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7. During the first three copulation tests stimulus females were made receptive by subcutaneous implants of EB. During all other tests the stimulus females were brought into estrus by injections of 50 μg of EB 48 hours before the test and 500 μg of progesterone 4 hours before the test (P. Perlman of Schering Corp., Bloomfield, N.J., contributed the hormone products).
8. Not all the spontaneously ejaculating females finished three copulatory series during individual observation periods. However, the data for the females completing less than three copulatory series were included, since deletion of the data for these females minimally altered the medians for the first and second copulatory series.
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10. Mount bouts are clusters of mounts and intromissions that are interrupted only by genital autogrooming or behavior oriented toward the stimulus female. Grooming any other part of the body or pointing the nose away from the stimulus female indicates that the preceding sexual act terminated the mount bout. For additional details, see B. D. Sachs and R. J. Barfield [*J. Comp. Physiol. Psychol.* **73**, 359 (1970)].
11. Male and perinatally androgenized female rats emit postejaculatory ultrasonic vocalizations [R. J. Barfield and L. A. Geyer, *Science* **176**, 1349 (1972); see also (*9*)]. Bat detector monitoring of the postejaculatory periods of three spontaneously ejaculating females indicated the absence of ultrasonic vocalizations. When the remainder of the spontaneously ejaculating females were observed during their postejaculatory periods, they did not display the stereotyped breathing pattern which normally accompanies postejaculatory vocalizations in the male rat.
12. Spontaneous pituitary tumors are common in older female rats, and the occurrence of pituitary tumors is further potentiated by chronic administration of estrogen [O. Mühlbock and H. G. Kwa, *Acta Unio Int. Contra Cancrum* **18**, 275 (1962)].
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22. Species of rodents differ in the periods in which exogenously administered androgens and estrogens are capable of potentiating the expression of the ejaculatory patterns in females. The postnatal period has been assumed to be the sensitive period for potentiation of the ejaculatory pattern in the rat [I. L. Ward, *Horm. Behav.* **1**, 25 (1969)]. Normal female rats may be exposed to prenatal androgen secreted by male siblings in utero. Female rats most proximal to male siblings in utero tended to show more mounting behavior in adulthood [L. G. Clemens, in *Reproductive Behavior*, W. Montagna and W. A. Sadler, Eds. (Plenum, New York, 1974), p. 23]. Natural in utero exposure to androgens may be involved in the reported adult display of the ejaculatory pattern in the prenatally untreated female guinea pig, a species in which the prenatal period is the sensitive period for differentiation of sexual behavior [R. W. Goy, C. H. Phoenix, R. Meidinger, *Anat. Rec.* **157**, 87 (1967)].
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Auditory Fatigue: Retrocochlear Components

Abstract. *Changes in auditory sensitivity were measured at the VIII nerve, cochlear nucleus, and inferior colliculus after a fatiguing sound exposure. Losses in sensitivity progressively increased from peripheral to central auditory sites. The results suggest that there is a retrocochlear component to auditory fatigue when it is induced by low-level sounds of short duration.*

Excessive acoustic stimulation can lead to auditory fatigue or a temporary threshold shift (TTS) in hearing. Although the definition of TTS is straightforward, its physiological basis appears to be complex, and several different physiological disorders in the cochlea have been implicated in TTS (*1*). These disorders presumably lead to a depression of the cochlear potentials; for example, during asymptotic TTS, Benitez *et al.* (*2*) reported a reduction in cochlear microphonic sensitivity that was 24 and 48 db for the second and third turns of the cochlea, respectively. They were unable to elicit the VIII nerve action potential.

