Consequences of cochlear damage for the detection of interaural phase differences

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Thresholds for detecting interaural phase differences (IPDs) in sinusoidally amplitude-modulated pure tones were measured in seven normal-hearing listeners and nine listeners with bilaterally symmetric hearing losses of cochlear origin. The IPDs were imposed either on the carrier signal alone—not the amplitude modulation—or *vice versa*. The carrier frequency was 250, 500, or 1000 Hz, the modulation frequency 20 or 50 Hz, and the sound pressure level was fixed at 75 dB. A three-interval two-alternative forced choice paradigm was used. For each type of IPD (carrier or modulation), thresholds were on average higher for the hearing-impaired than for the normal listeners. However, the impaired listeners' detection deficit was markedly larger for carrier IPDs than for modulation IPDs. This was not predictable from the effect of hearing loss on the sensation level of the stimuli since, for normal listeners, large reductions of sensation level appeared to be more deleterious to the detection of modulation IPDs than to the detection of carrier IPDs. The results support the idea that one consequence of cochlear damage is a deterioration in the perceptual sensitivity to the temporal fine structure of sounds.

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I. INTRODUCTION

The response of an auditory nerve (AN) fiber to a pure tone is normally phase-locked to the stimulus, as long as its frequency does not exceed a few kilohertz (Rose *et al.*, 1967). Owing to this phase-locking mechanism, information on the temporal fine-structure of sounds is conveyed to higher levels of the auditory system. Listeners with normal hearing do process that information. This is most clearly demonstrated by their ability to detect small interaural phase differences in binaurally presented pure tones, even in the absence of onset or offset cues (Hafter *et al.*, 1979). It is believed that, in addition to its important role in the localization of sounds, the peripheral encoding of temporal finestructure also plays a role in the perception of pitch (e.g., Moore, 1973) and the identification of spectral profiles such as those of vowels (Young and Sachs, 1979).

A few physiological studies have been devoted to the consequences of cochlear damage for the phase-locking capacity of AN fibers. Woolf *et al.* (1981) produced substantial destruction of outer hair cells in the cochleas of chinchillas, and found that this reduced significantly the precision of phase-locking in individual AN fibers. However, the results of Woolf *et al.* are at odds with those reported by Harrison and Evans (1979) and Miller *et al.* (1997), who found no loss in the quality of phase-locking following severe hair cell lesions due to the injection of kanamycin in guinea pigs (Harrison and Evans) or an acoustic trauma in cats (Miller *et al.*

al.). From the physiological literature, therefore, it is far from clear that human listeners with damaged cochleas should have a subnormal perceptual sensitivity to the temporal fine structure of sound waveforms. Yet, several psychophysical studies have suggested that this is the case.

Part of the psychophysical evidence comes from experiments on the detection of slow frequency modulation (Zurek and Formby, 1981; Moore and Glasberg, 1986; Lacher-Fougère and Demany, 1998; Moore and Skrodzka, 2002; Buss et al., 2004). For normal listeners, the perceptual detection of slow frequency modulation imposed on lowfrequency sinusoidal carriers seems to rest, at the AN level, on temporal cues rather than on tonotopic cues [see Moore and Sek (1996) or Lacher-Fougère and Demany (1998) for a review of the psychophysical arguments supporting that view]. In cases of cochlear damage, the detection thresholds of such modulations are generally elevated, and this elevation is very pronounced if the damage is severe. Recently, Moore and Moore (2003) have also argued that cochlear damage has a deleterious effect on the ability to discriminate the fundamental frequency of harmonic complex tones on the basis of cues related to the temporal fine structure of the waveform.

