threshold Level HTL_0, the noise exposure NE, and the post-exposure HTL_t can all be accurately measured, and the influence of extraneous factors on the threshold can be minimized (Ward, 1970).

For NIPTS, however, this is not the case, because NIPTS in humans cannot be produced in the laboratory, but must be inferred from field observations. In most attempts to measure NIPTS, the only measurement which can often be confident is the post-exposure threshold HTL_t, and even this will be true only when: (1) it has been measured correctly, i.e., using calibrated apparatus and standard procedures; (2) there has been no recent exposure to a TTS-producing noise; and (3) adequate steps have been taken to ensure that malingering will be detected, particularly when the results of the measurement are likely to be used to determine the amount of compensation to be awarded the worker for hearing loss.

Even when HTL_t has been accurately determined, PTS cannot be calculated unless HTL_0 is also known, and until recently this was seldom the case. Hearing was measured only *after* something had happened to it, not before. Next, in

order to determine how much of the PTS is a NIPTS, we must somehow correct it for *presbyacusis*, the gradual deterioration of sensitivity with age, and for *nosoacusis*, the effect, over the time concerned, of all damaging influences other than aging or noise: industrial chemicals like benzene, carbon disulphide, carbon monoxide, or aniline dyes (Lehnhardt, 1965); ototoxic drugs such as kanamycin and streptomycin; illnesses, especially chronic middle-ear infections; hereditary progressive hearing loss; and traumata such as blows to the head.

The final problem to be encountered is that in order to be of any practical use, the NIPTS must be related to the noise exposure NE, and characterization of NE poses formidable problems. People are exposed to noises of various levels, spectra, durations, and temporal patterns in the work situation, and then are also exposed to similarly diverse noises on the way to and from work, at home, and while engaging in hobbies. The term *sociacusis* is applied to hearing loss attributable to these noises of everyday life — i.e. those outside the work environment. The mere recording of the noises entering a single ear over a period of years would be nearly impossible (indeed, it has so far never been attempted), and even after that were done, one would still be faced with the problem of reducing all that information to a manageable number of significant parameters.

7.4.1 PTS from Steady Noise Exposures

As a result of this complexity, about all that is known about the relation between noise exposure and NIPTS in man concerns the effect of reasonably constant industrial noise environments on large numbers of workers who presumably started with normal hearing. Certain industries have used the same machines for many years, and in some of these industries the workers remain at the same job for a lifetime. It is therefore possible to select two groups of workers, one of which worked in this noise environment for a known number of years, while the other worked in 'quiet' conditions. If the ages of the groups are matched, then presbyacusis will have affected each group to the same degree. If, in addition, a questionnaire reveals that certain nosoacusic agents in the aggregate history of the groups are about equal, then it may be postulated that any difference in HTLs between the groups is due to their total noise exposure, and is in fact NIPTS. Finally, if it can be assumed or demonstrated that both groups are also equally exposed to noise outside the work situation, and so presumably have the same sociacusis, then the difference in HTLs can legitimately be called the *industrial* NIPTS (INIPTS), provided of course that one accepts the proposition that all of these influences are simply additive — i.e., that an individual's HTL_f is equal to the numerical sum of the changes of HTL, in decibels, that would have been caused by presbyacusis alone, the changes ascribable to nosoacusis alone, those attributable to sociacusis alone,

and those due to the industrial noise alone. There is no evidence, either theoretical or empirical, to support the notion of simple additivity; it has been accepted for so long, however, that its replacement by some other principle will require overwhelming evidence.

Passchier-Vermeer (1968) summarised in a single document the results of the cross-sectional (retrospective) hearing surveys that came close to meeting the stipulations listed above: steady and relatively invariant noise environments to which a significant number of workers had been exposed eight hour/day for many years. It must be pointed out, however, that even these few surveys did not use exactly the same rules for exclusion of individuals from analysis (other than a history of working at more than one noisy job), e.g., middle-ear problems or demonstrated conductive hearing loss, family history of hearing loss, military service, noisy hobbies, etc. So it is seldom really clear of what section these groups of workers were a representative sample. Furthermore, in some of these surveys adequate provisions were not made to ensure exclusion of TTS from the data (by measuring thresholds only before beginning work, or by requiring the use of ear-protectors until the audiogram was made). In others, there is no clear evidence that the testing environment was quiet enough so that if a worker happened to have better than 'normal' hearing, it could be measured. Finally, the control groups used by the authors of the surveys were often either inadequate or, in some cases, non-existent. In such cases, Passchier-Vermeer employed a set of 'average presbyacusis' curves developed by Spoor (1967) in a comparable synthesis of retrospective surveys of non-industrial, noise-exposed persons, in order to correct the data for aging plus some sociacusis and some nosoacusis. These average presbyacusis curves for males are shown in Figure 7.1, for females in Figure 7.2. Thus, for example, if a male worker of age 50 has a HTL at 6,000 Hz 20 dB higher than non-exposed males 25 years of age, his inferred industrial-noise-induced hearing loss would be zero. It must be emphasized that these curves represent neither the *sole* effect of age, completely untainted by sociacusis or nosoacusis, nor the *combined* effects of age, disease and non-industrial noises, but something between the two, probably closer to the latter. Robinson and Sutton (1979) have recently developed some equations that fit the Spoor curves fairly closely; however, it must be remembered, if these equations are actually adopted in an ISO document, that their use as a correction factor for industrial data will always leave the industrial-noise-induced loss still confounded with some degree of sociacusis and nosoacusis. It is unfortunate that there still exists no survey, performed under meticulously-controlled conditions, of the hearing of a random sample of the population of a particular country from which the *only* reason for exclusion was a 'yes' answer to the following question: 'Have you ever worked more than a few days in a noise so loud that you had to raise your voice in order to be heard?' Only with such a survey can the true combined effect of presbyacusis, nosoacusis and sociacusis be determined, and a realistic determination of industrial NIPTS be made.

