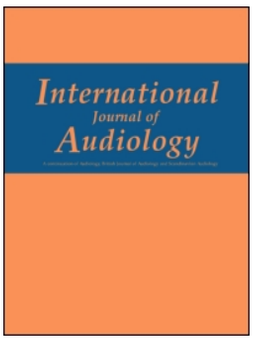
Taylor & Francis
Taylor & Francis Group



Audiology

ISSN: 0020-6091 (Print) (Online) Journal homepage: https://www.tandfonline.com/loi/iija19

Evoked-Response Forward-Masking Functions in Chinchillas with Noise-Induced Permanent Hearing Loss

Shalini Arehole, Richard J. Salvi, Samuel S. Saunders & Michael Anne Gratton

To cite this article: Shalini Arehole, Richard J. Salvi, Samuel S. Saunders & Michael Anne Gratton (1989) Evoked-Response Forward-Masking Functions in Chinchillas with Noise-Induced Permanent Hearing Loss, Audiology, 28:2, 92-110

To link to this article: https://doi.org/10.3109/00206098909081614

Published online: 07 Jul 2009.

Submit your article to this journal

Article views: 14

View related articles

Citing articles: 14 View citing articles

Full Terms & Conditions of access and use can be found at
https://www.tandfonline.com/action/journalInformation?journalCode=iija20

Audiology 1989;28:92–110

Evoked-Response Forward-Masking Functions in Chinchillas with Noise-Induced Permanent Hearing Loss

Shalini Arehole, Richard J. Salvi, Samuel S. Saunders, Michael Anne Gratton

Hearing Research Laboratory, Communication Disorders and Sciences, SUNY University at Buffalo, Buffalo, N.Y., USA

Key Words. Auditory-evoked response · Forward masking · Hearing loss · Hair cell loss · Inferior colliculus · Temporal resolution

Abstract. Evoked-response forward-masking functions were measured by chronic electrodes in the inferior colliculus of the chinchilla before and after exposure to an intense tone that produced a permanent hearing loss. Before exposure, the forward-masking time constants ranged from 50 to 90 ms. After exposure, the forward-masking time constants increased significantly in the region of hearing loss, but not in regions where hearing was normal. The effect of the hearing loss on the time course of forward masking was most pronounced once the hearing loss exceeded 20–25 dB. These physiological changes in the evoked-response forward-masking functions appear to parallel those observed psychophysically in human listeners.

Introduction

Many natural sounds such as speech vary rapidly in amplitude, and the ability of a listener to detect these amplitude fluctuations has ben referred to as auditory temporal resolution. In recent years, there has been a growing awareness that auditory temporal resolution may be compromised in hearing-impaired listeners. One measure of temporal resolution that has been shown to deteriorate in hearing-impaired listeners is gap detection. Normally, listeners can detect silent intervals, or gaps, in an acoustic waveform as short as 2–3 ms [Plomp, 1964; Smiarowski and Carhart, 1975; Giraudi et al., 1980]. However, gap detection thresholds of hearing-impaired listeners are longer than normal, particularly when the hearing loss exceeds 25–30 dB [Giraudi-Perry et al., 1982; Irwin et al., 1981; Fitzgibbons and Wightman, 1982; Boothroyd, 1973; Cudahy and Elliott, 1976]. Furthermore, the increase in gap detection thresholds appears to be strongly correlated with poor speech discrimination in hearing-impaired listeners [Tyler et al., 1982; Tyler, 1986; Dreschler and Plomp, 1985].

Previous work by Smiarowski and Carhart [1975] has indicated that there is a close relationship between gap detection and forward masking. Therefore, one might also expect to see alterations in the time course of forward masking in hearing-impaired subjects. Support for this contention comes from several recent studies showing a change in the time course of forward masking in hearing-impaired listeners [Trinder, 1979; Feston and Plomp, 1983; Dreschler and Plomp, 1985]. The works of Nelson and Turner [1980] and Cudahy [1982] are particularly relevant, since their results provide information about the time course of forward masking in different spectral regions. In their experimental paradigm, the listener adjusted the intensity of a tonal masker so that it just abolished the detection of a low-level probe tone having the same frequency as the masker. These measurements were then carried out over a range of masker-probe intervals in order to map out the time course of forward masking. The results obtained from hearing-impaired listeners indicated that the time course of forward masking was prolonged at frequencies with significant hearing loss and poor frequency selectivity, but was normal at frequencies where the threshold was normal. The increase in the forward-masking time constants was particularly noticeable once the hearing loss exceeded 25–30 dB.

Jesteadt [1980], on the other hand, has argued that the prolonged forward-masking time constants seen in hearing-impaired listeners may be the result of an abnormally rapid growth of masking rather than of a change in temporal resolution. Specifically, in hearing-impaired listeners, the amount of masking grows at a faster than normal rate when the masker level is increased. Consequently, only a relatively small increase in the masker level would be needed to mask the detection of a fixed-level probe, as the masker-probe interval increased; this in turn would increase the forward-masking time constant. These arguments illustrate the difficulty of separating the effects of abnormal temporal processing from abnormal response growth.

While the abnormally rapid growth of masking could potentially contribute to the prolonged forward-masking time constants in impaired listeners, it is not clear how this mechanism would account for prolonged gap detection thresholds, since equal-intensity signals surrounding the gap would be subjected to the same abnormally rapid growth of sensation [Irwin et al., 1981; Fitzgibbons and Wightman, 1982; Giraudi-Perry et al., 1982]. Thus, the prolonged gap detection thresholds in impaired listeners would seem to require abnormal temporal processing irrespective of any abnormally rapid growth of masking.

What neurophysiological mechanisms could be involved with the prolonged gap thresholds and forward-masking functions seen in hearing-impaired subjects? One mechanism that has been implicated is the neural recovery from short-term adaptation which is first expressed in the auditory pathway at the level of the auditory nerve. More specifically, if a unit is stimulated by two successive tone bursts, the firing rate to the second tone burst will be depressed below its normal level. However, as the time interval between the first and second tone bursts is increased, the firing rate to the second burst will gradually recover to its normal, unadapted firing rate by 100–200 ms [Smith, 1977; Harris and Dallos, 1979]. The neural

processes involved in short-term adaptation are generally thought to involve synaptic events between hair cells and auditory nerve fibers. Thus, one mechanism that could potentially account for the impairment in auditory temporal resolution is a delay in the recovery from short-term adaptation, i.e. an increase in the time it takes for the firing rate to return to its normal level following stimulation.

Several studies have suggested that the recovery from short-term adaptation in single auditory nerve fibers may be indirectly assessed by measuring the recovery in amplitude of the auditory nerve compound action potential [Abbas and Gorga, 1981; Harris and Dallos, 1979]. Recently, Gorga and Abbas [1981] compared the time course of recovery of the action potential in normal and noise exposed animals, using a forward-masking stimulus paradigm. They found no difference in the time course of recovery from short-term adaptation in normal and hearing-impaired animals. However, the growth of the response to the masker was reported to be steeper in the impaired than in the normal ears.

While the preceding action potential data indicate that the recovery from adaptation is unaltered in impaired ears, other single-unit studies suggest that the recovery process may be prolonged. Smith [1977] has stated that the recovery from short-term adaptation was abnormally long in single auditory nerve fibers obtained from animals in poor physiological condition. Evans [see discussion in Nelson and Turner, 1980] has also stated that the recovery from short-term adaptation is prolonged in auditory nerve fibers obtained from kanamycin-damaged ears. Although both investigators indicated that the recovery process was prolonged in impaired ears, neither presented data to support these claims.

