

SURVEY PAPER

Neural Correlates of Sensorineural Hearing Loss*

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EDITORIAL NOTE

At the 1981 meeting of the Editorial Board the idea of having Survey Papers was discussed and endorsed. The goal of a Survey Paper is to review recent activity in a specific area that is relevant to practicing clinicians, educators, scientists, and others who are interested in human hearing and its disorders. This paper by Salvi and his colleagues is the first Survey Paper that *Ear and Hearing* has published. How better a way to begin a series such as this than with a paper which summarizes the neurophysiological bases for the most prevalent type of hearing disorder, sensorineural hearing loss. It is hoped that readers will react favorably to this type of format, and will provide suggestions for future topics to be covered.

Ross J. Roeser, Editor-In-Chief

ABSTRACT

Sensorineural hearing loss is characterized by a relatively well defined set of audiological signs and symptoms such as elevated thresholds, abnormally rapid loudness growth, subjective tinnitus, poor speech discrimination, and a reduction in temporal summation of acoustic energy. Knowledge of the underlying neural mechanisms responsible for some of these auditory distortions has progressed substantially within the past 10 yrs as a result of physiological studies on hearing-impaired animals. Some of the important neurophysiological changes relevant to sensorineural hearing loss are reviewed. One important effect associated with sensorineural hearing loss is the broadening of the cochlear filtering mechanism which may influence loudness growth and the perception of complex sounds. The neurophysiological results may also provide new insights in interpreting traditional audiological data and help in developing more refined tests for fitting hearing aids or differentiating patients with sensorineural hearing loss.

A large number of patients referred to audiologists and otologists are those with sensorineural hearing loss; in many

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cases, the hearing loss is a result of exposure to intense noise. The clinical procedures for differentiating sensorineural hearing loss from other types of hearing impairment have been available for some time. Generally speaking, the diagnosis is straightforward, with the principal signs and symptoms of sensorineural hearing loss being elevated thresholds most often at the high frequencies, no difference between air and bone conduction thresholds, the presence of loudness recruitment, subjective tinnitus, disturbances in pitch perception, and poor speech discrimination particularly in a background of noise. When the hearing loss is severe enough to interfere with the perception of speech, a hearing aid may be considered; however, a listener's success with an aid is often difficult to assess accurately, particularly when the device is used in a noisy environment. Furthermore, listeners with essentially the same audiometric profile may perform quite differently when fitted with the same hearing aid. Thus, the clinician soon learns that fitting hearing aids is somewhat of an "art" and that the complete restoration of "normal" hearing through the use of an aid is seldom possible.

The extensive modifications in hearing performance that can occur with sensorineural hearing loss and the failure of hearing aids to compensate entirely for these changes suggests that there must be fundamental changes in the way the impaired ear processes acoustic information. Insights into neural processing in the impaired ear naturally begin in the auditory periphery, for it is here that anatomists have documented a wide variety of lesions to the hair cells, supporting cells of the organ of Corti, and neural elements in the spiral ganglion. These cochlear lesions presumably alter the way in which acoustic information is transformed into a pattern of neural activity. The auditory nerve is the only pathway through which acoustic information can enter the central auditory system. By recording the activity from many individual auditory nerve fibers it is possible to obtain a detailed yet comprehensive picture of the neural activity flowing into the central auditory system. Any changes in the pattern of neural activity resulting from the cochlear pathologies can be related to the audiological symptoms of sensorineural hearing loss in order to gain an understanding of neural basis for the alterations in hearing.

It is impossible, at present, to study the behavior of single

neurons in humans with sensorineural hearing loss. Thus, researchers have turned to animal models. Over the past 10 yrs, auditory physiologists have elucidated some of the fundamental changes that occur in the discharge patterns of VIIIth nerve fibers from animals with noise- or drug-induced hearing loss. A fundamental assumption of these studies is that the symptoms of sensorineural hearing loss in animals are the same as those in humans. This assumption seems reasonable because animal psychophysicists have used behavioral conditioning techniques to document some of the clinical symptoms of sensorineural hearing loss in animals.

Any serious discussion of sensorineural hearing loss would be incomplete without mentioning the anatomical changes, particularly the loss of inner and outer hair cells. It is popular to assume that the elevation of threshold sensitivity over the first 40 dB results exclusively from the loss of outer hair cells. However, single unit studies on hearing-impaired animals suggest that the relationship between elevated thresholds and cochlear pathologies is much more complex; loss of sensitivity may actually involve a wide variety of cochlear pathologies, some which are quite subtle.²⁶

The goal of this article is to summarize recent neurophysiological evidence relevant to understanding the psychophysical or audiometric symptoms of sensorineural hearing loss and to relate the results to the underlying cochlear pathologies. Particular attention will be paid to studies that involve a combination of psychophysical, physiological, and anatomical data. Before discussing the data from hearing-impaired animals, it is important to review some of the fundamental properties of VIIIth nerve fibers in normal animals.

NORMAL RESPONSE PROPERTIES OF THE AUDITORY NERVE

The auditory nerve, which consists of approximately 30,000 to 50,000 nerve fibers, is the only pathway for conveying acoustic information from the cochlea to the central auditory pathway. As Spoendlin^{33,34} has shown, approximately 90 to 95% of the nerve fibers innervate only 1 inner hair cell; the remaining 5 to 10% of the fibers project across the tunnel of Corti, spiral basalward, and innervate approximately 10 outer hair cells. Thus, each nerve fiber provides information regarding the status of a limited region of the cochlea.

Because the diameter of the nerve fibers exiting the internal auditory meatus is extremely small, on the order of 6 μ , one must use microelectrodes with tip diameters of less than 1 μ to record the all-or-none spike discharges from individual nerve fibers. If a microelectrode is advanced into the auditory nerve in the absence of any controlled acoustic stimulation, one finds that each nerve fiber will discharge spontaneously with an irregular temporal response pattern. Whereas the average spontaneous rate is relatively stable for a single fiber, the spontaneous rates vary from one unit to the next. If the spontaneous rates are measured from a large sample of auditory nerve fibers, the rates normally fall within the range from 0 to 100 spikes/sec. As seen in Figure 1, the distribution of spontaneous rates tends to be bimodal with many units below 20 spikes/sec and between

40 and 90 spikes/sec; few units have rates between 20 and 30 spikes/sec. An understanding of the normal pattern of spontaneous activity is particularly relevant to a discussion of sensorineural hearing loss because abnormally high rates of spontaneous activity have been suggested as the underlying mechanism for tinnitus.^{1,21}

When the ear is stimulated with tone bursts of the proper frequency and intensity, the unit's firing rate will increase above the spontaneous rate during the duration of the stimulus. The intensity which is required to raise a neuron's discharge rate just above the spontaneous rate is referred to as threshold. Threshold has generally been estimated with audiovisual criteria, but more recently, computers have been used to determine the intensity which produces a fixed increment in the firing rate above the spontaneous level. Each unit in the auditory nerve is most sensitive at one frequency, known as its characteristic frequency (CF); however, a unit will respond over a range of frequencies at higher intensities. A tuning curve refers to the frequency-intensity combinations capable of eliciting a threshold response from a cell. Figure 2 shows some typical tuning curves obtained from a chinchilla using audiovisual techniques for estimating threshold. The tuning curves of high CF units typically consist of two distinct segments; a low threshold, narrowly tuned region around CF and a high threshold, broadly tuned region in the tail of the tuning curve. Units with low CFs, on the other hand, tend to be relatively symmetrically shaped on a log frequency plot.^{7,8,18} Recent measurements of basilar membrane vibration strongly suggest that the shape of the tuning curve is closely linked to the mechanical vibration pattern of a particular point along the basilar membrane.¹⁶

In the cat¹⁹ and chinchilla, there appears to be a systematic relationship between spontaneous activity and threshold. As shown in the middle panel of Figure 3, units with high (>18 spikes/sec) spontaneous rates (approximately 66% of the population of units) tend to have the lowest threshold whereas those with the lowest (<1 spike

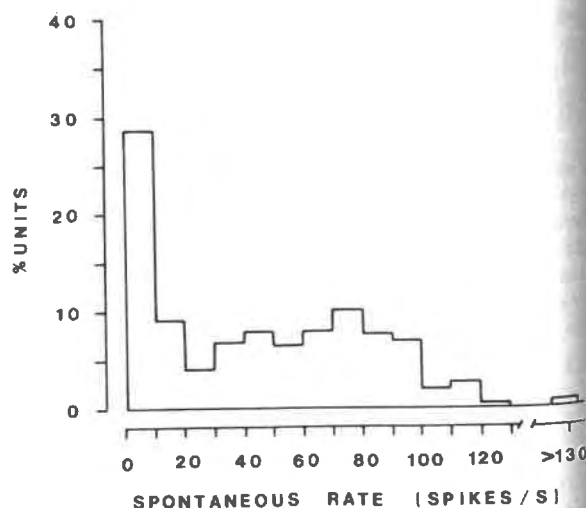


Figure 1. Histogram showing the distribution of spontaneous activity in a sample ($N = 144$) of auditory nerve fibers from normal animals with widths 10 spikes/sec (from Ref. 28, Tobias, J. V., and E. D. Schusterman, eds., *Hearing Research and Theory*, Academic Press, New York, 1968, reproduced by permission).

