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BIOLOGY OF HEARING AND NOISE-INDUCED HEARING LOSS

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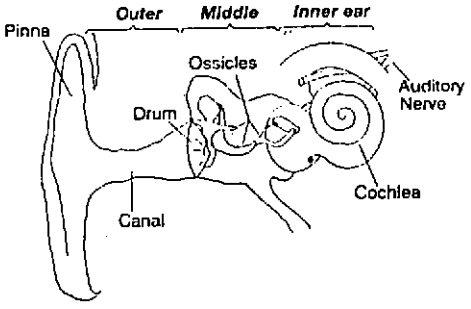
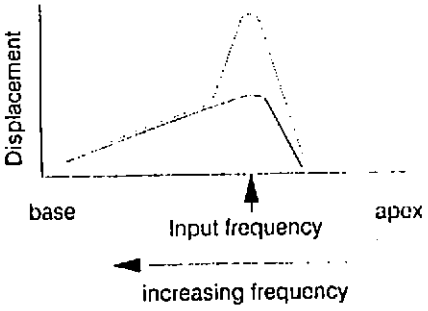
1. INTRODUCTION

In Thomas Kuhn's [1] book "The Structure of Scientific Revolutions" science is seen as developing in a step-wise function, where the step risers correspond to periods of immense development brought about by a fundamental and widely agreed re-assessment of the way scientists think about their subject. Most people working in hearing research would agree that their subject has passed through two such revolutions during the last 40 years. The first of these was the discovery during the '50s by von Bekesy [2] that sound coding in the inner ear, the cochlea, is achieved by a "place principle". Sound waves striking the ear drum produce maximum displacement of the basilar membrane at different distances along the length of the cochlea (Fig. 1). The position of maximum displacement dependends on sound frequency, with high frequencies represented at the base of the cochlea and low frequencies represented at the apex. The second revolution, and the one on which we concentrate in this paper, has occurred during the last 15 years, and has been the result of discoveries in several laboratories. These discoveries (collectively called "active processes", see ref. [3]) have demonstrated that the cochlea does not act as a simple, passive receiver. Rather, a specialised cell type within the cochlea, the outer hair cell (OHC), feeds energy into the cochlea to sharpen dramatically the frequency tuning of the mechanical response discovered by Bekesy.

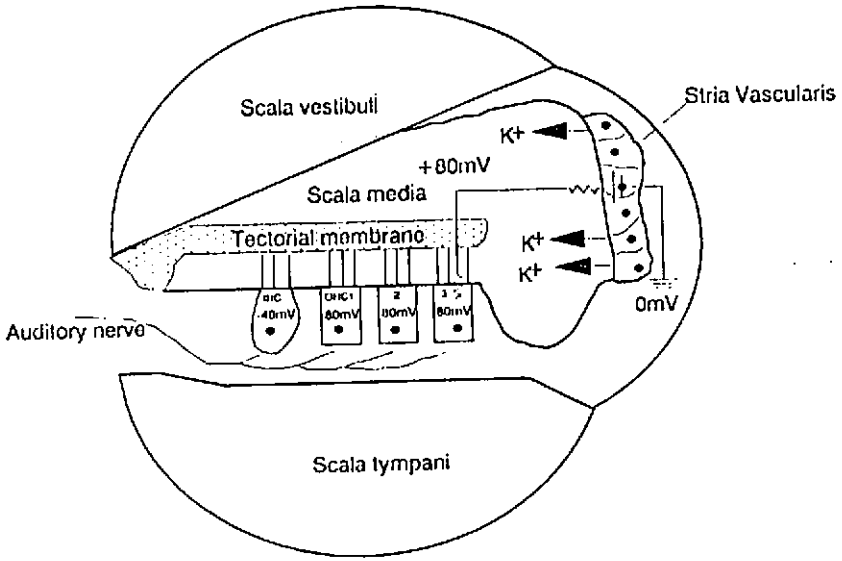
The discovery of active processes in the cochlea has led to a reevaluation of the mechanisms underlying noise-induced hearing loss and the properties of stimuli that can induce a loss. Our own interest in this subject derived partly from basic research on cochlear trauma, and partly from a concern that the "Noise at Work" regulations of the Health and Safety Executive (HSE;[4]) were being applied somewhat indiscriminately to stimulus situations other than those on which the research underlying the regulations was originally based. In this paper we outline the biology of noise-induced hearing loss and go on to consider the implications of that research for human exposure to a novel, but prevalent form of noise, reproduced music delivered via personal stereos.



