Paparella: Volume II: Otology and Neuro-Otology

Section 3: Diseases of the Ear

Part 4: Inner Ear

Chapter 45: Noise-Induced Damage

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A causal connection between loud noises and hearing loss has no doubt been recognized for thousands of years. It is unreasonable to think that no one ever noticed the partial deafness of metalsmiths as long ago as the Iron Age. Some of the ancient Greeks at least disliked the din; about 600 BC the Sybarites forbade metalwork involving hammering (and also the keeping of roosters) within their city limits. However, the earliest extant reference to the effect of noise on hearing appears to be an observation recorded in the first century AD by Pliny the Elder in his *Natural History*, when he noted that persons living near the cataracts of the Nile were "strucken deaf" (Bacon, 1627). Although few persons now live close to loud waterfalls, our industrial age has resulted in ever-increasing noise levels everywhere else - at work, at home, and at play. It is no wonder, then, that the problem of noise-induced hearing damage (NIHD) and its prevention are assuming greater and greater importance all over the world. Like presbycusis, NIHD cannot be undone - but unlike presbycusis, it can be prevented or at least minimized if reasonable precautions against noise exposure are taken.

Pathogenesis

Ideally, of course, our knowledge of the histologic and biochemical characteristics of NIHD should come from studies of individual human temporal bones. However, only rarely, if ever, can one be sure that a given patient is the victim *only* of NIHD; most of the material being tediously accumulated by the temporal bone banks around the country is heavily contaminated by presbycusis and the degenerative effects of diseases of one sort or another, to say nothing of ototoxic drugs and chemicals. In addition, the audiometric status of the patients at time of death is seldom known accurately. Relatively pure cases of NIHD can be found only in experimental animals, and so the following description of the pathogenesis of NIHD will be based on observations on cats, dogs, guinea pigs, and chinchillas. This need cause us little concern, however, because there is no reason to suspect that there is anything unique about the growth of NIHD in humans. It is true that different species may have different average sensitivities to noise damage - for example, Miller et al (1963) estimated that the intensity required to produce a given degree of NIHD in the cat is some 18 dB weaker than that necessary to cause the same NIHD in humans. The same seems to be true of the chinchillas (Mitchell, 1976), whereas the guinea pig apparently is no more susceptible than humans. Despite these differences, however, the morphologic changes seem to follow parallel courses in all animals studied thus far.

Acoustic Trauma

NIHD can be caused instantaneously by a single gunshot (or firecracker explosion) heard at close range or may result from long exposure to relatively moderate levels of sound. Since most of the experimental work, beginning with the pioneering study by Wittmaack (1907), has involved short, intense, uninterrupted exposures, the morphologic characteristics of such "acoustic trauma" will be discussed first.

Immediately after exposure, the anatomic changes range from a moderate disarray of the stereocilia of inner and outer hair-cells to complete absence of the organ of Corti and rupture of Reissner's membrane. No changes are ordinarily found in the bone, nerves, blood vessels, stria vascularis, spiral ligament, or limbus (Davis et al, 1953), although of course the endolymph may contain debris from destroyed hair-cells and other structures following very severe exposures. In such cases, pronounced swelling of various structures occurs with a few minutes (Thorne et al, 1986). In such cases, pronounced swelling of various structures occurs within a few minutes (Thorne et al, 1986), and edema of the stria vascularis appears shortly thereafter, which may persist for several days (Duvall et al, 1974). In short, the picture is of a mechanical system (the organ of Corti) that has been vibrated with too great an amplitude, with the severity of the resultant tissue injury related to both the exposure duration and the degree to which the amplitude exceeded a hypothetical tissue injury threshold. It has been speculated that much of the swelling observed is caused by an increase in potassium content of the perilymph, due to invasion of endolymph into the perilymphatic space when rupture of the structures that separate the two fluids occurs (Bohne, 1976).

Covell and coworkers (Davis et al, 1953; Eldredge et al, 1958, 1961) developed a nine-point scale that still appears reasonable for rating degree of injury from noise. Ratings of 1 and 2 represent normal and "within normal limits"; 3 and 4 are used to indicate a moderate swelling and pyknosis of the hair-cells and some disarray of their stereocilia, together with a slight displacement of the hair-cell nuclei, formation of small vacuoles in the supporting cells (Hensen's, Deiters', Claudius'), and some displacement of the mesothelial cells, a thin layer of cells on the bottom (scala tympani) surface of the basilar membrane. It is probable that these changes are all reversible, and represent part of the physiologic counterpart of auditory fatigue. Ratings 5 and 6 denote marked swelling, disintegration, pyknosis, and/or karyorrhexis of the outer hair-cells, marked displacement and fracture of stereocilia or their fusion into giant cilia, the formation of large vacuoles in and the partial separation of the supporting cells, and disappearance of a large proportion of the mesothelial cells. Beagley (1965) reported that extreme swelling and vacuolization of supporting cells sometimes results in so much pressure on the inner hair-cells that they apparently are avulsed from the organ of Corti, leaving holes in the reticular lamina that are replaced with a few hours by phalangeal scars (Bohne and Rabbitt, 1983). A rating of 7 is attached when some hair-cells are completely missing, Deiters' cells are detached from the basilar membrane, and the mesothelial cells are gone. A rating of 8 would indicate more severe loss of hair-cells, including even the inner hair-cells, and rupture of Reissner's membrane. A rating of 9 is given when all the hair-cells are destroyed and the organ of Corti is either completely collapsed, separated from the basilar membrane, or absent.

With destruction of the hair-cells, secondary degeneration takes place, so that if several weeks are allowed to elapse between trauma and sacrifice, ganglion cells and nerve fibers will

also be missing in a region where hair-cell destruction was complete (Kellerhals et al, 1968). If the hair-cells in an area are not all destroyed, however, the ganglion cells may appear quite normal, presumably because secondary degeneration will not occur as long as a few (possibly even one) of the original myriad nerve endings from a given ganglion cell terminate on a functional cell. As a matter of fact, Schuknecht (1953) reported that the best histologic correlation with the degree of ganglion degeneration was the severity of the changes in the supporting cells, particularly the pillar cells.

Two examples of "safety-valve" action in protecting the hair-cells from extremely high noise levels can be cited. One of these has been known for at least 80 years (Yoshii, 1909): if a given noise exposure or explosion has ruptured the eardrum, the auditory damage will be smaller than if the drum had been left intact. The rupture in this case has resulted in the transmission of less energy to the cochlea. Beagley (1965) reported an analogous phenomenon at the organ of Corti itself; he observed that in a group of guinea pigs exposed for 20 minutes to a 500-Hz tone at 128 decibels sound pressure level (SPL), the hair-cell damage was inversely related to the condition of the junction between the Hensen's and Deiters' cells. That is, if the junction were found to be intact, then a large amount of hair-cell damage could be expected, but when a clearly visible cleft between them had been produced, much less damage was seen.

