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Possible origins of the non-monotonic intensity discrimination function in forward masking

Fan-Gang Zeng *, Robert V. Shannon

House Ear Institute, 2100 West Third Street, Los Angeles, CA 90057, USA

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Abstract

A non-monotonic intensity discrimination function in forward masking has been recently reported [Zeng et al. (1991) Hear. Res. 55, 223–230; Zeng and Turner (1992) J. Acoust Soc. Am. 92, 782–787] in which just-noticeable-differences (jnds) in intensity are largest for midlevel tones and smaller for soft and loud tones following an intense narrow-band noise. One hypothesis was that this midlevel hump reflects the contribution of low-spontaneous rate (SR) neurons to intensity coding, based on the differential recovery from forward masking of low-SR and high-SR neurons [Relkin and Doucet (1991) Hear. Res. 55, 215–222]. The present study conducted three experiments stimulating different stages of the auditory system in an attempt to determine the peripheral and central origins of the midlevel hump. First, in two cochlear implant (CI) listeners, the forward masker produced a midlevel hump on the intensity discrimination function, suggesting that the synapses between the hair cell and the eighth nerve are probably not responsible for the hump, as they are bypassed and the eighth nerve is stimulated directly. Second, in auditory brainstem implant (ABI) listeners, the forward masker produced no midlevel hump, but the masked jnds were larger than those without a masker. The absence of the midlevel hump in the ABI listeners suggests that the occurrence of the hump requires physiological mechanisms in the auditory nerve transmission, or the intrinsic processing circuits of the cochlear nuclei, or both. Third, in normal-hearing listeners, an ipsilateral, 90 dB SPL, pure-tone forward masker produced a midlevel hump, which is similar to that using a narrow-band noise masker; whereas a contralateral forward masker produced essentially no midlevel hump, suggesting that binaural interactions at superior olivary complex and more central sites are probably not responsible.

Keywords: Intensity discrimination; Forward masking; Adaptation; Electric stimulation; Cochlear implant; Auditory brainstem implant; Contralateral stimulation

1. Introduction

A non-monotonic intensity discrimination function has been recently observed under conditions of forward masking (Zeng et al., 1991; Zeng and Turner, 1992). For 25-ms tones, the jnds in intensity are largest for midlevel tones and smaller for soft and loud tones following an intense narrow-band noise. This 'midlevel hump' effect has also been observed independently in other laboratories (Plack and Viemeister, 1992a; Carlyon and Beveridge, 1993). Based on a physiological finding that low-spontaneous rate (SR) neurons recover slower than high-SR neurons from forward masking (Relkin and Doucet, 1991), Zeng et al. suggested that this midlevel hump may reflect the reduced contribution of low-SR neurons to intensity discrimination. However, the specific physiological mechanisms are not clear (see the discussion of Zeng and Turner, 1992). Plack and Viemeister (1992a, b) reported that a notched-noise reduces the midlevel hump effect in forward masking and that backward masking also produces a midlevel hump. These results led Plack and Viemeister to suggest that the midlevel hump is of central origin and not determined by physiological process at the level of the auditory nerve.

The present study attempts to address the peripheral vs. central issue by taking advantage of the electric stimulation of hearing in both cochlea and cochlear nucleus. Our assumption is that the auditory system can be approximately represented by a series of se-

* Corresponding author. Fax: (213) 413-0950 Email: zeng@hei.org

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quential processing stages consisting of cochlea, hair-cell and nerve synapse, auditory-nerve transmission, cochlear nucleus, superior olivary complex, and other higher auditory centers. Our research strategy is to stimulate different stages of the auditory system and observe the effects of these different processing stages on the midlevel jnd hump. We will first review and discuss the possible role of each physiological site or processing stage in forward masking. We will then describe an experimental design using implant listeners and contralateral sound in normal-hearing listeners to deduce the possible sites at which the midlevel hump may occur.

Psychophysically, forward masking usually refers to shifted threshold and poor suprathreshold discrimination due to a prior sound stimulation. Depending on the level and the frequency of a specific forward masker and the delay between the masker and the following probe stimulus, forward masking can produce increased thresholds (e.g., Zwislocki et al., 1959), decreased thresholds (e.g., Rubin, 1960), enhanced loudness (e.g., Irwin and Zwislocki, 1971; Galambos et al., 1972), and shifted pitch (Rakowski and Jaroszewski, 1974). Many physiological mechanisms have been proposed to explain these psychophysical effects; the masker could change the firing rate and its variance to signal, or alter the signal excitation pattern. Recent physiological and psychophysical studies suggest that the mechanisms responsible for forward masking may be widely distributed in various stages of the auditory system. Let us review evidence from three physiological sites that may be involved in forward masking: (1) the synapse between hair cell and auditory nerve, (2) the auditory-nerve transmission process, and (3) the central auditory system from cochlear nucleus to cortex.

First of all, forward masking may be related to adaptation occurring in the hair cell and eighth-nerve synapse. In response to a continuous test tone, single auditory neurons have been shown to produce a reduced firing rate over time (Kiang et al., 1965; Smith and Zwislocki, 1975), an effect termed 'adaptation'. Although it is still controversial (Mulroy et al., 1974; Howard and Hudspeth, 1987), the cause of the adaptation has been generally assumed due to the generation and depletion of neural transmitters at the synapse (e.g., Schroeder and Hall, 1974; Ross, 1982; Schwid and Geisler, 1982; Smith and Brachman, 1982; Meddis, 1986). On the other hand, recent studies showed a more complicated patterns for the recovery from prior stimulation: (1) high-SR neurons recover faster than low-SR neurons (Rhode and Smith, 1985; Relkin and Doucet, 1991), (2) prior stimulation also produces a different effect on the rate-intensity functions of low-SR and high-SR neurons (Abbas, 1979; Relkin and Solessio, 1993). It is not yet known whether the differential effects of prior stimulation on low- and high-SR neurons are due to different synaptic structures or other mechanisms.