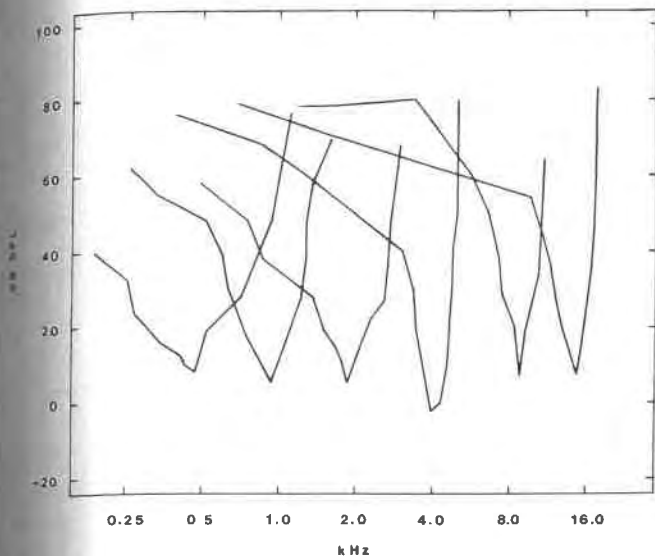


Figure 2. Solid lines show the frequency-threshold curves of six auditory nerve fibers. The tuning curves were obtained from normal chinchillas using audiovisual criteria.

sec) spontaneous rates (approximately 11%) have the highest threshold.^{20, 30} Units with intermediate (1 to 18 spikes/sec) spontaneous rates (approximately 23%) have thresholds between the low- and high-threshold groups. One relationship relevant to the interpretation of the audiogram is the correlation of the neural thresholds with the behavioral audiogram. Figure 3 (bottom) compares the mean thresholds at CF of units with high spontaneous rates and the mean behavioral thresholds at similar frequencies. At most frequencies, the mean behavioral threshold is within one standard deviation of the mean neural threshold; the comparison suggests that the most sensitive auditory nerve fibers in normal-hearing chinchillas are sufficiently sensitive to mediate the behavioral threshold. An important issue is whether this same relationship holds in animals with sensorineural hearing loss.

When a suprathreshold tone burst is presented, units in the auditory nerve will respond over the entire duration of the stimulus; however, the response pattern is probabilistic in nature and varies from one tone burst to the next. To characterize the "average" response patterns of a fiber, one can repeatedly present the same stimulus and, with the aid of a computer, store the time of arrival of each spike relative to stimulus onset in order to generate a poststimulus time histogram.¹¹ Figure 4 contains a series of poststimulus time histograms obtained over an intensity range of 75 dB using a 200-msec tone burst at the unit's CF. All units in the auditory nerve respond in the same general fashion as that shown in Figure 4.^{18, 32} Near threshold, the unit's firing rate is relatively low and approximately constant over the duration of the stimulus interval. As the intensity is increased, there is an increase in the unit's firing rate; however, the pattern of activity changes. Now the discharge rate is high near stimulus onset, but then quickly declines to an approximately steady level for the remainder of the stimulus. When the stimulus is terminated, the discharge rate falls below the normal level of spontaneous activity and then gradually recovers. Knowledge of the temporal char-

acteristics of the neural response patterns is relevant to understanding the process of temporal summation of acoustic energy at the threshold of hearing. In normal listeners, the threshold of a tone decreases approximately 10 dB as the duration of the tone increases from 10 to 500 msec. The improvement of threshold with duration is much less in listeners with sensorineural hearing loss.^{12, 15, 38} Although it is not used extensively, brief-tone audiometry can be used to assess the process of temporal summation.

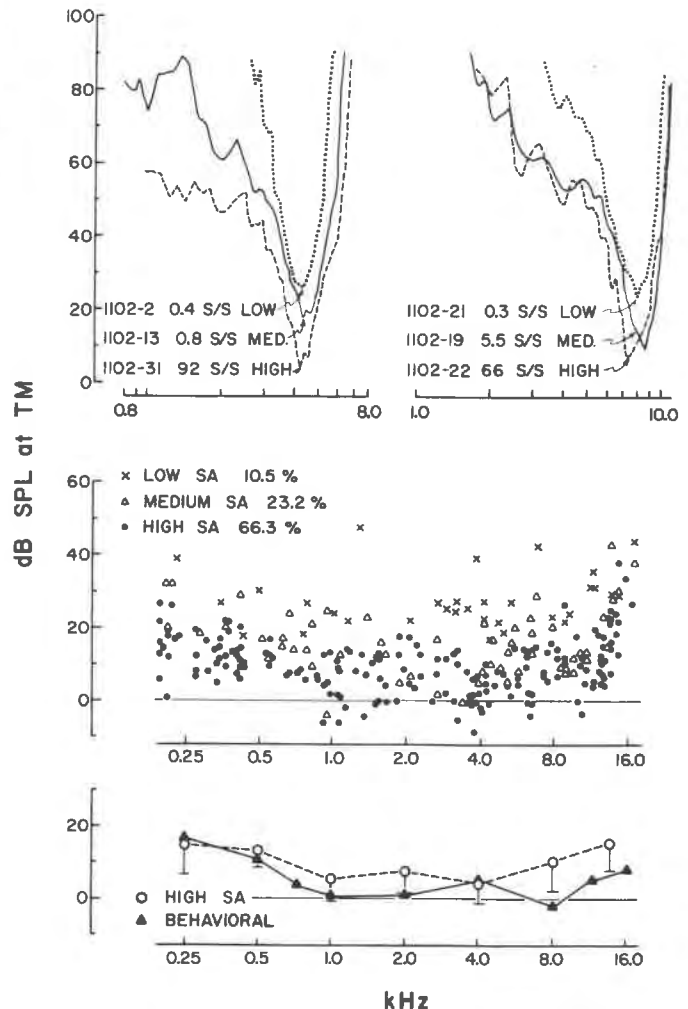


Figure 3. Top panel: examples of tuning curves obtained with a computer-automated threshold-tracking method. The units have CFs near 4 kHz (left) and 8 kHz (right) and have low (dotted lines), medium (solid lines), or high (dashed lines) rates of spontaneous activity (SA). Middle panel: distribution of neural thresholds plotted as a function of the unit's CF. The percentage of units with low (<1 spike/sec), medium (1 to 18 spikes/sec), and high (>18 spikes/sec) spontaneous rates are indicated in the figure. Bottom panel: filled triangles show the mean behavioral audiogram obtained from six normal chinchillas. The open circles and vertical bars show the mean thresholds and standard deviations of units within various octave and half-octave bands. The neural data are from units with high spontaneous rates. Both the neural and behavioral data have been expressed in dB SPL at the tympanic membrane to facilitate a comparison (from Ref. 30, Hamernik, R. P., D. Henderson, and R. J. Salvi, eds., *New Perspectives on Noise-Induced Hearing Loss*, Raven Press, New York, 1982, reproduced by permission).

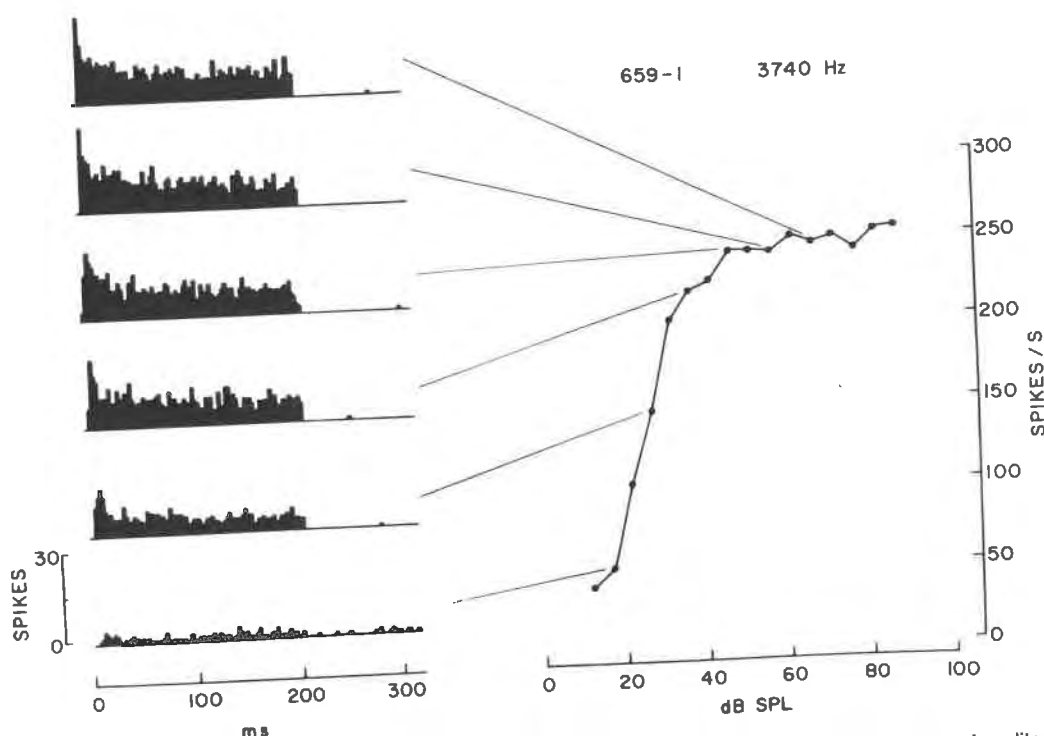


Figure 4. Poststimulus time histograms (left) and the discharge rate-intensity function (right) obtained from a normal auditory nerve fiber. The data were obtained with a 200-msec tone burst presented at the unit's CF over a 75 dB intensity range (from Ref. 28, Tobias, J. V., and E. D. Schubert, eds., *Hearing Research and Theory*, Academic Press, New York, 1983, reproduced by permission).

The right side of Figure 4 also provides information on the ability of individual nerve fibers to transmit information regarding stimulus intensity in terms of discharge rate. As stimulus intensity increases over a 75 dB range, there is a monotonic increase in the unit's discharge rate only over the first 35 dB above threshold; the discharge rate then saturates. The majority of units in the auditory nerve saturate within 20 to 50 dB of threshold and thus are able to encode changes in intensity over a relatively limited range.¹⁸ However, the discharge rate-intensity functions of some units in the cat reportedly exhibit a sloping saturation so that a few units are capable of encoding intensity over a somewhat greater range, about 60 to 70 dB.²⁴ However, even this range of intensity coding by a single fiber is still less than the 100 dB range over which listeners can make loudness judgments.³⁵ Presumably the coding of loudness involves some integration of neural activity across the population of nerve fibers. Given that loudness recruitment is a symptom of a hearing loss of cochlear origin, one might expect to find some change in the way individual neurons or the population of nerve fibers respond to changes in intensity.

