A search for central lateral inhibition

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(Received 5 July 1983; accepted for publication 11 October 1983)

A central spectrum explanation of the existence of the binaural edge pitch [Klein and Hartmann, J. Acoust. Soc. Am. **70**, 51–61 (1981)] appears to require central lateral inhibition in the human auditory system. We have looked for this effect in central masking experiments. Using the binaural-edge noise, which creates the binaural edge pitch, as a masker (in standard notation: No below and Nπ above a phase boundary frequency), we measured pulsation thresholds for sine tones in two frequency ranges where the binaural edge pitch exists. We also obtained reference data, using the same pulsation threshold technique, for standard binaural conditions NoSo, NoSπ, NπSo, and NπSπ. These data revealed masking level differences. Theoretically the difference between the binaural-edge thresholds and the reference data should show the peak and valley signature of lateral inhibition. No such structure was found. We suggest that this negative result does not exclude the possibility of central lateral inhibition, but that the time course of central lateral inhibition makes the pulsation threshold technique an inappropriate means for observing the effect.

PACS numbers: 43.66.Ba, 43.66.Hg, 43.66.Pn, 43.66.Rq [JH]

INTRODUCTION

A. Noise-band edge pitch and noise-edge masking

If one listens to a low-pass band of noise with a sharp spectral edge one hears a sensation of pitch, similar to the pitch of a weak sine tone of frequency lower than the noise-edge frequency. Similarly a high-pass noise band elicits a pitch sensation above the edge frequency of the noise (Small and Daniloff, 1967; Fastl, 1971; and the review by Bilsen, 1977). According to the most precise measurements of this effect (Klein and Hartmann, 1981) the pitch is shifted into the noise by about 4%–10% of the noise-edge frequency.

Small and Daniloff found an attractive explanation for this noise-edge pitch effect in the model of lateral inhibition among tuned neurons, championed by von Bekesy (1959, 1960). According to this model, a peak appears in the plot of neural firing rate versus tonotopic coordinate because there is a release from inhibition. The neurons which respond maximally to frequencies just outside the noise band are only weakly excited by the noise and hence fail to inhibit their neighboring neurons with characteristic frequencies just inside the noise band.

Lateral inhibition had been proposed originally as a neural sharpening mechanism, a process which appeared to be required to reconcile the sharp tuning of the auditory system as revealed psychophysically with the broadly tuned early measurements of basilar membrane displacement. The observation of the noise-edge pitch gave support to the hypothesis of lateral inhibition in hearing.

Given the hypothesized lateral inhibition and the support from the noise-edge pitch experiments it was natural to search for the effect in masking experiments (Carterette *et al.*, 1969; Rainbolt and Small, 1972). The experiments proceeded from the assumption that the noise masking at a given frequency should be proportional to the neural excitation created by the noise at the corresponding tonotopic coordinate. One therefore expected to see a peak in masked threshold for signals at the edge of noise bands. However, no reproducible demonstration of the expected masking effect was found until Houtgast (1972, 1974) employed nonsimultaneous masking techniques. In forward masking and, to a greater extent in pulsation threshold measurements, Houtgast found the structure expected from an excitation pattern which includes effects of lateral inhibition. The enhanced masking observed in these nonsimultaneous masking experiments was subsequently termed "suppression" because the psychoacoustical experiments did not, in fact, provide direct evidence of neural interaction. [The effects could well result from a nonlinear combination of spectral components in the mechanics at the organ of Corti (Sellick and Russell, 1979).]

B. Binaural edge pitch and binaural-edge masking

In 1981 Klein and Hartmann reported a dichotic pitch effect, similar to the Huggins pitch, in which a sensation of pitch is created from white noise having a special choice of interaural phase angles. The effect, called binaural edge pitch (BEP), was created by noise with an interaural phase of zero for noise components below a phase boundary frequency and with an interaural phase of 180° above the phase boundary frequency [Fig. 1(a)]. Pitch-matching experiments revealed a bimodal distribution of pitch matches, 4%–10% above and 4%–10% below the phase boundary.