More direct evidence has been provided by two studies on the detection of interaural time delays (ITDs) in binaural stimuli (Hawkins and Wightman, 1980; Buus *et al.*, 1984). In listeners with severe, wide-band, and bilaterally symmetric hearing losses of cochlear origin, the detectability of an ITD in a narrow-band noise centered at 500 Hz (Hawkins and Wightman, 1980) or in a 500- or 1000-Hz tone burst (Buus *et al.*, 1984) is strongly impaired. For the same listeners, in contrast, the detectability of an interaural intensity difference can be normal, according to Hawkins and Wightman. More-

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FIG. 1. Sinusoidally amplitude-modulated sinusoids. Two pairs of such functions are shown. In the top part (a), the two modulations are in phase but there is a 90° phase difference between the two carriers. In the bottom part (b), the two carriers are in phase but there is a 90° phase difference between the two modulations.

over, according to Buus et al., the detectability of an ITD in a 4000-Hz tone burst presented at a high SPL (100 dB) is in general nearly normal. In agreement with the latter finding, Smoski and Trahiotis (1986) reported that an ITD in a narrow-band sound centered at 4000 Hz is generally not harder to detect by impaired listeners than by normal ones when the stimulus is presented at a constant sensation level of 25 dB. In the case of a 500- or 1000-Hz tone burst, normal listeners are sensitive to the ongoing interaural phase difference produced by an ITD; but this is no longer true at 4000 Hz, in which case an ITD is detectable only by virtue of the delay in the amplitude envelope. Thus, the frequencyselective detection deficit observed by Buus et al. (1984) in listeners with cochlear damage suggests that such listeners have a subnormal sensitivity to temporal fine structure per se.

This suggestion is still not logically compelling, however. Besides, it should be noted that Hawkins and Wightman (1980) did not find a stronger deficit of ITD detection at 500 Hz than at 4000 Hz in their hearing-impaired subjects, although the audiometric deficit of some of these subjects was larger at 500 than at 4000 Hz. We reasoned that a more convincing demonstration might be provided by dissociating, within a given set of binaural stimuli, the interaural relations between the fine structures and the envelopes. In the present study, binaural stimuli consisting of sinusoidally amplitudemodulated pure tones were used and listeners had to detect interaural phase differences (IPDs) imposed either on the carrier signal alone-not the amplitude modulation-or vice versa (see Fig. 1). The carrier frequency was varied but kept within the range for which the binaural system is normally sensitive to fine-structure IPDs. The modulation frequency was always low enough to preclude cochlear resolution of the sounds' three spectral components. In our main experiment (experiment 1), the performance of normal listeners in the two tasks (carrier versus modulation, i.e., fine structure versus envelope) was compared to that of sensorineurally impaired listeners. Two additional experiments were conducted to determine if the impaired listeners' detection deficits in experiment 1 could simply originate from the fact that, for these listeners, the sensation level of the stimuli was abnormally low.

II. EXPERIMENT 1

A. Listeners

Sixteen listeners were tested. Seven of them (forming the normal group; age range: 24–45 yr) had, for each ear, absolute thresholds that did not exceed 20 dB HL (ISO 389 standard) from 250 to 8000 Hz. The other nine listeners (forming the impaired group; age range: 42–68 yr) had purely cochlear hearing losses which were similar for the two ears. The cochlear origin of their auditory deficits was established following a clinical examination including otoscopy, tonal and speech audiometry with air/bone gap measures, immitance audiometry, and BER recording (or MRI in one case). Their audiograms are presented in Table I. Listeners 1 and 5 were presbycusic. The hearing loss of Listeners 4 and 6 was congenital. The hearing loss of Listener 8 had been of the "sudden" type. For the remaining four impaired listeners, the origin of hearing loss was unknown.

B. Stimuli

We used 500-ms stimuli which were gated on and off with interaurally synchronous linear amplitude ramps of 50 ms. At each ear, before gating, the stimulus was an amplitude-modulated sinusoid defined by

TABLE I. Audiograms of the hearing-impaired listeners. In columns 2–7, the two numbers in each cell are the absolute thresholds for the left and right ears, in dB HL.

Listener (age)	250 Hz	500 Hz	1000 Hz	2000 Hz	4000 Hz	8000 Hz
1 (59)	5/10	10/15	20/20	15/25	45/65	65/90
2 (55)	15/20	20/20	20/20	30/25	45/35	50/30
3 (68)	25/20	30/20	35/30	45/30	55/50	55/40
4 (42)	30/20	30/30	40/40	50/55	60/65	70/60
5 (61)	20/20	25/35	45/50	70/60	65/55	75/65
6 (57)	30/25	50/35	65/55	65/70	70/95	80/100
7 (64)	25/35	35/35	40/40	35/35	35/25	35/20
8 (47)	45/40	50/40	40/45	45/45	50/60	50/60
9 (67)	40/45	55/50	55/60	50/45	25/15	35/20