Auditory nerve fibers respond in a consistent fashion when clicks are used as stimuli. The poststimulus time histograms obtained with click stimuli vary systematically with the CF of a neuron. The histograms from high-frequency fibers generally have a single large peak whereas those from low-frequency fibers have multiple peaks as shown in Figure 5. The latency to the first peak is related to the CF of the unit as illustrated by the data in Figure 6. High-frequency units have response latencies of approxi-

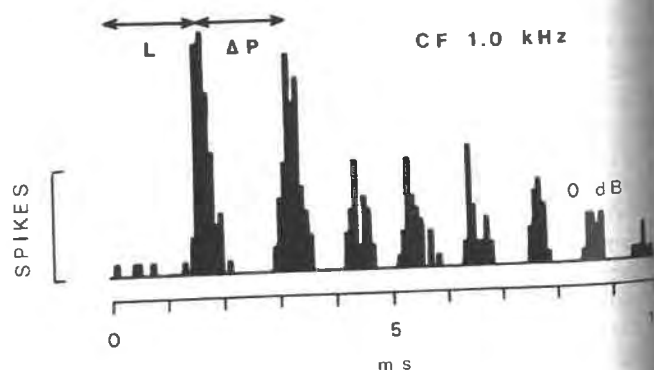


Figure 5. A poststimulus time histogram obtained from a normal auditory nerve fiber using an acoustic click as the stimulus (0 dB attenuation corresponds approximately to 100 dB peak SPL). The fiber had a CF of 1.0 kHz. The latency (L) and time between peaks (ΔP) are indicated (from Ref. 28, Tobias, J. V., and E. D. Schubert, eds., *Hearing Research and Theory*, Academic Press, New York, 1983, reproduced by permission).

mately 1 to 1.5 msec; as CF decreases there is a progressive increase in latency presumably because of the time required for mechanical events to propagate from the base to the apex of the cochlea. The latencies of individual nerve fibers obviously play an important role in determining the latencies of the gross neural potentials such as the action potential (AP) and wave I of the ABR. Because the latencies of the gross potentials are important in clinical diagnosis, it is important to determine whether sensorineural hearing

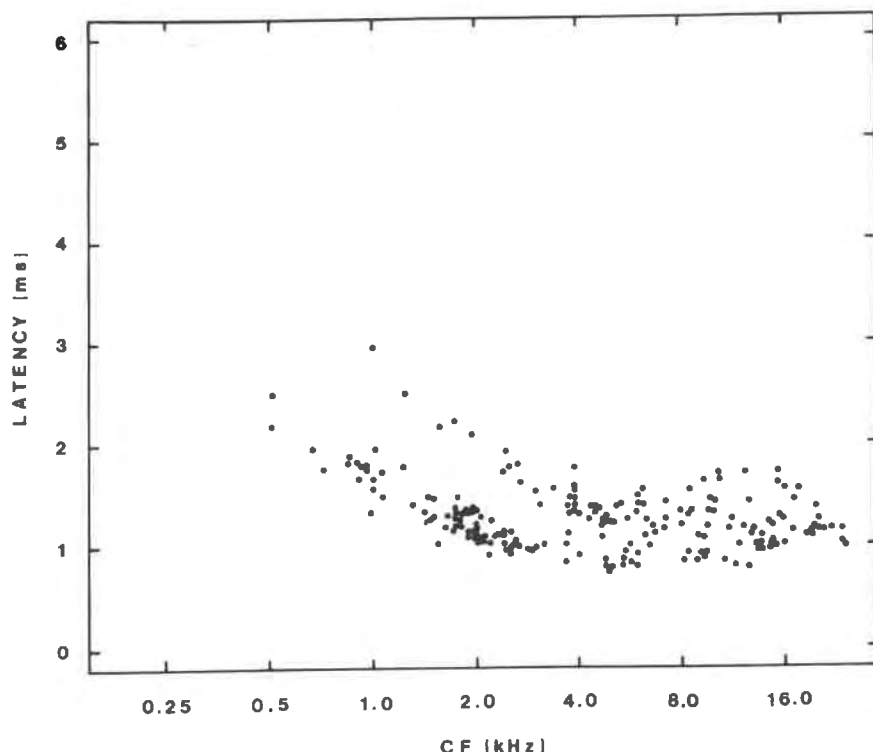


Figure 6. Scattergram showing the latency of individual nerve fibers as a function of the unit's characteristic frequency of the unit. The stimulus was an acoustic click having a peak SPL of 100 dB (from Ref. 28, Tobias, J. V., and E. D. Schubert, eds., *Hearing Research and Theory*, Academic Press, New York, 1983, reproduced by permission).

causes any systematic change in the latencies of the underlying neural elements.

NEURAL ACTIVITY WITH SENSORINEURAL HEARING LOSS

The preceding discussion has provided an overview of some of the fundamental response properties of auditory nerve fibers in normal animal. In the following section, a review will be made of the response properties of single units from animals with sensorineural hearing loss resulting from exposure to noise or ototoxic drugs. As the goal is to understand the symptoms of sensorineural hearing loss, special consideration will be given to animal studies which combine psychophysical, neural, and anatomical measurements.

Relationships between Neural and Behavioral Threshold

In normal animals, there is a close correspondence between the thresholds at CF and the behavioral audiogram; however, in animals with sensorineural hearing loss, the relationship can be disturbed to varying degrees which may lead to distortions in our auditory perceptions. Figures 7 and 8 contain data from two animals with high-frequency noise-induced hearing loss which may provide some insights into the alterations in hearing. Both animals were exposed for 5 days to an octave band of noise centered at 4 kHz and presented at 86 dB SPL. Using a shock-avoidance conditioning paradigm, the behavioral thresholds of the animals were obtained before, during, and after the exposure in order to estimate the amount of temporary (TTS) and

permanent threshold shift (PTS). After the behavioral testing was completed, the animals were prepared for single unit recordings from the auditory nerve. At the end of the physiological experiments, the animals were sacrificed and their cochleas were prepared for histological analysis. The bottom half of Figure 7 illustrates the pattern of TTS and PTS measured behaviorally in chinchilla 659 and also includes the threshold shifts at CFs for a large sample of auditory nerve fibers. During the exposure, the animal developed a TTS of approximately 70 dB at 4 kHz and above; however, the thresholds were essentially unaffected below 2 kHz so that the low frequencies can serve as a control region. After a 6-mo recovery period, the animal had a permanent hearing loss of 15 to 20 dB between 4 and 8 kHz, but had normal thresholds at lower and higher frequencies. The neural thresholds were also essentially normal among units with low CFs; but units with CFs near 5 kHz had thresholds which were elevated approximately 30 to 50 dB. The neural threshold shifts at CF were in reasonably good accord with the behavioral data over most of the frequency range; however, the neural thresholds were higher than one would expect from the behavioral audiogram in the 5 to 9 kHz region.

The top half of the Figure 7 shows the pattern of hair cell loss in chinchilla 659 plotted according to the frequency-place map for the chinchilla cochlea.⁶ Note that there is a complete loss of both inner and outer hair cells in the 8 kHz region which corresponds closely to the absence of units with CFs between 7.5 and 9 kHz; this result is consistent with the innervation pattern of the cochlea.^{33, 34} The lack of hair cells and absence of units with CFs near 8

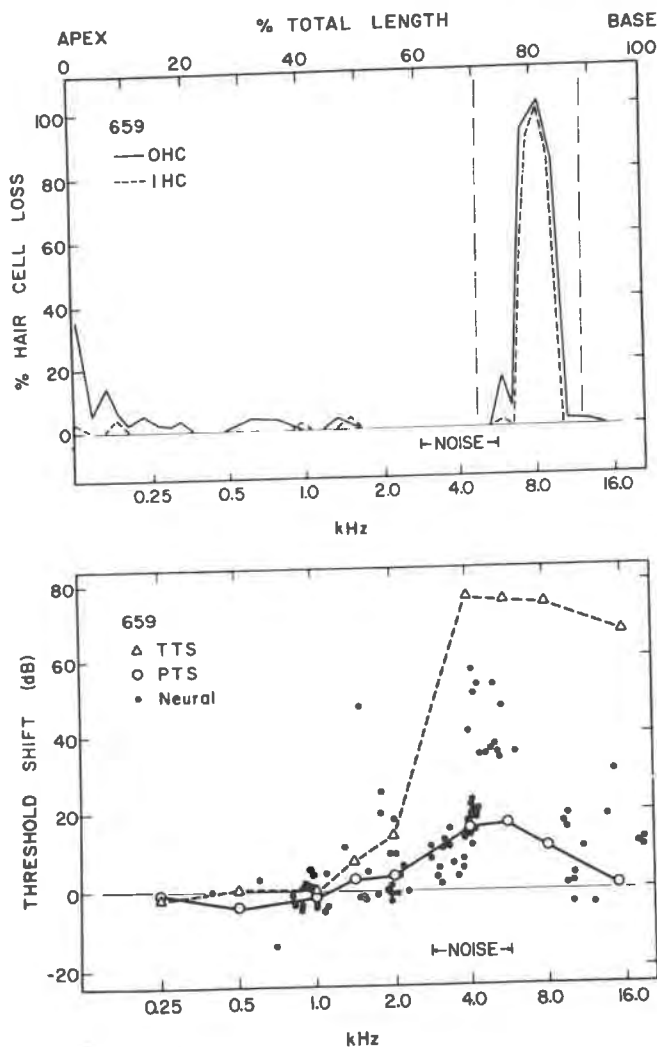


Figure 7. Top panel shows the percentage of outer (OHC) and inner hair cell (IHC) loss of chinchilla 659 plotted as a function of percent distance from the apex. Position in the cochlea has been transformed to frequency using the frequency-place map of Eldredge et al.⁶ The area between the dashed vertical lines indicates the region of the cochlea which contains a variety of subtle structural defects. Bottom panel shows the temporary (open triangles) and permanent (open circles) threshold shifts measured behaviorally. The filled circles show the magnitude of threshold shift at the unit's characteristic frequency. The neural threshold shifts were computed by normalizing the threshold of each unit in the noise-exposed animal to the average neural thresholds of normal units with similar CFs. The animal was exposed for 5 days to an octave band of noise centered at 4.0 kHz and having an SPL of 86 dB (from Ref. 30, Hamernik, R. P., D. Henderson, and R. J. Salvi, eds., *New Perspectives on Noise-Induced Hearing Loss*, Raven Press, New York, 1982, reproduced by permission).