Schematic diagram of human peripheral auditory structures.



Passive (solid) and active (dotted) travelling wave envelopes on the basilar membrane for given input frequency.



Cross section of a single turn of the mammalian cochlea.

2. ACTIVE PROCESSES

An early problem recognised from Bekesy's work was the apparent mismatch between the mechanical response of the basilar membrane and the exquisite ability of humans to discriminate pure tones. Under optimal conditions we can detect a change of less than 3Hz in a 1,000Hz pure tone [5]. In contrast, Bekesy's travelling wave envelopes had a half-power bandwidth of about one octave. Bekesy suggested that a sharpening of tuning occurred through neural processing, but the finding that auditory nerve fibres were sharply tuned cast doubt on this notion. If any neural sharpening occurred it would have to take place in the hair cells. Nevertheless, the idea of neural processing acting as a "second filter" gained favour, and for almost 20 years this was accepted doctrine.

In the early '70s, Russell and Sellick [6] performed the seemingly impossible task of recording the electrical activity inside hair cells in a living animal. The technical difficulty of this procedure may be appreciated by considering that the temporal bone, housing the cochlea, is the hardest bone in the body, and that the whole cochlea is about the size of a pea and has over a million essential moving parts [7]. Perhaps the most important result of Russell and Sellick's work was the finding that the tuning of inner hair cells (Fig. 1) was as sharp as that of auditory nerve fibres. This finding destroyed the notion of neurally-mediated sharpening, since the receptor potential is the very first stage of neural processing. The final death knell of the second filter hypothesis came in the early '80s when two groups independently demonstrated (see ref. [3]) that, using heroic tissue preservation techniques, the mechanical tuning of the basilar membrane could be as sharp as that of the nerve fibres and the hair cells (Fig. 1).

One of the main interests of recent studies of cochlear mechanics is not the occasional demonstration of sharp tuning, but the more common finding of broad tuning. Bekesy's measurements were made in cadavers (an obvious necessity in the case of his human studies!), but it gradually became clear that the physiological condition of the preparation was all-important in determining the sharpness of tuning. In particular, measurements made in the one subject before and after oxygen deprivation or death showed a broadening of mechanical tuning that was related to general pathology. These findings therefore suggested that the mechanics of the cochlea were somehow dependent on active physiological processes.

It is, of course, also possible that the differences observed between living and dead preparations were simply the result of post-mortem changes in tissue stiffness or some other passive property of the cochlea. However, in 1978, Kemp [8] showed that active processes must occur in the cochlea. By sealing a miniature

microphone in the ear canal, together with a sound delivery system, it was found that sound stimuli were followed by an acoustic signalthat emanated from the ear. Although these otoacoustic emissions (OAEs) were originally called cochlear echoes, they were not true echoes since they had larger than predicted latency variability from case to case, their waveform did not reflect that of the signal and, as shown in subsequent studies [9], they were physiologically vulnerable. Moreover, about 25-40% of normal human ears produce spontaneous (S)OAEs in the absence of stimulation. While the existence of SOAEs remains the most powerful and direct evidence for active processes in the cochlea, it does not in itself provide any indication of the site or nature of the energy input.

The transduction of mechanical into electrical energy in the inner ear has long been thought to involve a gating of currents into hair cells by modulation of a variable resistance between the positively charged scala media and the negatively charged interior of the hair cells (Fig. 1). The question remained, however, of the separate role played by the inner (IHC) and outer hair cells. The OHCs were known to be more vulnerable to trauma (eg. through noise, see below) than the IHCs, and combined anatomical and physiological experiments showed that damage to or loss of the OHCs was associated with a 30-40dB threshold increase and a broadening in frequency tuning of auditory nerve fibres. Thus, deliberate cochlear trauma, resulting in a selective lesioning of the OHCs, mimicked in auditory nerve responses the effects of inadvertent trauma seen in the mechanical response of the basilar membrane. These correlations raised the possibility that the OHCs might be critical in determining the mechanical response of the cochlea.