$$s(t) = \sin(2\pi \cdot F_{\text{car}} \cdot t + \varphi_{\text{car}}) \cdot [1 + \sin(2\pi \cdot F_{\text{mod}} \cdot t + \varphi_{\text{mod}})], \qquad (1)$$

in which F_{car} represents the carrier frequency–250, 500, or 1000 Hz–, F_{mod} the modulation frequency–20 or 50 Hz–, and *t* is time. The phases φ_{car} and φ_{mod} had a fixed value of 0° at the right ear. One of these two phases could be different from 0° at the left ear. In this case, it was always positive (without exceeding 180°). During the measurement of just-detectable IPDs, the sound pressure level was 75 dB at each ear. For the impaired listeners, this was always sufficient to make the stimuli clearly detectable at both ears (as confirmed by the listeners' verbal reports). The stimuli were generated via 16-bit digital-to-analog converters (Oros AU22), at a sampling rate of 19 kHz, and presented by means of TDH-39P earphones, in a doublewalled soundproof booth.

C. Procedure

Each listener took part in four experimental sessions of about 1 h, on different days. At the beginning of every session, before the measurement of just-detectable IPDs, the listener was required to perform a series of (typically six) across-ear intensity-matching trials. This test had two goals. The first was to check that, when the stimulus was diotic, the spatial position of the perceived sound was approximately central (rather than lateralized on the left or right due to an asymmetry of loudness). The second goal was to obtain information on the listener's sensitivity to interaural intensity differences. In each intensity-matching trial, the listener was repeatedly presented with a stimulus for which φ_{car} and φ_{mod} were 0° at both ears, F_{mod} was 20 Hz, and F_{car} was either 250, 500, or 1000 Hz. Consecutive stimulus presentations were separated by a 500-ms silent interval. The SPL was fixed at 75 dB at one ear (the left ear on about 50% of trials) and was variable at the other ear. Initially, the variable SPL differed from 75 dB by a random amount, within a range of ± 10 dB. The listener's task was to center the sound image, as accurately as possible, by adjusting the variable SPL. This could be done by steps of ± 1 or 4 dB, using four labeled buttons. The listener had an unlimited amount of time to perform his or her adjustment, and pressed a fifth button to record it when satisfied.

Detection thresholds for IPDs were then measured with an adaptive forced-choice method, the SPL being 75 dB at each ear. In a given block of trials, F_{car} and F_{mod} were fixed. On each trial, three successive stimuli separated by 500-ms silent pauses were presented. There was an IPD in only one of them: either the second or the third stimulus, at random and equiprobably. The listener's task was to identify the position of this stimulus by pressing one of two buttons. Visual feedback was provided by means of light-emitting diodes. Initially, the IPD was large. It was divided by the cube root of 1.5 following every correct response. Following a wrong response, it was multiplied by 1.5, or set to 180° if a multiplication by 1.5 produced an IPD exceeding 180° . Each block of trials ended after 14 reversals in the IPD variation. The geometric mean of the last 10 reversal points was taken as an estimate of the just-detectable IPD. In the absence of ceiling effects in the IPD variation, this threshold estimate corresponded to the 75% correct point of the psychometric function (Kaernbach, 1991). Overall, 24% of the threshold measurements forming the impaired listeners' raw data were biased by ceiling effects. Such effects occurred more frequently when the IPD was imposed on φ_{car} (30.5% of threshold measurements) than when the IPD was imposed on φ_{mod} (17.0%).

Thresholds were mainly measured for three combinations of F_{car} and F_{mod} : 500/20 Hz (in which case data were obtained from each listener), 1000/20 Hz (data obtained from four normal listeners and the entire impaired group), and 500/50 Hz (five impaired listeners and the entire normal group). Two normal and two impaired listeners were also tested using the 250/20-Hz combination. For a given $F_{\rm car}/F_{\rm mod}$ combination and a given type of IPD ($\varphi_{\rm car}$ or $\varphi_{\rm mod}$), the total number of threshold estimations per subject was typically equal to 7; however, it was sometimes smaller; its mean value was 6.3. As a rule, when more than four estimates had been obtained, only the last four were considered in the data analysis; their geometric mean was taken as the listener's threshold. For one impaired listener, however, only the last two estimates were averaged owing to the existence of a very strong practice effect (improvement of thresholds) in all conditions.