In summary, psychophysical studies indicate that temporal resolution may be impaired in listeners with sensorineural hearing loss; however, the neural mechanisms underlying these psychophysical changes are not yet understood. At present, the only systematic study carried out at the auditory neve indicates that the recovery from short-term adaptation is normal in noise-damaged ears [Gorga and Abbas, 1981]. Gorga and Abbas [1981] have suggested that the lack of correspondence between the physiological and psychophysical data in damaged ears may be due to neurophysiological changes occurring in the central auditory pathway. In order to explore the possibility of central changes, we measured the evoked-response forward-masking functions in the inferior colliculus of the chinchilla before and after inducing a noise-induced permanent threshold shift (PTS).

Methods

Subjects

Ten normal chinchillas (400–800 g) were used as subjects. Each animal was anesthetized (ketamine, 0.1 mg/kg; acepromazine, 0.03 mg/kg; Rompun, 0.1 mg/kg), and made monaural by surgical destruction of the left cochlea. An electrode for recording the evoked response was then stereotaxically implanted in the inferior colliculus along with a reference electrode just below the dura mater [Henderson et al., 1973; Salvi et al., 1982a]. Evoked-response testing began approximately 1 week after surgery. All measurements were carried out in a sound-attenuating booth lined with sound-absorbing foam. The animals were placed in a yoke-like restrainer with a nose bar that fixed the orientation of the animal's head within the calibrated sound field thereby minimizing fluctuations in sound intensity reaching the tympanic membrane [Blakeslee et al., 1978]. Furthermore, after adapting to the head

restraint, the animals remainded relatively motionless during testing, thereby reducing muscle and movement artifacts in the evoked response as well as any fluctuations in sound intensity.

Recording

The signal from the electrode was filtered (30–3 000 Hz), amplified (20 000×) and led to a 12-bit A/D converter of a computer (PDP 11/23) with artifact rejection capability. The response was sampled for 30 ms (600 points, 20 kHz sampling rate), beginning at the onset of the probe tone. A total of 125 samples was obtained per waveform, and the averaged waveform was stored on disk for later analysis.

Stimuli

The test stimuli were generated with standard audio equipment (oscillator, electronic switches, mixer, programmable attenuator, amplifier and loudspeaker). The speaker (Radio Shack Minimus 7) was positioned 61 cm directly in front of the subject. The stimuli were calibrated using a 12.7-mm condenser microphone and sound level meter (Bruël & Kjaer 2606). The sound field was calibrated with the microphone positioned at a point normally occupied by the center of the animal's head.

A probe tone (10 ms duration, 1 ms rise/fall time) was used to elicit the evoked response during the threshold and forward-masking procedures. For threshold determinations, the tone bursts were presented at the rate of 10/s. The forward-masking experiments were carried out using both a masker and probe tone of the same frequency (0.5, 2, 4, or 8 kHz). The masker was 100 ms in duration with a 1-ms rise/fall time. At each test frequency, the interval between masker offset and probe onset was varied from 2 to 100 ms. The masker-probe combination was repeated once every 400 ms. After each masker-probe trial, a control trial was run in which the probe was presented alone; this was done to obtain an estimate of the response to the probe in the unadapted state.

The hearing loss was induced with a 2-kHz pure tone having an SPL of 105 dB. The stimulus was generated using an oscillator (Tektronics FG506), amplifier (Technics SE9060), and loudspeaker (JBL 2445J, JBL 2360). During the 5-day exposure, 2 animals at a time were placed in wire cages (13×20×15 cm) suspended 91 cm beneath the horn of the loudspeaker. The animals were given free access to food and water during the exposure. The sound field was calibrated using a 12.7-mm condenser microphone and sound level meter (Bruël & Kjaer 2606). Measurements were obtained at 4 points within each cage and found to vary by less than ±2 dB.

Experimental Design

At the start of the experiment, the evoked-response thresholds were measured to ensure that they were within normal limits [Henderson et al, 1973; Salvi et al., 1982a] and to determine the level of the probe tone used in forward masking. The forward-masking functions were then determined at four frequencies (0.5, 2, 4, and 8 kHz). After establishing baseline measures for threshold and forward masking, the animals were exposed for 5 days to a 2-kHz tone having an SPL of 105 dB (re 0.0002 dyn/cm^2). The animals were removed from the exposure and allowed to recover for approximately 25 days before repeating all of the threshold and forward-masking measurements.

Threshold

Evoked-response thresholds were measured at octave intervals from 0.5 to 16 kHz. Sound intensity was varied in 5-dB steps over roughly a 40-dB range in order to obtain a series of evoked-response waveforms. Threshold was defined as a just noticeable deflection of the response from baseline activity. Three threshold measurements were obtained both before and after exposure, and the average difference between the two sets of measurements defined the magnitude of PTS resulting from the exposure.

Forward Masking

Forward-masking functions were obtained using a probe tone 10 dB above threshold in order to elicit a small, but consistent evoked response. At each masker-probe interval (2, 5, 10, 20, 40, 80 and 100 ms), the masker intensity was varied over a range of intensities that caused a significant reduction in the probe-evoked response. The peak-to-trough amplitude of the probe-evoked response obtained in the presence of the masker was normalized to the response obtained to the probe alone. The normalized amplitude was plotted as a function of masker level. A straight line was then fit to the steeply rising portion of the input/output function. The slope of the line was used to estimate the growth of masking as well as the masked threshold. The masked threshold was defined as the intensity needed to produce a 50% reduction of the probe-evoked response. Two complete sets of forward-masking data

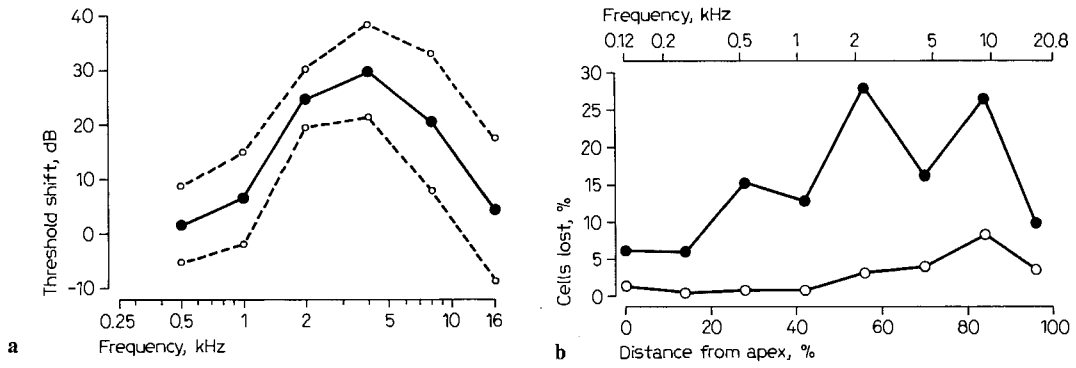


Fig. 1. (a) PTS pattern. Mean (n = 10) treshold shift in the auditory-evoked response (solid line) ± 1 SD (dashed lines). **(b)** Mean (n = 10) outer (= ●) and inner (= ○) hair cell loss averaged over octave intervals centered at 0.25, 0.5, 1, 2, 4, 8 kHz and over partial octaves at 0.125 and 16 kHz.

were obtained, and the mean value was computed. The mean masked threshold was plotted as a function of masker-probe interval in order to delineate the time course of forward masking. An exponential model of the form $Lm = Ae^{(DT/tau)}$ was used to fit the data by adjusting the constants A and tau to minimize the mean squared error term [Ryan et al., 1980]. Lm represents the intensity of masker, and DT is the time interval between masker offset and probe onset. This equation, which was used previously to describe the results from psychophysical studies of forward masking, has also been shown to provide a good fit to the evoked-response forward-masking functions in normal chinchillas [Vogten, 1978; Nelson and Turner, 1980; Cudahy, 1982; Arehole, 1986].