kHz raises an important issue, i.e., how was the animal able to detect an 8 kHz tone burst? The solution to the problem is apparent when one considers the shape of the neural tuning curves. Because the units with CFs near 10 kHz had normal thresholds, they will respond to tones of 8 kHz, but at intensities that are slightly higher than the threshold at CF (see Fig. 2). In other words, the detection of the 8 kHz tone is probably mediated by units with CFs above the location of the lesion; this occurs because the pattern of

mechanical vibration in the cochlea spreads predominantly toward the base of the cochlea as intensity is increased. A person with a lesion such as that seen in chinchilla 659 would most likely perceive an 8 kHz tone in a much different way than would a person with a normal complement of hair cells. In fact, individuals who advocate a strict place theory of pitch perception would almost certainly predict that the 8 kHz tone would be shifted upward in pitch in such an individual. The "predicted" change in pitch could be evaluated from binaural pitch matching

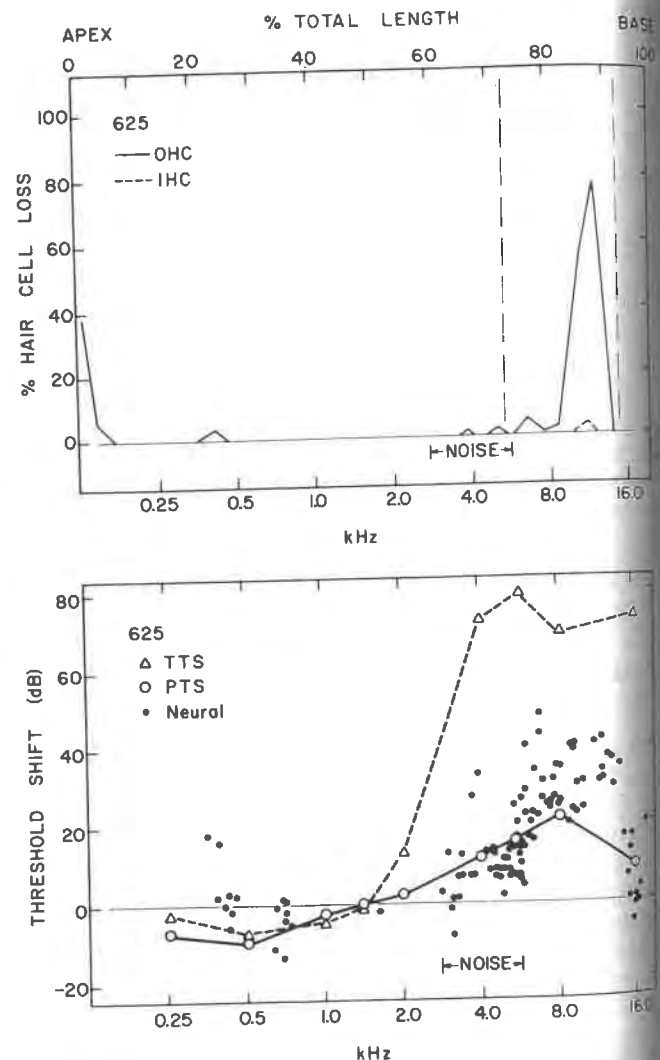


Figure 8. Top panel shows the percentage of outer (OHC) and inner hair cell (IHC) loss of chinchilla 625 plotted as a function of percent distance from the apex and as a function of frequency.⁶ The area between the dashed vertical lines indicates the region of the cochlea which contains a variety of structural defects. Bottom panel shows the temporary (open triangles) and permanent (open circles) threshold shifts measured behaviorally. The filled circles show the magnitude of threshold shift at the unit's characteristic frequency. The neural threshold shifts were computed by normalizing the threshold of each unit in the noise-exposed animal to the average neural thresholds of normal units with similar CFs. The animal was exposed for 5 days to an octave band of noise centered at 4.0 kHz and having an SPL of 86 dB (from Ref. 30, Hamernik, R. P., D. Henderson, and R. J. Salvi, eds., *New Perspectives on Noise-Induced Hearing Loss*, Raven Press, New York, 1982, reproduced by permission).

experiments using patients with a unilateral hearing loss. The pitch matching data might also provide a noninvasive method of evaluating the pattern of the cochlear lesion.

Clinicians familiar with the effects of acoustic trauma have frequently noted a peculiar relationship between the spectral characteristics of the exposure stimulus and the pattern of hearing loss. The frequency of maximum hearing loss is generally located $\frac{1}{2}$ to 1 octave above the center frequency of the noise exposure.^{5, 14} A half-octave shift was also seen in the PTS data of chinchilla 659. More importantly, the region of maximum hair cell loss and the CFs of units with maximum threshold shift were located $\frac{1}{2}$ to 1 octave above the center frequency of the noise. These results strongly suggest that the half-octave shift phenomenon has its origins in the cochlea.

Results from another animal exposed to the octave band of noise centered at 4 kHz are shown in Figure 8. The animal sustained a PTS between 4 and 16 kHz with the maximum loss occurring at 8 kHz, a full octave above the center frequency of the exposure stimulus. The neural threshold shifts at CF closely approximate the behavioral data. Note that the hair cell lesion is confined to the outer hair cells and that there are units with CFs extending across the region of cell loss as one might predict based on the innervation pattern of the cochlea. However, there is a slight discrepancy; the outer hair cell lesion is less than an octave wide, whereas the neural and behavioral thresholds are elevated over at least 2 octaves. This discrepancy is more apparent than real, however, if one considers other anatomical defects in the organ of Corti besides hair cell loss. The region between the dotted vertical lines in the cochleogram of Figure 8 contains a variety of anatomical defects which effectively extend the width of the lesion especially towards the apex of the cochlea. In the region from 5.6 to 8 kHz, where the hair cell loss is less than 10%, one can find a number of histopathologies which may affect its functional integrity. One prominent defect is the loss of outer pillar cells which influences the structural integrity of the cochlea. The loss of outer pillar cells is generally accompanied by the loss of outer hair cells; if the loss is severe, the reticular lamina may become distorted and the tunnel of Corti may undergo partial collapse. Such defects could affect the micromechanics of the cochlea, e.g., by changing the coupling between the tectorial membrane and the stereocilia on the hair cells.

Liberman and Kiang²⁰ have also studied the relationship between hair cell loss and neural thresholds in acoustically traumatized cats. In many cases there was close agreement between the location of the lesion and the units with elevated thresholds. However, in other animals, nearly all the hair cells were present yet the thresholds were elevated. The regions where thresholds were elevated were frequently associated with hair cells having abnormal stereocilia. In summary, there may be a wide variety of cochlear pathologies besides hair cell loss that contribute to auditory function.

Neural Tuning and Frequency Selectivity

Even at a noisy cocktail party, the normal-hearing listener is able to recognize complex sounds such as speech. By contrast, the listener with a sensorineural hearing loss per-

forms quite poorly even when fitted with a hearing aid. This remarkable ability of the normal-hearing listener to respond to selective frequency components of a complex sound has been referred to a frequency selectivity. There are a variety of ways of assessing frequency selectivity, but one of the most direct methods involves the measurement of psychophysical tuning curves. In obtaining a psychophysical tuning curve the listener is asked to detect a low-intensity probe tone which is fixed in frequency. A second tone is then introduced and its intensity is adjusted upward until the listener can no longer detect the low-intensity probe. When the procedure is carried out over a range of masker frequencies, one obtains a plot which shows the masked threshold as a function of frequency. Masked threshold is lowest near probe frequency and then increases with increasing separation between probe and masker frequency. These psychophysical tuning curves closely resemble the neural tuning curves shown in Figure 2. Inasmuch as patients with sensorineural hearing loss show poor speech discrimination in noise (i.e., an inability to segregate the frequency components), one might expect a change in their psychophysical and physiological tuning curves.

Psychophysical and neural tuning curves have been measured in chinchillas with noise-induced PTS. Figure 9 shows the magnitude of TTS and PTS sustained by chinchilla 695; the animal was exposed for 5 days to an octave band of noise centered at 500 Hz and having an SPL of 95 dB. The animal developed a midfrequency hearing loss of approximately 20 to 30 dB; notice that the neural threshold shifts at CF closely parallel the behavioral data. Figure 10 contains five psychophysical tuning curves from chinchilla 695 at the probe tone frequencies of 1.0, 2.0, 4.0, 8.0, and 11.2 kHz. The frequency of the probe tone is indicated by the open circle. A large sample of single unit tuning curves was obtained from the same animal. Several single unit tuning curves with CFs matching the psychophysical tuning curves have been drawn on the same figure to aid in the analysis. Note that the same psychophysical tuning curve appears three times in each row. Near 11 kHz, the neural and psychophysical tuning curves (Fig. 10, bottom panel) both have a narrowly tuned, low-threshold tip and a high-threshold broadly tuned, low-frequency tail. These tuning curves were obtained from a region where the behavioral and neural thresholds were normal and show extremely good frequency selectivity. As one moves upward in Figure 10 there is a progressive decrease in the best frequency of the neural and psychophysical tuning curves. The tips of the tuning curves become elevated as one approaches the region of maximum hearing loss near 2.0 kHz; in addition, the tuning curves become broader. The broadening occurs primarily because there is a greater loss of sensitivity in the tip of the tuning curve than in the tail, i.e., there is a general decrease in the systems frequency selectivity with increasing hearing loss. In the neural domain, this means that nearly all the frequencies below the high-frequency cutoff can excite the unit at roughly the same SPL. Likewise, in the psychophysical domain, nearly all the masker frequencies below the probe tone will mask the probe tone at roughly the same SPL.