Second, another possible physiological mechanism of forward masking may be inherent in the auditory-nerve transmission process as evidenced by results from the direct electric stimulation of the auditory nerve. Recent studies have shown some adaptation with electric stimulation, at least in a portion of recorded single nerve fibers (Javel et al., 1987; van den Honert and Stypulkowski, 1987; Javel, 1990; Dynes and Delgutte, 1992). Killian et al. (1992) also showed that the electrically-evoked compound action potential in guinea pig has a similar recovery to the forward masking recovery in human cochlear implant subjects. Perceptually, Shannon (1990) found that, once loudness is equated, cochlear implant listeners recover from forward masking with a time course similar to normal-hearing listeners. Thus the psychophysical forward masking cannot be solely due to synaptic adaptation because the hair cell and auditory-nerve synapse is bypassed in cochlear implants.

Third, the central auditory system may also play a role in forward masking as evidenced by psychophysical studies in electric and acoustic stimulation. In electric stimulation of human cochlear nucleus, Shannon and Otto (1990) found that the recovery function in forward masking is similar to that in acoustic stimulation. In acoustic stimulation, a preceding sound presented in the contralateral ear has been shown to affect the loudness perception in the ipsilateral ear (Galambos et al., 1972; Elmasian et al., 1980). This loudness effect suggests a central involvement in the recovery from forward masking because contralateral sound stimulation presumably either introduces central interactions occurring at superior olivary colliculus and more central sites (e.g., Zwislocki, 1971) or affects the ipsilateral cochlea directly through efferent innervation (e.g., Warren and Liberman, 1989).

The above review suggests that physiological mechanisms at both peripheral and central sites could contribute to forward masking. Although Zeng et al. (1991) hypothesized that the differential recovery between low- and high-SR neurons is responsible for the non-monotonic intensity discrimination function in forward masking, their study could not rule out the involvement of other physiological sites. In the present study, we will describe three experiments designed to test three hypotheses that the physiological site is at the hair cell synapse, in the auditory nerve transmission, and of the central origin. The hypothesis of the synaptic origin will be assessed using cochlear implant (CI) listeners, in which the hair-cell synapse is bypassed and the auditory nerve is directly stimulated. The auditory-nerve transmission hypothesis will be assessed using auditory brainstem implant (ABI) listeners, in which the auditory nerve is bypassed and the cochlear nu-

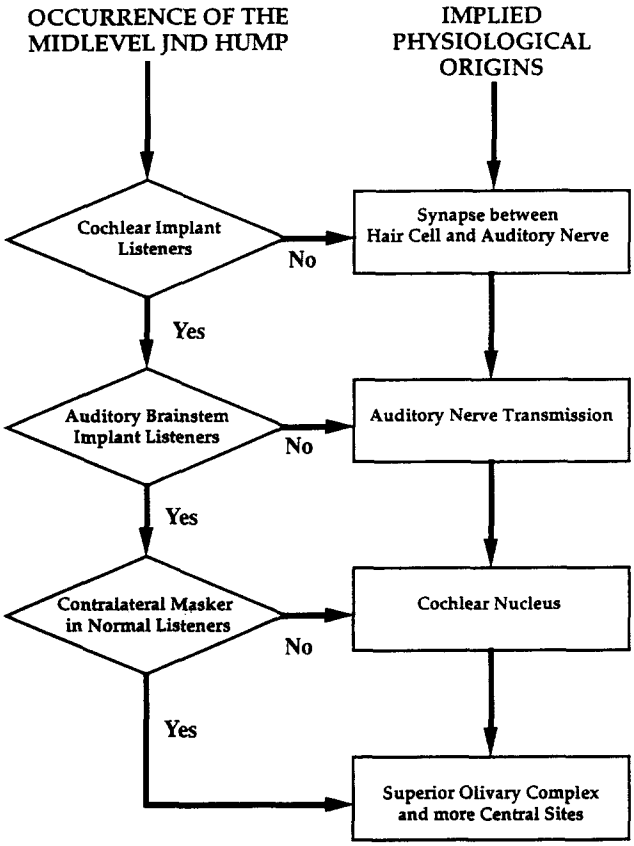

Fig. 1. Logic flow chart of the experimental design in the present study. See text for detailed description.

cleus is directly stimulated. The central origin hypothesis will be assessed using contralateral stimulus in normal-hearing listeners, in which physiological interactions occur at the superior olivary complex and higher centers.