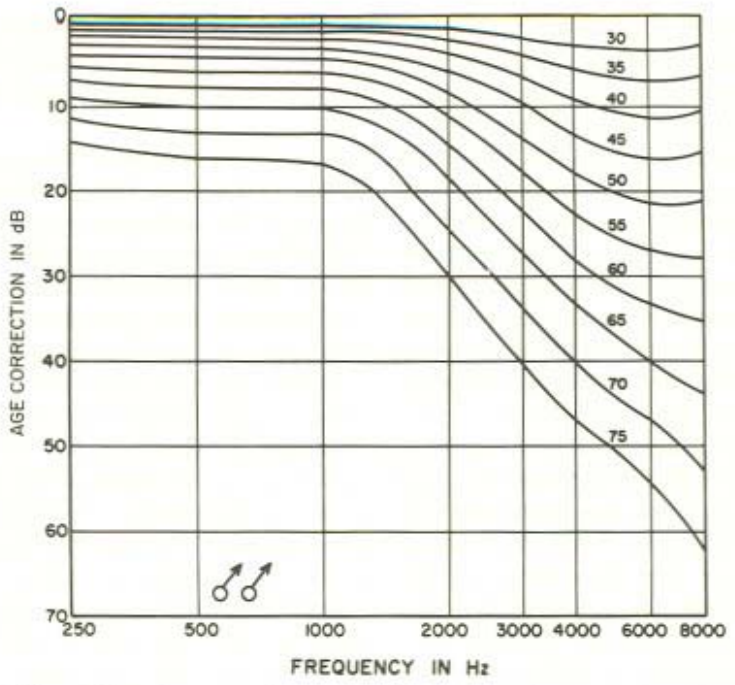

Figure 7.1 Age-correction curves for males, after Spoor (1967). The ordinate indicates the difference between the Hearing Threshold Level of a person of a given age (parameter) and that of an average 25-year-old, as a function of frequency (abscissa). These correction values incorporate not only the effects of the aging process *per se*, but also assume a moderate amount of sociacusis and nosoacusis

Despite all the deficiencies of the survey data, Passchier-Vermeer's analysis showed surprisingly consistent results. When the inferred INIPTS of the aggregate data, as determined by applying Spoor's correction for 'presbyacusis', were related to the overall A-weighted level of the noise, the results showed a clear linear relation. It must be mentioned, however, that a similar result was found when the exposure was expressed in terms of NR ratings, and Kraak (1979) has recently shown that these same data will fit his hypothesis that NIPTS is proportional to the integral of the sound pressure over time. Apparently many functions are consistent with this survey data, and determining the ultimate relation between exposure and resultant INIPTS is still to be accomplished.

However, the relation between A-weighted level and NIPTS, with duration of exposure held constant, is sufficiently simple that it lends itself well to the setting of exposure limits for industrial workers. In Figure 7.3, the ordinate shows the inferred INIPTS at 4 kHz, the frequency most severely affected by

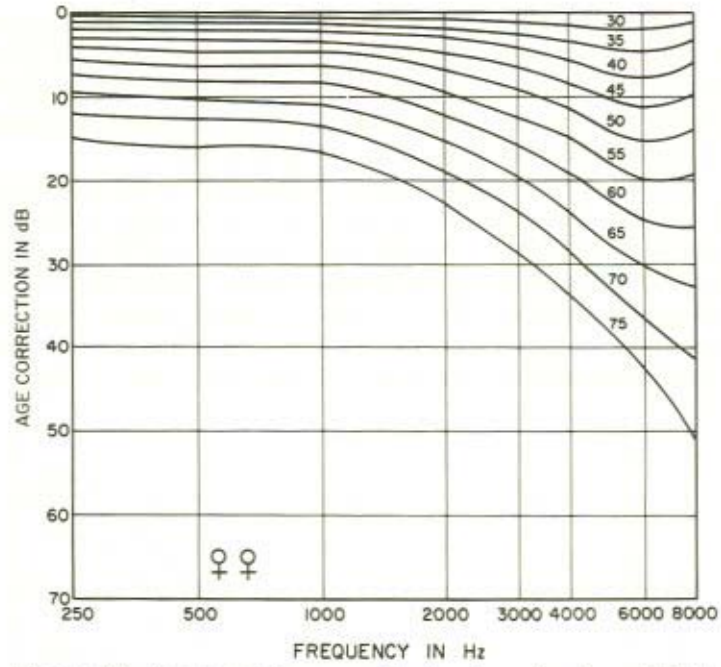

Figure 7.2 Age-correction curves for females, after Spoor (1967)