Acoustic trauma, then, essentially represents hair-cell destruction caused by physically exceeding what we might loosely term the "elastic limit" of the peripheral auditory mechanism, so that direct mechanical tissue injury results - either to the hair-cells themselves, or perhaps first to the supporting structures, with hair-cells degenerating later as a result of lack of this support.

Noise-Induced Hearing Damage from Habitual Exposure

The situation is not nearly so unequivocal in regard to the damage associated with years of exposure to more moderate noise, however. Two general, partly opposing points of view exist here, the micromechanical and the biochemical. Gravendeel and Plomp (1960) suggested that long-termed NIHD may represent a gradual accumulation of noise microtraumata. Noise is characterized by irregularity, so that in a noise with moderate *average* level, a peak many decibels higher will occasionally occur, and these infrequent peaks may irreversibly injure a hair-cell. Even in the most steady noises, the exposure to which a given ear is subjected will vary from time to time as the organism moves about in the noise field. One hair-cell may be lost today, another day after tomorrow, a third one next week, and so on, so that although one could not measure the effect of any single day's exposure, after several years the cumulative hair-cell loss from these microtraumata becomes significant.

An alternative point of view, and one historically much older, is that long-term NIHD is the outcome of a slowly accumulating exhaustion of metabolites at the cytochemical or enzymatic levels not involving direct gross tissue destruction (Vosteen, 1958). The notion here is that habitual exposure to noise gradually induces biochemical changes that eventually produce widespread hair-cell destruction only indirectly or, in essence, that auditory fatigue is the precursor of NIHD.

Actually the two hypotheses are not antagonistic as one might at first suppose. As we shall see, there is no doubt that steady exposure to a moderate intensity of sound does produce measurable changes in the underlying histochemistry of the cochlear partition. Furthermore, daily 5-hour exposure of dogs for up to 2 years to moderately high levels of sound (105 to 110 dB SPL) produced a sequence of eventual histologic changes that were quite indistinguishable from those observed following single short exposures at successively higher levels: a loss, first, of outer hair-cells, then inner hair-cells and supporting cells, followed by degeneration of nerve fibers and the spiral ganglion (Dieroff and Beck, 1964). In other words, for all types of NIHD, the condition of the hair-cells seems to be the crucial consideration.

The question is, however, how to discriminate cause and effect. Do the biochemical changes associated with mild exposures *produce* hair-cell degeneration or are they epiphenomena? Or, more likely, do such changes progressively alter the resistance of the hair-cell (or of its supporting cells) to damage when the unusually high energy peak comes along, so that as the workday wears on, the chance of losing a hair-cell or two becomes greater and greater? We do not yet know.

Biochemical Correlates of Auditory Fatigue and Incipient Noise Trauma

In the last 35 years, considerable effort has been devoted to the study of biochemical changes associated with sustained stimulation. The first experiments centered mainly on the question of the oxygen metabolism that must be involved in the maintenance of the cochlear partition. Since it was known that anoxia can produce irreversible damage to the hair-cells, this was a logical place to begin.

Early studies had shown that noise stimulation produced a decrease in the oxygen tension in the cochlear duct. Since noise also produces vasoconstriction of the extremities (Jansen, 1967, reviewed this evidence), it was reasonable to think that a similar effect might be involved here. However, Perlman and Kimura (1962), in direct observation of blood flow in the cochlea, saw neither vasoconstriction nor vasodilation until the sound level reached 120 dB SPL, whereupon the flow increased almost immediately. This implies that the decrease in oxygen tension associated with moderate exposure indicates only an increase in oxygen consumption, not necessarily a decrease in oxygen supply.

The link, if any, between hearing loss and the circulatory system remains obscure, as does, indeed, the whole area of the relation of biochemical processes in the cochlea to the development of NIHD. Although myriad metabolic changes - such as alterations in concentrations of RNA, DNA, succinic and lactic dehydrogenase, ATP, GABA and glutamate, various enzymes, and metabolites involved in maintenance of cochlear integrity, and changes in membrane permeabilities and transport processes - occur in the fluids and tissues of the cochlea in response to noise exposure, their precise role in producing eventual hair-cell degeneration is still essentially unknown (Thalmann, 1976; Schacht, 1982).

Localization of Effect

Whether NIHD is caused by mechanical overstimulation or by interference with metabolic processes, it is reasonable to first expect hair-cell damage at that point on the basilar membrane where the activity is greatest. And indeed, this is the case. A high-intensity pure tone gives rise to waves that travel from the oval window toward the apex of the cochlea, gradually increasing in amplitude as they proceed apically until a maximum is reached, whereupon the amplitude drops precipitously; thus the spatial distribution of amplitude of motion is quite asymmetric (Békésy, 1947). The locus of maximum amplitude depends on frequency: the maximum for high frequencies occurs toward the basal end, for low frequencies toward the apex. It is not surprising, then, that even the earliest studies showed that, in general, high-frequency sounds produce the most damage near the oval window, while low-frequency sounds affect the apical portions (Wittmaack, 1907). Furthermore, because of the asymmetry of the amplitude distributions, the spread of damage with continued exposure tends to be greater in the basal direction.

Narrow-band noises (noises with most of their energy in a single octave band) produce localized damage whose pattern is like that from pure tones. One might therefore expect that broad-band or "white" noise - noise that contains all frequencies in equal amounts - would produce uniform damage throughout the cochlea. Or, in view of the fact that a given region of the basilar membrane is stimulated by all frequencies lower than the frequency that produces maximum excitation at that region, one might predict that the most damage from white noise would be found immediately adjacent to the oval window, since that region is stimulated by all audible frequencies. However, this is not quite the case. Instead, the earliest and greatest damage appears a half-turn or so apically from the oval window - in humans, at a point about 10 millimeters along the cochlear partition, or a third of the way toward the apex. The reasons for this region of apparently greater susceptibility have been discussed for several generations, and several factors appear to be involved (Lehnhardt, 1966).