Two assumptions are made in the present study. One assumption is that the auditory system can be viewed as a cascade model and the deduction of the origins can be simplified as a bifurcated process. A second assumption is that the abnormal excitation pattern produced by electric stimulation in one stage does not affect the processing of intensity discrimination for the following stages. Fig. 1 demonstrates this reasoning process. The first decision node is based upon data from cochlear implant listeners. If CI listeners do not show a midlevel hump on the forward-masked intensity discrimination function, then the hair-cell and auditory-nerve synapse would be implicated as the physiological site responsible. This argument is based upon the difference between the CI and normal-hearing listeners: there is no hair-cell and auditory-nerve synapse present in the CI listeners. However, if CI listeners do show the midlevel hump, then we move to the second decision node which is based upon data from the ABI listeners. If the midlevel hump disappears in the ABI listeners, then the auditory nerve transmission and the intrinsic processing circuits in the cochlear nucleus are implicated because the main differences between the ABI and CI listeners are the missing auditory nerve and the nonspecific stimulation of the cochlear nucleus. If the midlevel hump is present in the ABI listeners, we then move to the third decision node which is based upon data from normal-hearing listeners with a contralateral forward masker. If the contralateral masker does not produce the midlevel hump in normal-hearing listeners, then the cochlear nucleus is implicated as the responsible site. Whereas if it does, then superior olivary colliculus and more central sites are candidate sites producing the midlevel hump.

2. Methods

Stimulus temporal paradigm was the same as in the previous studies (Zeng et al., 1991; Zeng and Turner, 1992). In contrast to the narrow-band masker used previously, the masker in the present study was a 1000-Hz sinusoidal stimulus for both electric and acoustic stimulation. The masker duration was 100 ms. The signal was a 25-ms, 1000-Hz sinusoidal stimulus. All stimuli had a 2.5-ms cosine ramp. The signal delay, as defined from the offset of the masker to the onset of the signal was 100 ms unless otherwise noted. All stimuli were generated digitally and output by a 16-bit D/A converter at a sampling rate of 20 kHz (TDT QDA2). The output stimuli were smoothed by an anti-aliasing filter (TDT FLT3) with a cut-off frequency of 8 kHz. The standard level and the signal level were separately controlled by two programmable attenuators (TDT PA3).

2.1 Electric stimulation in implant listeners

Two Richards Ineraid cochlear implant (Eddington et al., 1978) and six auditory brainstem implant (Eisenberg et al., 1987; Brackmann et al., 1993) listeners participated in the experiments. The brainstem implant subjects were patients with Neurofibromotasis Type 2, a decease characterized by the presence of bilateral acoustic tumors. They were one male and one female CI listeners, and one male and five female ABI listeners, aged 25 to 60 years old. All but two ABI subjects use their implants regularly. These two ABI subjects do not use the implants because they still had acoustic hearing in their contralateral ear at the time of testing. All subjects had previous experience in psychophysical experiments.

Only ipsilateral forward masking was performed in implant listeners. The forward masker was a 1000-Hz sinusoidal current-controlled stimulus and the masker was presented at about the 80% level of the subject's dynamic range in dB. The dynamic range was deter-

mined by the uncomfortable loud level minus the threshold in dB. The highest standard level used in each subject was 2 to 3 dB lower than the uncomfortable level of that subject. All levels were presented in dB re: 1 microamp. Electric stimulation was delivered through an optically-isolated constant-current source (Vurek et al., 1981). Stimulation mode was monopolar in all subjects. Detailed calibration of the electric stimulation has been described elsewhere (Shannon and Otto, 1990; Zeng and Shannon, 1992).

2.2. *Acoustic stimulation in normal-hearing listeners*

Four young, normal-hearing listeners (including the first author) served as subjects. They were two males and two females, aged 25 to 35 years old. All subjects had previous experience in psychophysical experiments.

A 1000-Hz sinusoidal stimulus was used as a forward masker in the present study. The 1000-Hz pure-tone forward masker was presented at 90 dB SPL in either the ipsilateral ear or the contralateral ear. The onset of the signal was 100 ms after the offset of the masker. All levels were expressed as values measured with TDH-49 headphones (MX41/AR cushion) in a NBS-9A coupler. The generation and the control of the stimuli were the same as in electrical stimulation, except that the final output was connected to a Crown D-75 amplifier.

2.3. *Procedure*

All subjects except the totally-deaf implant listeners were seated in a double-walled sound chamber (IAC). A 3-down, 1-up, 2IFC, adaptive procedure, which tracks the 79.4%-level of correct responses, was used in all experiments. Subjects received trial-by-trial feedback regarding the correct response. Twelve reversals were used for each run and the results were estimated from the mean of the last 8 reversals. In acoustic stimulation, the step size was 5 dB for the first four reversals and 2 dB after. In electric stimulation, the step size was 2 dB for the first four reversals and 0.5 dB after. The reported data in normal-hearing listeners were an arithmetic mean of four runs. Most data in implant listeners were an average of two runs. In the basal electrode condition in CI subjects, only one run was obtained due to time limitation. In cases where the standard deviation bar is shown, the data point was an average of three or four runs. The just-noticeable-difference (jnd) in intensity was reported as ΔI in dB or 20 LOG (1 + $\Delta L/L$), where L is the standard level and ΔL is the increment at which the subject achieved a 79.4% correct performance.