Another indirect line of evidence for this proposal came from purely anatomical studies of the cochlea. Each IHC was known to make contact with many auditory nerve fibres, whereas a single auditory nerve fibre contacted many OHCs. It therefore seemed that IHCs were especially important in the delivery of information to the brain. Conversely, the efferent fibres (those coming from the brain) were known primarily to contact the OHCs. This segregation of innervation suggested a segregation of function. The IHCs were seen as the true, sensory receptors, whereas the OHCs were effector cells, responsible for modulating the output of the IHCs.

The precise way in which the OHC modulation might be achieved is still a matter of conjecture. However, another major discovery of the last decade was that, in tissue culture, OHCs can be induced to move [10]. In this procedure, cells were dissociated by enzymatic digestion of connective tissue. The cells could then be kept in culture for some hours. A microelectrode applied to the cell membrane was used to inject current. OHCs could be induced either to elongate or contract (by up to 5%, ref. [11]), according

to the polarity of the injected current. In the living animal, the stimulus current for the contractions could be supplied by the modulation of the variable resistance across the OHC membrane, resulting in a bi-directional transduction (see above and Fig. 1). The contractions are thus seen as a means by which the OHCs could modulate the mechanical response of the cochlea to produce sharp tuning.

3. HEARING LOSS

Noise-induced hearing loss has usually been divided into temporary and permanent threshold shifts (TTS and PTS). As the names imply, TTS is a reversible phenomenon, whereas PTS is used in cases of indefinitely prolonged impairment. Since it is unclear to us that there is a qualitative difference between these phenomena we will refer to both simply as hearing loss, at least in this section dealing with the biological consequences of noise exposure in animals. The stimuli used to induce hearing loss have usually been relatively short (up to a few hours) periods of very high intensity, spectrally simple signals, such as pure tones or broad-band noise. The techniques used to study the effects of the loss have predominantly been light and electron microscopy, and physiological recording of cochlear potentials or auditory nerve fibre responses.

Structural cochlear changes after noise exposure have recently been discussed in this journal by Ade Pye [12]. The index of change used in these anatomical studies has traditionally been the number of hair cells, but more recent reports have used ultrastructural (ie. electron microscopic) indices, sometimes in conjunction with physiological measurements. Probably the most surprising aspect of this work has been the extreme variability between individual ears of the damage produced by noise, even when the stimulus conditions have been strictly controlled. Nevertheless, the mean degree and extent of damage increases with stimulus level, and several fairly distinct types of structural damage have been consistently reported. These include loss of hair cells, loss or damage of stereocilia, and swelling of the nerve endings underlying the base of the hair cells. OHCs are generally more severely affected than IHCs, and the first row of OHCs (OHC1, those closest to the IHCs; Fig. 1) are the most affected of all. Another surprising finding is that the region of the cochlea in which maximal damage occurs to the stereocilia is about 0.5 octaves higher than the frequency of the traumatizing tone. While most of the changes are observed immediately after presentation of the traumatizing stimulus, some only develop following a latent period of 10-30 minutes [12]. Finally, there is evidence of some recovery, particularly following damage that is limited to the stereocilia, after a further period of days or weeks. In certain non-mammalian animal species (eg.

birds) regeneration of lost hair cells and recovery of function has recently been shown, but this has not been found in mammals.

Functional changes following acoustic trauma are also extremely variable. Unlike the structural changes discussed above, it ispossible to follow the time-course of compound action potential (CAP) audiograms (reflecting neural activity in the auditory nerve), or auditory brainstem responses in one animal before, during and after exposure to the traumatizing stimulus. Physiological studies invariably show a recovery of function that is related to the level and duration of the stimulus. Early hearing loss ("TTS") occurs at all frequencies higher than that of the stimulus, whereas long-term loss ("PTS") is greatest at, or slightly above the stimulus frequency.