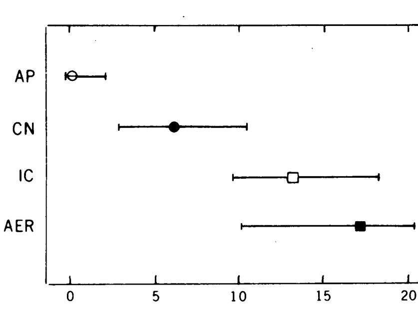

Fig. 1. The median threshold shift and interquartile range (horizontal bar) obtained after an 8-minute pure tone exposure at 95 db SPL. The open circle denotes the VIII nerve action potential (*AP*, $N = 41$); the closed square is the auditory evoked response (*AER*, $N = 30$). Threshold shifts in single neurons from the cochlear nucleus (*CN*, closed circle, $N = 51$) and inferior colliculus (*IC*, open square, $N = 12$) were based on the lateral shift in the function relating discharge rate to intensity.

Circumstantial evidence also links TTS to the cochlea. First, the TTS produced by a pure tone is distributed in a pattern that resembles the spread of mechanical vibration along the basilar membrane. Second, the audiological signs and symptoms associated with TTS (*3*) are consistent with a hearing loss of cochlear origin. Hence, the consensus has been that TTS is strictly a cochlear phenomenon.

A recent study, however, suggests that central auditory processes are involved with TTS (*4*). Our results not only show that there is a central component to TTS, but also demonstrate that there is a progressively larger loss in sensitivity from peripheral to central auditory sites.

Chinchillas were used as subjects in three series of experiments. Each animal was exposed to a standard TTS-producing tone 8 minutes in duration at 95 db SPL (sound pressure level relative to 0.0002 dyne/cm^2) near the tympanic membrane. The exposure was at a frequency between 0.4 and 8.0 khz.

In experiment 1, the chinchilla's auditory evoked response (AER) was recorded from a chronic electrode placed over the rudimentary tentorium. This potential was used to estimate the magnitude of TTS resulting from the exposure (*5*). Pre- and postexposure AER thresholds were measured at one of six test frequencies (0.5, 1.0, 2.0, 4.0, 6.0, and 8.0 khz, five animals at each frequency). The TTS exposure was always at the test frequency.

In experiment 2, the VIII nerve action potential and neurons from the cochlear

nucleus were measured from anesthetized (sodium pentobarbital) and tracheotomized chinchillas. The action potential visual detection level was measured with clicks (*6*). Microelectrodes were used to study neurons from the cochlear nucleus. Only neurons with characteristic frequencies between 0.4 and 8.0 khz were accepted for study. The pre- and postexposure protocol consisted of measuring spontaneous activity and the tone burst (50 or 100 msec duration) discharge rate as a function of intensity at characteristic frequency. Poststimulus time histograms were used to categorize units as Pauser, Chopper, Primary-like, Flat Primary-like, and On units (*7*). Each unit was given the TTS exposure at its characteristic frequency. Only one TTS exposure was used per ear.

In experiment 3, single units from the contralateral inferior colliculus were studied according to the procedures outlined in experiment 2.

Figure 1 summarizes the effects of the TTS exposure on various auditory potentials during the first 10 minutes postexposure. The VIII nerve action potential, which was measured approximately 7 minutes postexposure, has a median threshold shift of 0 db (N = 41, interquartile range 0 to 2 db). Single unit threshold shifts were defined from the lateral shift of the function relating discharge rate to intensity. Threshold shifts were averaged across units with different characteristic frequencies, since there did not seem to be any frequency effect. The units in the cochlear nucleus had a median threshold shift of 6.8 db (N = 51, interquartile range 2.9 to 10.4 db). The units in the inferior colliculus showed a median threshold shift of 13 db (N = 12, interquartile range 9 to 18 db). It is important to note that all units in the colliculus exhibited a threshold shift that was 6 db or greater, whereas a number of Pauser and Primary-like units in the cochlear nucleus failed to show any loss in sensitivity. The lack of a threshold shift in some cochlear nucleus units implies that the VIII nerve fibers innervating these cells were unaffected by the exposure. Finally, threshold shifts in the AER were nearly constant across all exposure frequencies, presumably because the sound pressure level was equalized at the tympanic membrane for different exposure frequencies (*8*). Consequently, the TTS values have been averaged across frequency. The median AER threshold shift was 17 db (N = 30, interquartile range 10 to 20 db). The sound exposure used here produces a progressively larger loss in sensitivity from the cochlea to more central auditory sites.