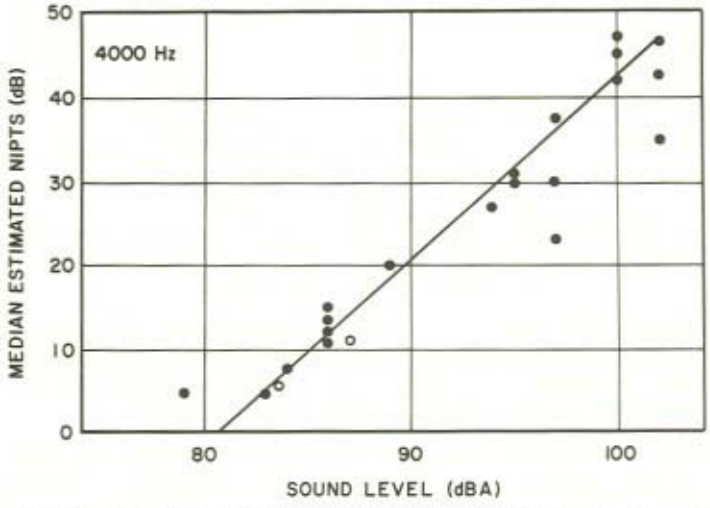

Figure 7.3 Estimated industrial-noise-induced permanent threshold shift at 4 kHz produced by 10 years or more of exposure to noise at the indicated A-weighted level. Solid points indicate values calculated from the literature by Passchier-Vermeer (1968), and open points represent recent studies by Robinson *et al.* (1973) and Yerg *et al.* (1978)

noise, after 10 years of exposure to a steady industrial noise environment whose A-weighted level is given on the abscissa. It can be seen that a reasonable fit to the data is given by a straight line passing through 0 dB at 80 dBA and 20 dB at 90 dBA. The point at 78 dBA can safely be ignored, as it represents only 14 ears of seven workers (Kylin, 1960).

The accuracy of this summarization is indicated by the two open circles, which represent the results of two subsequent large-scale studies of workers employed in levels of 80 to 90 dBA. Robinson *et al.* (1973) found that textile workers who had an 83-dBA environment showed a loss about 5 dB greater than a control group who worked in 70 dBA or below, and a recently concluded study involving several industries found a loss of 11 dB in workers whose daily A-weighted exposure levels were about 87 dBA (Yerg *et al.*, 1978).

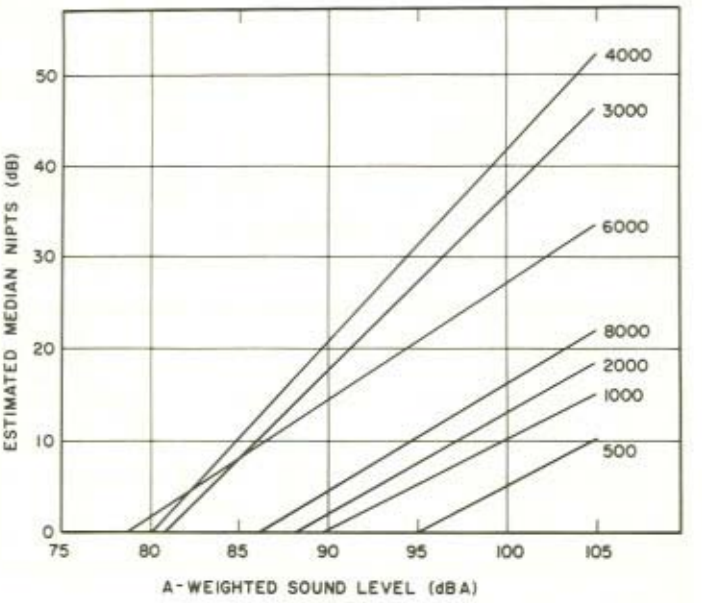

Figure 7.4 Estimated industrial-noise-induced permanent threshold shifts at various frequencies produced by 10 years or more of exposure to noise at the indicated A-weighted level. From data summarized by Passchier-Vermeer (1968)

The set of curves relating the growth of median INIPTS at all of the normal audiometric frequencies to the A-weighted level (eight hours per day steady noise, interrupted only by normal rest and lunch breaks, five days per week for 10 years or more) is shown in Figure 7.4. As a first approximation, it appears that: (1) 80 dBA is the level that can be regarded as being innocuous; (2) 85 dBA will result in a loss of about 10 dB at the most noise-sensitive audiometric frequencies of 3, 4, and 6 kHz (10 dB is the smallest loss in the individual case

that could be regarded as meaningful); (3) 90 dBA will generate a loss of 20 dB at these frequencies, although the traditional speech frequencies (500, 1,000, and 2,000 Hz) are still largely unaffected; only above 90 dBA does the noise adversely affect these lower frequencies; and (4) noises of 100 dBA, which are still common, will produce severe losses at high frequencies, and also moderate losses at the low frequencies — losses that, when added to the effects of presbyacusis, nosoacusis, and sociacusis, will produce a hearing handicap in a large proportion of those workers so exposed.

At present, 90 dBA has been adopted as the eight-hour exposure limit in most countries, although 85 dBA is advocated by those who feel that a 20-dB loss at high frequencies is too great to be tolerable. Those holding this view point out that the figure of 20 dB is only an average one, one that does not take individual differences into account. When the average INIPTS is 20 dB, a good many workers will have lose 30 dB, and a few even 40 dB (and, of course, some will have lose none at all). In order to protect the most sensitive individuals, it is contended, the exposure limit should be set at a lower value than merely what is necessary to protect the average worker. As the distributions of inferred NIPTSs were estimated by Passchier-Vermeer, her statistics can be used to predict what fraction of the exposed population will suffer any given amount of INIPTS, but at that point the issue ceases being a scientific one and becomes a political question, as the exposure limit adopted must depend on what INIPTS is deemed 'acceptable'. Millions of dollars have been squandered in arguments over what is acceptable.