As outlined above, a powerful approach has been the combined use of ultrastructure and physiological recording in the same animals. This approach has shown that structural and functional aspects of hearing loss are quite well correlated in individuals, despite the inter-animal variability of both measures. In one study [13] it was found that a CAP threshold decrement was always accompanied by stereocilia damage in OHC1. However, in some cases, stereocilia damage was not accompanied by a threshold loss. An important point that arises from these observations is that the indices used to monitor hearing loss can never give a complete picture of the full extent of the loss. In this case, structural damage was not accompanied by a threshold decrement, but it is entirely possible that some other functional measure (eg. frequency resolution, see below and [14]) may have been affected by the damaged stereocilia.

Recent studies of the biology of hearing loss have concentrated on the cellular mechanisms underlying the loss. In the most recent of those studies [15], a reduction in the AC current (the "cochlear microphonic" or CM) across the variable resistance of the OHCs (Fig. 1) was observed after overstimulation of the ear with a pure tone. Normally, the amplitude of the CM increases with increasing sound level until it saturates. This saturation is due to the finite number of receptor channels in the apex of the OHCs through which current can flow. Following the traumatizing stimulus, the amplitude of the CM response at all sound levels was reduced and the response saturated at a lower amplitude. The effect of acoustic overstimulation was thus to block transduction channels in the apex of the OHCs. This in turn reduced the electro-mechanical coupling between the basilar membrane and the OHCs (the bi-directional transduction). The end result was a reduction in the sharp tuning of the cochlea and the elevation of auditory nerve fibre thresholds for the frequencies affected by the traumatizing tone.

4. PERSONAL STEREOS

In a recent article in "Which?", the consumer magazine, it was claimed that personal stereos can damage your hearing [16]. The evidence cited for this claim was based on the HSE's Noise at Work regulations [4]. Those regulations specify the maximum permissable exposure to sound in working environments, and are themselves based on research mainly carried out in the '50s and '60s. Some of thatresearch (eg. Baughn [17]) attempted to assess the prevalence of hearing loss in humans working in various, 'typical' environments. Unfortunately, the research is questionable for a number of reasons, including the uncontrolled nature of the noise exposure, the assumptions made regarding the relation between TTS and PTS and the selection of the control (normal) population.

The HSE regulations invoke the "equal energy hypothesis" (EEH) to deal with noise durations shorter than 8 hours. The EEH allows for a halving of noise exposure time for every 3dB increase in sound level. Although the EEH has received some support from animal research, recent studies suggest that various forms of intermittent noise incorporating intense peak levels produce a greater degree of hearing loss than that produced by continuous sounds of the same total energy. These recent studies raise two problems. First, they question the use of short duration sampling times for application of the HSE regulations. Second, they suggest the need for separate research dealing with the effects of different noise types.

Given the prevalence of personal stereo use and, indeed, of exposure to all modes of recorded sound, it now seems timely to conduct some research specifically directed at the effects of that form of sound on the auditory system. Our feeling is that the methodological and ethical problems of studies in humans suggest the need for more animal research. In terms of stimulus control and assessment of the consequences of the stimulation, animals offer obvious advantages. The disadvantages that can be readily identified include possible differences between human and animal susceptibility to hearing loss, and the relative difficulty of assessing behavioural responses to sound in animals. Regarding the first of these points, it may be possible to apply filtering functions (such as the human 'a' weighting function) in order to present test sounds within the appropriate frequency range for the animal under test. Alternatively, we may choose an animal species having a frequency range similar to that of humans. Regarding behavioural testing, parallel physiological and audiometric tests on several animal species have confirmed the similarity between CAP and behavioural audiograms. However, these considerations raise again the question of the appropriateness of any form of audiometry as a test procedure. West and Evans [14] have recently shown that frequency resolution provides an earlier sign of the harmful

effects of amplified music than simple thresholds, and that high resolution audiometry provides earlier evidence than standard audiometry. We suggest that, although relatively simple audiometric tests should be applied initially to sort out the parametric aspects of the noise exposure, there will be a future need for more detailed and wider ranging test procedures, probably on a smaller sample of subjects.

5. REFERENCES

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