The binaural edge pitch found a ready interpretation by application of two hypotheses. First, according to the equalization–cancellation model (Durlach, 1972) the binaural system can add or subtract the noise signals to the two ears. The result of the equalization–cancellation process is to create a central spectrum with a sharp edge at the phase boundary frequency. If the signals to the two ears are added the central spectrum is a low-pass noise [Fig. 1(b)]; if the two signals are subtracted the central spectrum is a high-pass noise [Fig.

a) Work partially supported by the NIH, Grant NS 17917.
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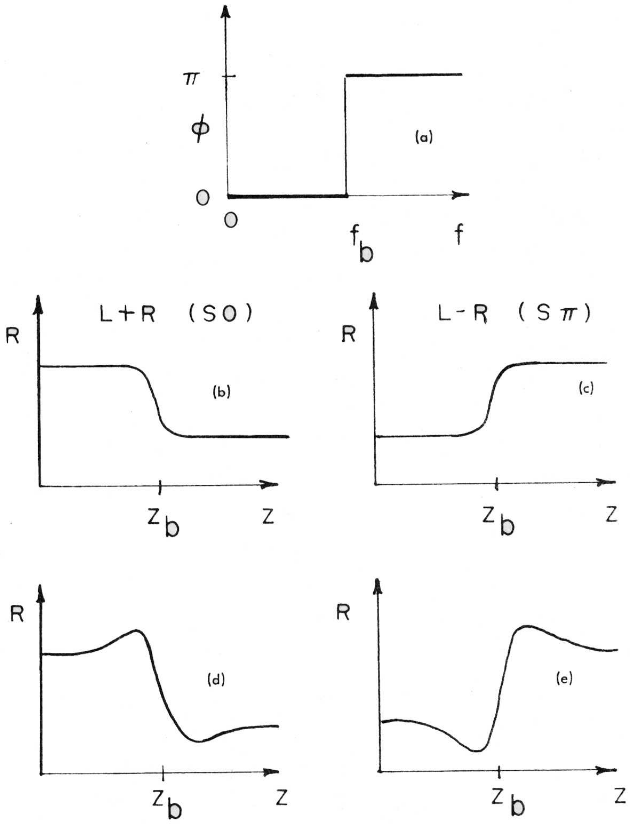

FIG. 1. (a) Interaural phase angle versus frequency for binaural-edge noise. (b) Central noise spectrum for homophasic signals according to the equalization–cancellation model. (c) Central noise spectrum for heterophasic signals according to the equalization–cancellation model. (d) Central noise spectrum of part (b) enhanced by central lateral inhibition. (e) Central noise spectrum of part (c) enhanced by central lateral inhibition.

1(c)]. The second step in the interpretation of the BEP was to invoke the hypothesis of lateral inhibition. According to that model neurons in the central auditory system interact so as to produce a central spectrum with a peak and valley structure as shown in Fig. 1(d) and (e). The bimodal nature of the experimental BEP matches was attributed to a fickle binaural system which sometimes adds and sometimes subtracts the signals to the two ears. Evidence in support of this explanation of the BEP came from the fact that the peaks of the bimodal distribution of the BEP coincided perfectly with the pitch matches to simple diotic or monaural low-pass and high-pass noise in the region of best binaural interaction, 300–800 Hz.

Given the evidence for central lateral inhibition from the BEP matching experiments, and given some physiological evidence that central lateral inhibition exists (Klinke *et al.*, 1968; Moore and Caspery, 1982), one may well ask whether the effect can be seen in a masking experiment. The resulting search for a manifestation of central lateral inhibition in masking is the topic of this paper.

I. METHOD

A. Procedure

Our method closely followed the procedure used by Houtgast (1974). A sine tone signal of given frequency f_s alternated with a noise. The subject's task was to adjust the level of the sine signal to the largest value for which the pulsating character of the tone disappeared and the tone sounded continuous.

The signal and noise had equal durations, 105 ms, and equivalent onsets and offsets, which were raised cosines of 20-ms duration. The beginning of the offset of the noise (signal) coincided with the beginning of the onset of the signal (noise). The overall cycle time for the signal and noise sequence was, therefore, 250 ms. Further, every fourth signal was omitted, a variation which Houtgast found to facilitate the level adjustment.