7.4.2 PTS from Intermittent Noise

Figure 7.4, regrettably, represents just about everything that is known about the relation between noise exposure and NIPTS in humans. Furthermore, it is unlikely that any further direct evidence will be gathered on the topic, except for exposures below eight hours at 90 dBA, because noise exposures considered at all hazardous are being eliminated rapidly from our industrial environment. We are left, then, with only the relation between habitual exposure to a steady noise for eight hours/day, five days/week, fifty weeks/year and the hearing loss that develops. Unfortunately, few noise exposures, industrial or otherwise, are for eight hours at a constant level. The problem of assessing the permanent effect of noise exposures that are intermittent, time-varying, impulsive, or merely shorter than eight hours can be solved only by extrapolating from temporary effects to permanent effects, by extrapolating from PTS in experimental animals to PTS in man, or by refusing to wait for development of enough evidence to make a decision and simply postulating that some arbitrary but simple rule must be used. Animal work is underway in several laboratories that will eventually provide at least

partial answers to the problem of intermittence, but in the meantime the most reliable evidence comes from studies of TTS.

7.4.3 TTS from Steady Noise in Man

As indicated earlier, TTS can be generated and measured under controlled conditions, so much is known about this phenomenon. Most work has been applied to the study of shifts persisting more than 2 min, as these are considered to indicate a true 'auditory fatigue'. As early as 1930 (Peyser, 1930) suggested that these longer-lasting effects, observed in industrial workers, might be classified as either 'physiological' or 'pathological', depending on whether or not full recovery from one day's exposure occurred before the next day's began. Pathological fatigue was, he proposed, the precursor of NIPTS. If this idea were correct, perhaps a reasonable 'critical value' of recovery time would be 16 h or about 1,000 min. TTSs recovering by this time would be considered physiological and therefore normal, while only those requiring longer times would be termed pathological. In general, the range of 'normal' would include values of TTS_2 (the TTS two min after cessation of exposure) up to 25 or 30 dB, at least when normal ears and steady uninterrupted daily exposures are involved.

This 'ordinary' TTS has been studied intensively in the laboratory because of the assumption that its dependence on the frequency, duration, and intensity of the exposure might reflect fairly accurately the dependence of NIPTS on these same parameters (Temkin, 1933). Although this assumption is currently being challenged, the evidence is still far from conclusive one way or the other, and thus some of the more established characteristics of TTS are worth recounting.

Both the growth of TTS_2 and its recovery are exponential processes; for all practical purposes, in a constant noise level an asymptote of TTS_2 is reached in eight to twelve hours. The magnitude of the TTS_2 grows approximately linearly with SPL once the SPL has exceeded some base value below which only short-duration effects (those running their course in two minutes or less) are produced. This basic SPL, termed 'effective quiet', appears, to be about 70 to 75 dB for octave bands of noise. That is to say, a 75-dB-SPL octave-band noise will not produce much TTS_2 no matter how long it is on; a 105-dB-SPL noise will generate about twice as much TTS_2 as one at 90 dB if duration of exposure is the same; and a 100-dB-SPL noise will produce a value of TTS_2 midway between those following 90 and 110 dB SPL. The actual rate of growth with level, of course, will depend on the duration and frequency characteristics of the fatiguing noise; high frequencies induce a more rapid rate of growth than low frequencies, even though effective quiet is relatively independent of frequency.

The foregoing principles are illustrated in Figure 7.5, which summarizes some experiments in which ten normal-hearing young adults were exposed for eight hours to various octave-band noises. The dashed line at the bottom shows the

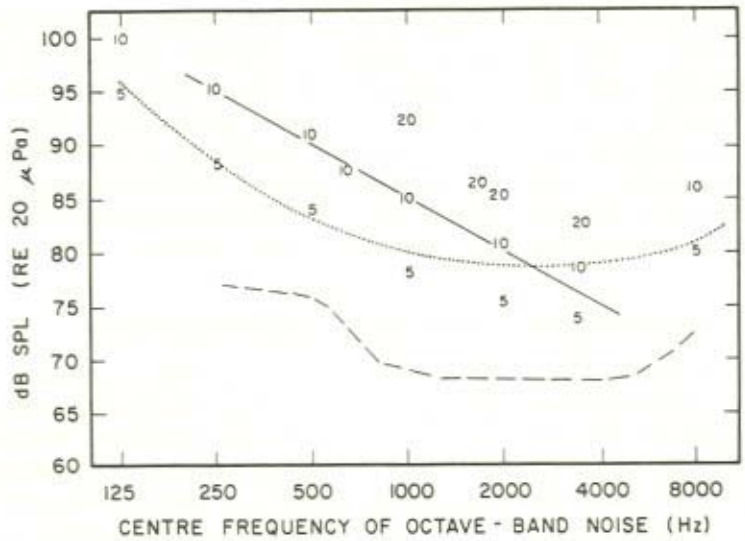

Figure 7.5 Octave-band sound pressure levels that will produce a 'particular' temporary threshold shift. The dashed line represents 'effective quiet'—i.e., that level that will just produce no TTS, nor retard recovery from TTS, in 95% of those exposed. Numerals denote the values of TTS_2 produced in the average listener by a continuous 8-hour exposure at the indicated frequency and SPL. The solid line, having a slope of −5 dB per octave, indicates 'magenta noise' that will produce approximately equal TTS_2 at all frequencies up to 4,000 Hz. The dotted line shows the locus of all SPLs having equal A-weighted levels, in this particular case an A-weighted octave-band level of 80 dBA. After Ward *et al.* (1976)