Histology

After all the evoked-response measurements were collected, the animals were killed by decapitation and their cochleas analyzed for sensory cell damage using the surface preparation technique [Engström et al., 1966]. The bulla was quickly removed and the cochlea exposed. The round window membrane was perforated and the stapes removed, after which cold glutaraldehyde (2.5%) in Veronal acetate buffer (pH 7.4) was gently perfused through the oval window. The cochleas were fixed overnight, and then postfixed in 1% osmium tetroxide in Veronal acetate buffer for approximately 15 min. Following dehydration by 70% ETOH, the cochleas were microdissected, and the organ of Corti and stria vascularis were mounted in glycerin on glass slides. The sensory epithelium was viewed at a magnification of 400 × with a Zeiss differential interference contrast microscope. Sensory cells were counted as present if the cell-body cuticular-plate complex was intact. Hair cell counts were averaged over 0.24-mm intervals and entered into a computer for analysis. The percentages of missing hair cells were plotted as a function of percent distance from the apex to obtain a standard cochleogram. The percent distance from the apex of the cochlea was converted to frequency, using the frequency-place map for the chinchilla developed by Eldredge et al. [1981].

Results

Threshold

The mean pre-exposure thresholds of the 10 animals used in the study ranged from 3 dB SPL at 0.5 kHz to 24 dB SPL at 16 kHz. The standard deviation at each frequency was less than 7 dB. These evoked-response thresholds are quite similar to those previously published for the chinchilla [Henderson et al., 1973; Salvi et al., 1982a]. Approximately 30 days after the exposure, the evoked-response thresholds were remeasured to determine the amount of PTS resulting from the exposure at 2 kHz. Figure 1a

shows the average hearing loss (±1 SD) resulting from the exposure. The hearing loss was primarily confined to the 2- to 8-kHz region, and ranged from 20 to 30 dB. As expected, the maximum loss occurred an octave above the exposure frequency, as has been noted previously [Davis et al., 1950; Hood, 1950]. It is important to note that the average threshold at 0.5 kHz was essentially unchanged; however, 1 animal in the group unexpectedly developed a 16-dB hearing loss at this frequency.

Hair Cell Loss

Figure 1b shows the average cochleogram for all 10 animals; this plot is intended to give the reader an overall perspective on the location and severity of damage in the cochlea. These results were obtained by dividing the cochlea of each animal into octave bands (partial octaves at the highest and lowest frequencies) using the frequency-place map developed for the chinchilla cochlea [Eldredge et al., 1981]. The mean loss in each band was computed using the data from all 10 animals. Outer hair cell damage was greatest in the 2- to 8-kHz region where the loss ranged from roughly 16 to 28%. The inner hair cell loss was negligible at the low frequencies, but increased slightly between 2 and 16 kHz, resulting in a maximum average loss of 8% at the 8-kHz location. An attempt was made to correlate the percentage of hair cell loss with the hearing loss in each animal. This was done by determining the inner, outer and total hair cell losses over octave lengths of the cochlea, centered at the place of either 0.5, 2, 4 or 8 kHz in the cochlea. The percent of inner, outer and total hair cell loss in each octave band was then correlated with the degree of threshold shift at the corresponding frequency. The correlation coefficients were generally positive, but low. The largest correlations were obtained between outer hair cell loss and hearing loss, but the correlation coefficients were not statistically significant. Thus, there was a relatively weak relationship between the pattern of hair cell loss and the pattern of hearing loss.

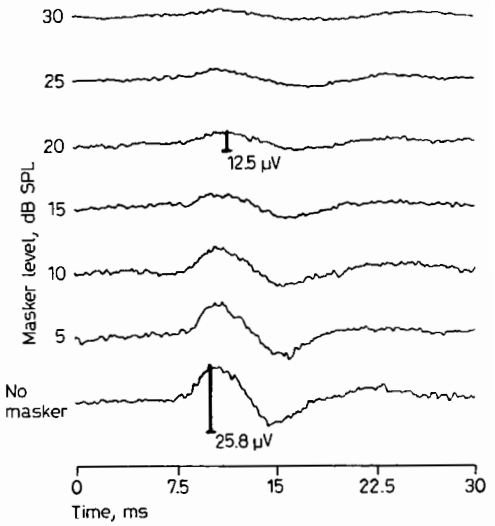


Fig. 2. Evoked-response waveforms obtained from one animal at 4 kHz in the absence of the masker (bottom trace) and preceded (DT=20 ms) by the masker (upper traces).

Forward-Masking Functions

Figure 2 shows a typical series of evoked-response waveforms obtained at 4 kHz using the forward-masking stimulus paradigm and a masker-probe interval of 20 ms. Time 0 corresponds to the onset of the electrical signal used to elicit the probe tone. The response is delayed by approximately 3 ms due to the rise/fall time of the stimulus and the transmission time from the speaker to the animal's head. The bottom trace shows

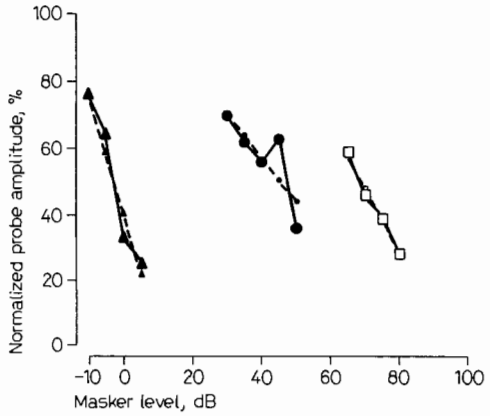


Fig. 3. Normalized probe amplitude plotted as a function of masker intensity at masker-probe intervals: ▲=5; ●=40; □=100 ms; all at a frequency of 4 kHz. Straight lines were fit to the data using linear regression (dashed lines).

the response in the absence of the masker. Waveforms obtained with the forward masker at levels between 5 and 30 dB SPL are shown above. As masker intensity increases, the amplitude of the response decreases so that by 20 dB SPL the response is reduced by approximately 50% of its normal amplitude.

The peak-to-trough amplitude of the evoked response obtained in the presence of the masker was normalized to the response obtained to the probe alone. The normalized amplitudes from 2 test sessions were averaged and then plotted as a function of masker intensity. Figure 3 shows the normalized amplitude-intensity functions for 1 animal at masker probe intervals of 5, 40 and 100 ms. The normalized amplitudes typically decreased linearly as the masker level increased. Using linear regression, a straight line (fig. 3, dashed line) was fit to the linear segment of the normalized amplitude-intensity function in order to estimate the slope of the masking function (% amplitude change/dB) and the intensity of the masker necessary to produce a 50% reduction in probe amplitude (i.e. the masked threshold). The masked thresholds were plotted as a function of masker-probe interval to obtain the forward-masking functions.

Figure 4 shows the pre-exposure evoked-response forward-masking functions obtained at four different frequencies from one representative chinchilla. Note that the masker-probe interval has been plotted on a logarithmic scale; this was done to allow a direct comparison with previously published psychophysical data [Vogten, 1978; Nelson and Turner, 1980; Cudahy, 1982]. The forward-masking functions show the same general trends at all four frequencies; that is, masked thresholds are lowest at short masker-probe intervals and then systematically increase as the time interval between masker and probe increases. The increase in the masked threshold is most noticeable once the masker-probe interval exceeds roughly 20 ms. In order to obtain quantitative estimates of the time course of forward masking, the exponential equation, $Lm = Ae^{(DT/tau)}$, was fit to the data. Lm and DT represent the masker level and the time interval between masker and probe, respectively. The constants A and tau were adjusted to fit the data using a least-squares procedure [Ryan et al., 1980]. The constant A represents the masked threshold at DT=0. The time constant tau determines the rate at which the exponential function rises. The time constants fit to the data in figure 4 ranged from 90 ms at 0.5 kHz to 45 ms at 2 kHz. There was a slight tendency for the time constant to be shorter at the mid-frequencies than at other frequencies; however,