These abnormally broad neural and psychophysical tuning curves indicate that a relatively low-frequency tone that is just above threshold can result in a rather widespread

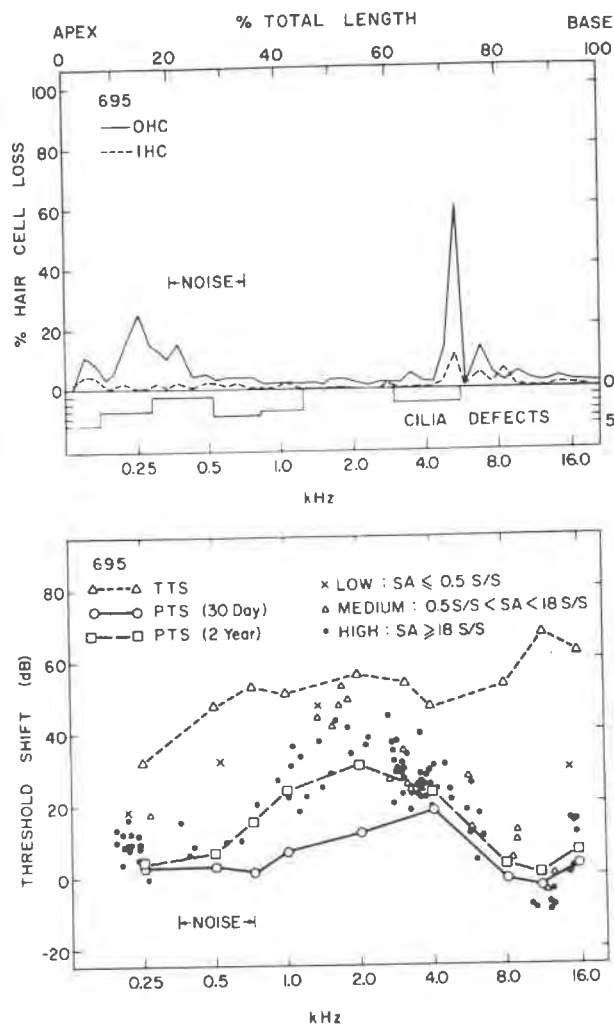


Figure 9. Top panel shows the percentage of outer (OHC) and inner hair cell (IHC) loss of chinchilla 695 plotted as a function of percent distance from the apex and as a function of frequency.⁶ The cilia defects were rated from 0 (normal) to 5 (grossly abnormal) and are indicated at the bottom of the cochleogram. Bottom panel shows the temporary (open triangles) and permanent threshold shift measured at 30 days postexposure (open circles) and at 2 yrs postexposure (open squares). The small dots, triangles, and squares show the neural threshold shifts plotted as function of CF (from Ref. 30, Hamernik, R. P., D. Henderson, and R. J. Salvi, eds., *New Perspectives on Noise-Induced Hearing Loss*, Raven Press, New York, 1982, reproduced by permission).

pattern of excitation in an ear with sensorineural hearing loss. This spread of excitation has important implications for those interested in fitting and developing hearing aids. Most aids simply amplify sounds, the amplification being greater at some frequencies than others. Although amplification can overcome the loss in sensitivity, it fails to restore the ear's frequency selectivity. Thus, listeners with hearing aids will often complain that they are unable to carry on a conversation in a noisy environment. They have no problem hearing sounds, but are unable to extract or separate out the important speech information from the background of noise. What is needed is a method of improving the signal (speech)-to-noise ratio. One device which is used to improve the signal-to-noise ratio in classrooms for the hearing im-

paired is the FM auditory trainer. The teacher speaks into a microphone and the speech sounds are then transmitted directly to the listener's aid uncontaminated by the background noise. Miniature transmitter and receiver circuits similar to this could be developed and incorporated into modern hearing aids to improve the signal-to-noise ratio for the hearing-impaired listener. However, even this technique does not correct for the breakdown in frequency selectivity.

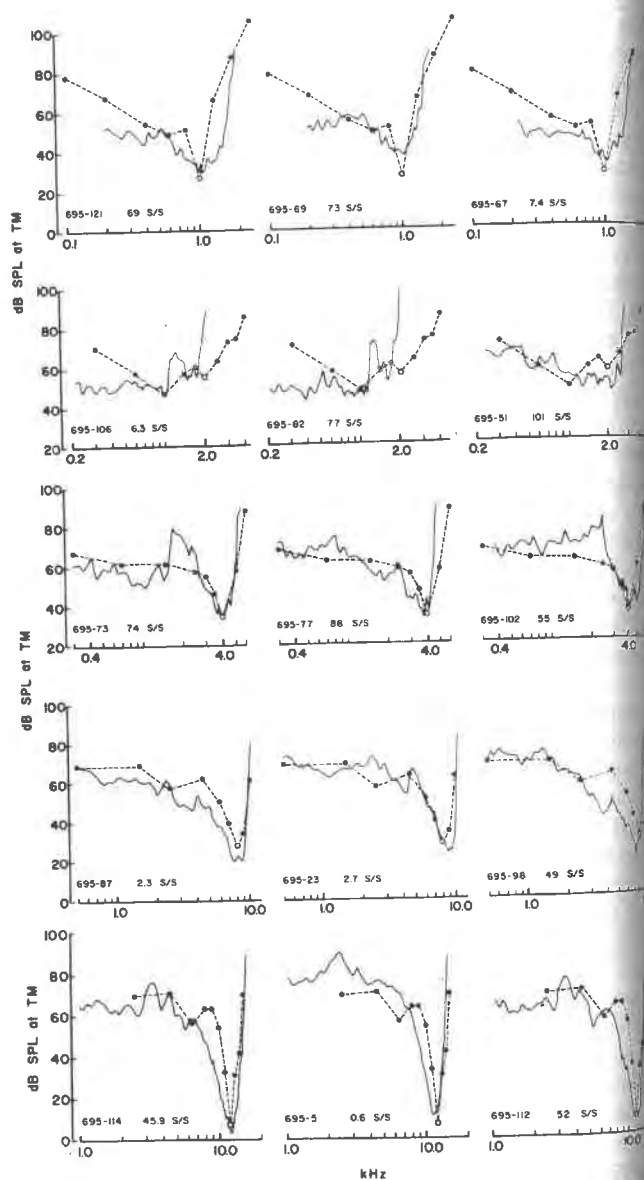


Figure 10. Dashed lines and filled circles show the psychophysical tuning curves obtained from chinchilla 695 using a simultaneous masking paradigm. The frequency of the probe tone is indicated by the open circles. The frequency of the probe tone increases from top to bottom. The psychophysical tuning curves have been repeated three times each row to aid in comparing the neural data. Solid lines show single neuron tuning curves from units with CFs approximating psychophysical data; the spontaneous rate of each unit is indicated (from Ref. 30, Hamernik, R. P., D. Henderson, and R. J. Salvi, eds., *New Perspectives on Noise-Induced Hearing Loss*, Raven Press, New York, 1982, reproduced by permission).

Loudness Recruitment

Recruitment, or the abnormally rapid growth of loudness with intensity, is a common disturbance of listeners with sensorineural hearing loss. One potential model for recruitment is based on a proportionality between intensity, the discharge rate of auditory nerve fibers, and the perception of loudness. The mechanism for explaining loudness recruitment involves a steepening of the slope of the function which relates the neural discharge rate to intensity. The change in neural activity predicted by this descriptive model is illustrated in the left panel of Figure 11. In the pathological ear, a small change in stimulus intensity produces an abnormally large increment in the neural firing rate which in turn is "coded" as a large change in loudness.

In order to test this model, chinchillas were exposed for 5 days to an octave band of noise centered at 0.5 kHz and having an SPL of 95 dB. At the end of the exposure, the rate-intensity functions were measured from auditory nerve fibers while the animals were in a state of TTS. The right panel of Figure 11 shows the slopes and saturation discharge rates obtained with tones at CF and plotted according to the threshold shift of the unit; the results are compared with similar data from normal animals. The slopes and saturation rates of units in the noise-exposed animals were not statistically different from those in normal animals even

though their thresholds were elevated approximately 50 dB. The noise exposure appears to have simply shifted the origin of the rate-intensity function without creating a recruitment-like change in the slope. Results similar to these have also been reported by Kiang et al.¹⁷ and Dallos and Harris⁴ in animals with sensorineural hearing loss induced by ototoxic drugs.

Another explanation of loudness recruitment, suggested by Kiang et al.¹⁷ and Evans,⁸ is based on the rate at which new units are activated as stimulus intensity is increased. The rate at which units are activated in the auditory nerve depends on the shapes of the neural tuning curves. A tone of low intensity will activate only a few units with CFs near the frequency of the stimulus. As stimulus intensity increases more units are activated, particularly those with CFs above the stimulus frequency, i.e., a high CF unit can be activated by frequencies located in the tail of the tuning curve, but only at high intensities. Thus, the difference in threshold between frequencies in the tip and tail of the tuning curve influences the rate at which new units are activated with increasing intensity.