values of SPL that produced no effect — that is, no TTS_2 — in any ear, even the most sensitive. The numerals indicate the average TTS_2 (i.e., 5, 10, or 20 dB) generated by the eight-hour exposure at the indicated frequency and level. It appears that to produce an equal TTS_2, a given frequency needs to be only about 5 dB less intense than the octave below it: the most hazardous noises are those in the 3,000-Hz range (the resonant frequency of the outer-ear canal).

Temporary threshold shifts are asymmetrically distributed relative to the exposure frequency. The maximum TTS_2 is found at a frequency half an octave above the frequency of the fatiguer in the case of a pure tone, or half an octave above the upper cut-off frequency of a fatiguer that is a band of noise.

It should be clear that this fact, in combination with Figure 7.5, implies that a broadband noise, one having significant energy through the entire spectrum, will tend to produce a maximum TTS at 4 or 6 kHz. In such a noise the greatest energy reaching the cochlea will be in the 3-kHz region, and this will therefore produce the most TTS half an octave to an octave higher.

At this point it is possible to see one of the reasons for adoption of

A-weighting for assessment of all steady noises. The dotted curve in Figure 7.5 portrays the relative weighting assigned to different frequencies by the A-weighting network of the sound level meter; that is, all points on that curve have equal weight in contributing to the overall output of the meter. Even though A-weighting was originally based on judgements of equal loudness of very weak sounds, it happened that it came the closest of the existing standard weighting networks to predicting equal TTS-producing capacity of different frequencies at high levels, a fortuitous but useful occurrence.

7.4.4 TTS from Intermittent Noise, and the Total-Energy Theory

Prediction of the TTS produced by time-varying and intermittent noise exposures is somewhat complicated, because of partial recovery during the quieter periods. However, for relatively rapid interruptions or fluctuations (periods of five minutes or less), the TTS_2 is proportional to the mean number of decibels by which the instantaneous sound level exceeds 'effective quiet' during the entire exposure. Thus, for example, if 'effective quiet' for that particular noise is 70 dB SPL, the same TTS_2 will be produced by eight-hour exposures to an 85-dB steady noise, or a 100-dB noise that is on half the time and off half the time (by 'off' is meant any level below 70 dB), or a noise that changes at regular intervals from 75 to 85 to 95 dB and back to 75 dB, or a noise that fluctuates irregularly but for which the average number of decibels by which 70 dB is exceeded is 15 dB.

The temporary threshold shifts produced by patterns of longer exposures and rest-periods can be calculated by means of empirical equations describing alternate growth and recovery. In either case, however, the TTS_2 is considerably less than the TTS_2 that would have been produced by a steady noise over the same time but whose level was such as to keep the total energy constant. For example, in the case of the noise that is on half the time at 100 dB and off the other half, the total energy of the exposure is the same as a 97-dB noise that is on all the time, yet it produces only the same TTS as an 85-dB noise, which is a significant difference. Thus TTS is not a function of the total energy or even the total A-weighted energy of the exposure; how the energy is distributed in time makes a considerable difference in the magnitude of the TTS produced.

7.4.5 TTS from Impulse Noise

The same is true for TTS from impulse noise, such as gunfire. Although 30 impulses of simulated gunfire at 150 dB SPL peak level may produce a TTS_2 of 20 dB in a particular ear, 300 impulses of the same pulse shape but at 140 dB SPL (i.e., the same total energy) will usually produce no TTS_2 whatever in this individual (McRobert and Ward, 1973). Here it appears that there is a critical

level (which, however, is a function of pulse duration) below which no effect is produced but above which the TTS increases with level. Unlike the growth of TTS_2 with time for steady or interrupted noise, the TTS_2 from impulses appears to be proportional to the number of impulses, as if each impulse produced the same amount of TTS in dB. However, the whole problem of impulse noise is vexed with confusion arising from the unavailability of an apparatus that can produce pulses whose rise-time, duration, and peak level can be independently varied over a large range; experiments from different laboratories can seldom be compared because they differ in more than one parameter.

If exposure is so long or intense that more than 40 dB of TTS_2 is produced, or if even somewhat lower values of TTS_2 have been developed by prolonged intermittent exposure to higher intensities (105 dB and above), recovery neither proceeds in the exponential manner described above nor is complete by the end of 16 hours of rest. Instead, this 'delayed recovery' proceeds in nearly a linear manner – that is, diminishing by a constant number of dB each day. Full recovery may require several days or, in unusually severe instances, weeks. Peyser's (1930) suspicion seems reasonable in this case – namely, that the hazard of incurring PTS will be increased if the auditory system is once again exposed to noise before full recovery has occurred. If this is true, then TTS_2 is probably not an appropriate index of noxiousness. Instead, hazard might better be based on the amount of TTS remaining after 16 hours of recovery.