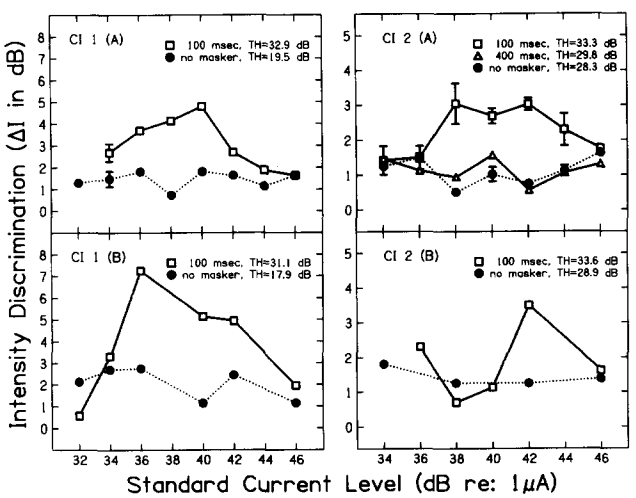

Fig. 2. Forward-masked intensity discrimination for two Richards Ineraid cochlear implant listeners. The upper two panels are data collected on the apical electrode and the lower two are on the basal electrodes. Note the signal delay and the threshold (TH) values on the upper-right corner in each panel.

3. Results

3.1. *Cochlear-implant (CI) listeners*

Fig. 2 shows intensity discrimination data measured from the two CI listeners. The upper two panels labeled CI 1 (A) and CI 2 (A) were results obtained with an apical electrode, while the lower two panels labeled CI 1 (B) and CI 2 (B) were results obtained with a basal electrode. For subject CI 1, the forward masker levels were 44 dB in the apical electrode and 46 dB in the basal electrode; while for subject CI 2, the masker levels were 45 dB on both electrodes. Four points are worth noting in Fig. 2.

First, note the threshold (as labeled in the upper-right corner of each panel) and dynamic range in the CI subjects. For subject CI 1 the unmasked thresholds were 19.5 and 17.9 dB on apical and basal electrodes; the dynamic range was about 30 dB (2–3 dB higher than the highest standard level, i.e., 46 dB). In the forward masking condition, the thresholds were 13 dB higher and the dynamic range was reduced to about 20 dB. For subject CI 2 the unmasked thresholds were 28.3 and 28.9 dB on apical and basal electrodes; the dynamic range was about 20 dB. In the forward masking condition, the thresholds were about 5 dB higher than the unmasked condition and the dynamic range was also about 5 dB less. In other words, forward masking appears to elevate the thresholds in terms of dB in CI listeners, but not their uncomfortable loudness level.

Second, note the jnds in the no-masker condition (filled circles). Both subjects were able to discriminate a level difference of 1 to 2 dB across their entire dynamic range. No difference was observed between apical and basal electrodes.

Third, note the jnds in 100-ms delay forward masking condition (open squares). It is obvious in all four

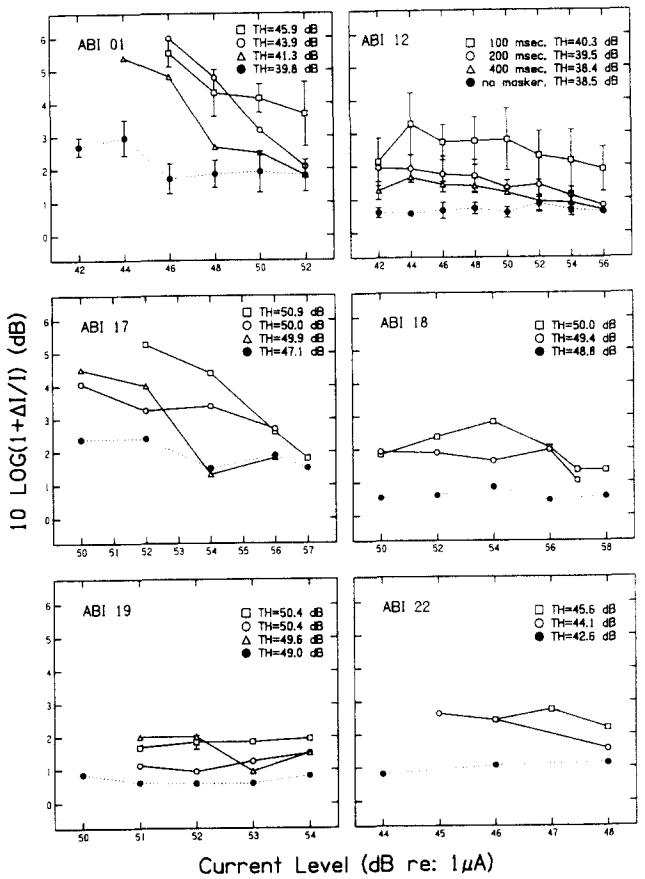

Fig. 3. Forward-masked intensity discrimination for six listeners with the auditory brainstem implants. Open squares, circles and triangles are the forward-masked data at signal delays of 100 ms, 200 ms and 400 ms, respectively. Filled circles are the controlled, unmasked data. Also note the threshold (TH) values for each condition on the upper-right corner in each panel.

panels that the forward masker produced a midlevel hump on the intensity discrimination function. The midlevel jnds were about 3–5 dB in most cases, except for the case of CI 1 (B) in which the greatest jnd was 7 dB. For standard levels close to the forward-masked thresholds, there were greater individual differences with some masked jnds being larger than unmasked values (CI 1 (A) and CI 2 (B)) and some being smaller (CI 1 (B)). Similar individual differences were observed in normal-hearing listeners (Zeng et al., 1991; Zeng and Turner, 1992).

Fourth, note the jnds at the 400 ms delay (open triangles) in panel CI 2 (A) only. The forward masker produced no midlevel hump and the masked jnds were not significantly different from the unmasked condition. It can also be noted that the threshold difference was much smaller (1.5 dB) at this delay. This set of data is different from normal-hearing listeners in which a small hump was present at the 400-ms delay.

