Recent studies of temporary threshold shift (TTS) and permanent threshold shift (PTS) in animals

William W. Clark
Central Institute for the Deaf, 818 South Euclid, St. Louis, Missouri 63155

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It is well known that excessive exposure to noise results in temporary and/or permanent changes in hearing sensitivity in both human and animal subjects. The purpose of this review is to describe the major findings from laboratory studies of experimentally induced hearing losses, both temporary and permanent, resulting from exposure to noise in animal subjects which have been published since the report of Kryter *et al.* (1966). The data reviewed support the following general statements: (1) The chinchilla is the most widely used and most appropriate animal model for studies of noise-induced hearing loss; (2) with continuous exposures to moderate-level noise, thresholds reach asymptotic levels (ATS) within 18–24 h; (3) permanent threshold shifts, however, depend upon the level, frequency, and the duration of exposure; (4) below a "critical level" of about 115 dB, permanent threshold shift (PTS) and cell loss are generally related to the total energy in continuous exposures; (5) periodic rest periods inserted in an exposure schedule are protective and result in less hearing loss and cochlear damage than equal energy continuous exposures; and (6) under some schedules of periodic exposure, threshold shifts increase over the first few days of exposure, then recover as much as 30 dB as the exposure continues.

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INTRODUCTION

It is well known that excessive exposure to noise results in temporary and/or permanent changes in hearing sensitivity in both human and animal subjects. Knowledge about damaging effects of noise, both functional and morphological, have come from a number of studies utilizing very different methodologies. Surveys of hearing levels of noise-exposed individuals in industrial environments can provide important, but limited, information about the relation between exposure to noise and hearing loss. Surveys are complicated by incomplete or nonavailable information about occupational noise exposure, nonoccupational noise exposure, pre-existing hearing loss due to other etiologies, and inconsistent test methods. Laboratory studies of hearing loss following exposure to noise in human subjects are limited to exposure conditions that produce only temporary hearing losses; recent concern about occult damage to the cochlea with functionally innocuous exposures makes these kinds of studies even more difficult to design and carry out safely.

Animals have been used as models in studies of noise-induced auditory effects for decades. For example, Davis (1953) and Wever and Lawrence (1954) investigated the effects of acoustic overstimulation on the cochlear microphonic response of the guinea pig cochlea. Although these studies did not include behavioral determination of hearing ability, they did incorporate an index of sensitivity, the elevation of the cochlear microphonic response, as the functional measure of impairment.

True behavioral measures of hearing sensitivity in animal subjects, despite some early reports in monkeys and chimpanzees (Wendt, 1934; Elder, 1934), did not become popular until the 1950s and 1960s. The development of these techniques allowed the assessment of correlations between functional deficit and histopathology after exposure to ototraumatic agents. Probably the most significant behavioral study before 1970 which addressed the issue of noise-induced temporary and permanent hearing loss in animal subjects was the Miller *et al.* report (1963). Miller *et al.*, used behaviorally trained cats to investigate hearing loss and cochlear damage after exposures to bands of noise on continuous and interrupted schedules. In the report of the NAS-NRC Chaba Working Group 46, "Hazardous exposure to intermittent and steady-state noise" (Kryter *et al.*, 1966), the Miller *et al.* study was the only study referenced that utilized animal subjects.

The purpose of this review is to describe the major findings from laboratory studies of experimentally induced hearing losses, both temporary and permanent, resulting from exposure to noise in animal subjects. The review is limited to studies that have been published since the report of Kryter *et al.* (1966). Studies that measured hearing directly, i.e, by behavioral methods, are given the greatest weight in the review. Many other methods for inferring hearing ability have been routinely used by auditory physiologists and morphologists; these indirect indices of auditory function have limitations that make them less precise at describing hearing loss than direct behavioral methods.

Due to limitations in scope, this review omits many important papers that are not directly concerned with temporary threshold shifts (TTS) or permanent threshold shifts (PTS). Among those studies omitted are important works that use noise as an ototoxic agent for studying physiological

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responses of the peripheral or central auditory nervous system (e.g., Liberman and Kiang, 1978); studies of morphological changes in the cochlea and auditory nervous system following exposure to noise; and all studies of impulsive noise.

The review is organized around four basic questions: (1) Is there an appropriate animal model for studies of NIHL? (2) what are the effects on hearing produced by continuous exposure to noise? (3) what is the role of periodic rest on hearing loss from noise exposure? (4) what is the relation between temporary and permanent hearing loss following exposure to noise?