7.4.6 The CHABA TTS-based exposure limits

Exposure limits based on TTS data were developed in the USA by the Committee on Hearing and Bioacoustics (CHABA) of the National Academy of Sciences (Kryter *et al.*, 1966), by making the assumption that a TTS_2 at the end of the work day will be completely reversible, and hence not hazardous *per se*, provided that it did not exceed 10 dB at 1,000 Hz and below, 15 dB at 2,000 Hz, or 20 dB at 3,000 Hz and above. Using the knowledge about the growth of TTS cited earlier, exposures that just produced those values of TTS_2 were calculated and presented in a very complicated set of charts that specified tolerable durations and levels for continuous exposures or those with various degrees of intermittence.

The CHABA contours for single continuous exposures are shown in Figure 7.6. It can be seen that the contours for two- to eight-hour exposures are enough like the inverse of the A-weighting network (Figure 7.5) that A-weighting has subsequently been widely supported as the best means of reducing spectral information to a single number, even though for shorter exposures, the low frequencies are less hazardous (at least if TTS is an adequate measure of hazard) to an even greater degree than an A-weighted measurement would imply. A series of equal-TTS contours for exposure to

intermittent noise was also developed, a different set of contours for each specific octave band.

These CHABA contours proved to be too complicated to gain general acceptance. In addition to the difficulty of interpretation of some dozen charts, doubts were raised as to the adequacy of TTS_2 as a predictor of NIPTS; a given TTS_2 may take longer than 16 hours to recovery (i.e., delayed recovery occurs) if it is caused by short bursts of high-intensity high-frequency noise, and hence must be regarded as more dangerous than the same TTS_2 caused by a continuous exposure at a lower level.

The problem of delayed recovery can be solved by taking into account not only the TTS, but also its duration. Kraak *et al.* (1977) have presented extensive evidence that the integral of TTS over time from onset of exposure to the end of recovery will be a more valid measure of hazard than mere TTS_2. With accumulation of more empirical evidence on the recovery from specific patterns and levels of exposure, a set of contours similar to the CHABA contours, but based on equal integrated TTS, could be established. However, this has not yet been done, probably because the contours would still be as difficult to use as those they will replace.

7.4.7 Simplifying Assumptions: Regulations by Decree

If the complexity of the relation between intermittence of exposure and TTS is so great that it is not judged to be usable in prediction of NIPTS, then another solution is to make simplifying assumptions. Because of the resemblance of the equal-TTS curves of Figure 7.6 to the A-weighting network, almost all bureaucratic agencies concerned with establishment of exposure limits have, in the interests of simplicity, simply decreed that A-weighting shall be used, thus 'solving' the complex problem of differential effects of different frequencies at various levels. Then another arbitrary decision is made: how much NIPTS, as indicated by the data of Figure 7.4, is tolerable, and therefore what eight-hour exposure level is to be the limit. A further simplification can be effected by pretending that it makes no difference whether the daily noise exposure comes in one continuous segment or in bursts, so that effectiveness of a noise can be assessed simply by adding up periods of noise. The final thrust at the Gordian Knot is then administered by postulating some simple relation between level and duration for equal effect, so that an increase in level of some fixed number of decibels must be balanced by a decrease in permitted duration of exposure.

A wide range of exposure regulations, with different standard exposures and trading relations, can be found throughout the world. Even in a single country, several different exposure limits may apply. In the USA, for example, the official (OSHA) standard is a 90-dBA limit for eight hours, a trading relation of 5 dB per halving time, and a prohibition against levels over 115 dBA as measured with an ordinary sound level meter or against peak levels of 140 dB.

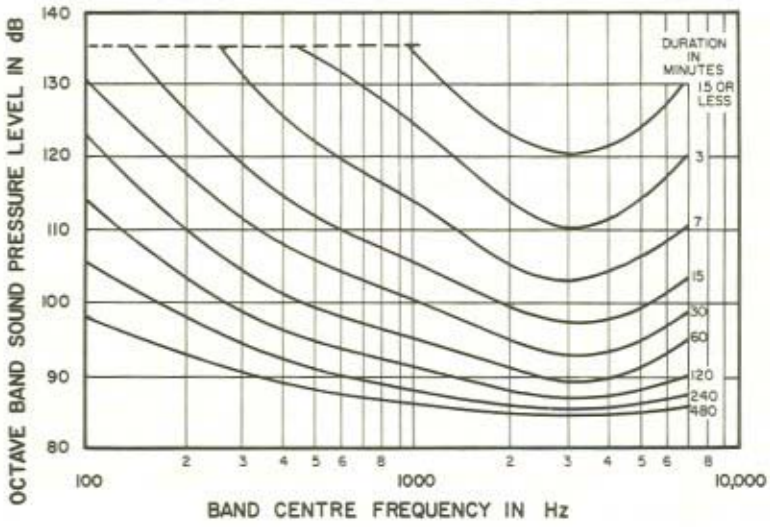

Figure 7.6 CHABA damage-risk contours for single continuous noise exposures. These contours indicate those combinations of octave-band SPL (ordinate), frequency (abscissa) and duration (parameter) that will just produce a temporary threshold shift, measured 2 min after exposure, of no more than 10 dB at 1,000 Hz or below, 15 dB at 2,000 Hz, or 20 dB at 3,000 Hz or above, in the average normal listener. Adapted from Kryter *et al.* (1966)

However, another government agency (NIOSH) is promoting a change of the eight-hour limit to 85 dBA, a suggestion adopted by the US Army, and the Environmental Protection Agency seeks to reduce it to 80 dBA, maintaining that 75 dBA is really necessary to protect everyone against the slightest NIPTS. EPA also believes that the trading relation should be 3 dB per halving of duration, in accordance with a proposed ISO standard. This amounts to simply integrating the sound intensity (after A-weighting) over time, thus calculating total energy. Because of the controversy between the proponents of the 3-dB and 5-dB rules, the US Air Force has adopted a 4-dB rule, beginning at 84 dBA for eight hours (Ward, 1977). It must not be thought that the list of possibilities is yet exhausted: Kraak contends that the best predictor of damage based on measurement of the exposure itself rather than on integration of the TTS it produces, is the integral of the instantaneous *pressure* over time, which leads to a 6-dB trading relation.