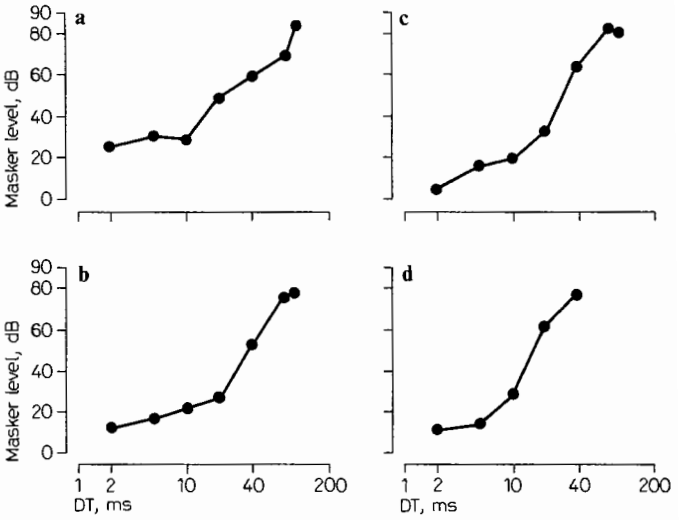


Fig. 4. Typical pre-exposure evoked-response forward-masking functions obtained at 0.5 kHz (**a**; tau 90.09), 2 kHz (**b**; tau 45.55), 4 kHz (**c**; tau 55.87) and 8 kHz (**d**; tau 66.67) from one chinchilla.

this difference was not statistically significant [Arehole, 1986]. Finally, the degree of variance accounted for (R^2) by the model ranged from 0.64 to 0.99 across all 10 subjects. Thus, the model appears to provide a reasonably good fit to the data.

After the exposure, the forward-masking functions were altered in frequency regions where there was a significant hearing loss. This is illustrated in figure 5a–c by comparing the pre- and postexposure forward-masking function for a typical animal. Figure 5a shows the pre- and postexposure forward-masking functions at 2 kHz where the threshold shift was 28 dB. Two important changes can be seen. First, the postexposure forward-masking function is shifted upward due to the hearing loss. Second, the postexposure forward-masking function rises more slowly than the pre-exposure function. Consequently, after the exposure, the masked threshold increased by only 40 dB, when the masker-probe interval increased from 2 to 100 ms, whereas before the exposure the masked threshold increased by 60 dB over the same time interval. This change in the time course of forward masking is reflected as an increase in the time constant. In this case, the time constant increased from approximately 81 to 143 ms. Figure 5b, c shows the time course of forward masking at 0.5 and 4 kHz, where the hearing loss was 5 and 20 dB, respectively. Note that the 4-kHz forward-masking function has been altered, resulting in an increase in the time constant from approximately 63 to 114 ms (fig. 5c). Consequently, when the masker-probe interval increased from 2 to 100 ms, the masked threshold increased by 40 dB in the impaired ear, whereas the masked threshold increased by roughly 75 dB when hearing was normal. A similar increase in the time constant was also seen at 8 kHz where the threshold shift was 27 dB. By contrast, there was essentially no hearing loss at 0.5 kHz and little or no difference be-

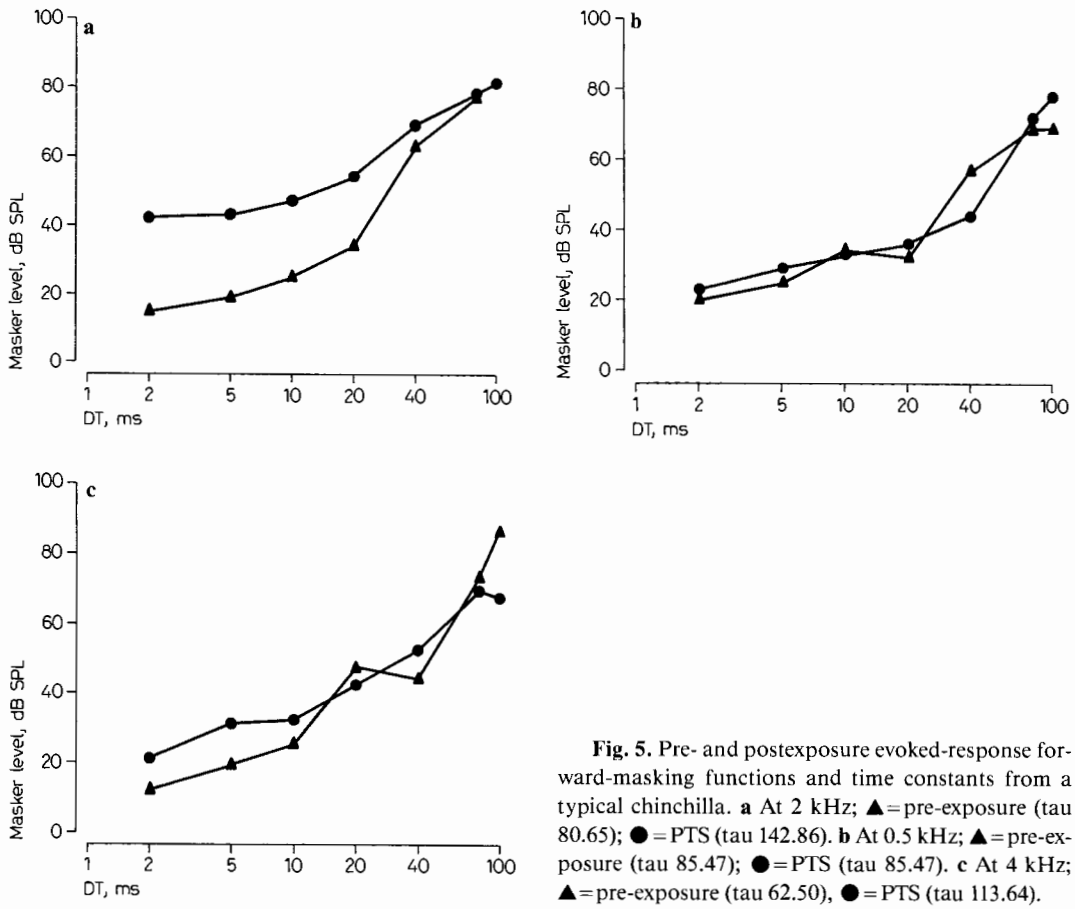


Fig. 5. Pre- and postexposure evoked-response forward-masking functions and time constants from a typical chinchilla. **a** At 2 kHz; ▲ = pre-exposure (tau 80.65); ● = PTS (tau 142.86). **b** At 0.5 kHz; ▲ = pre-exposure (tau 85.47); ● = PTS (tau 85.47). **c** At 4 kHz; ▲ = pre-exposure (tau 62.50), ● = PTS (tau 113.64).

tween the pre- and postexposure forward-masking functions (fig. 5b).

The changes in the forward-masking functions illustrated above were fairly representative of the overall trends in the data. However, the forward-masking time constants in 2 animals also increased at 0.5 kHz. In 1 animal, the increase in the time constant (80–154 ms) was correlated with a 17-dB increase in the threshold at 0.5 kHz; thus, this change is consistent with the increase in tau seen at other frequencies where the threshold was elevated (2–8 kHz). However, in the second animal, the time constant at 0.5 kHz increased by roughly a factor of 2, yet there was no evidence of hearing loss or hair cell loss in the 0.5-kHz region. Thus, the results from 1 animal are not representative of the group.

The trends outlined above can also be seen in the group data shown in table 1. The constant A which represents the masked threshold at DT = 0, increased in proportion to the hearing loss as one would expect. Thus, increases were seen at 2, 4 and 8 kHz where the thresholds were elevated. The time

constant also increased after the exposure. The time constants increased by a factor of 2.3, 2.8 and 1.6 at 2, 4 and 8 kHz, respectively. Note that the increase in the time constant is related to the magnitude of the hearing loss. There was also a slight increase (1.3 ×) in the mean time constant at 0.5 kHz, even though the average threshold shift was negligible here. A statistical analysis revealed that the overall difference between the pre- and postexposure time constants was statistically significant ($p < 0.01$) [Zar, 1974]. An analysis of individual frequencies indicated that the increase in tau was statistically significant at $p < 0.01$ for 2, 4 and 8 kHz, whereas the increase at 0.5 kHz was significant at $p < 0.05$.