In a given experimental run the noise was always fixed. The sine tone frequency was the experimental variable. The frequency was not swept; it increased by equal steps on a logarithmic scale, from trial to trial. Each run comprised 22 adjustment trials for 22 different signal frequencies. For each noise and signal combination there were five runs, done successively. The data given in Sec. II for each subject represent averages over the five runs.

B. Stimuli

The sine signal and the noise were generated digitally by the 16-bit 4C synthesizer at the Institut de Recherche et Coordination Acoustique/Musique. The synthesizer was run by a PDP 11/34 computer, which also collected the response data. The noise consisted of 1000 equal-amplitude random-phase components equally spaced in frequency by 3.9 Hz. The largest frequency was thus 3900 Hz. The sampling rate was 16 kHz. The stimuli were low-pass filtered at 8 kHz and routed to a Neve console, which permitted control of channel level and phase. Subjects listened to the stimuli over AKG K242 headphones. The noise spectrum level was 34 dB.

C. Subjects

Three subjects B, D, and S participated in the experiments. Subjects were males with normal hearing and were experienced in psychoacoustical tasks. All subjects could match a binaural edge pitch with a standard deviation of only a few percent. Subject B was the author.

D. Experiments

To obtain the data for the analysis we did experiments as follows:

1. Low-pass noise masking experiments

The low-pass noise experiments were essentially replications of the pulsation threshold experiments of Houtgast (1974), except for the frequency of the noise edge. Noise and signal were both homophasic. The noise was cut off sharply at an edge frequency of 600 or 300 Hz, to make a low-pass masking band. In practice the low-pass noise was generated by adding the two channels of the Bo noise (see below). The resulting noise spectrum had a discontinuous 40-dB drop at the boundary frequency, i.e., over a range of 3.9 Hz.

The low-pass noise experiments do not play an essential role in our search for central lateral inhibition. We ran the experiments because pulsation thresholds for some subjects do not exhibit suppression effects (Houtgast, 1982). We wanted to make sure that none of our subjects was in that category.

2. Masking level difference (MLD) experiments

There is a sizable literature on MLDs in forward and backward masking conditions. A list of references is given in the paper by Yost and Walton (1977). We are aware of only one report on MLD measured by pulsation threshold, an abstract by Soderquist (1981). Our pulsation threshold MLD runs are, therefore, of some interest in themselves, though their primary purpose in the present context is to serve as normalization for the binaural-edge experiments. Experimental conditions, in standard notation, were: NoSo, NoSπ, NπSo, and NπSπ.

The MLD experiments were done in two experimental ranges, 600 and 300 Hz. The noise was identical for those two ranges, only the signal frequencies, chosen to match the frequencies of the binaural-edge experiments below, were different. Only two subjects participated in the runs in the 300-Hz range. In the paragraphs which follow we use the term "MLD experiment" to refer to measurements of pulsation threshold made with a noise masker which was either homophasic or heterophasic at all frequencies.

3. Binaural-edge masking experiments

The binaural-edge masking experiments provide primary data for our analysis. They were identical to the MLD experiments, with the exception that the noise masker was the dichotic noise which produces the binaural edge pitch [Fig. 1(a)]. Noise condition Bo was defined as a dichotic noise in which spectral components below the phase boundary frequency were homophasic and components above the phase boundary frequency were heterophasic. The phase discontinuity, therefore, was 180°/3.9 Hz. Condition Bπ was obtained from condition Bo by reversing the phase of the noise to one ear. Components below f_b were heterophasic, components above f_b were homophasic. The experimental conditions were: BoSo, BoSπ, BπSo, and BπSπ. The binaural-edge experiments were done with two values of the phase boundary frequency, 600 and 300 Hz. Only two of the subjects participated in the 300-Hz experiment.