First, and most important, the ear canal acts as a resonator, amplifying frequencies between 2 and 5 kHz by 10 dB or more (Wiener and Ross, 1946). This "resonance" effect has a maximum in the neighborhood of 2500 Hz. Thus, pure tones (and, of course, components of noise) in the frequency region from 2 to 3 kHz reach the inner ear at a higher intensity than do tones at higher or lower frequencies (assuming the intensity at the entrance to the outer ear to be constant across frequency) and these 2- to 3-kHz tones produce their maximum effect in the 10-millimeter area. A second consideration is that the middle ear, because of its construction, also transmits some frequencies to the cochlea more readily than others (Lehnhardt, 1966). Finally, the basilar membrane is very near the bony lamina for the lower part of the first turn, so that perhaps the amplitude of motion of the organ of Corti is thereby limited from the oval window to a point in the neighborhood of the area cited (Kelemen, 1962). The asymmetry of motion of the basilar membrane, the restriction of movement of the extreme basal end, and the selective attenuation (or lack of amplification) of incoming sound above 4000 Hz and below 1000 Hz - these all conspire together to produce the greatest amplitude of motion of the organ of Corti in this one spot, so it is no wonder that the greatest injury from most types of noise is found there.

Measurement

The most salient result of overstimulation of the organ of Corti, then, is destruction of hair-cells at the region of maximum amplitude of motion of the basilar membrane, with the outer hair-cells usually succumbing first, the inner hair-cells either later or as the result of more intense exposure. This is followed by degeneration of nerve fibers and ganglion cells in areas from which all the hair-cells have been eliminated, but not where the hair-cell damage is only partial.

Thus, the basic datum in NIHD has traditionally been the condition of the hair-cells. In experiments with animals that can be sacrificed, one can observe this directly by means of ordinary light microscopy. In assessing NIHD in living organisms, however, some indirect method of measurement must be employed.

Pure-Tone Threshold Shifts

The most commonly used method of inferring NIHD is by measuring the absolute threshold to pure tones (that is, the weakest tone that can just barely be heard). Unlike the situation for a strong pure tone, a weak tone will produce motion of the basilar membrane only in a very circumscribed area. Indeed, a weak tone appears to actually trigger, in the normal cochlea, the release of energy at this point, so that the resultant vibration has an amplitude greater than would be expected from the extremely minute amount of acoustic energy that enters the ear (Sellick et al, 1982). For example, a weak 4000-Hz pure tone will elicit a neural response only from those nerve fibers associated with hair-cells in the 10-millimeter region (ie, 10 millimeters from the oval window); for this reason, this hair-cells are commonly called the "4000-Hz receptors", and the associated nerve fibers are called the "4000-Hz nerve fibers".

In the normal cochlea, approximately the same small amount of energy will be needed at all frequencies between 500 and 6000 Hz to stimulate the corresponding receptors and thus give rise to perception. However, if the hair-cells in a particular area are damaged or missing, or if the nerve fibers themselves have degenerated, then at the appropriate frequency more energy will have to be supplied before the motion of the basilar membrane is sufficient either to cause the injured hair-cells to trigger the "correct" nerve or to arouse responses from the hair-cells and nerve fibers in the regions adjacent to the affected area. The difference in the energy thus required, when expressed in decibels, is called the noise-induced hearing loss (NIHL).

As indicated just above, the area most easily affected by noise, that is, about 10 millimeters from the oval window, is the 4000-Hz receptor region. The audiometric frequency most often affected by noise, therefore, is 4000 Hz, and audiograms of noise-exposed workers will generally show a maximum loss at this frequency, although an individual ears maxima at 3000 and 6000 Hz are not at all uncommon. Furthermore, if a noise has most of its energy in a particular frequency region, then the maximum damage will be manifested at an audiometric frequency that is slightly higher - that is, at a frequency about half an octave above that of the noise (50 per cent higher). Thus, a noise with most of its energy at 1000 Hz would be expected to produce more damage at 1500 Hz than at higher or lower test frequencies.

The typical audiogram of an ear in a noise-exposed worker, then, will usually show a maximum loss at 4000 Hz. The four curves illustrate the gradual growth of NIHL as reflected in the audiogram of a hypothetical worker who worked for 40 years in a 120-dB noise environment without ear protection. Audiogram A is a typical beginning "boilermaker's notch", or, in Europe, "C5 dip" (the musical tone C5, the topmost key on the piano, is about 4000 Hz) that would develop in a few weeks. The 40-dB loss of hearing at 4000 Hz would never be detected by the worker himself, unless the loss was found in only one ear, in which case he might have noticed that the tick of his watch appeared somewhat duller in that ear. Probably this condition represents the loss of at least the outer hair-cells at the 4000-Hz receptor region.

With continued exposure the notch becomes deeper and wider, with a temporal plateau at about 60 dB hearing level (HL). Schuknecht (1963) speculated that this flattening at 60 dB may indicate the point at which all the outer hair-cells over a large range have been destroyed, so that they can no longer facilitate the firing of inner hair-cells, which, because of their orientation on the basilar membrane, have higher natural thresholds. As the latter also become affected, the thresholds once again become less sensitive, until high-frequency perception is completely gone. After this, the low frequencies are more and more affected, until eventually a state of near-deafness exists.

Although this description may appear straightforward, the picture is not really this clear. Unfortunately, studies relating hair-cell damage to behaviorally determined thresholds on an individual basis present a most confusing picture. Chinchillas whose auditory sensitivity has essentially returned to normal following a noise exposure in our laboratory that was severe enough to produce a temporary loss of 60 dB or more may nevertheless display extensive hair-cell destruction. In humans, also, many individual hair-cells may be missing without a correlated hearing loss (Bredberg, 1968). On the other hand, a pronounced permanent notch of as much as 80 dB may be correlated with an apparently normal set of hair-cells in both the cat (Elliott, 1961) and the monkey (Hunter-Duvar and Elliott, 1972).

Perhaps, then, other possible methods of determining or inferring noise-induced damage to the auditory system should be considered.

Cochlear Microphonics

It is not easy to determine the auditory threshold of animals other than humans; they must be trained to perform some more or less simple task when they hear a tone, and both training and testing require an inordinate amount of time and effort, at least at the present state of the art. One of the earliest attempts to infer the state of the hair-cells without either major surgery or tedious behavioral tests was through the measurement of cochlear microphonics (CM) at the round window, or, under special conditions, at the eardrum. However, it is clear that the round window CM reflects primarily the state of the basilar membrane within a few millimeters of the round window (Simmons and Beatty, 1962). If one is to make inferences from CM about one particular area, therefore, one must use differential electrodes placed very near the portion of the organ of Corti concerned - which of course requires complicated surgery and causes irreversible damage. So, while CM from differential electrodes may enable us eventually to understand the relations among the mechanical, chemical, and electrical events involved in the process of sound transduction and the

production of auditory fatigue, it offers no hope as a means for determining details of the state of the auditory mechanism in the intact organism.