As mentioned above, the results from one animal were somewhat anomalous at 0.5 kHz, i.e. there was no evidence of hearing loss or hair cell loss, yet the time constant in this animal had increased substantially. If we treat the results from this one animal as aberrant, and eliminate the data at 0.5 kHz from the comparison, then the pre- and postexposure time constants at 0.5 kHz would not be statistically different.

Growth of Masking

The growth of masking at 4 kHz was compared before and after the exposure by measuring the percent reduction in the probe response per decibel increase in masker level, that is, the slope of the masking function. Before the exposure, the slopes ranged from approximately 3 to 6% per decibel, as shown in figure 6. The slopes tended to decrease with an increasing masker-probe interval up to approximately 20 ms. In order to determine if the hearing loss had altered the slopes of the masking functions, the pre- and postexposure slopes were compared using analysis of variance [Zar, 1974]. As shown in table 2, the pre-and postexposure slopes for the growth of masking were not statistically different from one another at $p < 0.05$.

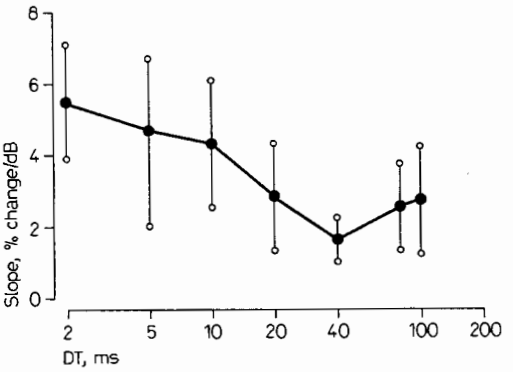


Fig. 6. Mean slope of the pre-exposure masking functions at the seven masker-probe intervals (DT). Vertical bars indicate 1 SD above and below the mean.

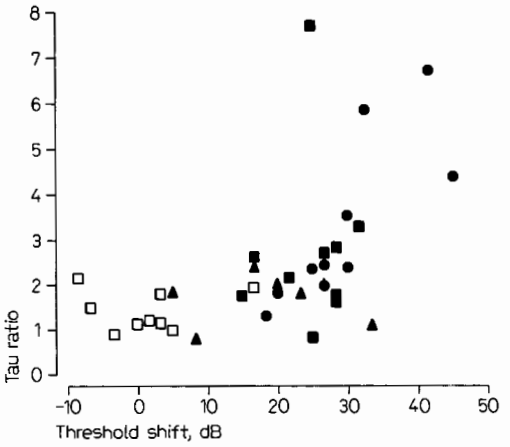


Fig. 7. Tau ratio plotted as a function of threshold shift at each frequency. □ = 0.5 kHz (r = 0.003); ■ = 2 kHz (r = 0.105); ● = 4 kHz (r = 0.807); ▲ = 8 kHz (r = 0.233).

Table 1. Forward-masking parameters (mean ± SD)

	0.5 kHz	2 kHz	4 kHz	8 kHz
Condition: normal				
Tau, MS	89 ±20	55 ±17	52 ±18	68 ±22
A, dB	29 ± 1.2	18 ± 1.7	13 ± 1.9	22 ± 1.4
R^2	0.77 ± 0.08	0.81 ± 0.11	0.82 ± 0.10	0.88 ± 0.08
Condition: PTS				
Tau, MS	120 ±45	127 ±44	141 ±31	107 ±46
A, dB	31 ± 1.2	43 ± 1.0	41 ± 1.2	43 ± 1.2
R^2	0.91 ± 0.07	0.91 ± 0.06	0.91 ± 0.04	0.92 ± 0.06

Table 2. Growth of masking slope analysis. One-way ANOVA comparing normal to PTS masking slopes

DT	Source	Sum of squares	DF	Mean squares	F[1]	Probability $p < 0.05$
2 ms	Group	0.0086	1	0.0086	0.803	4.00
	Error	0.6391	60	0.0107		
5 ms	Group	0.0086	1	0.0086	0.804	3.98
	Error	0.7151	67	0.0107		
10 ms	Group	0.0328	1	0.0328	3.52	4.00
	Error	0.5873	63	0.0093		
20 ms	Group	0.0469	1	0.0469	3.69	4.00
	Error	0.8257	65	0.0127		
40 ms	Group	0.0101	1	0.0101	0.973	3.98
	Error	0.7360	71	0.0104		
80 ms	Group	0.0106	1	0.0106	0.882	4.00
	Error	0.7935	66	0.0120		
100 ms	Group	0.0043	1	0.0043	0.260	4.03
	Error	0.8432	52	0.0162		

[1] F values are not significant.

Hearing Loss and Time Constant Changes

The results presented in figure 5 suggest that there may be a relationship between the change in the time constant and the degree of hearing loss. In order to examine this relationship in more detail, the ratio of the post- to pre-exposure time constant (tau ratio) was computed and plotted as a function of hearing loss at each frequency (fig. 7). As the threshold shift increased from 0 to 25 dB, the tau ratio increased slowly from approximately 1 to 2 indicating that small amounts of hearing loss have little or no effect on the forward-masking time constants. However, once the hearing loss exceeded approximately 25 dB, the tau ratio increased rapidly. In fact, there were several instances where the

time constant increased by more than a factor of 4. At 4 kHz, where the hearing loss was greatest, the increase in the time constant was closely related to the degree of hearing loss, as indicated by the high correlation coefficient (0.81). However, the correlation coefficients were low or negligible at the other three frequencies where the hearing loss was typically less than 25 dB.

Hair Cell Loss

One question that arises from the preceding analysis is whether the change in the tau ratio is linked to the degree of hair cell loss. In order to explore this question, the inner, outer and total hair cell losses were computed over lengths of the cochlea corresponding to octave intervals centered at 0.5, 2, 4 and 8 kHz. The tau ratio was then correlated with the percent loss of outer, inner or combination of inner and outer hair cells at each frequency. The correlation coefficients obtained at all four frequencies, although positive, were low and statistically insignificant. A similar attempt was made to relate the degree of hair cell loss in each octave band to the magnitude of permanent threshold shift. These correlation coefficients were also positive, but low and statistically insignificant.

Discussion

The results of the present study indicate that the time course of the evoked-response forward-masking function can be altered by a noise-induced PTS. In terms of the exponential model used to describe the data, the hearing loss resulted in an increase in the time constant of forward masking. This increase was especially noticeable once the threshold shift exceeded 20 dB. The tendency of the evoked-response time constant to increase with hearing loss is consistent with the results of several recent psychophysical studies. Feston and Plomp [1983] used a fixed-level masker, and reported that the slopes of the forward-masking functions were shallower than normal in subjects with 30–60 dB of hearing loss. Furthermore, the decrease in slope was positively correlated with the degree of hearing loss.

Several aspects of the present study are also interesting to compare to the psychophysical results of Nelson and Turner [1980], since their stimulus paradigm is similar to our own. First, our pre-exposure evoked-response forward-masking time constants are comparable to those obtained from their normal-hearing human listeners. Second, our forward-masking time constants increased with hearing loss, particularly after the hearing loss exceeded 25–30 dB. This increase parallels the changes seen psychophysically [Nelson and Turner, 1980; Cudahy, 1982]. Furthermore, the increases in the psychophysical time constants were roughly in the same order of magnitude as those in our physiological study. In summary, the evoked-response forward-masking data are consistent with similar psychophysical results obtained from human listeners.