The top half of Figure 12 illustrates how the model works in normal and pathological ears. The dashed vertical line indicates the frequency of the stimulus and the series of V-shaped solid lines are hypothetical tuning curves of units with different CFs. A unit begins to contribute to the active

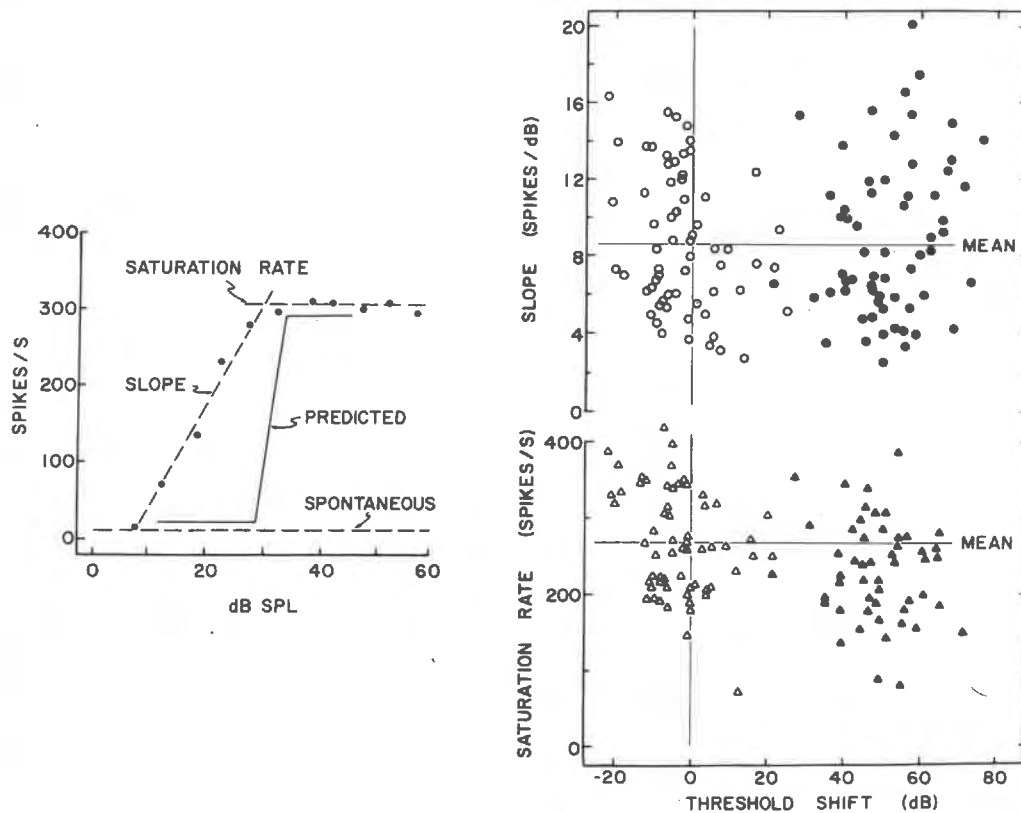


Figure 11. The dashed line and solid circles in the left panel show the slope and saturation discharge rate of the rate-intensity function of a normal auditory nerve fiber and the predicted rate-intensity function from a unit in a noise-exposed animal. The predicted increase in the slope of the rate-intensity function presumably would account for loudness recruitment. The right panels show the actual slopes and saturation discharge rates obtained from units in normal (open symbols) and noise-exposed (filled symbols) chinchillas; the data have been plotted as a function of the unit's threshold shift at CF. There is no statistically significant difference between the results from the normal and noise-exposed chinchillas (from Ref. 13, Alberti, P., ed., *Personal Hearing Protection in Industry*, Raven Press, New York, 1982, reproduced by permission).

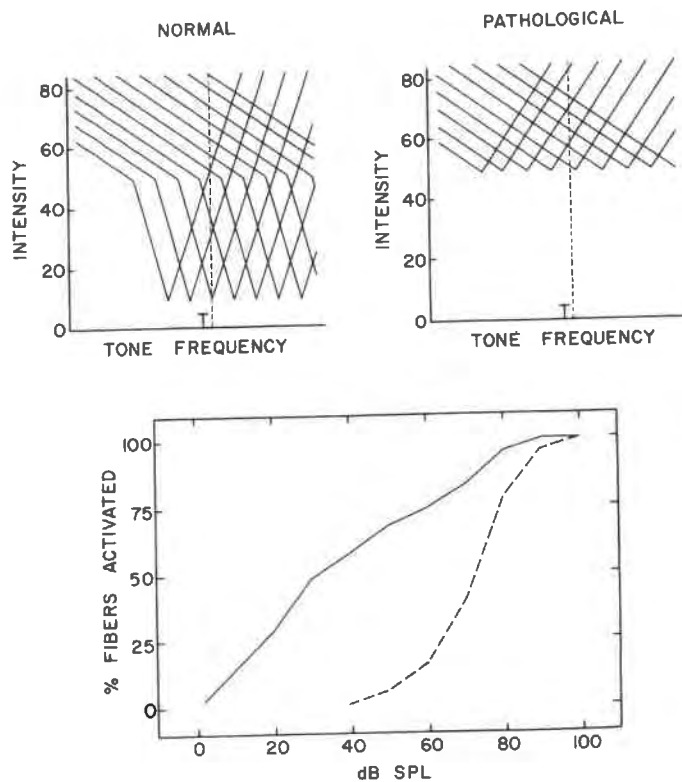


Figure 12. Idealized tuning curves in normal and pathological animals according to Evans.⁹ Units begin to respond to the tone (dashed vertical line) at the intensity where the tone cuts across the tuning curve. The bottom panel shows the percentage of units in the normal (solid line) and the noise-treated (dashed line) chinchillas that were activated as the intensity of a 2 kHz tone was increased in intensity. Note that the slope is steeper in the noise-exposed group than in normals (from Ref. 13, Alberti, P., ed., *Personal Hearing Protection in Industry*, Raven Press, New York, 1982, reproduced by permission).

population when the vertical line cuts across the tuning curve. As intensity is increased, additional units are activated. At high intensities, many units are being activated by tones located in the tails of the tuning curves. Note that in the pathological population, more units are activated per decibel change of intensity than in the normal population; this occurs because the tuning curves are broader than normal in subjects with sensorineural hearing loss.

The bottom half of Figure 12 shows the results of applying the model to normal and noise-exposed animals. The solid line shows the percentage of units in our sample of 144 normal auditory nerve fibers that were activated by a 2 kHz tone as intensity was increased. The dashed line shows similar results for 96 units from animals with a relatively flat hearing loss of 50 dB. Note that units from the noise-exposed animals begin to respond at a level which is approximately 40 dB higher than that in the normal group. Between 40 and 60 dB SPL, the slope of function of the noise-treated group is shallower than that for normals. However, between 60 and 80 dB the slope is much steeper and consequently the function from the noise-exposed animals "catches up" to the one from normal animals. Thus, there appears to be a "recruitment-like" phenomenon in the population response which results from the spillover of neural activity toward higher CF regions of the cochlea.

Although this model provides an appealing explanation for loudness recruitment, there are certain psychophysical data on loudness growth that pose a serious problem for such models.³⁷

Brief-Tone Audiometry and Temporal Summation

It has long been known that the threshold for detecting a tone improves as stimulus duration increases out to approximately 300 to 500 msec.^{15, 38} The improvement in threshold is on the order of 10 dB and is a reflection of the underlying process of temporal summation. In listeners with sensorineural hearing loss, the difference in threshold between long and short duration tones is consistently reduced^{12, 15, 39}; consequently, a clinical procedure known as brief-tone audiometry was developed to capitalize on this effect.

One explanation for the reduction in temporal summation in listeners with sensorineural loss is based on the assumption that there is an abnormally rapid decay over time in the neural output of the cochlea at intensities near threshold.³⁸ The lower left panel of Figure 13 is a schematic which shows the normal discharge pattern of a unit to a long duration tone burst and also the neural response predicted for an ear with sensorineural hearing loss. Note that the firing rate in the impaired ear shows an abnormal amount of decay over time. Thus, the detector mechanism in the central auditory pathway of the impaired ear has fewer neural discharges to integrate over time compared to the normal ear.

The poststimulus time histograms shown in the right half of Figure 13 were obtained from a chinchilla with 50 to 60 dB of noise-induced TTS. The unit was stimulated with tone bursts at CF over a range of intensities. At intensities near threshold, the poststimulus time histograms are nearly flat and are similar in shape to those from normal animals. Thus, the data from noise-exposed chinchillas do not provide any support for the notion of an abnormally rapid decay in the output of the auditory nerve that could be responsible for the breakdown in temporal summation observed with sensorineural hearing loss. It is not yet clear what neural mechanisms lead to a breakdown in temporal summation; perhaps it is due to changes in neural processing that take place in the central auditory pathway.

Tinnitus

Listeners with sensorineural hearing loss frequently complain of tinnitus, i.e., a buzzing or ringing sensation in the absence of any external sound. There have been many attempts at treating this phenomenon, e.g., masking and drug therapy.^{2, 3, 36} However, effective treatment often requires knowledge of the underlying mechanisms of the disorder. One of the most popular explanations for tinnitus assumes that a subpopulation of neurons in the auditory nerve develops abnormally high rates of spontaneous activity as a result of sensorineural hearing loss.^{1, 9, 21} The pitch of the tinnitus is presumably correlated with the CFs of the units with elevated spontaneous rates and the loudness of the tinnitus is related to the rate of spontaneous activity. This hypothesis can be evaluated by comparing the spontaneous rates of units from normal and noise-exposed animals. Unfortunately, the data on this subject are mixed, but nevertheless quite interesting.