Figure 2 shows the effects of the exposure on the function relating discharge rate to intensity. The intensity functions from cochlear nucleus units were generally displaced to the right, but the slope and maximum discharge rate of the functions were unaltered (Fig. 2A). The intensity functions of most units in the inferior colliculus were displaced to the right as in Fig. 2B.

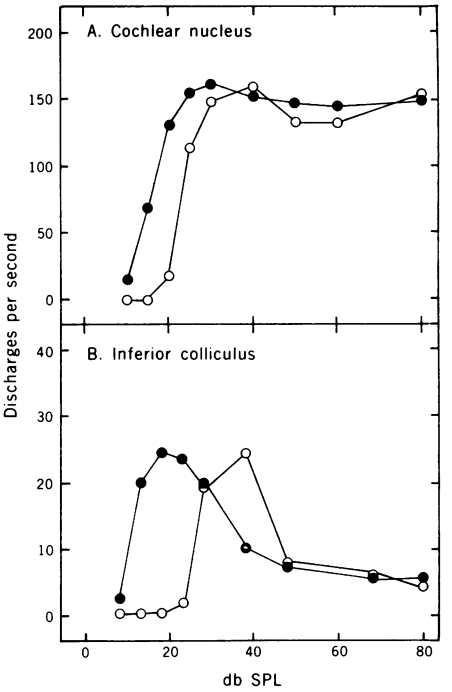

Fig. 2. The preexposure (closed circles) and postexposure (open circles) functions relating discharge rate to intensity for a typical neuron in the cochlear nucleus (A) and inferior colliculus (B). The unit from the cochlear nucleus was classified as a Chopper neuron based on its tone burst poststimulus time histogram. The neuron from the inferior colliculus was an On unit.

The remaining units showed a lateral shift of the function plus a significant reduction in the maximum discharge rate. The loss in sensitivity is almost certainly a consequence of the pure tone exposure, because the units in the cochlear nucleus and inferior colliculus tended to recover after the exposure. Thus, each preparation served as its own control and the immediate loss in sensitivity following exposure cannot be attributed to a general physiological deterioration of the animal or cell. Postexposure spontaneous activity was also reduced in all of the cochlear nucleus units except in the On units; their spontaneous rates were equal to or greater than preexposure levels. Similar effects were observed in the inferior colliculus.

In summary, our measurements indicate that low-level, short-duration sound exposures produce a progressively larger loss in sensitivity from peripheral to central auditory sites. These results suggest that there is a retrocochlear component to TTS. Results from Benitez *et al.* (*2*) and others imply that TTS is strictly a cochlear phenomenon. These seemingly opposite positions can be reconciled if we consider the auditory system as being organized as a series of cascaded stages. With low-level, long-duration exposures (for example, asymptotic TTS) there can be considerable fatigue at the first stage (that is, the sensory cell). Thus, the output of the first stage will be low even though the input signal (basilar membrane motion) is being maintained at a high level. A reduced output at the first (or preceding) stage reduces the possibility of fatigue at the second (or more central) stage. Thus, with low-level, long-duration exposures, the loss of sensitivity at the first stage (or cochlea) will tend to determine the system's overall loss in sensitivity.

The system will respond differently to a low-level, short-duration exposure. Such an exposure will produce a high output at the first stage, second stage, and so on, because none of the stages can be completely fatigued during the exposure. This leads to a small amount of fatigue at each stage of the system. The system's overall loss in sensitivity will therefore depend on the net loss at each stage; consequently, the measured loss in sensitivity will increase from the cochlea to more central auditory sites. In summary, different TTS exposures are likely to produce different physiological changes even though the final behavioral measure of TTS is the same.

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