I. ANIMAL SUBJECTS AND METHODS OF STUDY

Noise-induced hearing loss has been investigated in a number of animal subjects. By far, the largest representation in the literature comes from mammals, mainly rats (Borg, 1982a; Sullivan and Connoly, 1988; Lenoir *et al.*, 1978; Borg, 1987), mice (Yanz and Abbas, 1982; Henry, 1983), guinea pigs (Cody and Johnstone, 1982; Cody and Robertson, 1983; Buck, 1982; Petersen *et al.*, 1977; Robertson and Johnstone, 1980; Maehara *et al.*, 1984; Ising *et al.*, 1982; Stopp, 1983; Goulios and Robertson, 1983; Conlee *et al.*, 1986; Syka and Popelar, 1980; Canlon *et al.*, 1988; Buck *et al.*, 1984; Pye, 1978; Pye, 1973); and chinchillas (Clark *et al.*, 1987; Ward *et al.*, 1981; Salvi *et al.*, 1983; Clark and Bohne, 1978; Clark *et al.*, 1974; Bohne and Clark, 1982; Miller, 1970; Salvi *et al.*, 1978; Mills, 1973; Mills, 1973; Mills *et al.*, 1973; Mills and Talo, 1972; Saunders *et al.*, 1977; Carder and Miller, 1972; Borg and Engstrom, 1983). A few studies have investigated hearing loss in primates (Lonsbury-Martin and Martin, 1981; Moody *et al.*, 1978; Lonsbury-Martin and Martin, 1981; Nielsen *et al.*, 1984; Nielsen *et al.*, 1978; Stebbins *et al.*, 1979; Lonsbury-Martin *et al.*, 1987), but the expense of maintaining a primate colony and practical and ethical considerations make the primate a difficult subject at best. Studies of hearing loss following noise exposure in non-mammalian species include the lizard (Mulroy, 1986) and the parakeet (Saunders and Dooling, 1974).

The assessment of hearing sensitivity is made either directly, by behavioral methods, or indirectly, by analysis of electrical potentials such as the averaged evoked response (AER) from the brain stem, which provide a measure of sensitivity of some portion of the auditory system. The chief advantage to the electrical methods is that they do not require the extensive training time that is common with behavioral procedures. They also allow frequent assessment of thresholds, for example, in following the growth of hearing loss over periods of minutes or hours. However, these measures provide only an indirect assessment of thresholds, which may not always co-vary with behavioral sensitivity. Furthermore, evoked response procedures utilize short-duration signals (approximately 20 ms). Henderson (1969) has shown that the AER thresholds of the chinchilla are approximately 20 dB less sensitive than behavioral thresholds; this can be accounted for by considering the temporal integration of the signal. Clark and Bohne (1986) have shown that the temporal integration function flattens in chinchillas with 10–15 dB of threshold shift for long duration (>1 s) signals; this alteration of the function leads to an underestimate of threshold shift when short-duration signals are used as estimators. Despite these limitations, AER audiometry has been used widely in studies of noise-induced hearing loss in animal subjects.

Obviously, a true measure of shifts in auditory sensitivity obtained behaviorally is preferred if it is possible. A variety of behavioral techniques have been employed to evaluate auditory sensitivity in animal subjects; these include conditioned inhibition of reflex activity, such as conditioned shiver audiometry in refrigerated guinea pigs (Anderson and Wedenberg, 1965). The conditioned suppression technique, which uses a conditioned aversive stimulus (tone followed by shock) to interrupt an ongoing behavioral response, such as licking a drink tube for water reinforcement, has been successfully used to measure auditory sensitivity in a number of species, including rabbits (Engstrom and Borg, 1982; Fay, 1988).

The behavioral procedure most commonly used in animal studies is the double-grill avoidance procedure. This technique requires an animal to cross a barrier in the presence of an acoustic signal to avoid a shock. The animals are tested in free-field conditions, and the method of constant stimuli is used to assess thresholds. The other behavioral procedure, commonly used in primates, guinea pigs, and chinchillas, uses a positive reinforcement paradigm to maintain the behavioral response (see Clark *et al.*, 1974, for details). Both behavioral procedures have advantages and disadvantages. Which technique is most appropriate depends upon the experimental question being addressed. For example, the positive-reinforcement procedure is inappropriate for short-term studies, because of the long (months) training time required. In addition, because the animal becomes satiated during experimental sessions, threshold shift behavior cannot be followed for long periods. For studies that require frequent monitoring of thresholds, even strong proponents of the positive reinforcement procedure must use an avoidance task (Stebbins *et al.*, 1973). On the other hand, the avoidance paradigm has been criticized because it uses aversive stimuli, is less humane, is subject to artifact (Ward *et al.*, 1981), and is not completely objective (Saunders *et al.*, 1977). In spite of these differences, hearing sensitivity determined by either procedure is quite similar.

Over the past 20 years, the chinchilla has emerged as the "standard" laboratory animal for the behavioral and anatomical study of noise-induced hearing loss. These animals are tractable, usually free from upper respiratory or middle ear infections, and can be maintained economically. The lifespan of the chinchilla is about 20 years; this makes the animal an ideal subject for long-term chronic studies of noise-induced hearing loss.

II. HEARING LOSS FROM CONTINUOUS EXPOSURE

Several reports in the literature have detailed the effects of exposure to high-level (>100 dBSPL) tones and bands of noise for short durations (<6 h) in behaviorally trained animals (e.g., Ward and Duvall, 1971; Hunter-Duvar and

Elliot, 1974; Clark *et al.*, 1974; Lonsbury-Martin and Martin, 1981; Borg, 1982a,b; Buck *et al.*, 1984). Generally, the noise exposures used in these studies is sufficient to produce permanent threshold shifts (PTS) after only a few minutes or a few hours of exposure. In general, these reports: (a) confirmed that maximum temporary threshold shifts occurred at frequencies approximately 1/2 oct above the center frequency of the exposure band; (b) found significant permanent threshold shifts associated with exposures that produced TTS of more than 60 dB; and (c) revealed a correlation between the measured permanent hearing loss and the extent and location of damage to sensory cells.