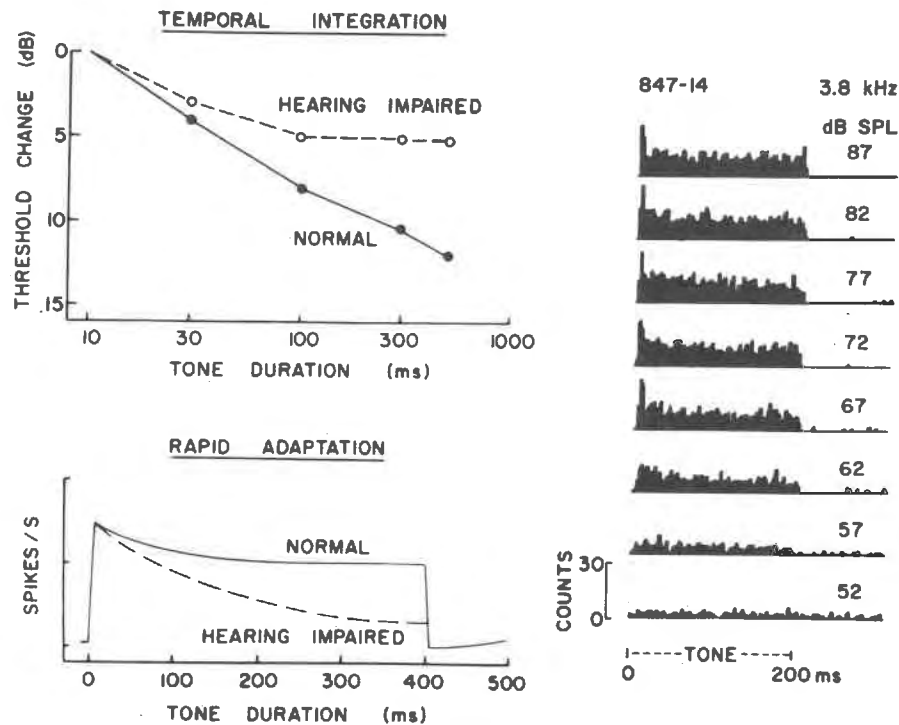


Figure 13. Top left panel shows typical psychophysical data which illustrate the improvement in thresholds with increasing signal duration for normal and hearing-impaired listeners. The bottom left panel illustrates the predicted firing pattern over time near threshold in a unit from a normal (solid line) and noise-exposed (dashed line) animal. The right panel shows the actual firing patterns obtained from an animal with a noise-induced threshold shift of approximately 50 dB. The poststimulus time histograms were obtained at the unit's characteristic frequency (from Ref. 13, Alberti, P., ed., *Personal Hearing Protection in Industry*, Raven Press, New York, 1982, reproduced by permission).

Several investigators have found a decline in the spontaneous discharge rates among units with elevated thresholds after the administration of ototoxic drugs.^{17,31} These results were contrary to the original model and led to an alternative explanation by Kiang et al.,¹⁸ who suggested that tinnitus was not due to the absolute rate of activity, but rather to a sudden change in spontaneous activity across the distribution of CFs. Some support for this model is provided by the data in Figure 14 which were taken from chinchillas exposed to an octave band of noise centered at 4 kHz.²⁵ The animals developed a significant TTS at the high frequencies, but thresholds remained normal at the low frequencies. Recordings were made from units in the cochlear nucleus of a second group of animals exposed to the same noise; the median spontaneous rates are shown in Figure 14 for units with CFs in different octave bands. The spontaneous rate is highest in the region where threshold is lowest, but the spontaneous rate systematically declines as the region of maximum hearing loss is approached. Thus, there is an "edge" in the distribution of spontaneous activity that corresponds to the boundary of the hearing loss. According to the "edge effect" model, the pitch of the tinnitus should correspond to the 3 to 4 kHz region where there is a sharp drop in spontaneous activity.

Pitch-matching studies of tinnitus cannot be done with animals; however, human data are available that are relevant to the edge effect model. When humans are exposed to a high-frequency band of noise, the tinnitus induced by the noise has a pitch which is matched to a frequency just below the region of maximum hearing loss.^{1,21} Extrapolat-

ing to our animal data, one would predict the pitch of the tinnitus to be approximately 4 kHz, i.e., corresponding to the region where there is a precipitous drop in spontaneous activity.

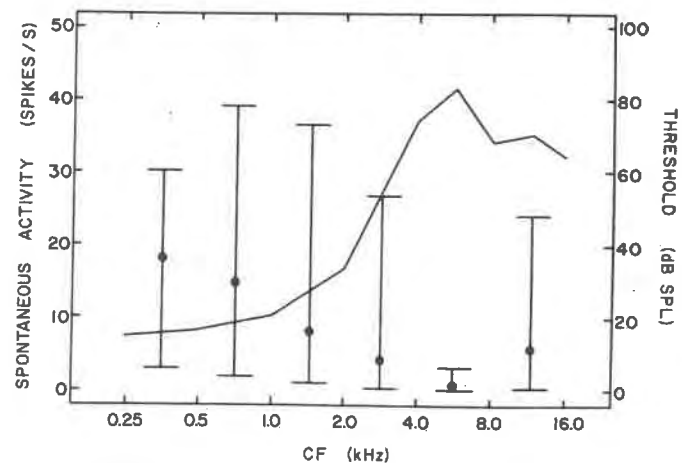


Figure 14. Distribution of spontaneous activity across a population of units in the cochlear nucleus during the recovery from a noise-induced asymptotic threshold shift. The median (filled circles) and interquartile range (vertical bars) were computed for units having characteristic frequencies in different octave bands. The solid line shows the behavioral thresholds of the chinchillas during the asymptotic threshold shift. The animals were exposed for 5 days to an octave band of noise centered at 4 kHz and having an SPL of 86 dB (from Ref. 13, Alberti, P., ed., *Personal Hearing Protection in Industry*, Raven Press, New York, 1982, reproduced by permission).

As was mentioned earlier, the model relies on a change in spontaneous rate. Thus, studies showing an increase in spontaneous activity could also be consistent with the model provided there is an edge. Figure 15 shows the distribution of spontaneous activity across CF for a population of VIIIth nerve fibers. The recordings were obtained from chinchillas that had developed a permanent hearing loss of approximately 20 dB at frequencies above 2 kHz after exposure to an octave band of noise centered at 4 kHz. At the low frequencies the neural and behavioral thresholds were completely normal (see Fig. 7 and 8 for other details). A cursory inspection of the data reveals that few units with CFs below 2.8 kHz have spontaneous rates exceeding 100 spikes/sec. However, above 2.8 kHz there appears to be a sizeable number of units with rates above 100 spikes/sec. Such high rates are rarely seen in normal animals.^{4, 20, 31} Our visual examination, which suggests an abrupt increase in spontaneous activity, was also confirmed statistically. Again, an edge effect model would predict that the tinnitus would be localized around 3 kHz near the edge of the hearing loss. Penner²³ has recently provided psychophysical data that complement our neural results. Her patients with sensorineural hearing loss reported that the pitch of their tinnitus was localized to frequencies along the edge of the high-frequency hearing loss.

Although the comparisons between tinnitus and spontaneous activity are intriguing, they are based only on correlational evidence. The models for tinnitus are also extremely simplistic, e.g., an edge effect model would have difficulty

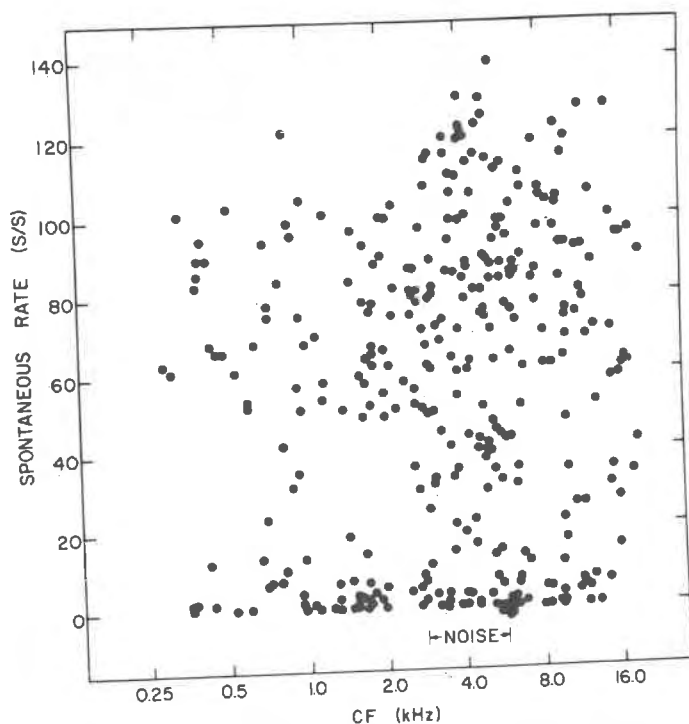


Figure 15. Scattergram showing the spontaneous discharge rate of each unit as a function of its characteristic frequency. The data were obtained from animals with a 15 to 30 dB PTS near 5.6 kHz. The animals had been exposed for 5 days to an octave band of noise centered at 4 kHz and having an SPL of 86 dB (from Ref. 28, Tobias, J. V., and E. D. Schubert, *Hearing Research and Theory*, Academic Press, New York, 1983, reproduced by permission).

accounting for tinnitus that is matched to a broad spectrum signal such as a hiss or buzz. One must also consider mechanisms in the central auditory pathway, as some cases of tinnitus are most effectively masked by stimuli presented to the ear contralateral to the ear in which the tinnitus is perceived.⁹

Neural Latencies

Clinical procedures for assessing auditory pathology have progressed to the stage where evoked potentials can now be used as a routine part of the audiometric test battery. The schemes for quantifying the brain stem and cochlear potentials have focused heavily on certain aspects of the latency and amplitude of the electrical waveforms. The latencies of the waveforms have proved to be extremely helpful in differential diagnosis. However, because the clinician seldom has direct access to the underlying neural events which give rise to these gross potential waveforms, it is sometimes difficult to understand or interpret the significance of a change in latency with various pathologies.

Single unit studies performed on animals with sensorineural hearing loss can provide some insights into the latency changes that occur in wave I of the brain stem response and the gross AP of electrocochleography. The single unit latency data shown in Figure 16 were obtained from a group of chinchillas that had been exposed for 5 days to an octave band of noise centered at 500 Hz.²⁹

The animals exposed to the noise developed a TTS of approximately 50 to 60 dB across the entire range of hearing; thus, all units showed a substantial loss in sensitivity. An acoustic click similar to that used in human evoked potential studies was used to obtain the AP latency data. There are two possible ways of comparing the latency data from normal and noise-exposed animals. One way is to compare the neural latencies at some fixed level above threshold. Figure 16 (bottom) compares the latencies of units from normal and noise-exposed animals at threshold, i.e., at a constant neural response level. With this type of comparison, the latencies of units from the noise-exposed animals are consistently shorter than those of normal units over nearly the entire range of CFs. A second way to compare latency values is at a constant SPL. The top half of Figure 16 shows the latency values obtained with a click having a peak SPL of approximately 100 dB. Under these conditions, the latencies of units in the normal and noise-exposed groups are virtually identical. Essentially the same result was found with the gross AP recorded from the round window. As shown in Figure 17, the AP latencies of the noise-exposed animals were virtually identical with those from normal animals even though there is a difference in sensitivity of approximately 50 to 60 dB. In the exposed animals, the latency of the AP and the single units does not show the same pattern of decrease with increasing intensity above threshold as occurs in normal animals; instead the latencies are strictly related to the level of the stimulus.