3.2. Auditory brainstem implant (ABI) listeners

Fig. 3 shows intensity discrimination data measured under forward masking in six ABI listeners. The forward masker levels were 50, 54, 58, 57, 55, 47 dB for subjects ABI 01, 12, 17, 18, 19, 22, respectively. Thresholds without masking were 10 to 20 dB higher than the CI listeners' thresholds, as previously reported by Shannon and Otto (1990). The dynamic range was about 10 dB or less in ABI subjects, except in subject ABI 12 who had a range of about 20 dB. The threshold difference between masked and unmasked conditions in ABI subjects was smaller than CI subjects and ranged from 2 to 5 dB.

Similar to the CI listeners, the jnds in the no-masker condition were 1 to 2 dB across the entire dynamic range (filled circles). Unlike the CI listener, forward masking produced no midlevel hump at the 100 ms delay (open squares) in ABI listeners. However, the forward-masked jnds were 1 to 4 dB greater than the unmasked values across the entire dynamic range. Intensity jnds were measured as closely as possible to the threshold and the uncomfortable levels in forward masking condition to ensure that there would not be any improvement of the jnds at these extreme standard levels.

A similar pattern was observed for the 200 (open circles) and 400 (open triangles) ms delay conditions, except that the difference between the masked and the unmasked jnds were smaller at 400 ms delay. Also note the smaller threshold shifts at these two delays, compared with 100 ms delay. In some subjects (ABI 01, 12, and 17), the jnds were recovered to the unmasked values at high standard levels, but no midlevel hump was apparent.

3.3. Normal-hearing listeners

All four subjects exhibited a similar pattern, so only arithmetic mean results are reported. Fig. 4 shows

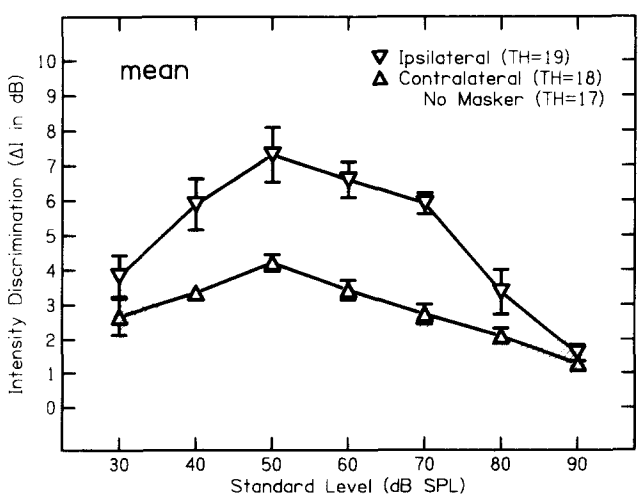

Fig. 4. The average data from four normal-hearing listeners in ipsilateral and contralateral forward masking conditions. Inverted triangles are from the ipsilateral masker and triangles are from the contralateral masker. The hatched area is the unmasked mean, plus and minus a standard deviation.

threshold and jnd data in conditions of no masking, ipsilateral and contralateral forward masking. As indicated in the upper-right corner of Fig. 4, there was little threshold difference across conditions. The shaded area represents the unmasked jnd data plus and minus a standard deviation. The unmasked intensity discrimination function shows the so-called near miss to Weber's law; jnds gradually improve as a function of standard level.

The ipsilateral, 90 dB SPL, pure-tone forward masker produced a midlevel jnd hump (inverted triangles), which is both qualitatively and quantitatively similar to that produced by a narrow-band noise masker (Zeng et al., 1991). This result shows that at the 100-ms delay there is little or no difference between noise and sinusoidal maskers. The same masker presented in the contralateral ear produced a much smaller jnd difference at the midlevels (2 dB or less, regular triangles). There was little or no effect of masking on intensity discrimination at low and high standard levels.

4. Discussion

4.1. Comparisons between acoustic and electric stimulation

In order to properly compare psychophysical results between acoustic and electric stimulation, we must first consider several significant differences in the two stimulation modes. One concern is loudness growth in electric hearing. By directly balancing loudness between acoustic and electric stimulation in the same subjects, a monotonic, logarithmic loudness balance function was found between the acoustic amplitude and the electric amplitude (Eddington et al., 1978; Zeng and Shannon, 1992; Dorman et al., 1993). It has been established that for the present 1000-Hz stimulus, loudness is an exponential function of electric amplitude (Zeng and Shannon, 1994).

Another concern with electric hearing is the reduced dynamic range, measured by the level difference between threshold and uncomfortable loudness level. In particular, the dynamic range is typically 10 dB in ABI listeners and 20 dB in CI listeners. This reduced dynamic range makes it difficult to compare the midlevel hump between acoustic and electric hearing; for example, one may argue that a 10 dB increment in the midlevel jnds in normal-hearing listeners only corresponds to a 1 dB effect in implant listeners, given the compression of the 100-dB normal range to the 10-dB electric range. In two of the six ABI listeners (ABI 12 and 18), there was a 1 dB mean difference between the highest jnd value at midlevels and the jnd values at low and high levels for the 100-ms masking condition. Does this 1-dB difference indicate the presence of a midlevel hump?