Other studies that include exposure to high-level stimuli include the Moody *et al.* (1978) report of hearing loss in primates following repeated exposures to octave bands of noise (OBN) from 0.5–8.0 kHz of up to 120 dB SPL, for periods of 8 h per day indicated relatively stable TTS values during exposure, and significant PTS for all exposure bands, although less TTS and PTS was observed for the 0.5-kHz OBN than for the higher frequency exposures.

The most systematic and complete work concerning the development of threshold shifts during and after continuous noise exposure in animal subjects came from Miller, Mills, Eldredge, and associates at the Central Institute for the Deaf between 1968 and 1973. Reports from these projects provide a thorough description of the development and progression of hearing loss from continuous exposure to octave bands of noise; recovery of hearing after noise exposure, and relations between electrophysiological and anatomical correlates of the behaviorally measured hearing loss. All the studies utilized the double-grill avoidance procedure developed for the chinchilla by Miller.

The audibility curve of the chinchilla, measured with a double-grill avoidance procedure, was reported by Miller (1970). A comparison of Miller's data for monaural chinchillas (contralateral ear surgically deafened) with monaural minimum audible field data for young, normal human ears (Sivian and White, 1933) is shown in Fig. 1. In general, the curves are quite similar. The chinchilla, unlike other small mammals, has a hearing bandwidth quite similar to that reported for humans. Absolute sensitivity is similar to human hearing with the exception that the low-frequency sensitivity of the chinchilla ($f < 1.0$ kHz) is somewhat better than man, high-frequency sensitivity is somewhat worse, and the chinchilla lacks the sensitivity notch noted for human ears around 4.0 kHz.

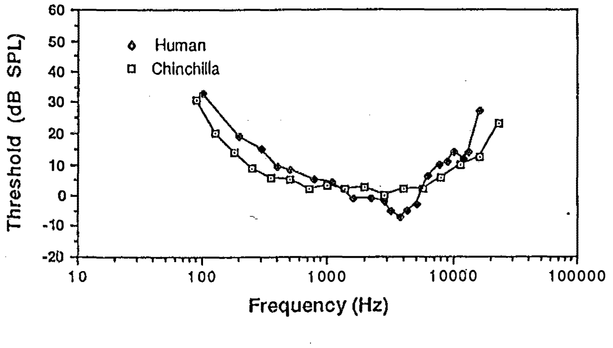

FIG. 1. Comparison of absolute sensitivity (monaural, minimum audible field) in chinchillas (Miller, 1970) and humans (Sivian and White, 1933).

Miller *et al.* (1971) investigated growth and decay of TTS, small PTSs, and cochlear injury in two chinchillas exposed to an OBN (300–600 Hz) at 100 dB SPL. This study showed the growth of TTS measured 4 min after the animal was returned to the quiet grew to about 60 dB at 715 Hz during the first 24–48 h of exposure; extending the exposure to 7 days did not increase the TTS. Miller *et al.* suggested that, after 20 h, the thresholds reached asymptotic levels and, after the noise exposure, thresholds returned to near-baseline levels within 5 days; small permanent injuries were noted in the cochlea.

Carder and Miller (1972) studied the relationship between octave band level (OBL) and asymptotic threshold shift in a group of chinchillas exposed to an OBN centered at 0.5 or 4.0 kHz for periods of 2 to 21 days. The OBL ranged from 65–105 dB SPL. The main findings of the Carder and Miller study are summarized in Figs. 2 and 3. It was found that TTS increased for the first 24–48 h of exposure and then reached an asymptote and remained constant for as long as the exposure continued. At asymptote the relation between ATS and OBL was well described by the formula

$$\text{TTS 4 min} = 1.6(\text{OBL} - 65), \tag{1}$$

where TTS was measured at 0.715 kHz, one-half oct above the center frequency of the exposure band, 4 min after the animal was removed from the noise exposure. Decay of TTS after exposure was approximately exponential with time; the course of decay of TTS was independent of the duration of exposure once asymptote had been reached.

In a series of studies, Mills and colleagues (Mills and Talo, 1972; Mills *et al.*, 1973; Mills, 1973a,b) provided