All of these simple standards are of course to some extent incorrect; non linear functions cannot be changed to linear ones by executive fiat, although in the case of the total-energy rule (3 dB per halving), that fact is regrettable, because the total A-weighted energy of an individual's exposure can be readily and reliably measured. Indeed, it is because of its simplicity that

the total-energy principle has been adopted by ISO, not because there was any good evidence in its favour. Some proponents of the principle go so far as to postulate not only the pattern of exposure during the day is irrelevant, but even that the distribution over the lifetime of the individual can be disregarded in predicting the NIPTS. In such systems, the concept of the 'safe exposure' is non-existent: every erg of acoustic energy that enters the cochlea makes an equal contribution to hearing loss, and the apparent recovery of threshold sensitivity is only illusory.

The simplicity of the total-energy theory is so attractive that there is a real danger that it may be accepted in its most extreme form as just expressed; its supporters point out, correctly, that it errs only by being too conservative. However, the costs of overprotection can be astronomical, especially when, as in the USA, use of hearing protectors is officially regarded as permissible only as a last resort, when it is not 'feasible' to reduce the noise at the source. The urgent need for valid evidence from controlled animal experimentation relative to the effect of intermittence is obvious.

7.4.8 Animal Research on the Total-Energy Theory

Animal experimentation on the effects of long exposures at moderate levels is not very exciting, so data are only slowly beginning to accumulate. It is already clear, however, that the total-energy principle is not correct, just as one would predict from the behaviour of other biological systems. For example, a 220-min exposure of chinchillas to a particular noise produced markedly less PTS and cochlear destruction when the exposure came in the form of 22 10-min exposures administered twice a week rather than a single uninterrupted exposure (Ward and Turner, 1980). However, one such datum is not sufficient to establish any sort of simple rule by which the true hazard of any particular pattern can be predicted. It may turn out, of course, that the total-energy principle can be used as the basis for predicting the effect of single uninterrupted exposures, regardless of length; indeed, the animal experimentation just cited showed that the same NIPTS and cochlear damage as that caused by 220 min at 112 dB SPL was produced by 2,200 min at 102 dB, by 22,000 min at 92 dB, and by 220,000 min (150 24-hour days) at 82 dB. But in order to be useful in predicting the effect of repeated exposures, the effective total energy must be adjusted by applying some sort of correction factor. It can only be hoped that such a system of correction factors, when finally determined, will be sufficiently simple that it will be accepted and used.

7.4.9 Critical Intensity

The same chinchilla study also showed that at some 'critical level' the amount of damage increases precipitously (Ward *et al.*, 1981). When the intensity level

was 120 dB for a 22-min exposure (which therefore contained somewhat *less* energy than the 112-dB 220-min exposure), the cochlear damage increased from about 10% hair-cell destruction to 70%, and the NIPTS from about 20 dB to more than 50 dB. This change illustrates what has long been termed the 'critical intensity', an intensity somewhat lower than the critical intensity for impulses mentioned earlier. Although the underlying physiological explanation of the phenomenon is not clear, it is generally presumed that it represents the point at which damage changes from being primarily metabolic to primarily structural — from a problem in depletion of energy stores or a build-up of toxic products to one of mechanical damage to the organ of Corti, damage that may permit the intermixing of perilymph with endolymph, a situation that appears to be toxic to hair cells. This critical level is not completely independent of duration, however, as incorrectly assumed by the earlier theorists in this area. Considerable work remains to be done in defining critical levels for particular doses for both continuous and impulse noises in a variety of animals if there is to be any hope of generalizing the results to man.

4.10 Summary

At present then, the curves of Figure 7.4 indicate the only reasonably certain relation between noise exposure and resultant hearing loss, a relation that is specific to eight-hour exposures to continuous noises having average spectral characteristics. If the exposure is intermittent instead of steady, less NIPTS will be produced, although it is not yet clear how much less hazardous a particular pattern of intermittence will be. On the other hand, if the energy of the noise were all concentrated in the 3-kHz region, considerably more NIPTS would be generated. Eventually the results of animal experimentation may permit development of more accurate predictors of damage; in the meantime it must be remembered that any schemes that use a simple relation between exposure and loss, such as the total-energy or total-pressure theories, cannot be correct.

7.5 INDIVIDUAL SUSCEPTIBILITY

The foregoing discussion has dealt with averages. However, average results seldom occur in individuals, and the present situation is no exception. Even in a population of workers of the same age, whose work exposure histories are known and nearly identical, the HTLS, and so the inferred NIPTSs, will vary over a wide range. No matter how innocent the noise exposure history, there will always be some individuals with severe losses; conversely, even among a group of boilermakers, someone will have completely normal hearing even though he has never used ear protection. Some of these differences, of

course, can be ascribed to differences in nosoacusis and sociacusis, and to errors in estimating exposure. However, no one doubts that individual differenes in susceptibility to hearing loss also play a large role.