There are two measures that are indicative of a midlevel hump in normal hearing listeners as seen in Fig. 4: (1) a significant difference between the jnd values at midlevels and the values at two extremes; (2) a little or no difference between the masked and unmasked jnd values at the low and high standard levels. We examined the subject (ABI 12) in which both the mean and the variance data are available, and found that both measures suggest an absence of the midlevel hump. First, a Student's t-test shows, for the 100-ms masking condition, that there is no significant difference between the jnd values at the lowest standard level (42 dB) and the 44-dB midlevel ($t = -0.68$, $df = 4$, $P > 0.25$) and between the 44-dB and the 56-dB standard levels ($t = 0.85$, $df = 4$, $P > 0.20$). Second, there is a significant 1–2 dB difference (perhaps corresponding to 10–20 dB in normals) in the jnd values between the 100-ms delay masked and the unmasked conditions at the low and high standard levels.

4.2. Possible origins of the midlevel jnd hump in forward masking

The most important result in this paper is the presence of the midlevel hump in cochlear implant (CI) listeners and its absence in brainstem implant (ABI) listeners. Because the obvious difference between the CI and the ABI listeners is that the ABI listeners lack both the auditory nerve transmission and the intrinsic processing in the cochlear nucleus, according to Fig. 1, this pattern of results suggests that the midlevel hump is likely due to physiological mechanisms in the auditory nerve transmission and the cochlear nucleus processing circuits. However, in ABI listeners, the jnds do not recover to the unmasked values, indicating that cochlear nuclei and higher centers may still be involved in forward masking.

The small contralateral forward masking effect in normal-hearing listeners also suggests limited central contribution. Because we used circumaural headphones in the acoustic stimulation experiments, the small jnd shifts in the contralateral masking condition may be due to acoustic cross-talk and not a true contralateral effect. Assuming that the interaural attenuation is 40–50 dB, the 2-dB contralateral masking effect may be explained by the equivalent ipsilateral masker at 40–50 dB SPL (Zeng and Turner, 1992). An alternative explanation is the efferent inhibition effect of the contralateral masker on the auditory nerve, particularly on the low-SR neurons (Warren and Liberman, 1989). The present study cannot distinguish these two mechanisms.

4.3. Physiological mechanisms

Let us consider possible physiological mechanisms that could explain the observed pattern of results. At the level of the auditory nerve, if the jnd is determined by a statistically significant increment in firing rate as a function of stimulus level, a large jnd could be caused

either by a shallower slope of the rate growth function or by an increased variance in firing rate. Consider the following two possible mechanisms that could produce a midlevel hump. First, suppose that the overall rate of high- and low-SR neurons is the relevant cue for the jnd measurement. It is also known in the 100-ms delay forward masking condition that thresholds for low-SR neurons with high thresholds are selectively elevated (Relkin and Doucet, 1991) and their rate-intensity functions are also shifted towards higher levels (Relkin and Solessio, 1993). Both the threshold elevation and the up-shifted rate-intensity function for the low-SR neurons would result in a shallower slope of the overall rate growth function at the midlevels.

Second, an increment in the variance of firing rate in forward masking can also produce a midlevel hump. We are not aware of any data examining the variance of firing rate in forward masking, but an indirect study has successfully related the neural variance to a midlevel jnd hump. In order to explore the physiological mechanisms of a similar midlevel jnd hump for click signals (Raab and Taub, 1969), Taub and Raab (1969) recorded both the amplitude and the standard deviation of the compound action potential for the clicks and found that the variability of the compound action potential was the principal factor correlating with the click's midlevel jnd hump.

At the cochlear nucleus level, two potential physiological mechanisms may also contribute to the difference (the presence and absence of the midlevel jnd hump) between the CI and the ABI listeners. Recent physiological studies have suggested that both the auditory nerve and cochlear nucleus innervation and the intrinsic neural circuits in cochlear nuclei play an important role in auditory information processing (e.g., Young et al., 1988). In particular, Blackburn and Sachs (1990) showed that the 'chopper' cells can listen selectively to low-SR neurons. Stimulating the auditory nerve (the CI case), the brain may take advantage of both the partially preserved innervation between the auditory nerve and the cochlear nuclei and the processing ability of the intrinsic circuits in cochlear nuclei. However, in the ABI case, the cochlear nuclei are stimulated nonspecifically; the brain may not be able to make use of these two mechanisms. The different effect between the CI and ABI listeners suggests that the midlevel hump is likely due to physiological mechanisms in the auditory nerve transmission and the cochlear nucleus processing circuits. This suggestion, however, is limited to the lack of relevant electric stimulation data at both the auditory nerve and the cochlear nucleus levels.

4.4. Recovery from forward masking for low- and high-SR neurons

The present data may also shed light on the differential recovery from forward masking between low- and high-SR neurons. Two morphological differences between low- and high-SR neurons have been observed between hair-cell synapse and cochlear nucleus and related to their differential recovery from forward masking. First, low-SR neurons have been shown to be smaller in diameter of both their dendrite and axon (Liberman, 1982; Liberman and Oliver, 1984), and a smaller diameter leads to a higher threshold and slower conduction speed (e.g., Koester, 1981). Second, the dendrites of low-SR neurons have been shown to receive more innervation by the lateral olivocochlear portion of the efferent system (Liberman, 1980). Since the efferent effect is usually inhibitory and takes several hundred milliseconds to develop and decay (e.g. Warren and Liberman, 1989), it is possible that the efferents are involved in the recovery from forward masking, particularly, for low-SR neurons.