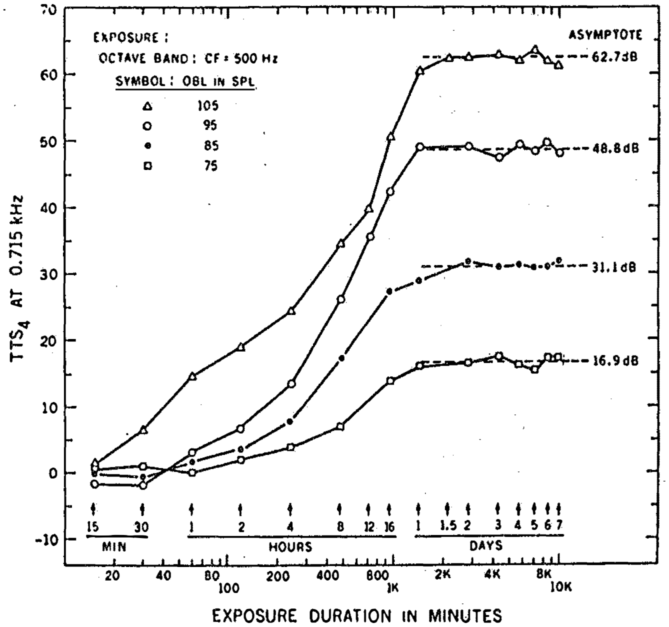

FIG. 2. Growth of TTS measured 4 min after exposure at 0.715 kHz as a function of exposure duration for chinchillas. Parameter is the level of noise exposure (from Carder and Miller, 1972).

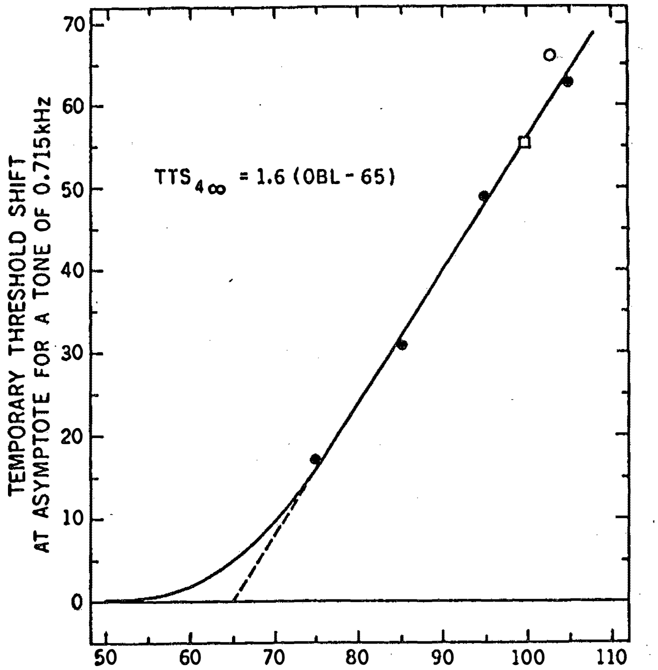

FIG. 3. Relation of TTS4 at 0.715 kHz at asymptote to the octave-band level of the noise with a center frequency of 0.5 kHz (from Carder and Miller, 1972).

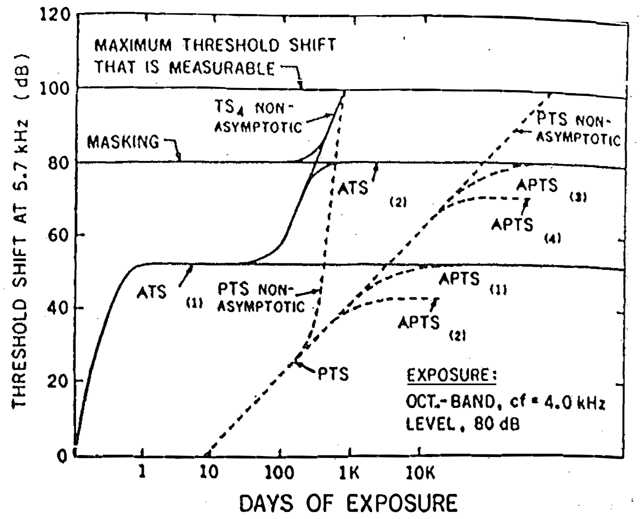

FIG. 4. Hypothetical relations between ATS, PTS, and duration of exposure (from Mills, 1976).

further support for the concept of asymptotic threshold shift for exposures to octave bands of noise (OBNs) at 0.5 or 4.0 kHz at levels ranging from 57–98 dB SPL. It was shown that the level of the shift at asymptote increased by 1.6–1.7 dB for every 1-dB increase in noise above a certain level. This level, the subtractive constant in Eq. (1) above, is 65 dB SPL for an OBN centered at 0.5 kHz, and decreases by 18 to 47 dB SPL for a noise centered at 4.0 kHz. The 18-dB difference was explained on the basis of the acoustic characteristics of the chinchilla external ear canal and the differences in critical ratio for 0.5 and 4.0 kHz.

In one of the experiments (Mills, 1973b), threshold shifts produced by exposure to an OBN at 80 dB SPL for 5 days were compared in a group of normally hearing chinchillas and a group that had previously sustained high-frequency PTSs of approximately 30 dB. It was found that the animals with pre-existing losses had lower threshold shifts than the controls; however, the sound-pressure levels required for detection, that is, the shifted thresholds rather than the threshold shifts, were the same in both groups. These data provide strong support for the hypothesis that the ear with sensorineural hearing loss is not more susceptible to the effects of noise than is the normal ear.