II. RESULTS

Our analysis of the data was done in the following way: From the five runs for each condition we found a mean threshold and a variance for each frequency. To represent threshold differences between two experimental conditions we subtracted the means, and found the square root of the sum of the variances for each frequency to estimate the standard deviation of the difference. Plots of the resulting differences were used to draw the inferences in the paragraphs which follow. For presentation in this paper the data from different subjects or different conditions appear on the same graph with only a single error bar for each subject and condition. The lengths of these error bars are twice the standard deviation, as computed above, averaged over the signal frequencies. The actual errors varied rather randomly over the signal frequencies, but tended to grow with increasing signal frequency so that errors at the highest frequency were 1.5 or 2 times larger than errors at the lowest frequency. However, the average error shown in the graphs gives an adequate image of the detail in which inferences can be made.

The actual experimental signal frequencies were equally spaced on a logarithmic scale. The graphs shown here have a linear frequency scale for the horizontal axis, a scale which gives a clearer image of the data.

A. Low-pass noise masking

Pulsation thresholds for low-pass noise with edge frequencies of 600 and 300 Hz are shown in Fig. 2. Thresholds are given with respect to thresholds for broadband noise, the NoSo condition. The spectrum level was 34 dB in all cases except for subject D in the 600-Hz range; there the spectrum level was 24 dB.

Figure 2 shows clear evidence of lateral suppression. The peaks in the pulsation threshold data are larger than the standard deviations in each case.

B. Masking level differences

Figures 3–5 show pulsation threshold MLDs as a function of frequency, all referenced to NoSo. Values of NoSo itself in dB SPL for the leftmost point on each graph were as follows: for the 600-Hz region, subject B, 51.4; subject D, 62.8; subject S, 56.1; for the 300-Hz region, subject B, 53.4; subject S, 53.8. Figure 3 shows NoSo − NoSπ. The average behavior appears to resemble the MLDs for simultaneous masking collected by Durlach (1972) except that the pulsation threshold MLDs are about 5 dB smaller.

Figure 4 shows NoSo − NπSo. The average behavior resembles the MLDs for simultaneous masking collected by Durlach except that the pulsation threshold MLDs are

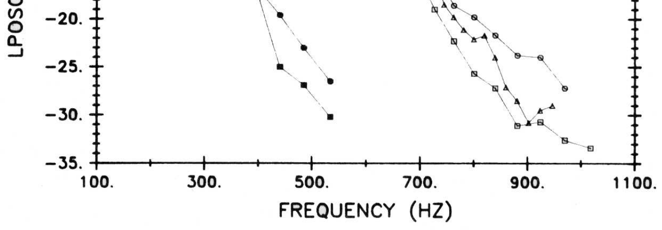

FIG. 2. Pulsation thresholds versus signal frequency for subject: B—circles, D—triangles, S—squares, for low-pass noise with edge at: 600 Hz—open symbols, 300 Hz—filled symbols. Data are referenced to thresholds for a white noise masker (NoSo). The spectrum level was 34 dB except for open triangles where the spectrum level was 24 dB.

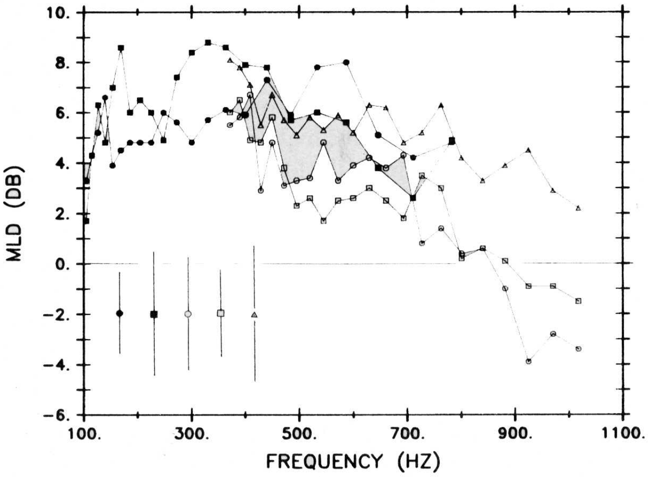

FIG. 3. Masking level difference NoSo − NoSπ versus signal frequency. Symbols are those from Fig. 2. All spectrum levels were 34 dB.

about 3 dB lower. For all subjects in all conditions NoSπ results in a larger MLD than does NπSo; the average difference is 2.7 (1.0) dB. Both NoSπ and NπSo curves show a peak. For NoSπ the peak is near 300 Hz; for NπSo the peak is near 500 Hz.

