Peer Commentary on 'Anatomical and Physiological Consequences of Acoustic Trauma: A Short Review' by R. V. Harrison

A View From Within Will Be Needed to Understand Acoustic Trauma

The review by Robert Harrison focuses in a very lucid way on two aspects of the cochlear damage produced by overexposure to sound. One aspect is the view from outside, using scanning electron microscopy to visualize the cilia of the hair cells; the other is the view from a distance using single auditory nerve fiber recordings to probe the functional changes in the damaged regions of the cochlea. Correlations between the findings of the two approaches have taught us a great deal about the importance of the integrity of the outer hair cells, including the early signs of damage in the cilia, for the sensitivity and frequency selectivity of the cochlea. Despite these advances in our knowledge, we still do not know why sometimes there is recovery from noise exposure and sometimes there is not, and in general, we also do not know very much about the inter-individual variability of noise exposure.

For some time, it has been known that metabolic factors are important to understand the effects of noise trauma. Drescher (1976) showed that noise was less damaging in the cochleas that were hypothermic (29 C instead of 38 C) suggesting that the reduced metabolic activity protects the cochlea from the energy demanding over stimulation. Axelsson and Dengerink (1987) showed considerable evidence for a significant reduction in cochlear blood flow during noise exposure, again pointing to changes in cochlear metabolism. This probably will have a much larger impact on the outer hair cells whose function is not only transduction (as it is for the inner hair cells) but also to supply energy to the basilar membrane thereby enhancing its vibration amplitude and fine tuning. Recently, it has been reported (Branis & Burda, 1988) that the application of ascorbic acid reduces the percentage of outer hair cell loss during noise exposure. Does this mean that a lack of vitamin C potentiates the effects of noise exposure?

All this suggests that a real understanding of the damaging effects of noise exposure probably will come only from a biochemical and/or intracellular recording approach to changes that occur in outer hair cells. It is a view from within that is required to understand the many similarities and differences between the various ototoxic agents, of which overexposure to sound is only one.

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In this article, Harrison has described some of the underlying anatomical and physiological consequences of acoustic trauma, alluding to possible behavioral manifestations of these effects. In my commentary, I would like to expand on the implications of reduced tip-to-tail differences of FTC's for speech perception by impaired listeners.

In normal ears, there typically is a 40–60 dB difference between threshold at CF and on the tail of the FTC (see Figures 6, 7, and 8 in this article). Still, even a fiber with a high CF (i.e., one that innervates the basal end of the cochlea) will respond to intense low frequency stimuli. Psychophysically, this effect underlies upward spread of masking.

In an impaired ear, this tip-to-tail threshold difference is invariably reduced, such that thresholds are more similar as a function of frequency. In some cases, one may observe abnormally sensitive thresholds to low frequency stimuli (see Figures 7 and 8 in the article). These findings are consistent with the observation of greater upward spread of masking in listeners with cochlear hearing loss (see, for example, deBoer & Bouwmeester,1975; Leshowitz & Lindstrom, 1977; Gagne, 1982) and led to speculation that the poor speech discrimination ability (in noise) of some impaired listeners may be due to reduced tip-to-tail differences in their FTC's (Gorga & Abbas, 1981).

Recently, Stelmachowicz, Jesteadt, Gorga, and Mott (1985) measured speech perception ability in normal and impaired listeners in the presence of low-pass and broadband noise, and correlated these results with various aspects of the psychophysical tuning curve (PTC). (PTC's are derived from masking experiments that attempt to measure the behavioral equivalent of the single fiber FTC.) Although normal and impaired listeners performed similarly in the presence of broadband noise, the impaired listeners performed more poorly in the presence of low-pass noise. Furthermore, their performance in the presence of low-pass noise was correlated most with the tip-to-tail difference on their PTC's, which were reduced compared to normal.

In summary, the observed changes in single unit FTC's led to predictions regarding the underlying mechanisms for poor speech perception in noisy environments by impaired listeners. Recent psychophysical data support these predictions. Understanding the mechanisms responsible for the performance deficits experienced by impaired listeners may lead to the development of better rehabilitative techniques, including improved design of amplification systems.

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Harrison's review of the contemporary research on the anatomical and physiological effects of acoustic trauma is enlightening to all who are interested in hearing loss. Recent studies have shown us that the effects of even subtle changes in the structure of the cochlea can result in hearing loss. I would like to emphasize also the work of Sellick, Patuzzi, and Johnstone (1982) and of Leonard and Khanna (1984) who have demonstrated that the mechanics of the basilar membrane can change following injury to the cochlea and that some types of sensorineural hearing loss may occur in conjunction with a reduced basilar membrane vibration (relative to normal) for a given sound level. This suggests that sensorineural hearing loss may have a cochlear (mechanical) component in addition to the sensory cell damage described by Harrison. Many theorists today propose an active role for the hair cells in the mechanical vibration of the cochlea.