On the basis of the numerous studies carried out at Central Institute in the early 1970s, Mills (1976) formalized two previously stated hypotheses concerning the progression of hearing loss during continuous noise exposure and the recovery after cessation of exposure. These hypothetical possibilities are summarized in Fig. 4, which is developed for an exposure to an OBN centered at 4.0 kHz at 80 dB SPL. Consider first the solid lines, which delineate three possibilities for the progression of threshold shift measured 4 min after exposure (TS4). The first, labeled "ATS(1)," suggests that the ATS value obtained is sustained indefinitely. If true, this finding would indicate that during exposure an ear would reach a state of "equilibrium," where the restorative processes would just balance the degenerative ones, and that this process would continue indefinitely. The second possibility, labeled "ATS2," is that, after about 30 days of asymptotic behavior, thresholds would begin a secondary elevation which would be limited by the masked threshold of a signal whose spectrum level was identical to the exposure stimulus. The third, and most ominous possibility, is that TS increases indefinitely and is presumably causally related to continuing degeneration of sensory elements within the cochlea.

As acknowledged by Mills, the hypothetical possibilities for the PTS functions are even more tenuous than those for the ATS functions. However, there are some logical possibilities, which, of course, depend upon the ATS function. The first is that PTS will continue to grow without bound, that is, there would be no ATS. The second suggests that PTS will reach the ATS value, that is, ATS would set an upper bound on PTS. Finally, Mills suggested that some functional recovery after termination of exposure would always remain and the PTS function would also asymptote, but at a level lower than the corresponding ATS.

Three published studies and one continuing investigation address the progression of threshold shifts with long-duration exposures. Mills (1976) and Moody *et al.* (1976) exposed animals to moderate levels of noise for 90 days. In both studies, it was noted that beyond the 15th day of exposure, there was a 5- to 10-dB increase in thresholds over the initial asymptotic level. Mills thought that the elevation was within the precision of the measurement and therefore might not be significant, Moody *et al.* suggested that the thresholds had initially reached a plateau rather than a true asymptote.

On the basis of their previous anatomical and behavioral studies involving chinchillas exposed for periods of 9 or 18 days, Bohne and Clark (1982) predicted that, for continuous exposure of long-duration threshold shifts would remain constant only until sensory cell loss reached a "critical" level. They reported that chinchillas exposed to an OBN centered at 0.5 kHz, 95 dB SPL, for 108 days did not sustain any additional threshold shifts after the first few days of exposure; in order to test the critical level hypothesis, it was suggested that even longer exposures were needed.

Clark and Bohne have undertaken a continuing investigation of growth of threshold shift in chinchillas for exposures of extremely long duration. Their preliminary report of this study (Clark and Bohne, 1987) indicated that thresholds in chinchillas exposed for more than 3 years to the 0.5-kHz OBN at 95 dB SPL remained stable over the entire period (Fig. 5). The animals were removed from the noise for 2-week periods during the 3rd year of exposure; during these periods, thresholds decreased by 5–20 dB at all test frequencies (see Fig. 6). These observations suggest that even after chronic exposure for long periods, PTSs are not asymptotic with the ATS; the measured threshold shift is still a compound threshold shift (CTS), with both a temporary and permanent component.

Other studies in addition to those listed above have reported ATSs or ATS-like behavior. For example, Syka and Popelar (1980) described asymptotic shifts in behaviorally trained guinea pigs exposed to an OBN centered at 2 kHz, 100 dB SPL for 5 days. ATSs were also reported for the monkey by Moody *et al.* (1978) in the study reviewed above. On the other hand, Nielsen *et al.* (1984) failed to observe asymptotic thresholds in squirrel monkeys exposed to an OBN centered at 0.5 kHz, 95 dB SPL, for 96 h. Given the weight of the evidence cited above, it is likely that the Nielsen *et al.* study would have observed ATS-like behavior if the duration of the exposure was extended to several days.

III. HEARING LOSS FROM INTERRUPTED EXPOSURE TO NOISE

A small number of papers have addressed the role of intermittence on hearing loss, both temporary and permanent, produced by exposure to noise. Ward and colleagues have been conducting a series of studies of equal-energy exposures in chinchillas and assessing hearing loss and cochlear damage for a number of years (see Ward and Turner, 1982). The results of this investigation, which utilized several combinations of level, duration, and schedule of exposure with equal energy, generally support the equal-energy concept for continuous exposures less than a "critical level" of approximately 115 dB SPL for the chinchilla; interruptions in the exposure reduced the effects significantly. Ward's studies showed that outer hair cell loss was related more directly to exposure energy than PTS. Buck *et al.* (1984) evaluated threshold shifts of compound action potentials in guinea pigs exposed to iso-energetic noises, which ranged from 101 dB for 1 h to 127 dB for 9 s. They found a nonmonotonic function, with no effect observed between 9 s and 1 min or for durations longer than 30 s, and maximal shifts of 40 dB for exposures of 104–113 dB SPL.

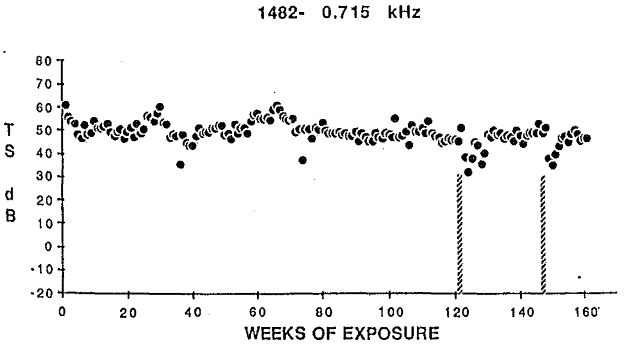

FIG. 5. Weekly average threshold shifts measured at 0.715 kHz for a chinchilla exposed continuously to an OBN centered at 0.5 kHz, 95 dB SPL. Vertical bars represent 2-week periods in which the noise was turned off.