D. Results

The data collected during the intensity-matching trials are summarized in Fig. 2. For a given listener and F_{car} , we computed: (i) the absolute value of the mean of the adjusted interaural intensity differences-an index called "absolute shift" (from 0, i.e., no ear asymmetry); (ii) the standard deviation of the adjusted interaural intensity differences-an index called "random error." The top panel of Fig. 2 displays the mean value of the absolute shifts measured in the normal group (open circles) and the impaired group (closed circles), as a function of F_{car} . What must be noted here is the absence of a definite difference between the two groups. A two-way analysis of variance (ANOVA) with listeners as the random factor confirmed that the "group" factor had no significant main effect [F(1,42) < 1] and did not interact significantly with the "frequency" factor [F(2,42) < 1]. This outcome implies that, as we wished, the detection of IPDs by the hearing-impaired listeners was not liable to be significantly disrupted by abnormal asymmetries in loudness. The apparent absence of such asymmetries is not very surprising since, up to 2000 Hz, the two monaural audiograms of the impaired listeners were closely matched: the average interaural difference between the absolute thresholds at a given frequency was only 5 dB (cf. Table I).

In the bottom panel of Fig. 2, it can be seen that there was also no pronounced difference between the two groups with respect to the within-subject variability of the adjustments. An ANOVA performed on these data showed that the main effect of group was only marginally significant [F(1,42)=2.92; P=0.095]. This result is consistent with

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FIG. 2. Results of the intensity-matching test. Open and closed circles, respectively, represent normal and hearing-impaired listeners. Top panel: mean absolute shifts. Bottom panel: mean random errors. The error bars represent standard deviations.

Hawkins and Wightman's (1980) finding of normal interaural intensity difference thresholds in listeners with sensorineural hearing loss.

In Fig. 3, we have plotted the mean IPD thresholds measured in the normal group. For the fine-structure task (detection of interaural differences in φ_{car}), the mean thresholds increased very slightly from 250 to 500 Hz, and to a larger extent from 500 to 1000 Hz; at 500 Hz, no effect of F_{mod} was



FIG. 3. IPD thresholds measured for normal listeners in experiment 1. For comparison, the fine-structure IPD thresholds displayed by Durlach and Colburn (1978, p. 417) are also plotted; these thresholds represent a synthesis of data published by Klumpp and Eady (1956) and Zwislocki and Feldman (1956). A logarithmic scale is used on both axes.



FIG. 4. IPD thresholds measured in experiment 1 for each member of the two groups of listeners. The four panels correspond to the four combinations of $F_{\rm car}$ and $F_{\rm mod}$. Open and closed symbols, respectively, represent normal and impaired listeners. Diamonds symbolize pairs of thresholds which are both underestimated due to ceiling effects. When only the fine-structure threshold is underestimated, the symbol used is a triangle pointing to the right. The oblique line displayed in each panel has a slope of 1 and goes through the centroid of the normal listeners' data.

found. As shown in Fig. 3, our results are similar to those reported by Durlach and Colburn (1978) for unmodulated pure tones at 50 dB SL. For the envelope task (detection of interaural differences in φ_{mod}), the effect of F_{car} on thresholds was different: thresholds were highest at 250 Hz and almost the same at 500 and 1000 Hz; but at 500 Hz, again, no marked effect of F_{mod} was found. At 1000 Hz, essentially identical mean thresholds were obtained for the two tasks.

Figure 4 shows the IPD thresholds measured for each member of the two groups of listeners. The four panels correspond to the four combinations of F_{car} and F_{mod} . Open and closed symbols, respectively, represent normal and impaired listeners. Diamonds symbolize pairs of thresholds (finestructure and envelope) which are both underestimated due to ceiling effects (cf. Sec. II C). When only the fine-structure threshold is underestimated, the symbol used is a triangle pointing to the right. Globally, thresholds were poorer in the impaired group than in the normal group for both tasks, but the impaired listeners' deficit was larger for the fine-structure task. This was true for every F_{car}/F_{mod} combination. In each panel is drawn an oblique line which has a slope of 1 and goes through the centroid of the normal listeners' data. If the impaired listeners had been equally deficient in the finestructure task and the envelope task, then their data points would have fallen equally often above and below the oblique lines. It can be seen that this was not the case. Overall, out of the 25 relevant data points, only three fall above the line. This asymmetry is statistically significant (P < 0.001, binomial test). Moreover, 7 times out of 25, an impaired listener's fine-structure performance was strongly subnormal while his or her envelope performance was normal. The converse was