Tone Decay

A curious phenomenon discovered by Lord Rayleigh (1882) is that under certain conditions a sustained pure tone at a constant intensity may at first be quite audible but later disappear; it may be necessary to raise the intensity in steps by as much as 50 dB before the tone remains audible for a full minute (Schubert, 1944). However, the locus (or, more probably, loci) of this particular type of fatigue is still obscure, and despite widespread use of such "tone decay" tests, the phenomenon seems to have no relation to NIHL. Kowalik (1965) found some tone decay in 194 of 290 patients with presumptive noise-damaged ears, and none in 96. Similarly, in an intensive study of losses probably caused both by steady noise and by gunfire, no relation between degree of tone decay and hearing loss was found (Ward et al, 1961). Although abnormal tone decay may be found more often in persons with NIHL than in normal individuals, it seems unlikely to provide any more dependable indication of cochlear damage than the pure-tone threshold.

Recruitment

On the other hand, in the last study cited (Ward et al, 1961) every ear tested demonstrated *recruitment:* as one raises the intensity of a tone whose threshold is elevated, the perceived loudness grows more rapidly than in a normal ear (or than at a frequency, in the same ear, whose absolute threshold is normal), so that by the time the intensity reaches 100 dB SPL, the loudness is as great as normal. But, unfortunately, recruitment also accompanies other forms of end-organ dysfunction (Eby and Williams, 1951). For example, the recruitment functions in the study by Ward and colleagues (1961) were completely indistinguishable from those found in Ménière's disease (Hallpike and Hood, 1959).

Although recruitment, therefore, cannot be used to discriminate NIHL from other types of end-organ dysfunction such as those caused by (1) Ménière's disease, (2) hereditary progressive high-frequency loss (Huizing et al, 1966), (3) ototoxic drugs of various types, and perhaps by (4) some toxic industrial chemicals, (5) carbon monoxide poisoning, and (6) certain diseases such as dysentery (Lehnhardt, 1966), its absence does have value in discriminating these hearing losses from those associated with more central problems such as acoustic tumors. And, although this possibility does not seem to have yet been studied, one may speculate that perhaps that portion of old-age hearing loss associated with end-organ damage that may have been caused either by industrial noise or by the typical auditory hazards of modern life (sociocusis) might be discriminated from the central degeneration accompanying general aging (true presbycusis) by determining the relative completeness of recruitment.

There are two models to explain recruitment. One is that the hair-cells or the ganglion cells in a particular region are only relatively insensitive, so that as one raises the intensity, the elements eventually all fire in their normal way (Davis et al, 1949). This is probably the case in Ménière's disease and may also be true for temporary threshold shifts. However, this explanation can hardly hold for recruitment at a tonal gap, if a large block of hair-cells and ganglion cells is missing. In this case, the intensity of the tone must be raised until the hair-

cells adjacent to the damaged area are stimulated sufficiently to initiate neural excitation. As this elevated threshold is exceeded by raising the intensity further, more and more of the unaffected sensory units are stimulated. The relative contribution to the normal total firing by the "dead" area, therefore, becomes proportionately smaller and smaller, finally becoming negligible at high levels.

Pitch Shifts

Because of the asymmetry of the pattern of motion of the basilar membrane, as one raises the intensity of a tone at which a threshold hearing loss exists, the intact fibers on the oval window side of the area of hair-cell loss will be stimulated before those on the apical side. Thus the pitch of the percept will be higher that it should be (Ward, 1969). Therefore, if a tonal gap is found at the frequency involved in only one ear of the patient, one can estimate the degree of this pitch shift by comparing the percept with that aroused in the normal ear; the difference in pitch indicates the degree of "diplacusis", and the range of frequencies that are affected should correlate with the extent of the cochlear lesion. Usually, of course, hearing loss will be found in both ears, so diplacusis measurements will be indeterminate. In that case a special technique for inferring pitch shifts relative to a single frequency in the same ear would have to be used, a method applicable only to musically trained persons (Ward, 1954).

Another unexplored possibility exists, however. If, say, all the 4000- to 700-Hz receptors are gone, then at threshold all tones in this frequency region should appear to have the same pitch. The ability to discriminate a 5000-Hz from a 6000-Hz tone, provided the intensities have been adjusted to give the same loudness, should be lost. Thus the "difference limen for pitch" - the ability to discriminate differences between tones on the basis of pitch alone - should be elevated over the region of damage. Again, however, the tests concerned would have to be preceded by a tedious series of judgments by the patient in order that one could set the tones to be compared so that they have equal loudness.

Miscellaneous Psychoacoustic Indices of Damage

In the last decade, considerable effort has been expended in an attempt to find some other measurable aspect of human perception that is more sensitive to hair-cell damage than the behavioral audiogram. Usually these studies assume that a person with a noise-damaged ear, as revealed by a significant NIHL, probably has damaged hair-cells at lower frequencies, even though the threshold is still within normal limits at those frequencies. Therefore comparisons are made, between such ears and those that are normal at all frequencies, of such indicators as difference limens for frequency and intensity (Turner and Nelson, 1982), masking (Festen and Plomp, 1983), production of aural harmonics and combination tones (Schmiedt, 1986), temporal integration (Hall and Fernandes, 1983), and perception of tone glides (Collins, 1984). However, these efforts have met with little success. Although some of the results have been promising, in the last analysis the problem is one of validation: the existence of diffuse hair-cell destruction or dysfunction can be confirmed only in experimental animals; and in these, the determination of reliable estimates of these psychoacoustic indicators is difficult if not completely impracticable.

Evoked Responses

A possible tool for circumventing behavioral thresholds is the average evoked response (AER). The brain potentials generated at the surface of the skull by a series of tone pulses at a given intensity are averaged by means of a computer, so that random electrical activity in the brain (ie, that portion of the recorded potentials not linked in time with the pulses) tends to be canceled out, leaving only the AER. However, although a good correlation has been reported, under optimal conditions, between the behavioral threshold and the detectability of a cortical AER in humans (Davis et al, 1967), there seems to be a significant difference between threshold and brain stem AER for the chinchilla (Henderson et al, 1969). In the latter case a detectable AER can be seen only when the intensity is some 15 to 20 dB greater than that to which the chinchilla is able to respond in a behavioral threshold test. Nevertheless, because of the advantage of AER over behavioral testing of laboratory animals in terms of efficiency in time and effort, and because the AER is more "objective", this technique has been widely used in the last quarter century to study both temporary and permanent effects of noise on neural activity.

At present, therefore, it appears that we have no better method of inferring cochlear damage than from pure-tone threshold shifts, measured either directly or indirectly. However, it must be kept in mind that rather extensive damage to a particular area may well exist before it is reflected in a measurable tonal gap. On the other hand, it may nevertheless be true that a measurable pure-tone threshold loss is necessary before any other impairment of function is detectable. For example, in view of the fact that we still do not know what role is played by the outer hair-cells in the process of transformation of acoustic energy to neural impulses, it may turn out that their condition is quire irrelevant until the structural damage is very extensive.