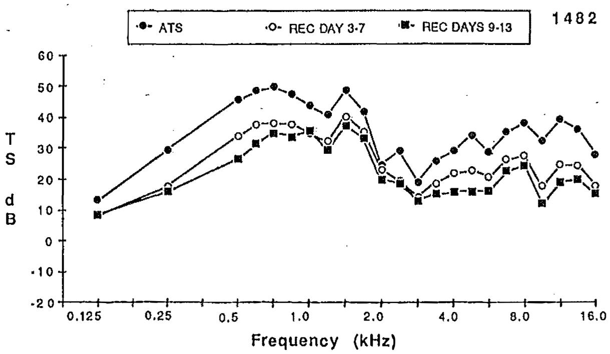

FIG. 6. Average threshold shifts as a function of test frequency for a chinchilla determined after 161 weeks of continuous exposure to an OBN centered at 0.5 kHz, 95 dB SPL. Closed circles represent ATS measured during exposure, and open circles and closed squares represent threshold shifts averaged over days 3–7 and 9–13 in quiet, respectively.

In summary, there are some data which suggest that, within some quite circumscribed limits of exposure level and duration, damage to the cochlea, and perhaps hearing loss can be predicted by the total energy in an exposure.

Saunders *et al.* (1977) addressed the issue of whether asymptotic threshold shifts could be observed in chinchillas periodically exposed to noise. Trained chinchillas were exposed to an octave band of noise (OBN) centered at 4.0 kHz 6 h per day, followed by 18 h of quiet, for 9 days. Thresholds were assessed with the avoidance paradigm (0.5–8.0 kHz) immediately before and after each day's exposure. The decay of threshold shift was followed after the ninth exposure until it stabilized; the procedure was then repeated for six exposure levels (57, 65, 72, 80, 86, and 92 dB). Care was taken to insure that PTS-producing exposures (the 86 and 92 dB ones) were the last in the sequence for the two groups of animals used in the study.

The major results of the study are depicted in Fig. 7. Shown are the TS4 min and TS18 h data points obtained at 5.7 kHz (1/2 oct above the center frequency of the exposure band) during the exposure and the recovery functions obtained thereafter. The data obtained during exposure suggest that TS4 and TS18h thresholds reach asymptotic values after a few days of exposure and the amount of TS is clearly dependent upon OBL. Furthermore, small PTSs were observed after the exposures at 86 and 92 dB; these PTSs were

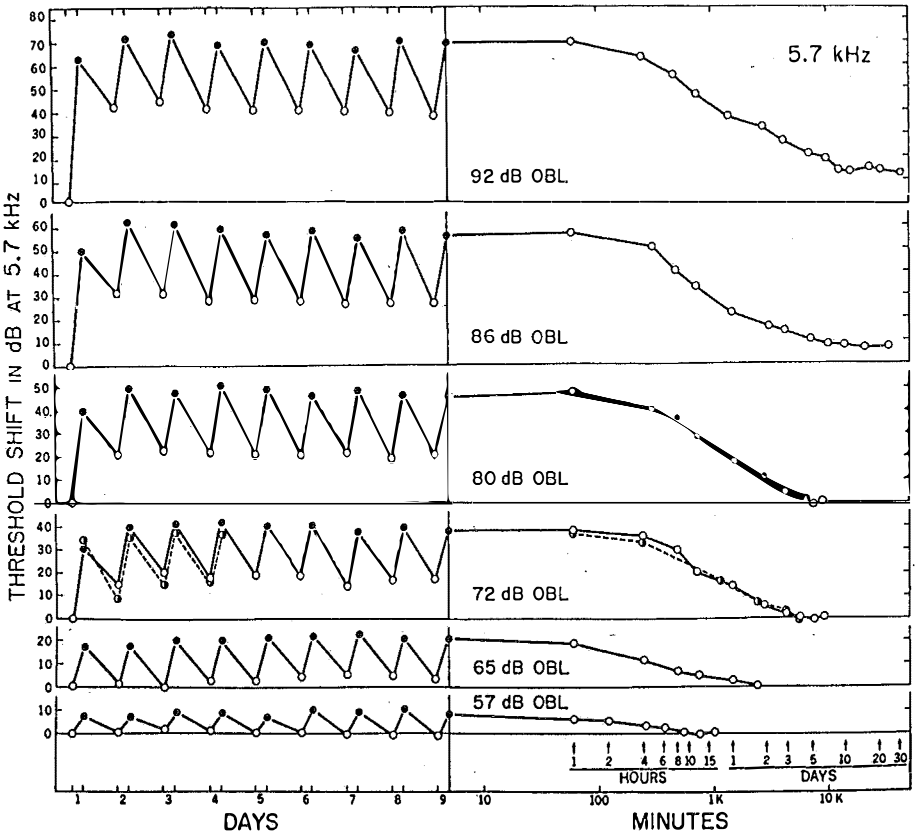

FIG. 7. Threshold shift during and recovery following a 9-day exposure to an octave band of noise centered at 4.0 kHz. Exposure was 6 h per day. The parameter is the OBL of the noise. Closed symbols represent thresholds obtained 4 min after each exposure (TS$_{4\,\mathrm{min}}$); open symbols represent thresholds obtained 18 h after each daily exposure (TS$_{18\,\mathrm{h}}$) (from Saunders *et al.*, 1977).

less than those reported for continuous noise at the same levels (Mills, 1973a).