TABLE II. Geometric means of the individual thresholds measured (in degrees) for the two groups of listeners, and ratios of the two means (impaired/normal) obtained for each condition.

	Fine-structure thresholds				Envelope thresholds			
$F_{\rm car}/F_{\rm mod}$	250/20	500/20	500/50	1000/20	250/20	500/20	500/50	1000/20
Normal-hearing group	3.37	3.75	3.82	7.89	19.79	7.80	9.72	7.36
Impaired-hearing group	66.28	24.50	34.35	67.30	68.54	22.25	39.29	29.85
Ratio	19.7	6.5	9.0	8.5	3.5	2.9	4.0	4.1

never observed. For each task and $F_{\rm car}/F_{\rm mod}$ combination, we indicate in Table II the geometric mean of the thresholds measured in each group, as well as the ratio of the two means (impaired/normal). The ratios have a geometric mean of 9.9 for the fine-structure task, and 3.6 for the envelope task.

For the three main F_{car}/F_{mod} combinations (500/20, 500/50, and 1000/20 Hz), the impaired listeners' IPD thresholds are replotted in Fig. 5 as a function of the absolute threshold (for the ear with greater loss in case of inequality) at F_{car} . Here, digits identify the listeners. In the upper left panel, it can be seen that the fine-structure IPD threshold measured in a given listener for the 500/50-Hz combination (small digits) was generally similar to his or her finestructure threshold for the 500/20-Hz combination (large digits). There was also a within-subject similarity of the envelope thresholds measured for these two F_{car}/F_{mod} combinations (lower left panel). However, the fine-structure thresholds were not significantly correlated with the absolute thresholds at 500 Hz (r=0.09 for $F_{mod}=20$ Hz; r=0.26 for $F_{\rm mod}$ =50 Hz). This result conflicts with the rather high (approximately 0.7) correlations between ITD thresholds and absolute thresholds observed by Hall et al. (1984) for 500-Hz tone bursts at 70 dB SPL and by Hawkins and Wightman (1980) for 85-dB narrow-band noises in the same



FIG. 5. IPD thresholds of the impaired listeners as a function of their absolute threshold (for the ear with greater loss in case of inequality) at $F_{\rm car}$. Each listener is identified by a digit consistent with Table I. Large digits are used for $F_{\rm mod}$ =20 Hz, smaller digits for $F_{\rm mod}$ =50 Hz.

spectral region. On the other hand, our data confirm previous evidence (Hawkins and Wightman, 1980; Smoski and Trahiotis, 1986) that, in listeners with bilateral cochlear hearing losses at high frequencies but normal absolute thresholds at low frequencies, the ITD threshold at low frequencies may be abnormally large: at 500 Hz, Listener 1 had abnormal fine-structure IPD thresholds, but normal absolute thresholds and normal envelope IPD thresholds. At 1000 Hz, however, we found a higher and significant correlation (r=0.68, P =0.04) between fine-structure IPD thresholds and absolute thresholds. Similar correlations were found, at 500 and 1000 Hz, between the envelope IPD thresholds and the absolute thresholds (average r: 0.62). Note finally that there was no significant correlation between the impaired listeners' ages and their fine-structure thresholds (average r: 0.35) or envelope thresholds (average r: 0.32).

III. EXPERIMENTS 2 AND 3

A. Rationale and method

In experiment 1, the hearing-impaired listeners showed a larger deficit for the fine-structure task than for the envelope task. Could this be due to the fact that the sensation level of the stimuli (which had a fixed SPL, 75 dB) was generally lower for the impaired listeners than for the normal ones? To answer that question, we performed two experiments assessing, in normal listeners, the effect of a reduction in sensation level on the detectability of fine-structure and envelope IPDs.