Other measures of temporal resolution appear to be affected by sensorineural hearing loss in much the same way as forward masking. For example, backward masking extends over much longer time intervals, and appears to recover more slowly in hearing-impaired subjects than in normals [Elliott, 1975; Cudahy, 1982]. The detection of amplitude-modulated noise also provides a metric of auditory temporal acuity. After

noise exposure, Henderson et al. [1984] reported that the amplitude modulation thresholds of the chinchilla increased at high-modulation frequencies. Gap detection thresholds, another measure of temporal resolution examined in the chinchilla, also increase with hearing loss, but only after the loss exceeds approximately 30 dB [Giraudi-Perry et al., 1982]. Masking-period histograms have also been used to estimate temporal resolution in hearing-impaired listeners. Ludvigsen [1985] reported an approximately linear relationship between the deterioration in temporal resolution and the degree of hearing loss for subjects with 30–60 dB of threshold shift.

In contrast to the preceding studies, several investigators have reported normal temporal resolution in hearing-impaired subjects. Jesteadt et al. [1976], for example, used the discrimination of Huffman sequences as an index of auditory temporal resolution. The majority of hearing-impaired subjects exhibited normal or better than normal temporal resolution; only 2 of 10 subjects demonstrated an impaired ability to discriminate waveform shape, and this did not appear to be related to the degree of hearing loss. McFadden et al. [1984] examined forward-masking functions in listeners with temporary threshold shift induced by high doses of salicylates. Little or no change in the time course of forward masking or gap detection was reported. One interpretation of these results is that salicylates may not interfere with temporal resolution. However, it is important to note that the hearing loss resulting from salicylate intoxication was 17 dB or less; consequently, the loss may have been less than that needed to cause a significant change in temporal resolution.

Growth of Masking

Jesteadt [1980] has argued that the prolonged time course of forward masking seen in hearing-impaired subjects may be due to the abnormally rapid growth of masking rather than to any inherent change in temporal processing. That is, the effectiveness of the masker presumably increases at a faster than normal rate in impaired listeners as the masker intensity increases, resulting in an increase in the time constants fit to the data. This argument is appealing because loudness recruitment is a common symptom in hearing-impaired listeners; however, it may not be an entirely adequate explanation for several reasons. First, in many gap detection studies, signals of equal intensity are presented on both sides of the gap, therefore, both signals are subjected to the same 'recruitment-like' effect. Thus, if temporal resolution were truly unaffected by hearing loss, one would predict that gap detection thresholds in hearing-impaired listeners would be normal. In general, this does not appear to be the case. Most studies indicate that subjects with threshold shifts greater than 30 dB have gap detection thresholds which are longer than normal, when the results are compared at equivalent sensation levels [Giraudi-Perry et al., 1982; Fitzgibbons and Wightman, 1982]. The results of Giraudi-Perry et al. are particularly important, because they obtained gap detection thresholds from the same animal before and after the hearing loss, but were unable to obtain normal gap thresholds at high signal levels once the hearing loss exceeded 25–30 dB. Buus and Florentine [1985] have found similar results in 3 of 7 hearing-impaired subjects. However, their 4 remaining subjects had gap thresholds within normal limits at high stimulus intensities. Thus,

most gap detection studies indicate that there is a loss of temporal acuity in hearing-impaired listeners. It is also important to note that the slopes of the evoked-response masking functions failed to increase after the hearing loss as one might expect from Jesteadt's argument[1980].

Physiological Results

The alterations in the time course of forward masking seen in the present study are based on recordings from the inferior colliculus; thus, there is some question as to whether the effect originates at the colliculus or more peripherally. Some insights into this issue may be gained from single-unit recordings from the auditory nerve. Smith [1977] studied the degree to which the firing rate to a probe tone was depressed by a forward masker. While this study focused on the results from normal animals, Smith indicated that the recovery from short-term adaptation was prolonged in animals in poor physiological condition. Evans has also stated that the recovery from short-term adaptation in single auditory nerve fibers may be prolonged in kanamycin-treated guinea pigs [see discussion following Nelson and Turner, 1980]. Recently, we obtained data from a large sample of auditory nerve fibers in noise-exposed chinchillas (40–60 dB of threshold shift) using a forward-masking stimulus paradigm [Salvi et al., 1986]. The single-unit forward-masking time constants measured in the noise-exposed animals were longer than normal. To determine if this increase in the time constant was due to an abnormally high firing rate of the masker, the rate-intensity functions of the masker discharge in the noise-exposed animals were compared to those obtained from normals. No differences were seen between the rate-intensity functions obtained from units in normal and noise-exposed ears in agreement with earlier reports [Kiang et al., 1970; Dallos and Harris, 1978; Salvi et al., 1983a]. Thus, the prolonged time constants seen in the present study may be due in part to physiological changes occurring in the cochlea.

The auditory nerve compound action potential data of Gorga and Abbas [1981] appear to conflict with the preceding findings. They employed a forward-masking paradigm, and studied the time course of recovery of the action potential in a group of normal and noise-exposed animals. In contrast to the present study, they found that the forward-masking time constants from noise-exposed animals were within normal limits. However, they noted that growth of masking of the action potential was abnormally rapid in the noise-exposed animals. This abnormally rapid growth is consistent with the abnormally steep rate-intensity functions of discharge seen in some, but not all, hearing-impaired animals [Evans, 1974; Schmiedt et al., 1980; Schmiedt and Zwislocki, 1980; Harrison, 1981]. Thus, these results appear to differ from those obtained in the chinchilla.

It is unclear why our results differ from those of Gorga and Abbas [1981]. One possibility is that there may be important differences in the type of cochlear damage, which in turn may lead to different physiological effects, as noted previously [Liberman and Kiang, 1976; Liberman and Mulroy, 1982; Liberman et al., 1986; Schmiedt et al., 1980; Schmiedt and Zwislocki, 1980]. Unfortunately, we cannot evaluate this possibility, since Gorga and Abbas did not obtain histological data from their animals. Even if such data were available, it is unlikely that the dis-

crepancy between the two studies could be resolved due to the poor correlation between forward masking and hair cell loss data in our study. Another factor that needs to be considered in comparing these results is the type of experimental design. Gorga and Abbas compared the forward-masking time constants from their noise-exposed animals with those from control ears. Our comparisons, on the other hand, were made within the same animal thereby eliminating between-subject variability. Thus, our experimental design may be more sensitive to detect changes in temporal processing. Finally, one must consider other factors such as differences in the recording site, type of noise exposure and species which could potentially lead to differences between our results and those of Gorga and Abbas [1981].

Intensity Effects

Since the probe intensity was maintained at a level 10 dB above threshold, the absolute intensity level of the probe would be much higer – in the region of hearing loss – after the exposure than before. Consequently, if the forward-masking time constants were to increase with probe SPL, this could lead to an increase in the time constants in the region of hearing loss. Several lines of evidence argue against this. First, psychophysical forward-masking functions from normal listeners show little or no change with increasing probe intensity [Nelson and Turner, 1980; Cudahy, 1982]. Second, forward-masking time constants of action potential do not appear to change when probe intensity increases [Gorga and Abbas, 1981]. Similarly, pilot studies using the evoked response from the inferior colliculus indicate no significant change in the time constant, when the probe intensity was increased from 10 to 25 dB above threshold. Thus, it is difficult to attribute the increase in the forward-masking time constant to an increase in the absolute intensity of the probe tone.