The ATS was calculated for each level of noise by averaging the TS4 values over the last six exposures and a regression function obtained for the line shown in Fig. 8. This graph compares the ATS function for intermittent noise with that previously described by Mills and colleagues for continuous noise. The function for intermittent noise has a slope of 1.7 and an intercept of 52 dB. Therefore, the ATS produced by 6-h exposures an octave band of noise centered at 4 kHz can be predicted from the formula

$$\mathrm{ATS4} = 1.7\times(\mathrm{OBL} - C).$$

For the 4 kHz OBN, C is equal to 52 dB. The similarity with the continuous noise function led Saunders *et al.* to propose the "equivalent power hypothesis." Stated simply, this hypothesis suggests that ATS4 for intermittent exposures is reduced from that produced by continuous exposures by the ratio of the exposure time, Te, to the integration time for ATS, Ti. For the chinchilla, the integration time for ATS is 18–24 h; although the data from the Saunders *et al.* report are better fit by assuming a Ti of 18 h, utilizing 24 h as the base allows easy computation of ATS for interrupted exposures: One simply increases the value of C by 10 log (Te/Ti). Therefore, the effective OBL of an interrupted noise is reduced 3 dB from its OBL for each halving of the duration of exposure below 24 h. The ATS4 then is reduced by 1.7 dB for each 1-dB reduction in effective OBL.

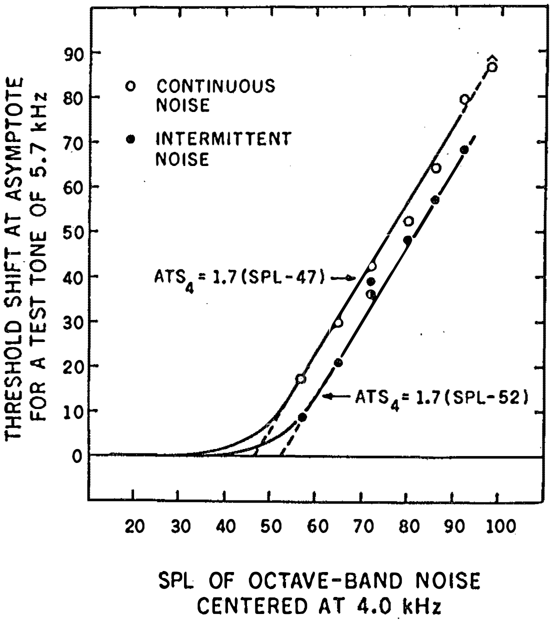

FIG. 8. The dependence of ATS at 5.7 kHz on the level of the octave band noise with a center frequency of 4.0 kHz for continuous exposure and for 6-h exposures repeated daily (from Saunders *et al.*, 1977).

Thus, for a constant level of exposure, ATS4 should be 8–10 dB less than for continuous exposures when Ti is 18–24 hours. The data contained in the Saunders *et al.* report were consistent with these predictions.

The generality of the equivalent power hypothesis depends not only upon an appropriate estimate of Ti for various octave bands of noise, but also upon evaluation of a number of schedules of exposure that contain equivalent en-

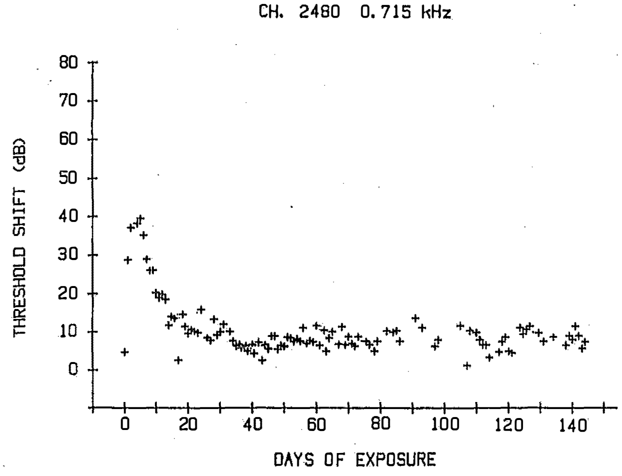

FIG. 9. Threshold shift and recovery during and following an exposure to an OBN centered at 0.5 kHz, 95 dB SPL, 6 h per day for 36 days (from Clark *et al.*, 1987).

ergy. Clark *et al.* (1987) evaluated threshold shifts in groups of chinchillas exposed to an OBN centered at 0.5 kHz, 95 dB SPL, on two schedules: 6 h per day for 36 days, and 15 min per hour for 144 days. Both types of exposures produced an initial shift of thresholds of 35–45 dB; however, after a few days of exposure, thresholds began to decline and eventually recovered to within 10–15 dB of original baseline values even though the exposure continued. Data from the two exposure schedules are shown in Figs. 9 and 10. An additional finding was that exposures on these schedules produced less PTS and cochlear damage than continuous exposures of equal energy.