The stimuli used in experiment 2 were identical to those used in the 500/20-Hz condition of experiment 1. They thus had a fixed SPL of 75 dB. They were presented either alone or together with a masker which reduced their sensation level. The masker was a diotic and continuous white noise low-pass filtered at 1250 Hz. It was produced by an analog generator (Brüel & Kjaer, WB 1314) and presented at an SPL of 69.5 dB. In the presence of this masker, for the authors, the sensation level of the stimuli was about 22 dB. Three listeners were tested. Two of them—the authors—had previously served as subjects in experiment 1. The third listener—a student with normal hearing—was trained during four 1-h sessions before data collection began.

In experiment 3, one of the two conditions of testing was again identical to the 500/20-Hz condition of experiment 1. In the other condition, the sensation level of the stimuli was reduced not by the addition of noise but simply by a 40-dB decrease of intensity: the stimuli were presented at 35 dB SPL. The three listeners who served as subjects included author L.D. and two audiometrically normal students, who were initially trained for 3–4 h.



FIG. 6. IPD thresholds measured in experiment 2 (upper panel) and experiment 3 (lower panel). The five listeners are represented by different symbol shapes.

B. Results

The data collected in experiment 2 consisted of five threshold estimates in each cell of the design [3 subjects $\times 2$ IPD types (fine structure versus envelope) $\times 2$ contexts (no noise versus noise)]. The geometric means of these estimates are displayed in the top panel of Fig. 6. Adding noise to the stimuli had similar consequences for the three listeners. This produced a degradation of thresholds for the envelope task, but had no effect for the fine-structure task. A three-way ANOVA performed on the logarithms of the threshold estimates confirmed the existence of a significant interaction between the "IPD type" and "context" factors [F(1,48)=36.3, P<0.001].

In experiment 3, ten threshold estimates were obtained for each cell of the design [3 subjects \times 2 IPD types (fine structure versus envelope) \times 2 intensities]. The results, displayed in the bottom panel of Fig. 6, were similar to those of experiment 2: reducing the sensation level of the stimuli did not markedly affect performance for the fine-structure task, but was definitely deleterious for the envelope task. An ANOVA confirmed the existence of a significant interaction between the "IPD type" and "intensity" factors [F(1,108)= 56.2, P < 0.001].

C. Discussion

In a previous study by Smoski and Trahiotis (1986), the ability of normal listeners to detect ITDs has been assessed using narrow-band sounds spectrally centered at 500 or 4000 Hz and presented either at 80 dB SPL—i.e., about 60 dB SL—or at 25 dB SL. At both frequencies, it was found that detection thresholds were poorer for the lower SL. However, the SL effect appeared to be markedly larger at 4000 than at 500 Hz. The latter finding is qualitatively consistent with our own results insofar as ITDs at 4000 and 500 Hz are, respectively, detected on the basis of envelope and fine-structure cues.

From the present results, two conclusions can be drawn with regard to the source of the hearing-impaired listeners' deficits observed in experiment 1. First, their deficit in the detection of envelope IPDs is probably due, at least in part, to the fact that the stimuli had, for them, a lower SL than for the normal group. Second, their larger deficit in the detection of fine-structure IPDs must originate, at least in part, from factors other than the elevation of their absolute thresholds.

IV. GENERAL DISCUSSION

The aim of this study was to test the hypothesis that one consequence of cochlear damage is a deficit in the sensitivity to the temporal fine structure of sounds. This hypothesis was tested by comparing the effects of cochlear damage on the detection of fine-structure IPDs and envelope IPDs. By making such comparisons in common spectral regions, using identical standard stimuli, we ensured that the two detection tasks would recruit the same cochlear cells for a given listener. In addition, it is reasonable to conjecture that the central mechanisms involved were also the same for both tasks. In support, two points should be made. First, at least for normal listeners and near threshold, the subjective cue permitting identification of the target stimulus presented on a given trial was spatial position for both tasks-a lateralization of the target on the left. Second, Colburn and Esquissaud (1976) and Bernstein and Trahiotis (2002) have explicitly suggested that ITDs in the fine structure of sound wave forms and in their envelopes are processed by one and the same binaural mechanism. Bernstein and Trahiotis (2002) argued that the detection of both types of ITDs can be accounted for by a model based on normalized interaural correlations computed subsequent to known stages of peripheral auditory processing (augmented by a realistic low-pass filtering of envelopes). It should be pointed out, however, that Stellmack et al. (2005) recently questioned the validity of this model for the detection of envelope IPDs.