When recording the AP response in patients with sensorineural hearing loss, there may be instances where AP latency appears prolonged, but then suddenly catches up with the normal function.³⁹ What significance can be attributed to this "recruitment-like" behavior in latency? Based on our single unit results from noise-exposed animals, it seems reasonable to assume that when the AP response

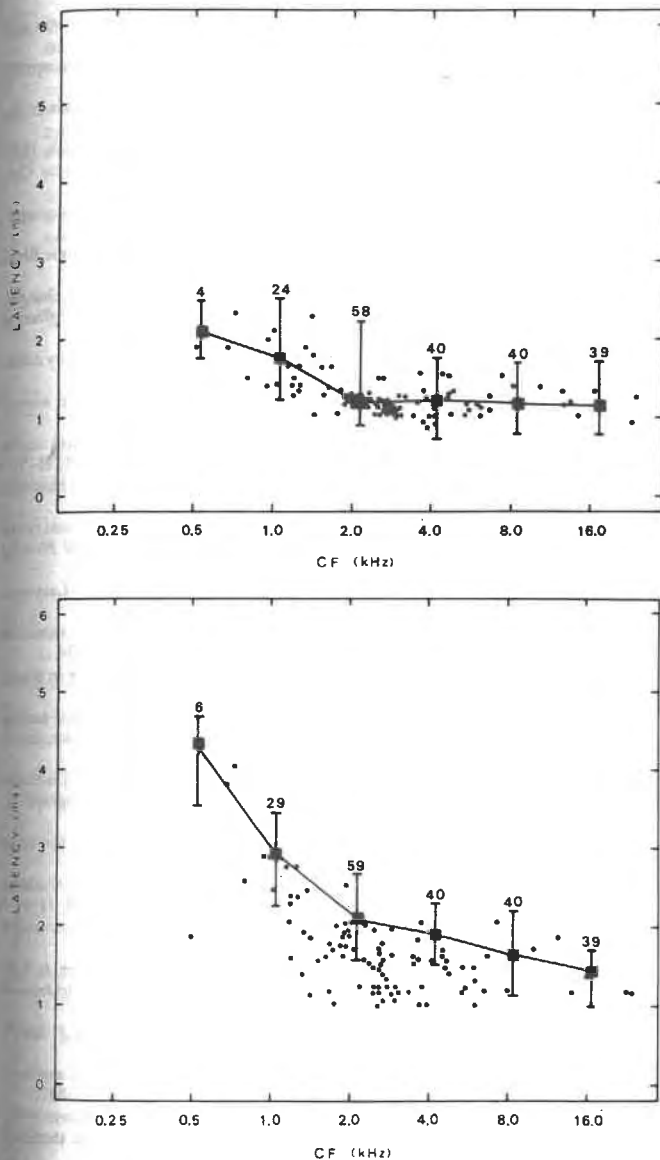


Figure 16. The top panel shows the single fiber latencies obtained with clicks having an SPL of approximately 100 dB. The bottom panel shows the single fiber latencies obtained at the unit's threshold for click stimulation, i.e., the stimulus level varied. The median (filled squares) and range (vertical bars) show how the latency values vary among units in different octave bands in the normal chinchilla. The latency data from individual units from noise-exposed animals are plotted as a function of the characteristic frequency of the unit (filled circles) for comparison (from Ref. 27, Salvi, R., D. Henderson, and R. P. Hamernik, Single auditory nerve fiber and action potential latencies in normal and noise-treated chinchillas, *Hear. Res.* 1, 237-251, 1979, reproduced by permission).

delayed, it is originating from regions of the cochlea apical to those regions that typically generate the response in normal ears. The sudden "catch up" that occurs as intensity is raised presumably occurs when the neural activity spills over into adjacent high-frequency regions.

SUMMARY

The symptoms of sensorineural hearing loss have been recognized for some time, but little has been known about

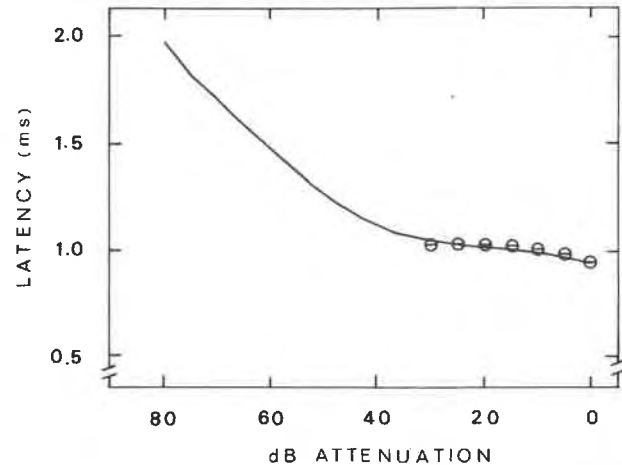


Figure 17. Mean latency of the N_1 AP as a function of click attenuation in normal (line) and noise-treated (open circles) chinchillas. The peak SPL is approximately 100 dB at 0 dB attenuation (from Ref. 27, Salvi, R., D. Henderson, and R. P. Hamernik, Single auditory nerve fiber and action potential latencies in normal and noise-treated chinchillas, *Hear. Res.* 1, 237-251, 1979, reproduced by permission).

the underlying physiological changes. Recent single unit studies have begun to provide insights into the mechanisms responsible for the symptoms and have helped to sharpen our concepts on how acoustic information is processed in both normal and hearing-impaired ears. Single unit studies performed on animals with noise- or drug-induced hearing loss have revealed significant changes in neural tuning and neural sensitivity. In addition, the timing of the neural responses is also altered if the data are evaluated in terms of intensity above threshold. There is also some indication that the pattern of spontaneous activity across the distribution of nerve fibers may be affected by sensorineural hearing loss. However, other measures such as the neural discharge patterns over time and the discharge rate-intensity functions at the characteristic frequency do not seem to be altered in pathological animals.

The frequency selectivity of the normal ear is remarkable in comparison to most electronic filters. These neural filters play a key role in processing complex acoustic signals such as speech. Unfortunately, one of the most profound changes that occurs with sensorineural hearing loss is the broadening of these neural filters. Thus, when complex sounds are presented at suprathreshold levels, the individual neural elements within the system are unable to respond to selective components of the signal. Low-frequency components pose the most serious problem when there is a significant loss of tuning; excitation will now readily spread toward the high frequencies. A conventional hearing aid cannot restore normal frequency selectivity; however, one way in which the spread of excitation might be minimized in a hearing aid is to limit the maximum output of low-frequency sounds, e.g., by independently controlling the maximum output (automatic gain control) of each channel of a series of band-pass filters; the output of each of the channels could then be recombined and used to drive the hearing aid. At the low frequencies, the maximum output could be restricted to intensities near threshold to prevent the spread of excitation toward the high-frequency regions of the cochlea.

The audiogram often plays an important role in selecting a hearing aid with the appropriate frequency response characteristics. The assumption is that the aid will overcome the loss in sensitivity of a particular region of the cochlea. The assumption may be reasonably valid as in Figure 8 where the behavioral threshold shifts are in close accord with the neural threshold shifts and pattern of cochlear damage. However, in some cases, the activity from a region of the cochlea may be completely eliminated, as in Figure 7, yet the behavioral response is maintained due to the spread of excitation. The point of comparing Figures 7 and 8 is that the audiograms of the two animals are quite similar yet the pattern of cell loss is much different. Consequently, one would expect the two animals to perceive an 8 kHz tone in much different ways. It should not be too surprising then that two listeners with the same audiogram will perform quite differently with the same hearing aid. One may be a successful hearing aid user and the other may not. In summary, the audiogram may provide a rough estimate of the underlying pattern of cochlear damage. If one wishes to obtain a clearer picture of the region of the cochlea mediating the behavioral response, one might consider measuring psychophysical tuning curves. Presumably, the location of the tip provides an indication of the region of the cochlea generating the responses. Zwicker⁴⁰ has developed a relatively efficient method of measuring psychophysical tuning curves in humans using a modified Bekesy audiometer; such a technique might prove useful clinically.

Because sensorineural hearing loss frequently has been correlated with damage in the cochlea, one might assume that all the symptoms of sensorineural hearing loss have a neural correlate in the output of the auditory nerve. One symptom which has defied an immediate explanation at the level of the auditory nerve is the breakdown in temporal summation. The working assumption outlined earlier has been that the input to the physiological summator was abnormal and that the centrally located summation process was unaffected by peripheral damage. Recent evidence suggests that these assumptions may be incorrect. First, anatomists have recently discovered that degeneration occurs in central auditory nuclei after damage to the cochlea.²² Second, Gerken¹⁰ has measured the behavioral thresholds for electrical stimulation using electrodes in the central auditory pathway. The electrical stimulus bypasses the cochlea, thus, the threshold-duration function obtained with electrical stimulation represents a central auditory process. After destroying the cochlea, there was a drastic reduction in temporal summation for electrical stimulation. Apparently, peripheral damage can alter the central process involved with the temporal summation of electrical stimuli. These central changes could account for some of the symptoms associated with sensorineural hearing loss. Additional single unit studies in the central auditory pathway using animals with cochlear lesions could provide important insight into the mechanism for temporal summation in both normal and hearing-impaired animals.

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Announcement of Annual Meeting and Call for Papers

The 10th Annual Meeting of the American Auditory Society will be held in Cincinnati, November 17, 1983. Interested persons are invited to submit five copies of a 300-word abstract with a brief summary by June 1 to: Robert W. Keith, Ph.D., Department of Otolaryngology, Mail Location No. 528, University of Cincinnati Medical Center, Cincinnati, OH 45267. Acceptance notices will be sent August 15, 1983.