Hair Cell Loss

The 2-kHz exposure produced the maximum amount of hair cell loss in the 2- to 8-kHz region of the cochlea which corresponds to the frequencies with the greatest hearing loss (fig. 1). The average hair cell loss in this frequency region, however, was relatively small, amounting to less than 8% of the inner and less than 30% of the outer hair cells. While there were positive correlations between the degree of hearing loss and hair cell loss, the relationships were weak and statistically insignificant. Positive correlations were also found between the increase in forward-masking time constants and the degree of hair cell loss, but, again, the relationships were weak and statistically insignificant. Other investigators have also reported poor correlations between noise-induced threshold shift and degree of hair cell loss [Lindquist et al., 1954; Hunter-Duvar and Elliott, 1972; Schuknecht, 1974]. We have also seen single-unit and behavioral threshold shifts near 2 kHz, but found little or no evidence of hair cell loss in the corresponding region of the cochlea [Salvi et al., 1982b]. More recently, Liberman and Kiang [1976] and Liberman et al. [1982, 1986] reported significant threshold shifts in single auditory nerve fibers innervating regions of the cochlea with minimal hair cell loss. Careful examination of the hair cells by light and electron microscopy revealed a variety of stereocilium defects ranging from clumped stereocilia to missing cilia and frac-

tures in the cilia rootlets near the cuticular plate; these defects appeared to be correlated with the loss in sensitivity. Thus, the poor correlation between hair cell loss and the functional measures used in this study may be due to subtle defects of sensory cells that cannot be adequately evaluated using the conventional surface preparation technique. One possible method for improving upon these correlations in the future would be to embed the cochleas in plastic and to evaluate the condition of the cilia along the entire length of the cochlea as Liberman and Mulroy [1982] have done. However, a light-microscopic analysis of the cilia would not be suitable for detecting fractures in the cilium rootlets [Liberman et al., 1986].

Mechanisms

The mechanical properties of the basilar membrane in pathological ears could also potentially account for abnormal auditory temporal processing. For example, if the basilar membrane exhibited more ringing in pathological ears, it would be more difficult to resolve the silent interval between masker and probe. Unfortunately, the opposite effects have been seen in experimental studies. Specifically, there is less ringing in the mechanical response of the basilar membrane [Robles et al., 1976] and less ringing in the neural response of single auditory nerve fibers [Salvi et al., 1980].

Hair cell stereocilia have traditionally been viewed as static structures; however, recent studies have indicated that the stiffness of stereocilia can change dynamically. Saunders et al. [1986], for example, showed that the stiffness of isolated inner and outer hair cell stereocilia progressively decreased over a 10-min period of overstimulation with a water jet. During the recovery period, the stereocilia stiffness gradually increased to normal values. Thus, Saunders et al. have suggested that the change in stereocilium stiffness could play a role in the recovery from temporary threshold shift. One might take this reasoning a step further and envision a change in stereocilium stiffness contributing to the recovery from short-term adaptation in normal and pathological ears. A serious limitation of such a mechanism, however, is that the time course of the observed change in stiffness is longer than the recovery from short-term adaptation.

Acute studies of acoustic trauma have also provided some interesting data on the relationship between structure and function. Liberman and Mulroy [1983] have reported a relationship between threshold shifts of auditory nerve fibers and profuse vacuolizations in the neuropil beneath inner hair cells. Since short-term adaptation is thought to reflect synaptic processes between hair cells and auditory nerve fibers [Furukawa and Matsuura, 1978], the pathologies seen in the neuropil beneath the inner hair cells could provide an anatomical substrate for the prolonged forward-masking functions seen in the present study.

In summary, noise-induced hearing loss appears to alter the time course of the evoked-response forward-masking functions, particularly in subjects in which the hearing loss exceeds 20–25 dB. Thus, the present results from the inferior colliculus are consistent with the psychophysical results seen in hearing-impaired listeners. Recent single-unit data from our laboratory [Salvi et al., 1986] suggest that the present results could be due, at least in part, to physiological changes occurring at the periphery.

Acknowledgements

This work was partially supported by the following grants: NIH R01NS16761 and R01 NS23894 and NIOSH 5R01OH1152. We wish to thank K. Rockwood and G. Appleby for their generous donation of chinchillas.

Fonctions de masquage antérieur des réponses évoquées auditives chez le chinchilla présentant une perte auditive permanente après exposition au bruit

Les fonctions de masquage antérieur des réponses évoquées auditives furent mesurées par des électrodes chroniques implantées dans le colliculus du chinchilla avant et après exposition à un son pur intense entraînant une perte auditive permanente. Avant l'exposition, les constantes de temps du masquage antérieur étaient de 50 à 90 ms. Après l'exposition, les constantes de temps augmentèrent d'une façon significative dans la région de la perte auditive, mais non pas dans les régions où l'audition était normale. L'effet de la perte auditive sur les constantes de temps du masquage antérieur était maximum quand la perte auditive dépassait 20–25 dB. Ces changements physiologiques dans la fonction de masquage antérieur des réponses évoquées auditives semblent comparables à ceux que l'on observe en psychophysique chez l'homme.

References

Abbas, P.J.; Gorga, M.P.: AP responses in forward-masking paradigms and their relationship to responses of auditory-nerve fibers. J. acoust. Soc. Am. *69:* 492–499 (1981).

Arehole, S.: Evoked response 'forward masking' functions in normal and hearing-impaired chinchillas; PhD diss., University of Texas at Dallas, Dallas (1986).

Blakeslee, E.A.; Hynson, K., Hamernik, R.P.; Henderson, D.: Asymptotic threshold shift in chinchillas exposed to impulse noise. J. acoust. Soc. Am. *63:* 876–882 (1978).

Boothroyd, A.: Detection of temporal gaps by deaf and hearing subjects; SARP No. 12, Clarke School for the Deaf, Northampton, Mass. (1973).

Buus, S., Florentine, M.: Gap detection in normal and impaired listeners: the effect of level and frequency; in Michelsen, Time resolution in auditory systems (Springer, Berlin 1985).

Cudahy, E.: Changes in the temporal processing of acoustic signals in hearing-impaired listeners; in Hamernik, Henderson, Salvi, New perspectives on noise-induced hearing loss (Raven Press, New York 1982).

Cudahy, E.A.; Elliott, L.L.: Temporal masking patterns for hearing-impaired and normal listeners. J. acoust. Soc. Am *59:* S2 (1976).

Dallos, P.; Harris, D.: Properties of auditory nerve responses in the absence of outer hair cells. J. Neurophysiol. *41:* 365–383 (1978).

Davis, H.; Morgan, C.T.; Hawkins, J.E.; Galambos, R.; Smith, F.W.: Temporary deafness following exposure to loud tones and noise. Acta oto-lar., suppl. 88, pp. 1–57 (1950).

Dreschler, W.A.; Plomp, R.: Relations between psychophysical data and speech perception in hearing-impaired subjects. Part II. J. acoust. Soc. Am *78:* 1261–1270 (1985).

Eldredge, D.H.; Miller, J.D.; Bohne, B.A.: A frequency-position map for the chinchilla cochlea. J. acoust. Soc. Am. *69:* 1091–1095 (1981).

Elliott, L.L.: Temporal and masking phenomena in persons with sensorineural hearing loss. Audiology *14:* 336–353 (1975).

Engström, H.; Ades, H.W.; Anderson, A.: Structural pattern of the organ of Corti (Almqvist and Wiksell, Stockholm 1966).

Evans, E.F.: Auditory frequency selectivity and the cochlear nerve; in Zwicker, Terhardt, Facts and models in hearing (Springer, New York 1974).

Feston, J.M.; Plomp, R.: Relations between auditory functions in impaired hearing. J. acoust. Soc. Am. *73:* 652–662 (1983).

Fitzgibbons, P.J.; Wightman, F.L.: Temporal resolution in normal and hearing-impaired listeners. J. acoust. Soc. Am. *72:* 1387–1393 (1982).

Furukawa, T.; Matsuura, S.: Adaptive rundown of excitatory postsynaptic potentials at synapses between hair cells and eighth nerve fibres in the goldfish. J. Physiol. *276:* 193–209 (1978).

Giraudi, D.M.; Salvi, R.J.; Henderson, D.; Hamernik, R.P.: Gap detection by the chinchilla. J. acoust. Soc. Am. *68:* 802–806 (1980).

Giraudi-Perry, D.M.; Salvi, R.J.; Henderson, D.: Gap detection in hearing-impaired chinchillas. J. acoust. Soc. Am. *72:* 1387–1393 (1982).