7.5.1 TTS Predictor

Unfortunately, susceptibility is still little more than a *post-hoc* hypothetical construct. Although one can have faith that such characteristics as the size of various elements of the ear, the mass of the ossicles, the stiffness of the cochlear partition, and the density of cochlear blood vessel contribute to susceptibility, few such indices can be measured in the intact organism. Some 50 years ago, Temkin (1933) reasoned that the same factors that determine susceptibility to PTS should determine susceptibility to TTS. For the next 40 years a 'TTS-test' was sought that would predict 'susceptibility to PTS'. It was finally realized that this was a hopeless quest because the ear most susceptible to TTS from high-frequency noise was not necessarily the most susceptible to low-frequency noise, and neither of these bore a significant relation to susceptibility to TTS from impulse noise. Accordingly, the search for a test of 'overall' susceptibility has largely been abandoned; it has become clear that the only likely candidate for a successful susceptibility test based on TTS is one that involves an exposure to a noise having the same spectrum as the noise to which the individual will be primarily exposed at work. Such tests are being given in some industries; Kraak *et al.*, for example, cite a thesis of Richartz in which the integral over time of TTS (during both growth and recovery) produced by the first day of work in an actual industrial environment was used as the TTS index, successfully predicting PTSs produced by two years of work. Temkin may eventually be proven correct after all.

7.5.2 Drugs and Chemicals

Obviously there are certain conditions under which susceptibility — even in the global, general, sense — can be affected. Noise exposures that cause no damage in the normal experimental animal may have a pronounced effect when the animal is being administered ototoxic drugs (e.g., kanamycin, neomycin) at dosages that are, by themselves, subtoxic. It also seems likely that the ear's resistance to PTS could be reduced by certain mineral and vitamin deficiencies, or by illnesses that affect the blood-flow to the cochlea or produce a biochemical imbalance in the auditory system. However, the evidence relating to this topic is either anecdotal or so inferential that little confidence can be placed in its validity.

The same is true of medications administered in hopes of decreasing susceptibility or hastening the process of recovery from exposure. Although the literature abounds with articles extolling the virtues of vitamin A, nicotinic

acid, procaine, nylidrin, adenosine triphosphate, brain cortex gangliosides, carbogen (95% oxygen, 5% carbon dioxide), and dextran, to name a few, in the treatment of acoustic trauma, there is still no convincing evidence that a placebo would not produce just as much effect as any of the substances cited (Ward, 1980). The most recent example of negative results can be seen in a report by Eibach and Berger (1980) involving more than half a dozen of these substances.

7.5.3 Miscellaneous Factors Affecting Susceptibility

Women usually have average Hearing Levels that are slightly better than those of men working in the same noise environment, as Figures 7.1 and 7.2 imply. Although this could mean that women have 'tougher' ears than men, alternative explanations are equally tenable — i.e., that women are exposed to less sociacusic influence, or that they have a higher absentee rate (hence, less exposure), or that they are freer to quit a job that involves noise so loud that it bothers them. Only if these possibilities are eventually excluded by proper controls should we conclude that only women should be hired for noisy jobs.

The same line of argument applies to reports of differences between workers with brown eyes and those with blue, and between black and white workers. Although melanin in the cochlea may have something to do with susceptibility, equally likely alternative explanations exist.

There is also little evidence that very young or very old ears, or those already damaged by noise, are especially susceptible. Similarly, no convincing experimental support exists for the notion that the auditory systems of those who work in noise gradually become 'toughened' and so less susceptible. On the other hand, it has been clearly demonstrated that middle-ear problems, such as chronic otitis media, generally reduce the transmission of energy to the cochlea and so reduce 'susceptibility' in the same way as earplugs.

7.6 SUMMARY

No index of noise exposure has been devised that can succinctly characterize the relative noxiousness of different noise exposures. Although, on the average, the threshold limiting value (TLV) for measurable damage from steady industrial noise is about 85 dBA for an ordinary eight-hour exposure, the relative hazard associated with briefer or interrupted exposures cannot yet be determined. Even the eight-hour TLV is questionable because of the impossibility of eliminating sociacusic and nosoacusic influences from the data on hearing loss in industry. Prospects are dim for identifying individuals, in advance of employment in noisy industries, who are unusually susceptible to permanent hearing loss by means of any index based on temporary threshold shifts.

A vast amount of data has been published on NIPTS, but most of it is scientifically useless. Furthermore, although an extensive fund of information exists on the temporary effects of noise, the relation between temporary and permanent effects is still unknown. Finally, since no satisfactory method of characterizing noise exposures has been devised, we are in the uncomfortable position of having an undefined independent variable, a non-dependable dependent variable, and many potent irrelevant variables.

It appears that the only viable solution is to continue intensive studies of the development of NIPTS in animals whose environments can be subjected to the necessary strict control, and hope that the results can legitimately be generalized to man by appropriate corrections for differences of species. Only then can a realistic criterion for control of exposure to noise be developed —one that is stricter than the present standard, which permits the development of significant hearing losses in the more susceptible individuals, and yet not as strict as limits advocated by anti-pollution activists who fail to realize that 'noise pollution' differs from air and water pollution in that, although our stomachs were not designed to digest mercury compounds nor our lungs to absorb sulfur dioxide, the normal function of our ears is to process sounds.

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