I also would like to propose to the reader that the temporal variation in real-world noise exposures may be more troubling than the spectral variation because the A weighted spectrum tends to be a good first approximation of the relative noxiousness of various frequencies. It is not known precisely why A weighting reflects the potential for damage of various sounds. Perhaps it is because it mimics the middle ear transfer function. On the other hand, the variation of temporal patterns of sound exposures has no such simple rule to equate exposures; the equal-energy theory seems to hold only for steady-state exposures (Ward & Turner, 1982). Finally, many laboratories have demonstrated that the individual variability in amount of damage between animals who have been exposed to precisely the same noise is quite large; this same variability also appears to exist in humans.

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Response to Commentaries by Drs. Eggermont and Gorga

In my short review, I chose (because of space limitations) to focus on only two areas pertaining to acoustic trauma (cochlear anatomy and electrophysiology). The commentaries by Drs. Eggermont and Gorga raise important issues in areas not covered in detail in the review, namely, the metabolic/biochemical alterations induced by acoustic trauma and the psychophysical consequences of cochlear damage, respectively.

I agree with Eggermont that cellular dysfunction or degeneration in the cochlea is ultimately caused by a change to biochemical events within the cell. Indeed, all our observations of morphological changes and degraded electrophysiological responses are higher level expressions or sequelae to such basic changes. Some might go further in the reductionist tradition and claim that our understanding (of the effects of cochlea trauma) will ultimately come from knowledge of the genetic mechanisms that control cell development, maintenance, and death. "The view from within" is clearly necessary and is a research area that must take on increasing importance in the future. However, on a philosophical note, I would add that, although the elucidation of very fundamental processes is essential, there always will be the formidable task of linking basic biochemical changes with events observed on a higher level of complexity. Thus, what are required are studies that attempt to span levels of complexity so as to correlate, for example, reduction in cochlear threshold sensitivity with changes in stereociliar mechanics and, after that, with interruptions in the biochemical pathways involved in the maintenance of hair cell protein structure.

At the other end of the biological complexity scale, we see another example of the problem of spanning organizational levels. What is the correlation between specific neural events observed in the brain and complex psychological tasks? Michael Gorga's commentary provides a good example of such a physiological-psychophysical correlation in relation to how changes in cochlear neuron tuning properties relate to degraded speech intelligibility. We know that cochlear hearing loss involves degradations other than just threshold sensitivity; some of these are outlined in the review, and Gorga mentions an important factor not discussed, that of the reduced tip-to-tail threshold difference.

In extrapolating from physiological data to complex behaviour, there are (at least) two stages: first, between the physiological measures made in animal models (e.g., tuning curves) and their most direct psychophysical equivalents (psychophysical tuning curves, critical band measures, etc.); secondly, between those basic psychophysical factors and more complex human behaviour (speech intelligibility). Gorga cites some interesting studies with respect to this latter comparison that show that reduced tip-to-tail threshold differences result in poor speech intelligibility in the presence of certain types of background noise. I concur with his final point that we must determine the basic factors (either physiological or psychophysical) that contribute to degraded speech intelligibility in order to rationally design prosthetic devices to aid the hearing impaired.

Response to Commentary by Dr. Turner

Christopher Turner has introduced a different perspective on the effects of injury to the cochlea by drawing attention to the changed mechanical properties of the basilar membrane. However, changes to basilar membrane mechanics do not always imply that the basilar membrane itself has suffered some mechanical injury. As Turner correctly points out, there is much evidence (e.g., oto-acoustic emissions) for an active biomechanical feedback system in the cochlea, with the basilar membrane, the hair cells of the organ of Corti, and the tectorial membrane being mechanically coupled; some events initiated at the hair cell level will eventually be reflected in measurements of basilar membrane displacement. Thus, the measure-

ments of sharply tuned basilar membrane mechanics probably reflect the tuning properties of hair cells; damage to hair cells, particularly the stereocilia, will be seen in the mechanical measurements. In mild acoustic trauma or other cochlear insults that primarily affect the sensory epithelium, there may be little value in separating sensory and mechanical components to the resulting dysfunction. However, in acoustic trauma in which there are gross mechanical lesions to the cochlear partition, even the (passive) mechanical properties of the basilar membrane will be changed. Turner's suggestion of separating out such mechanical influences is very appropriate. (Such a concept has been applied to the endolymphatic hydrops in Meniere's disease.)

I like Turner's proposal concerning the temporal variation in real-world noise exposure. There is a tendency for our concepts regarding acoustic stimuli in general to be governed by the physical devices that we have available to us for their measurement. Perhaps, indeed, we need to concentrate on temporal properties of sound rather than just their spectral components.

In response to Turner's final comment concerning individual variability in acoustic trauma, I would like to refer back to the initial comments made by Eggermont that ultimately cochlear dysfunction relates to changes at the biochemical level within cells. Even with careful standardization of the physical parameters of a traumatizing agent and its presentation to the cochlea, there is still much individual variation in degree of damage. It is useful in this respect to think of all the local variations that may affect the biochemistry of individual cells including, for example, local electrolyte and hormonal conditions, efferent activation, circadian rhythms, and a whole host of other factors yet to be discovered. This variability,which in the past has been a nuisance for workers attempting to find unifying theories concerning acoustic trauma, may well become a useful source of new insight into the mechanisms of acoustic trauma.

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