Figure 5 shows NoSo − NπSπ. According to simultaneous masking data collected by Durlach and according to the equalization–cancellation model this difference should be zero. Our data are not inconsistent with the expectation that this difference is, in fact, zero.

C. Binaural-edge masking

1. Model

Our report of the results for the binaural-edge masking experiments is based upon a heuristic model of the supposed lateral inhibition and its effect upon pulsation threshold. The model is illustrated by Fig. 6, where the four graphs show the possible combinations of binaural-edge noise and signal. The horizontal axis shows signal frequency f_s, and the vertical axis shows pulsation threshold according to the

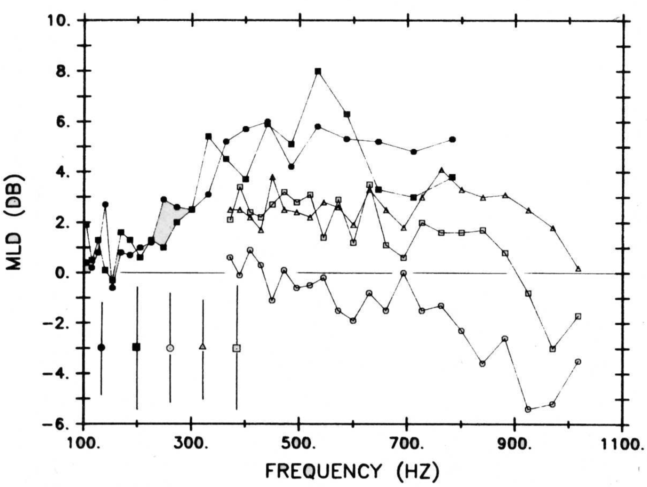

FIG. 4. Masking level differences NoSo − NπSo versus signal frequency. Symbols are those from Fig. 2. All spectrum levels were 34 dB.

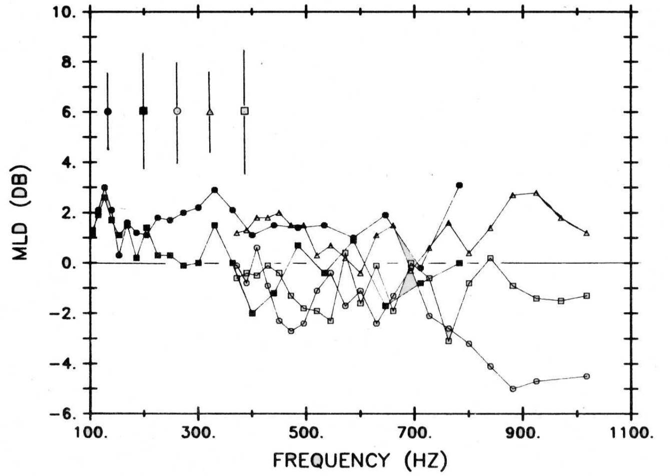

FIG. 5. Masking level diference NoSo − NπSπ versus signal frequency. Symbols are those from Fig. 2. All spectrum levels were 34 dB.

model. The model includes the assumption that if the signal is So then the binaural system must add the signals to the two ears to obtain optimum signal-to-noise ratio. Similarly, if the signal is Sπ, the binaural system must subtract. Thus, unlike the binaural edge pitch experiment, a binaural-edge masking experiment forces the binaural system into one mode or the other. Because our masking experiment employed nonsimultaneous masking, it is not obvious that this assumption is correct. However, the very existence of binaural MLDs in forward and backward masking, and of MLDs in pulsation thresholds, as reported above, suggests that the assumption is a reasonable one.