Gorga, M.P.; Abbas, P.J.: AP measurements of short term adaptation in normal and in acoustically traumatized ears. J. acoust. Soc. Am. *70:* 1310-1321 (1981).

Harris, D.M.; Dallos, P.: Forward-masking of auditory nerve fiber responses. J. Neurophysiol. *42:* 1036–1044 (1979).

Harrison, R.V.: Rate versus intensity functions and related AP responses in normal and pathological guinea-pig and human cochlea. J. acoust. Soc. Am. *70:* 1036–1044 (1981).

Henderson, D.; Hamernik, R.P.; Woodford, C.; Sitler, R.W.; Salvi, R.: Evoked-response audibility curve of the chinchilla. J. acoust. Soc. Am. *54:* 1099–1101 (1973).

Henderson, D.M.; Salvi, R.; Pavek, G.; Hamernik, R.: Amplitude modulation thresholds in chinchillas with high-frequency hearing loss. J. acoust. Soc. Am. *75:* 1177–1183 (1984).

Hood, J.D.: Studies in auditory fatigue and adaptation. Acta oto-lar. *92:* 1–57 (1950).

Irwin, R.J.; Hinchcliff, L.K.; Kemp, S.: Temporal acuity in normal and hearing-impaired listeners. Audiology *20:* 234–243 (1981).

Hunter-Duvar, F.M.; Elliot, D.N.: Effects of intense auditory stimulation: hearing losses and inner ear changes in squirrel monkey. J. acoust. Soc. Am. *52:* 181–1192 (1972).

Jesteadt, W.: Effect of masker level and signal delay on forward masking in normal and impaired listeners. J. acoust. Soc. Am. *68:* S28 (1980).

Jesteadt, W.; Bilger; R.C.; Green, D.M.; Patterson, J.H.: Temporal acuity in listeners with sensorineural hearing loss. J. Speech and Hearing Res. *19:* 357–370 (1976).

Kiang, N.Y.S.; Moxon, E.C.; Levine, R.A.: Auditory-nerve activity in cats with normal and abnormal cochleas; in Wolstenholme, Knight, Sensorineural hearing loss (Churchill, London 1970).

Liberman, M.C.; Dodds, L.W.; Learson, D.A.: Structure-function correlation in noise-damaged ears: a light- and electron-microscopic study; in Salvi, Hamernik, Henderson, Colletti, Basic and applied aspects of noise-induced hearing loss (Plenum Press, New York 1986).

Liberman, M.C.; Kiang, N.Y.S.: Acoustic trauma in cats: cochlear pathology and auditory-nerve activity. Acta oto-lar., suppl. 358, pp. 1–63 (1976).

Liberman, M.C.; Mulroy, M.J.: Acute and chronic effects of acoustic trauma: cochlear pathology and auditory nerve pathophysiology; in Hamernik, Henderson, Salvi, New perspectives on noise-induced hearing loss (Raven Press, New York 1983).

Lindquist, S.E.; Neff, W.D.; Schuknecht, N.F.: Stimulation deafness: a study of hearing losses resulting from exposure to noise of blast impulses. J. comp. physiol. Psychol. *47:* 406 (1954).

Ludvigsen, C.: Relations among some psychoacoustic parameters in normal and cochlearly impaired listeners. J. acoust. Soc. Am. *78:* 1271–1280 (1985).

McFadden, D.; Plattsmier, H.S.; Pasanen, E.G.: Aspirin-induced hearing loss as a model of sensori-neural hearing loss. Hear. Res. *16:* 251–260 (1984).

Nelson, D.; Turner C.W.: Decay of masking and frequency resolution in sensorineural hearing impaired listeners; in Van den Brink, Bilsen, Psychophysical, physiological and behavioral studies in hearing (Delft University Press, Delft 1980).

Plomp, R.: Rate of decay of auditory sensation. J. acoust. Soc. Am. *36:* 277–282 (1964).

Robles, L.; Rhode, W.S.; Geisler, C.D.: Transient response of the basilar membrane measured in squirrel monkeys using the Mössbauer effect. J. acoust. Soc. Am. *59:* 926–939 (1976).

Ryan, A.; Joiner, B.L.; Ryan, B.F.: Minitab reference manual (Duxbury Press, North Scituate 1980).

Salvi, R.J.; Ahroon, W.A.; Perry, J.W.; Gunnarson, M.S.; Henderson, D.: Comparison of psychophysical and evoked-potential tuning curves in the chinchilla. Am. J. Otolaryngol. *3:* 408–416 (1982a).

Salvi, R.J.; Hamernik, R.P.; Henderson, D.: Response patterns of auditory nerve fibers during temporary threshold shift. Hearing Res. *10:* 37–67 (1983a).

Salvi, R.; Henderson, D.; Hamernik, R.P.; Parkins, C.: VIII nerve response to click stimulation in normal and pathological cochleas. Hearing Res. *2:* 335–342 (1980).

Salvi, R.J.; Perry, J.; Hamernik, R.P.; Henderson, D.: Relationships between cochlear pathologies and auditory nerve and behavioral responses following acoustic trauma; in Hamernik, Henderson, Salvi, New perspectives on noise-induced hearing loss (Raven Press, New York 1982b).

Salvi, R.J.; Saunders, S.S.; Ahroon, W.A.; Shivapuja, B.G.; Arehole, S.: Psychophysical and physiological aspects of auditory temporal processing; in Salvi, Hamernik, Henderson, Colletti, Basic and ap-

plied aspects of noise-induced hearing loss (Plenum Press, New York 1986).

Saunders, J.C.; Canton, B.; Flock, Å.: Mechanical changes in stereocilia following overstimulation: Observations and possible mechanisms; in Salvi, Hamernik, Henderson, Colletti, Basic and applied aspects of noise-induced hearing loss (Plenum Press, New York 1986).

Schmiedt, R.A.; Zwisłocki, J.J.: Effects of hair cell lesions on responses of cochlear nerve fibers. II. Single- and two-tone intensity functions in relation to tuning curves. J. Neurophysiol. *43:* 1390–1405 (1980).

Schmiedt, R.A.; Zwisłocki, J.J.; Hamernik, R.P.: Effects of hair cell lesions on responses of cochlear nerve fibers. I. Lesions, tuning curves, two-tone inhibition, and responses to trapezoidal wave patterns. J. Neurophysiol. *43:* 1367–1389 (1980).

Schucknecht, H.F.: Pathology of the ear (Harvard University Press, Cambridge 1974).

Smiarowski, R.A.; Carhart, R.: Relations among temporal resolution, forward masking, and simultaneous masking. J. acoust. Soc. Am. *57:* 1169–1174 (1975).

Smith, R.: Short-term adaptation in single auditory nerve fibers: some poststimulatory effects. J. Neurophysiol. *40:* 1098–1112 (1977).

Trinder, E.: Auditory fusion: a critical interval test with implications in differential diagnosis. Br. J. Audiol. *13:* 143–147 (1979).

Tyler, R.S.: The relationship between speech perception and psychoacoustical measurements in noise-induced hearing loss subjects; in Salvi, Hamernik, Henderson, Colletti, Basic and applied aspects of noise-induced hearing loss (Plenum Press, New York 1986).

Tyler, R.S.; Summerfield, A.Q.; Wood, E.J.; Fernandes, M.A.: Psychoacoustic and phonetic temporal processing in normal and hearing-impaired listeners. J. acoust. Soc. Am. *72:* 740–752 (1982).

Vogten, L.L.M.: Low-level pure-tone masking: a comparison of 'tuning curves' obtained with simultaneous and forward masking. J. acoust. Soc. Am. *63:* 1520–1527 (1978).

Zar, J.H.: Biostatistical analysis (Prentice Hall, Englewood Cliffs 1974).

Received: January 23, 1987
Accepted: September 27, 1988

Richard J. Salvi, PhD
Hearing Research Laboratory
215 Parker Hall
SUNY University at Buffalo
Buffalo, NY 14215 (USA)