Factors Affecting the Growth of Noise-induced Hearing Damage

It is not possible in a single chapter to cover the entire field of industrial hearing loss in detail; therefore, only a few of the more important issues will be treated here. Amplification of the principles discussed below can be found elsewhere (Ward, 1984).

Frequency

Frequencies in the 2- to 3-kHz region produce more auditory damage than either higher or lower frequencies, for the reasons discussed earlier. That is, the NIHL in decibels, at the frequency most affected, will be greater from noises in the middle frequencies than from other frequencies, if the overall noise intensity and the duration of exposure are constant. Ultrasound (frequencies above 20.000 Hz, and not ordinarily audible) must exceed levels of 110 dB SPL before they are at all dangerous (Acton, 1968); such levels are quite rare in industry.

Intensity, Time, and Intermittence

The average NIHL in a group of workers will increase with both intensity of noise and time of exposure. Noises below about 80 dBA* are quite safe, as indicated by the fact that hearing losses among workers who spend 8 hours per day in that level are no greater than

those found in non-noise-exposed persons (Passchier-Vermeer, 1968), even after many years of exposure. Slight auditory fatigue, more commonly termed temporary threshold shift (TTS), from the noise leaves no permanent effects. If the 8-hour exposure level exceeds 80 dBA, however, the hazard increases with level in a reasonably linear fashion, whether "hazard" is defined either as the average hearing loss of a large group of workers or as the percentage of workers who reach some fixed value of hearing loss such as the average HL at 500, 1000, 2000, and 3000 Hz of 25 dB that is considered to constitute the beginning of auditory handicap. Habitual exposure to 100 dBA for 8 hours per day will produce, after 10 years of regular exposure, a median NIHL of some 40 dB at 3, 4, and 6 kHZ, although only 10 to 15 dB at the lower frequencies.

* The term "dBA" indicates the overall SPL measured with the "A" network of the sound level meter. With this network, low and extremely high frequencies are weighted much less heavily than those in the middle range, in general accordance with the relative noxiousness of the frequencies, as described earlier.

In a work situation in which intensity of noise is constant, the growth of NIHL with time approximates an exponential function. That is, the damage grows rapidly at first but then gradually slows down. The figure shows this exponential increase of HL at 4 kHz in men who worked in four different environments of unusually uniform noise (Glorig et al, 1961). In each case the growth of HL in a nonindustrial-noise-exposed sample of men having the same average age is also shown. It is clear that most of the NIHL at 4 kHz is produced in the first 5 years of exposure - indeed, for the two highest levels, nearly half the NIHL in decibels appears in the first 2 years. At lower frequencies, however, the growth is much slower and continues for many years.

What has not yet been firmly established is the trading relation between intensity and time. When the daily exposure to noise is less than 8 hours, higher sound levels can be tolerated. But how much higher? This problem would be solved if it were true that damage depended only on the amount of acoustic energy that had entered a given worker's ear during the day. Because this energy is equal to the product of intensity and time, then if the duration were cut in half, the intensity could be doubled without increasing the hazard. For example, if this "equal energy" principle were correct, then whatever danger was posed by 8 hours (480 minutes) of exposure at 90 dBA would be the same as the risk associated with a 93-dBA exposure for 4 hours, a 96-dBA exposure for 2 hours, a 48-minute exposure at 100 dBA, a 4.8-minute exposure at 110 dBA, and so on.*

* It will be recalled that the decibel scale is a logarithmic one, so that each tenfold change in the intensity (energy per second) of a sound represents a 10-dB change in intensity *level*, a 20-dB change represents a hundredfold increase or decrease in intensity, 30 dB is a thousandfold change, etc. Doubling the intensity increases the level by 3.01 dB, or, as a first approximation, 3 dB.

Present evidence indicates that the equal-energy principle does apply, at least in the chinchilla (Ward et al, 1983), but only for single, uninterrupted exposures such as those just specified. If, instead, the daily exposure is broken into several exposures with appreciable quiet periods between them, the damage is reduced. When, for example, a daily exposure of 48 minutes to a 100-dBA noise was changed to a series of 40 noise bursts, each of 1.2

minutes in duration, but spaced throughout the workday, the NIHL and cochlear damage were both considerably less. The reduction was even greater when the noise level was 110 dBA. The reduction was even greater when the noise level was 110 dBA. Not only do periods of relative quiet between noise bursts permit some recovery from auditory fatigue to occur, but they also apparently allow the middle ear muscles to recover their strength, thereby increasing the amount of reduction of sound reaching the cochlea caused by their contraction. The net result of these two factors is that considerably more energy can be tolerated if exposure is intermittent rather than steady.

The foregoing results provided experimental confirmation of the empirical fact that, in real life, intermittent exposures produce less hearing loss than would be expected on the basis of the equal-energy principle. For example, jet aircraft commonly develop intensities above 130 dBA and, in the past, maintenance personnel were exposed for several minutes to such noise. Fortunately, however, the exposures were irregular enough that no NIHL was produced (Sataloff, 1953; Kopra, 1957; Ward, 1957), despite the fact that the energy in a single exposure of only 3 seconds to 130 dBA is equal to the energy of 80 hours at 80 dBA. Intermittence must also be the explanation for the lack of NIHL among some glass blowers who work all day with machines that generate over 105 dBA of noise while they are operating (Sanderson and Steel, 1967), or among rock musicians.

Although the relation between temporary and permanent threshold changes is not yet clear, it seems likely that what is important in avoiding NIHL from habitual exposure is that at the beginning of the workday no residual auditory fatigue from the previous day exists. Frequent interruptions of the workday tend to keep the auditory fatigue down to a value from which 16 hours of "quiet" (anything weaker than about 70 dBA) permits full recovery.

A "correction" for the effect of intermittence in reducing hazard is embodied in regulations developed by the Department of Labor to protect workers from NIHL (OSHA, 1983). The permissible daily exposure for 8 hours is limited to 90 dBA (thereby permitting a small amount of NIHL to be eventually produced), but instead of allowing only a 3-dBA increase in level for each successive halving of the exposure duration, a 5-dBA increase is permitted. Thus, in a 195-dBA noise, 4 hours per day of exposure is allowed, or 2 hours at 100 dBA, and so on, up to 15 minutes at 115 dBA. Provided that those 15 minutes are actually broken up into several shorter bursts during the day, this regulation will probably provide the protection against the development of large amounts of NIHL that was the expressed goal of the regulation.