The model in Fig. 6 incorporates the following ideas. When the signal frequency is well away from the phase boundary frequency, the threshold is simply the threshold from the MLD experiment. We suppose that NoSo and NπSπ thresholds are the same, and we have, for the moment, neglected any frequency dependence of the MLD experiment thresholds. Near the phase boundary there are peaks and valleys caused by four lateral inhibition effects as follows:

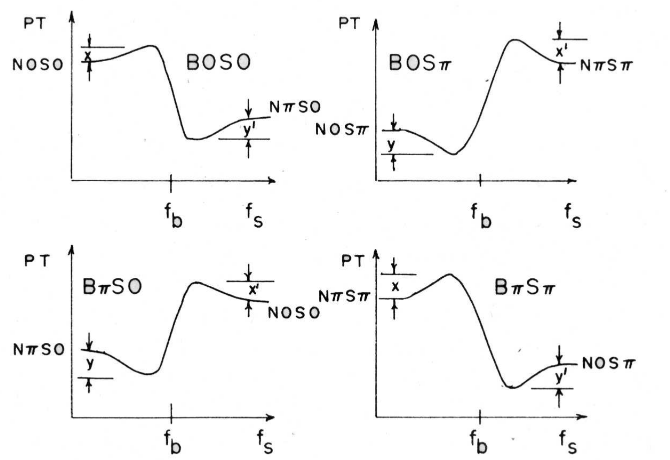

FIG. 6. Expected pulsation thresholds versus signal frequency for four conditions of binaural-edge masker and signal, derived from a model of central lateral inhibition (see text).

x: release from inhibition by absence of noise energy above c.f.

x′: release from inhibition by absence of noise energy below c.f.

y: inhibition by noise energy above c.f.

y′: inhibition by noise energy below c.f.

2. Normalized graphs

We define a normalized graph as the difference between pulsation thresholds with a binaural-edge masker and pulsation thresholds with a masker having no phase boundary, the normalizer. Interaural phases for the noise and signal of the normalizer are chosen to correspond to either the low-frequency or the high-frequency limit of the experiment with the phase boundary.

Each of the four binaural-edge masking experiments thus requires two normalized graphs. For example, the normalized graphs for the BoSo condition are BoSo − NoSo below the phase boundary frequency and BoSo − NπSo above the phase boundary frequency. If there is central lateral inhibition then the first of these graphs should have a peak just below the phase boundary frequency, caused by the release from inhibition of central fibers by the lack of in-phase noise excitation above the phase boundary for noise Bo. Similarly the second normalized graph should have a valley just above the phase boundary frequency caused by inhibition of central fibers by the presence of in-phase noise excitation below the phase boundary. Such in-phase excitation is present in the case of BoSo and not present in the case of the normalizer, NπSo. The normalized graphs are as follows:

$N1$: BoSo − NoSo

$N2$: BoSo − NπSo

$N3$: − BoSπ + NoSπ

$N4$: − BoSπ + NπSπ

$N5$: − BπSo + NπSo

$N6$: − BπSo + NoSo

$N7$: BπSπ − NπSπ

$N8$: BπSπ − NoSπ.

The odd-numbered graphs ($N1$, $N3$, ...) are expected to have a peak below the phase boundary. The even-numbered graphs should have a valley above the phase boundary.

We plotted the normalized graphs for the five experiments, subjects B, D, and S at 600-Hz phase boundary and subjects B and S at 300 Hz. Of the 40 graphs 13 had a peak or a valley as expected, 12 had no peak or valley, and 15 had curvature opposite to the structure expected. Successes and failures in this box score appeared to be randomly distributed among subjects and conditions. Normalized graphs for subject B for both phase boundary frequencies are shown in Fig. 7; they show the absence of any significant structure near the phase boundaries.

III. DISCUSSION

A. Low-pass noise masking

All of the pulsation threshold curves in Fig. 2 show evidence of suppression. The suppression peaks are 3–5 dB in height. These can be compared with the 10-dB peaks obtained by Houtgast (1974) with a 1100-Hz noise-edge frequency, a spectrum level of 36 dB, and timing parameters similar to ours. One expects the spectrum level to be an important factor in determining the size of the suppression peak because of the nonlinear growth of suppression with masker level. We have found it possible to obtain suppression peaks as high as 10 dB (subject B) in our experiment by raising the noise spectrum level by 10 to 44 dB. Further, the smallest peak in Fig. 2 occurs for subject D, where the spectrum level was 24 dB, 10 dB lower than for the other experiments. Our suppression peak frequencies correspond well with Houtgast's results; they are about 0.7 critical-bandwidth units below the noise-edge frequency.