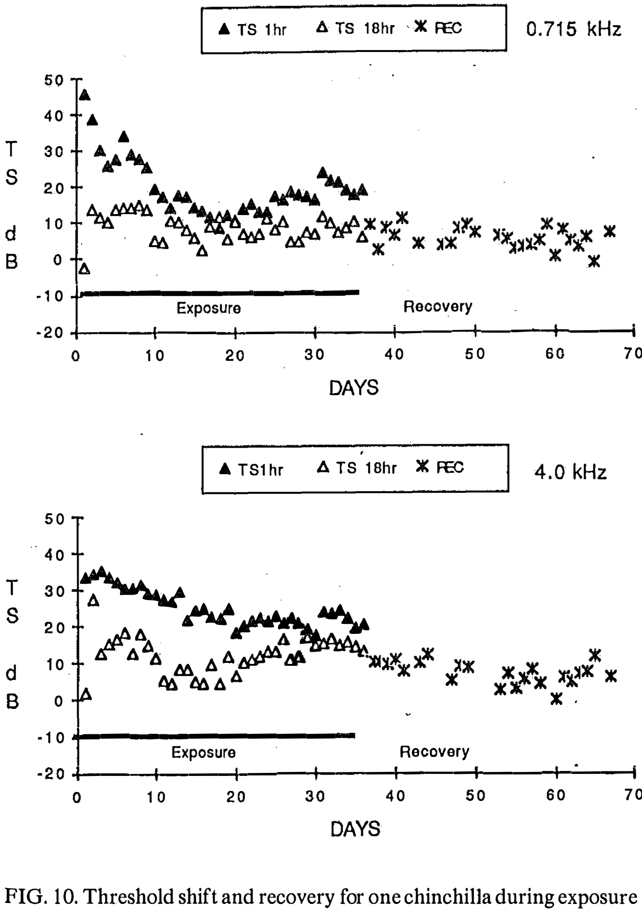

FIG. 10. Threshold shift and recovery for one chinchilla during exposure to an OBN centered at 0.5 kHz, 95 dB SPL, 15 min per hour for 144 days (from Clark *et al.*, 1987).

Clark *et al.* suggested that there was no contradiction between their findings and those reported by Saunders *et al.* (1977). Rather, it was suggested that the 9-day exposure paradigm utilized by Saunders *et al.* was simply not long enough to observe the recovery of thresholds during exposure. The recovery phenomenon, reported by Clark *et al.* for behavioral thresholds, was confirmed in a followup study of whole-nerve action potentials and single auditory-nerve fiber responses in chinchillas exposed on the same schedules (Sinex *et al.*, 1987).

The findings of the Clark *et al.* study show that the ear can recover as much as 30 dB of sensitivity even while it is being damaged by noise exposure and that the locus of the phenomenon is peripheral, probably at the level of the hair cell complex. This suggests that, after a few days of periodic exposure, the surviving cells are "toughened" or at least can adjust their operating point to maximize sensitivity. Further study is needed to test the generality of the recovery phenomenon, including determination of boundary conditions (level, frequency, scheduling, and duration of rest periods) of the effect.

The finding that the ear is somewhat protected from noise trauma by interrupting exposures with rest periods has been reported by other investigators (*i.e.*, Miller *et al.*, 1963; Bohne *et al.*, 1987). Canlon *et al.* (1988) hypothesized that, because the outer hair cells may serve a "musclelike" role in cochlear physiology, it might be possible to exercise or train the cochlea to be able to tolerate acoustic overstimulation. The goal of the project was to pre-expose subjects to a nontraumatic acoustic stimulus prior to receiving an exposure known to produce PTS.

Auditory brain-stem response thresholds were used to evaluate sensitivity in two groups of guinea pigs. The experimental group was exposed to a 1-kHz tone, 81 dB SPL continuously for 24 days; this exposure did not alter ABR thresholds in the experimental group. Following the pre-exposure regimen, the experimental and control groups were exposed to a 1.0-kHz tone at 105 dB SPL for 72 h. The control group had threshold shifts that ranged from 33–53 dB across frequencies; threshold shifts in the experimental group were significantly lower (12–25 dB) than the controls. The PTS function was obtained 8 weeks after the traumatic exposure. Permanent threshold shifts for the control group ranged from 14–35 dB. In contrast, all thresholds in the experimental animals returned to pre-exposure levels.

The findings summarized above have theoretical and practical importance. Obviously, the mechanism by which the sensory receptor mechanism can regain 30 dB of sensitivity between traumatic noise exposures needs to be identified. Furthermore, the observation that TS can recover during exposures complicates the relations between TS measures and noise exposure, since "more" noise can produce "less" TS. It also suggests that caution should be used in exposing human subjects to schedules of noise that may, in fact, be hazardous, but that produce little or no TS.

ACKNOWLEDGMENTS

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