The Critical Exposure

If the daily exposure is continuous, then the equal-energy principle should be invoked in setting exposure limits, but only up to a certain point. In a series of experiments on the chinchilla, in which the intensity of the noise was systematically increased while the total energy of the exposure was held constant by decreasing the duration commensurately, although equal moderate amounts of damage were produced by 150 days at 82 dBA, 15 days at 92 dBA, 1.5 days at 102 dBA, and 0.15 day (220 minutes) at 112 dBA, a 22-minute exposure at 120 dBA resulted in near-complete destruction of the cochlea and an NIHL of 50 dB (Ward et al, 1981). For that particular energy, some critical point was apparently exceeded when the level was raised from 112 to 120 dB, even though the duration was

shortened appropriately. In effect, what occurred can be described as defining the transition from "ordinary" NIHL to acoustic trauma - that is, the point at which the "elastic limit" of the organ of Corti was exceeded. However, this point is not an ordinary type of elastic limit, because it depends on both the intensity and the duration and not just on the intensity alone, which would have to be the case if one wished to speak of a "critical intensity" of stimulation. Although 120 dB is above such a critical point for a 20-minute exposure, subsequent experiments showed that it was below critical for exposures of less than about 7 minutes (Ward and Henderson, 1986; Duvall and Robinson, 1987). Data on damage in the guinea pig indicate a similar dependence of the critical point on both level and duration, such that the extensive damage is approximately the same if the product of the duration and the square of the intensity are held constant (Eldredge and Covell, 1958; Spoendlin and Brun, 1973; Erlandsson et al, 1980). In the guinea pig, the critical level associated with a 7-minute exposure is around 135 dB (Spoendlin and Brun, 1973), which suggests that the chinchilla is considerably more susceptible to acoustic trauma than is the guinea pig.

If the human auditory system is as susceptible to damage as that of the guinea pig, so that a 7-minute exposure at 135 dB is the dividing line between moderate NIHL and acoustic trauma, then acoustic trauma could be expected to occur in humans only after around 40 seconds of exposure at 140 dB, 4 seconds at 145 dB, or 0.4 second at 150 dB. Actually, when young healthy air force personnel were exposed for 0.4 second to noise at 153 dB, only slight amounts of even temporary threshold shift were produced (Sommer and Nixon, 1973), indicating that this exposure was below critical.

The present OSHA regulation avoids the problem of the critical exposure by stipulating that no exposure over 115 dBA is to be permitted (OSHA, 1983). This highly overprotective measure was instituted because of an erroneous belief in the existence of a critical intensity, independent of duration. Although additional information on single exposures that are hazardous is still needed, it is clear that there is no such magic number.

Impulse Noise: Gunfire

The extreme instance of short, intense noise is, of course, the impulse noise generated by explosive sounds such as gunfire. As indicated above, in most situations the hazard to hearing can be estimated from the exposure level and duration; if the level is fluctuating, then the average level is sufficient to specify the hazard. However, if impulses with peaks above 140 dB are involved, then the level, duration, and frequency of occurrence of these single peaks must be determined, since safety limits depend on all of these (Coles et al, 1968).

In those persons habitually exposed to gunfire, such as military ordnancemen and policemen, hearing losses that develop are in no way distinguishable from those caused by industrial noise (Ward et al, 1961). The typical NIHL begins and is generally most severe in the same region as the NIHL from steady noise, namely 4000 to 6000 Hz. Apparently the "whipping" of the cochlear partition produced by the traveling wave associated with a single impulse reaches its maximum amplitude in the 10-millimeter region also.

In gunfire-induced hearing loss, it is seldom clear to what extent the losses grow relatively gradually, as the "microtrauma" hypothesis would predict; often the loss can reasonably be attributed to a particular incident of unusual severity which was followed by

several days of ringing in the ears (tinnitus), a feeling of fullness, and a temporary loss so severe that the listener noticed that sounds appeared muffled. (For an example of a case of documented damage from a single firecracker explosion, see Ward and Glorig, 1961.) However, lest one attach too much importance to such retrospective reports in attempting to determine the cause of a particular hearing loss, it must be emphasized that these same symptoms are often reported by gunners with completely normal hearing (Ward, 1957).

Head Blows

As far as the ear is concerned, a severe head blow amounts to the same thing as an explosion and, therefore, can produce the same type of loss (Rüedi and Furrer, 1946). In either event, the stapes moves suddenly inward, sending an impulse traveling down the cochlear partition - in the one case because of an air pressure condensation, in the other because of inertia of the ossicular chain. That is, when the head is struck the skull moves, but the ossicles do not because they are not connected directly to the skull. Hence the cochlea, for all practical purposes, is in this case driven into the stapes! Apparently an inward movement of the stapes is more damaging than an equal outward movement, because a blow to one side of the head usually produces more loss in the contralateral ear. This underscores the importance of the direction of the head blow in determining the degree of damage and probably explains why Kumagai (1959), in a study of 100 cases of auditory disturbance following head injury, found no relation between duration of unconsciousness and subsequent HLs.

Individual Susceptibility

That not everyone is equally susceptible to NIHL was noticed at least 160 years ago by Fosbroke (1830), who wrote the first major review in English of the problem of NIHL. He ascribed the difference to constitutional factors - specific weaknesses, such as those responsible for hemophilia. Even now, it is clear that structural details - the static and dynamic characteristics of the middle and inner ears - would indeed be expected to determine how much exposure to noise a given ear can tolerate before NIHL occurs. Such characteristics as the stiffness of the cochlear partition, thickness of the basilar and tectorial membrane, blood supply to the cochlea, rate of oxygen metabolism, and density of afferent and efferent innervation must be important. Unfortunately one cannot measure these in the intact organism, so again indirect measurement must be used to identify the more noise-susceptible individuals before NIHL is incurred.

On face validity grounds, the most likely overall predictor of susceptibility to NIHL should be auditory fatigue (Peyser, 1930). The middle ear characteristics that determine how much sound reaches the inner ear should affect temporary and permanent effects similarly, and many of the structural details of the cochlear partition and blood supply should be relevant for both phenomena.

However, to date the use of fatigue tests as susceptibility predictors is not practicable. The correlation among individual differences in auditory fatigue produced by low-, medium-, and high-frequency noise is not high, and none bears any relation to differences in auditory fatigue from impulses (Ward, 1968). It is, therefore, clear that if auditory fatigue is to be of any predictive value whatever, it should be the fatigue cause by a subtraumatic exposure to

a noise having the same characteristics as the noise to which the individual is to be exposed (and the utility of even so specialized a test is still to be demonstrated).