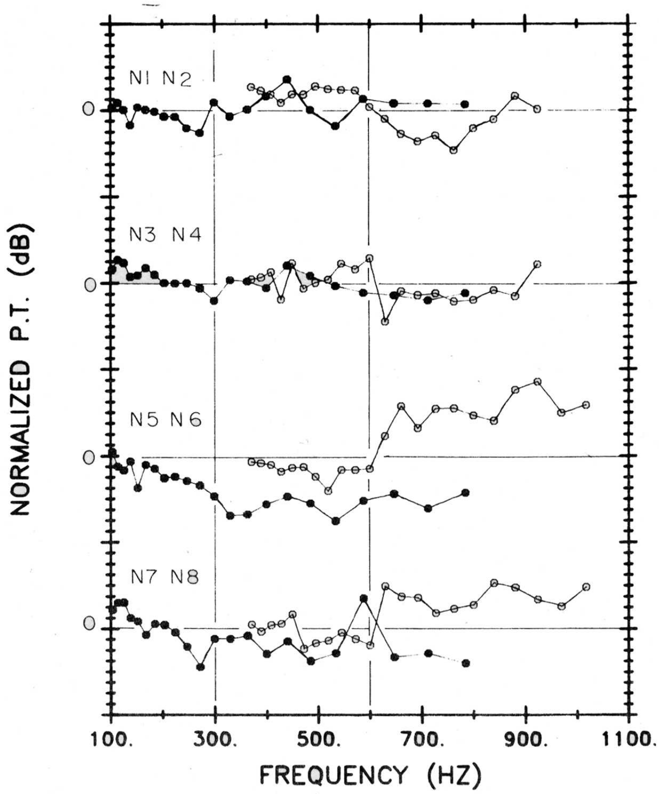

FIG. 7. Normalized graphs for subject B, the difference between pulsation thresholds for binaural edge noise and pulsation thresholds for an MLD experiment with signal and noise conditions corresponding to the low- and high-frequency limits of the binaural edge experiment. Normalizing data sets from the MLD experiments are switched at the phase boundary frequencies. Filled symbols and open symbols are for phase boundary frequencies of 300 and 600 Hz, respectively. For definitions of the normalized graphs $N1$–$N8$ see the text. Each vertical division equals 1 dB. Average errors are about 3 dB at the lowest frequencies and about 6 dB at the highest frequencies.

B. Masking level difference

The appearance of Figs. 3–5 for the pulsation threshold MLDs is somewhat marred by the data of subject B. Figure 4 for NπSo actually shows a negative MLD for almost all frequencies. The origin of the problem is an anomalously low threshold for a block of runs for NoSo, which, as the common reference, afflicts all the MLD graphs. Apart from this offset, B's data have the same frequency-dependent shape as the data for the other subjects.

There is a consistent discrepancy for $N\pi So$ between thresholds in the 300-Hz range and thresholds in the 600-Hz range for subjects B and S. There is no reason why this should be the case; noise and signal parameters were identical for both ranges.

In general, however, the MLD data are consistent with what one would expect. The thresholds themselves in dB SPL agree with those found by Soderquist (1981) at 500 Hz. The MLDs are 3–6 dB smaller than those obtained with simultaneous masking, a result which is consistent with other studies of MLDs in noise-temporal experiments, e.g., Small *et al.* (1972).

Although there are differences among subjects in the actual values of the MLDs the frequency dependence is rather similar in all cases. Smooth curves drawn through the data in Figs. 3 and 4 for NoSo $-$ NoSπ, and NoSo $-$ NπSo have broad maxima at 300 and 500 Hz, respectively. What is remarkable about these data is their close resemblance to the frequency dependence found in simultaneous masking [see Durlach's (1972) Figs. 4 and 12, respectively]. The fact that the frequency-dependent signature of the MLDs survived the radical change in method strikes us as little short of amazing, and indicates the robust nature of the MLD effect. Our data for the NoSπ condition show a decrease in MLD for frequencies below 200 Hz, a result in conflict with the equalization–cancellation model, but in agreement with some of the simultaneous masking data reviewed by Durlach.