In our group of hearing-impaired listeners, we found a large variability of performance, in line with previous studies of binaural processing by similar populations (e.g., Hawkins and Wightman, 1980; Gabriel *et al.*, 1992). We also found a global deficit in the detection of envelope IPDs, which could be ascribed in part to the elevation of these listeners' absolute thresholds. However, a markedly larger deficit was observed for the detection of fine-structure IPDs, and this was not expected on the basis of the elevation in absolute threshold. Therefore, it does seem warranted to conclude from the present research that cochlear damage produces, independently of its deleterious effect on absolute thresholds, a deterioration in the monaural encoding of temporal finestructure. A similar suggestion had been made before, on the basis of different data and more speculatively (Hall *et al.*, 1984; Buus *et al.*, 1984; Lacher-Fougère and Demany, 1998; Moore and Skrodzka, 2002; Buss *et al.*, 2004).

In our experiments, the IPD-type factor (fine structure versus envelope) was combined with a periodicity factor: the fine-structure cycles (1–4 ms) were always shorter than the envelope cycles (20–50 ms). This was essentially unavoidable. Should one interpret the main finding as an effect of periodicity rather than as an effect of IPD type? Against such a view, the deficits observed in the impaired listeners for the detection of fine-structure IPDs were not stronger at 1000 Hz than at 500 or 250 Hz (see Fig. 4 and Table II). This suggests that the crucial factor was IPD type *per se*. In cases of co-chlear damage, presumably, pure tones tend to have an abnormal temporal representation at the AN level.

How could this happen? A straightforward idea is that, in consequence of cochlear damage, the precision of phaselocking in individual AN fibers is reduced. However, as pointed out in Sec. I, the results of two physiological studies (Harrison and Evans, 1979; Miller et al., 1997) do not support this notion. Thus, another possible scenario should be looked for. In this regard, Buss et al. (2004) suggested that cochlear damage is rather often associated with a reduction in the number of inner hair cells that are responsive to sound, and that reductions in performance are due to the fact that there are fewer channels providing information. A decrease in the number of responsive AN fibers could indeed lead to a poorer sensitivity to temporal fine structure because the responses of distinct AN fibers to a tone are statistically independent point processes (Johnson and Kiang, 1976): if, for instance, one fiber does not respond to a given cycle of the tone, a neighboring fiber may not miss this cycle. The inputs to the binaural neurons do not come from AN fibers but from the anteroventral cochlear nuclei. At this level, remarkably, Joris *et al.* (1994) have found that phase-locking is typically more accurate than in AN fibers. This improvement presumably requires a convergence of AN inputs, which might be reduced in case of damage to the inner hair cells.

In addition, as pointed out by Moore and Skrodzka (2002), an optimum combination of the fine-structure temporal information conveyed by separate AN fibers may require a specific traveling wave pattern on the basilar membrane, and cochlear damage is liable to modify significantly the normal pattern. The second part of this hypothesis is consistent with the results of recent studies concerning the consequences of cochlear damage for the masking of pure tones by harmonic complexes with low fundamental frequencies (Summers and Leek, 1998; Oxenham and Dau, 2004). In normal listeners, the magnitude of masking is strongly dependent on the relative phases of the masker's components, and the phase relationships producing maximum and minimum masking apparently correspond to uniform phase curvatures of the masker. In cochlear hearing-impaired subjects, on the other hand, the effect of masker phase curvature on the magnitude of masking is much weaker. This might simply originate from a reduction of cochlear compression in case of cochlear damage. However, Oxenham and Dau (2004) proposed an interesting alternative interpretation. They argue that, in hearing-impaired listeners, the phase response of the cochlea itself could be markedly nonuniform. If this were true, large variations in masking would not be expected from maskers with variable but always uniform phase curvatures.

The validity of the above-mentioned hypotheses is uncertain. Further research is obviously needed to determine precisely why cochlear lesions affect the perceptual sensitivity to the temporal fine structure of sounds.

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