Since auditory neurons have essentially no spontaneous activity in electric stimulation of cochlea, we can no longer assume any relationship between their recovery from forward masking and spontaneous activity. However, the morphological differences between low-SR and high-SR neurons should still be preserved in cochlear implant subjects and thus affect electric excitability of these two populations differentially. In addition, recent anatomical evidence indicates that low-SR neurons are segregated in the auditory nerve bundle and have a path closest to the scala vestibuli (Leake and Snyder, 1989; Liberman, 1991). This means that for intracochlear electrode inserted in scala tympani, the distance between the electrode and the axon is longer for low-SR neurons than for high-SR neurons and the longer distance to the electrode would result in higher threshold for low-SR neurons (e.g. van den Honert and Stypulkowski, 1987). Therefore, the partial preservation of normal morphology and the electrode position may result in a midlevel hump in cochlear implant listeners. On the other hand, such morphological differences between low- and high-SR neurons are obviously absent in the ABI listeners, and thus no midlevel jnd hump should be observed.

5. Summary of results

Intensity discrimination functions in forward masking have been obtained in cochlear implant listeners, auditory brainstem implant listeners, and normal-hearing listeners. Although there are significant individual differences, the overall pattern of results suggest a possible peripheral origin of the midlevel hump. Specifically, the forward masker produced a similar midlevel hump in normal-hearing and cochlear implant listeners, suggesting that the hair-cell synapse is not the likely origin. The forward masker produced no hump in auditory brainstem implant listeners, suggest-

ing the hump is due to some mechanism either peripheral to or in the cochlear nucleus. The contralateral forward masker produced a small hump in normal-hearing listeners, further suggesting that central interactions at superior olivary complex and higher are not the likely origin of the midlevel hump.

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References

Abbas, P.J. (1979) Effects of stimulus frequency on adaptation in auditory-nerve fibers. J. Acoust. Soc. Am. 65, 162–165.

Blackburn, C.G. and Sachs, M.B. (1990) The representation of the steady-state vowel sound /e/ in the discharge patterns of cat anteroventral cochlear nucleus neurons. J. Neurophysiol. 63, 1191–1212.

Brackmann, D.E. et al. (1993) Auditory brainstem implant: I. Issues in surgical implantation. Otolaryngol. Head Neck Surg. 108, 624–633.

Carlyon, R.P. and Beveridge, H.A. (1993) Effects of forward masking on intensity discrimination, frequency discrimination and the detection of tones in noise. J. Acoust. Soc. Am. 93, 2886–2895.

Dorman, M.F., Smith, L. and Parkin, J.L. (1993) Loudness balance between acoustic and electric stimulation by a patient with a multichannel cochlear implant. Ear Hear. 14, 290–292.

Dynes, S.B.C. and Delgutte, B. (1992) Phase-locking of auditory-nerve discharges to sinusoidal electric stimulation of the cochlea. Hear. Res. 58, 79–90.

Eddington, D.K., Dobelle, W.H., Brackmann, D.E., et al. (1978) Auditory prostheses research with multiple channel intracochlear stimulation in man. Ann. Otol. Rhinol. Laryngol. 87, Suppl. 53, 5–39.

Eisenberg, L.S., Maltan, A.A., Portillo, F., Mobley, J.P. and House, W.F. (1987) Electrical stimulation of the auditory brain stem structure in deafened adults, J. Rehab. Res. Rev. 24, 9–22.

Elmasian, R., Galambos, R. and Bernheim, A. (1980) Loudness enhancement and decrement in four paradigms. J. Acoust. Soc. Am. 67, 601–607.

Galambos, R., Bauer, J., Picton, T., Squires, K. and Squires, N. (1972) Loudness enhancement following contralateral stimulation. J. Acoust. Soc. Am. 52, 1127–1130.

Howard, J. and Hudspeth, A.J. (1987) Mechanical relaxation of the hair bundle mediates adaptation in mechanoelectrical transduction by the bullfrog's sacular hair cell. Proc. Natl. Acad. Sci. 84, 3064–3068.

Irwin, R.J. and Zwislocki, J.J. (1971) Loudness effects in pairs of tone bursts. Percept. Psychophys. 10, 189–192.

Javel, E. (1990) Acoustic and electric encoding of temporal information. In: J.M. Miller and F.A. Spellman (Eds.), Cochlear implants. Models of the electrically stimulated ear. Springer Verlag, New York.

Javel, E., Tong, Y.C., Shepherd, R.K. and Clark, G.M. (1987) Responses of cat auditory-nerve fibers to biphasic electrical current pulses. Ann. Otol. Rhinol. Laryngol. 96, (Suppl. 128), 26–30.

Kiang, N.Y.S., Watanabe, T., Thomas, E.C. and Clark, L.F. (1965) Discharge Patterns of Single Fibers in the Cat's Auditory Nerve. MIT Press (Research Monograph No. 35), Cambridge, MA.

Killian, M.J.P., Klis, S.F.L. and Smoorenburg, G.F. (1992) Similarities between forward masking in cochlear implant patients and recovery from adaptation of the electrically evoked VIIIth nerve compound action potential in guinea pig. Proc. 29th Inner Ear Biology Workshop, Engelberg, Switzerland.

Koester, J. (1981) Functional consequences of passive electrical properties of the neuron. In: E.R. Kandel and J.H. Schwartz (Eds.), Principles of neural science. Elsevier/North-Holland, New York, 44–52.

Leake, P.A. and Snyder, R.L. (1989) Topographic organization of the central projections of the spiral ganglion in cats. J. Comp. Neurol. 281, 612–629.

Liberman, M.C. (1980) Efferent synapses in the inner hair cell area of the cat cochlea: an electron-microscopic study of serial sections. Hear. Res. 3, 189–204.