Sex

While we apparently cannot measure the functional characteristics that are important to individual differences in resistance to NIHL, the possibility remains that more readily measured factors are important. For example, the degree of hearing loss is smaller in women than in men, even when they all work in the same noise (Flodgren and Kylin, 1960; Dieroff, 1961). However, this does not necessarily mean that women are more resistant to NIHL than men and should therefore be given the noisier jobs (although it is possible). Women are exposed much less to the potentially damaging noises of everyday life, especially gunfire, they show a higher absentee rate (giving the ear more recovery), and, until recently, were freer to leave a job that was unpleasantly loud to them. These facts may account for the difference.

Age and Experience

Although one could argue that the "young, tender" ear of a teenager will probably be more easily damaged than the ear of a middle-aged worker, it is just as sensible to suppose that the "young, resilient" ear of the teenager will be *less* easily damaged. There is about the same amount of inconclusive evidence for both views. Laboratory studies suggest that, at least in the guinea pig, the very young auditory system is more susceptible to damage (Coleman, 1976; Danto and Caiazzo, 1977), but the issue is far from settled. Similarly, no convincing experimental support exists for the notion that the ears of persons who work in noise gradually become "toughened" and so more resistant to NIHL.

One example should suffice to illustrate the ambiguity of the results of most field studies bearing on this question. Kup (1966) divided the audiograms from 1198 German ironworkers (hearing was measured at least 16 hours after the last noise exposure) into two groups - those from men who began working in noise before or after age 35. Each group was then further divided into four subgroups according to years of noise exposure. In men with between 6 and 15 years of exposure, there was a slightly higher average HL for those who began working after age 35, which led Kup to conclude that the older ear is more susceptible. However, the fact that there was no difference in HL between the two corresponding subgroups composed of men with 1 to 5 years of noise exposure (when most of the loss appears) implies that some other causal factor may have been acting. This confounding variable could well have been exposure to military noise. That is, men who began working in noise at age 40, and who at the time of the survey (1965) had worked 10 years, would then be 50 years old. On the other hand, if they entered at age 25, they would then be only 35. In 1945 (the end of World War II) the first group would have been 30 years old, whereas the second would have been 15. Obviously the first group will, on the average, have suffered more damage from exposure to noise from gunfire and tank noises. This confounding effect of military service all too often leads to inconclusive results in such longitudinal surveys (Wagemann, 1966).

Existing Noise-Induced Hearing Loss

Is an ear that has already suffered NIHD more susceptible to further injury than a normal ear? At first glance, the results of a 7-year longitudinal study of the hearing of steelworkers who wore no ear protection despite work levels ranging up to over 100 dBA (Howell, 1978) would appear to answer the question in the negative: the number of decibels of additional hearing loss over the 7 years was the same in men whose initial HL was 25 dB or greater as in those with normal hearing initially. But if the HL in an already damaged ear progresses from 40 dB to 50 dB, is this 10-dB additional loss the "same" injury as a 10-dB shift from 0 to 10 dB HL in an initially normal ear? Not really; if the individual already has a loss great enough to interfere with perception of speech, then a 10-dB additional loss will produce an exaggerated effect, while no one would ever notice the effect of a difference between 0 and 10 dB HL. So perhaps the study could be regarded as showing that the damaged ear is indeed more susceptible.

On the other hand, certain evidence implies that the damaged ear will be *less* susceptible - for example, the fact that workers habitually exposed to a given noise environment show the greatest damage in the first few years. If the damaged ear were as susceptible as a normal ear, then one would expect the damage to increase at a constant rate; if 10 dB were lost the first year, then the (damaged) ear should lose 10 dB more the second year, and so on. This does not occur. Furthermore, Pye (1974) demonstrated that an existing loss in one frequency region has no effect on susceptibility to damage in a different frequency region. Therefore, preventing men with moderate existing losses from working in noisy area would eliminate an unnecessarily high number of qualified workers. On the other hand, men with losses that are already handicapping (that is, having average HLs at 500, 1000, 2000, and 3000 Hz of over 25 dB) should be excluded from all further hazardous noise exposure, because at this point overconservativism is not only medically desirable but also economically necessary, because any further deterioration will automatically be compensable.

Middle Ear Disorders

Most middle ear problems will reduce the flow of energy to the cochlea and therefore diminish the amount of NIHL produced by a given noise. Thus, in cases of unilateral conductive loss, less NIHL will be found in the "protected" ear (Dieroff, 1964; Chung, 1978). A chronic perforation of the eardrum also seems to protect the hearing (Dohi, 1953). Conversely, it would be expected that diseases involving paralysis of the middle ear muscles would tend to make the ear more susceptible to NIHL, because such an ear is more susceptible to auditory fatigue (Zakrisson, 1979); however, only seldom will a patient with such a paralysis be working in noise anyway, so no direct data are available.

Although otosclerotic ears are partially protected from hearing loss, operations to alleviate the conductive loss may occasionally cause a sensorineural loss, either directly from the drilling noises that are conducted to the organ of Corti during stapedectomy, or indirectly from serofibrinous labyrinthitis somehow induced by the operative procedure (Schuknecht, 1962). Mastoidectomy may also produce NIHL (Palva and Sorri, 1979).

The possibility also exists that chronic otitis media may cause a sensorineural loss through invasion of the cochlea by bacteria or viruses through the round or oval windows.

Such losses have been produced in laboratory animals; however, the evidence that this occurs in humans is somewhat inferential. It cannot happen very often; in a study of 64 men with long-standing unilateral otitis media, Reker (1975) found that in every case, the threshold at 4000 Hz, measured by means of bone conduction in order to assess the state of the cochlea, was better in the ear with otitis media than in the other ear, the average difference being 30 dB. The otitis media, by reducing the energy reaching the cochlea, clearly prevented more loss of hearing than whatever it produced by direct action.

Drugs and Chemicals

It is well established that drugs that can produce hair-cell damage may enhance NIHL. That is, noise exposures that cause no damage in the normal animal may have a pronounced effect when the animal is being given subtoxic doses of oleum chenopodil antihelminthics, salicylic acid, quinine or dihydrostreptomycin (Sato, 1957), kanamycin (Darrouzet and de Lima Sobrinho, 1962), or neomycin (Voldrich, 1963). Persons taking such drugs probably should be warned against exposure to noise.

Miscellaneous

As in the case for all human afflictions, attempts have been made to link hearing loss from noise, or susceptibility to such loss, to nearly every type of human frailty, such as smoking, use of social drugs or stimulants, artificial food additives, lack of exercise, over-exercise, over-eating, vitamin deficiency, poor posture, promiscuity, sexual inactivity, and so on. Such studies, however, are seldom adequately controlled; when they are, the results usually deny a significant relation.

A case in point is smoking. Because smoking has an effect on the circulatory system (of the ear as well as the general body), numerous studies have attempted to show a causal relation between smoking and hearing loss. Although Chung and colleagues (1982), in an analysis of 5440 audiograms of men exposed to 90-dBA noise, found a significantly higher HL in heavy smokers than in nonsmokers in Canada, a random 7913-person sample of the population in the USA indicated that female smokers had an average blood pressure that was *lower* than that of nonsmokers (Singer et al, 1981). Cunningham and associates (1983) found no difference in high-frequency (8 to 18 kHz) thresholds between smokers and nonsmokers, and Dengerink and coworkers (1984) actually demonstrated that smokers developed less auditory fatigue than did nonsmokers after a 5-minute exposure to white noise of 110 dB SPL. Obviously, smoking is not a very important factor in determining susceptibility to hearing loss.

General Health

Although it seems reasonable that the ear's resistance to NIHL 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, the evidence on this topic is either anecdotal or so inferential that little confidence can be placed in its validity. A typical result is that of Willson and colleagues (1979), who measured 28 indices of general health (blood pressure, blood and urine components, and so forth) but could find no relation between health and hearing loss.

Nevertheless, differences in susceptibility to damage among people, and in vulnerability from day to day in a given individual, play an important role in the occurrence of hearing loss. Many cases of "sudden deafness", for example, seem to be precipitated by a noise exposure that has little effect on most people and that had been experienced in the past by the individual concerned. An illustration of this is provided by the hearing losses apparently produced in a few of the individuals who were exposed to the ringing of a cordless telephone. It seems clear that a fraction of a second of exposure to a 138-dB telephone bell whose energy is mostly at 800 to 100 Hz caused a slight to moderate hearing loss at the appropriate frequency (1000 to 1500 Hz) in at least three persons whose hearing status was known before the incident (Orchik et al, 1987); however, this same sound has had no effect on innumerable other people. Indeed, as mentioned earlier, Sommer and Nixon (1973), in a study of the possible auditory effects of the air-bag restraint system for automobiles (a study in which the intensity was gradually raised in successive tests), exposed 10 young, healthy men to a 153-dB noise for 0.4 second and found only a small temporary hearing loss that disappeared in a few minutes. It may be that those few persons who suffer damage from this particular exposure are unusually vulnerable because of some other type of illness or infirmity. Only careful accumulation of health data on such persons for whom a pre-exposure audiogram is available can be expected to solve the riddle of susceptibility.

Treatment

Prophylaxis

Evidence that NIHL can be retarded by medication is even more meager than that on its enhancement, despite a voluminous literature on substances that have been tried, generally without any attempt at experimental controls. For example, for some time it was thought that massive doses of vitamin A might decrease the susceptibility to noise (Rüedi, 1954), but controlled studies of auditory fatigue with and without vitamin A gave negative results (Ward and Glorig, 1960), and Dieroff (1962) reported that in a group of 15 machinists given vitamin A for a year, the increase in NIHL was no different from that observed after a year in which no vitamin A was administered. So although it is possible that a vitamin A deficiency may increase susceptibility, an excess is no better than a normal supply.

Similarly, although suggestive indication of a slight ameliorative action for such substances as nicotinic acid, vitamin B_1, hydrochloric papaverine, nylidrin, thioctic acid, and chlorpromazine has been reported in Japan (Nakamura, 1964), for adenosine triphosphate in Czechoslovakia (Faltýnek and Veselý, 1964), for ephedrine (Stange and Beickert, 1965) and gingko-biloba extract (Stange and Benning, 1975) in Germany, and for amino-oxyacetic acid in the USA (Bobbin et al, 1976), considerable effort must still be expended before one can be confident that any of these will be really beneficial.

Even in cases of acoustic trauma, in which victims may be treated shortly after the incident, the best treatment may consist of inserting an ear plug in the affected ear in order to reduce auditory stimulation to a minimum. Voldrich (1979) exposed guinea pigs for 5 minutes to a level of 145 dB, thus inducing acoustic trauma in each animal. However, while half of the animals were then put into quiet surroundings, the other half were subjected for 30 minutes to a 90-dB noise before they too were placed in quiet surroundings. The extent of cochlear damage was about three times as great in the animals in which acoustic trauma

was followed by the otherwise innocuous 90-dB exposure. This result is consistent with the hypothesis that rips or holes in the reticular lamina that are induced by the 145-dB exposure allow mixture of endolymph and perilymph, poisoning the hair-cells, and that these membrane ruptures will heal more slowly while the cochlea is in even moderate motion.

If more than a few hours have elapsed since the acoustic trauma, however, the use of agents that presumably increase the blood circulation and hence hasten or augment the recovery process is the only program that sometimes may prove useful. At the moment, the most viable contenders for the title of anti-noise elixir are breathing carbogen or injecting dextran. Both have shown a slight advantage, in the treatment of either acoustic trauma or sudden deafness, over placebo substances in some studies (Hippert and Pfalz, 1970; Martin and Jakobs, 1977; Joglekar et al, 1977; Brown et al, 1982; Pigramm and Schumann, 1986), but not in others that seem to have been as well controlled (Eibach and Börger, 1980; Dauman et al, 1985). As in the case in which the effect of general health on susceptibility was of concern, only carefully controlled studies can be expected to show any beneficial effect of medication on either NIHL or sudden deafness, although treatment with procaine hydrochloride (Novocain), ergoloid mesylates (Hydergine), vasodilators, vitamin E, dextran, and carbogen or by stellate block no doubt will continue to be used by the practicing physician (Niemeyer, 1962), despite the fact that there really is no convincing evidence that a placebo would not produce just as much effect as any of these.

Reduction of Noise

There is only one course of action that can be taken in advising a person who has suffered NIHL: he or she should be told to limit noise exposure, either by changing jobs or by wearing suitable ear protection in order to reduce the effective noise level (if possible, to below 80 dBA). Although there is no further deterioration of hearing once the person has been removed from the noise, it is also clear that very little improvement can be expected, since hair-cells do not regenerate. Ordinary hearing aids are also of limited benefit in sensory losses such as NIHL; selective amplification of the high frequencies in an attempt to restore perception of, say, 4000 Hz, cannot be expected to succeed if the 4000-Hz receptors have all been destroyed. Another possibility is just beginning to be explored - the shifting of energy from the high-frequency region downward. By electronic means, one can drop all frequencies either by a fixed amount or, better, by a common ratio, so that, for example, a 4000-Hz tone becomes a 2000-Hz one, 3000 Hz is changed to 1500 Hz, and so on. Whether, with a suitable relearning period during which they hear *only* this shifted sound, persons with severe noise-induced high-frequency losses may benefit from such devices is still to be determined.