Liberman, M.C. (1982) Single-neuron labeling in the cat auditory nerve. Science 216, 1239–1241.

Liberman, M.C. (1991) Central projections of auditory-nerve fibers of differing spontaneous rate. I. Anteroventral cochlear nucleus. J. Comp. Neurol. 313, 240–258.

Liberman, M.C. and Oliver, M.E. (1984) Morphometry of intracellularly labeled neurons of the auditory nerve: Correlations with functional properties. J. Comp. Neurol. 223, 163–176.

Meddis, R. (1986) Stimulation of mechanical to neural transduction in the auditory receptor. J. Acoust. Soc. Am. 79, 702–711.

Mulroy, M.J., Altmann, D.W., Weiss, T.F. and Peake, W.T. (1974) Intracellular electric responses to sound in a vertebrate cochlea. Nature 249, 482–485.

Plack, C.J. and Viemeister, N.F. (1992a) The effects of notched noise on intensity discrimination under forward masking. J. Acoust. Soc. Am. 92, 1902–1910.

Plack, C.J. and Viemeister, N.F. (1992b) Intensity discrimination in backward masking. J. Acoust. Soc. Am. 92, 3097–3101.

Raab, D.H. and Taub, H.B. (1969) Click-intensity discrimination with and without a background masking noise. J. Acoust. Soc. Am. 46, 965–968.

Rakowski, A. and Jaroszewski, A. (1974) On some secondary masking effect (Pitch shifts in post-stimulatory masking). Acoustica 31, 325–329.

Relkin, E.M. and Doucet, J.R. (1991) Recovery from prior stimulation. I. Relationship to spontaneous firing rates of primary auditory neurons. Hear. Res. 55, 215–222.

Relkin, E.M. and Solessio, E. (1993) Effects of a preceding high-intensity tone-burst on rate-intensity functions of low spontaneous-rate auditory neurons. Abstr. Assoc. Res. Otolaryngol. 93.

Rhode, W.S. and Smith, P.H. (1985) Characteristics of tone-pip response patterns in relationship to spontaneous rate in cat auditory nerve fibers. Hear. Res. 18, 159–168.

Ross, S. (1982) A model of the hair cell-primary fiber complex. J. Acoust. Soc. Am. 71, 926–941.

Rubin, H. (1960) Auditory facilitation following stimulation at low intensities. J. Acoust. Soc. Am. 32, 670–681.

Schroeder, M.R. and Hall, J.L. (1974) Model for mechanical to neural transduction in the auditory receptor. J. Acoust. Soc. Am. 55, 1055–1060.

Schwid, H.A. and Geisler, C.D. (1974) Multiple reservoir model of neurotransmitter release by a cochlear inner hair cell. J. Acoust. Soc. Am. 72, 1435–1440.

Shannon, R.V. (1990) Forward masking in patients with cochlear implants. J. Acoust. Soc. Am. 88, 741–744.

Shannon, R.V. and Otto, S.R. (1990) Psychophysical measures from electrical stimulation of the human cochlear nucleus. Hear. Res. 47, 159–168.

Smith, R.L. and Brachman, M.L. (1982) Adaptation in auditory nerve fibers: A revised model. Biol. Cybernet. 44, 107–120.

Smith, R.L. and Zwislocki, J.J. (1975) Short-term adaptation and incremental response of single auditory-nerve fibers. Biol. Cybernetics 17, 169–182.

Taub, H.B. and Raab, D.H. (1969) Fluctuations of N1 amplitude in relation to click-intensity discrimination. J. Acoust. Soc. Am. 46, 969–978.

van den Honert, C. and Stypulkowski, P.H. (1987) Temporal patterns of single auditory nerve fibers elicited by periodic electrical stimuli. Hear. Res. 29, 207–222.

Vurek, L.S. et al. (1981) Opto-isolated stimulators used for electrically evoked BSER. Ann. Otol. Rhinol. Laryngol. 90, Suppl. 82, 21–24.

Warren, E.H. and Liberman, M.C. (1989) Effects of contralateral sound on auditory-nerve responses. I. Contributions of cochlear efferents. Hear. Res. 37, 89–104.

Young, E.D. et al. (1988) Response properties of cochlear nucleus neurons in relationship to physiological mechanisms. In: G.M. Edelman, W.E. Gall and W.M. Cowan (Eds.), Auditory functions: Neurobiological bases of hearing. John Wiley & Sons, New York, pp. 277–312.

Zeng, F.-G. and Shannon, R.V. (1992) Loudness balance between electric and acoustic stimulation. Hear. Res. 60, 231–235.

Zeng, F.-G. and Shannon, R.V. (1994) Loudness coding mechanisms inferred from electric stimulation of the human auditory system. Science 264, 564–566.

Zeng, F.-G. and Turner, C.W. (1992) Intensity discrimination in forward masking. J. Acoust. Soc. Am. 92, 782–787.

Zeng, F-G., Turner, C.W. and Relkin, E.M. (1991) Recovery from prior stimulation. II. Contribution to intensity discrimination. Hear. Res. 55, 223–230.

Zwislocki, J.J. (1971) A theory of central auditory masking and its partial validation. J. Acoust. Soc. Am. 52, 644–659.

Zwislocki, J.J., Piroda, E. and Rubin, H. (1959) On some poststimulatory effects at the threshold of audibility. J. Acoust. Soc. Am. 31, 9–14.