C. Binaural-edge masking

Experiments with the binaural-edge masker are of paramount interest because they test the hypothesis of central lateral inhibition.

1. Normalized graphs

The normalized graphs, as defined in Sec. II, reveal the differences between the thresholds in a binaural-edge masking experiment and the thresholds obtained in a standard MLD experiment, where the signal and noise phase conditions of the MLD experiment correspond to the high- and low-frequency limiting conditions of the binaural-edge experiment. The normalized graphs are so named because the frequency dependence of the MLD is automatically canceled in the differencing operation. The normalized graphs are as close as we can come to the alternative pulsation threshold experiment in which the signal frequency is fixed and the phase boundary frequency is varied. (Note: The advantage of our constant phase boundary frequency is that the inhibition region, which we are trying to map with the sine probe, is a constant one throughout the experiment.)

The summary of the 40 normalized graphs at the end of Sec. II indicates that for every normalized graph which showed the expected peak or valley structure, there was another normalized graph which showed the opposite structure, and still another which showed no structure. We conclude that the observed structure represents only experimental error. The normalized graphs suggest that the effects of central lateral inhibition are nowhere present in our data.

2. Difference graphs

From the model curves of Fig. 6 it is clear that the most sensitive test for central lateral inhibition is to take differences between data such that the expected inhibition valleys are subtracted from the peaks, where there is release from inhibition. This procedure involves no normalization by data from the MLD experiments. For example, a graph of BoSo $-$ BoSπ should have a peak below the phase boundary frequency, equal to $(x + y)$ and a valley above the phase boundary, equal to $(x' + y')$. We referred to graphs constructed by this differencing procedure as "difference graphs." There are four possible difference graphs, and we studied them in some detail for the three subjects in the 600-Hz range and the two subjects in the 300-Hz range. Some of the peaks and valleys predicted by the model illustrated in Fig. 6 appeared as expected and others did not. There was considerable uniformity among subjects, for a given frequency range, regarding the appearance or nonappearance of the expected structure.

It eventually became clear that the structure observed in the difference graphs could be explained without the hypothesis of central lateral inhibition. The explanation involved two ideas. First, a binaural-edge masking experiment presents a smooth transition from one form of MLD experiment to another. Second, each form of MLD experiment retains its frequency-dependent signature when incorporated into a binaural-edge masking experiment. The smooth transition represents the fact that for target tones near a phase boundary frequency some of the masking noise is homophasic and some of it is heterophasic. To fit our data we required a transition region width of about 2 critical bandwidths, which seems reasonable in view of the reported enlargement of critical masking bands for binaural effects, e.g., Sever and Small (1979).

There are essentially two different MLD frequency dependences involved, occurring in MLDs of different sizes. When these were subtracted appropriately on either side of the phase boundary we obtained a reasonable fit to the structure observed in the difference graphs. Thus, in the end, the difference graphs, like the normalized graphs, provided no evidence of central lateral inhibition.

IV. CONCLUSION

The clear conclusion of our search for lateral inhibition is that we have not seen the anticipated effect. The normalized graphs revealed only random error, the difference graphs revealed only artifacts associated with the frequency dependence of the MLD.

The easiest interpretation of our results is that the effect simply does not exist. There is no central lateral inhibition. The difficulty with this interpretation is that the binaural edge pitch clearly does exist. The perfect coincidence between the BEP and monaural noise-edge pitches suggests that the BEP may be derived from a central spectrum which is created by the equalization cancellation process. However, that explanation requires that there be central lateral inhibition.

There are alternatives to the equalization–cancellation central spectrum model for explaining the detection of the BEP. The neural cross correlator suggested by Licklider in 1959 is such a possibility, but, as noted by Klein and Hartmann (1981), that model in its present form does not predict the pitch shift away from the phase boundary frequency. It may be that an improved cross-correlator model, incorporating physiologically realistic coincidence cells rather than mathematically ideal cells, could predict the observed bilateral frequency shifts.

The tapped-interaural-delay-line model, introduced by Bilsen (1977) and Raatgever and Bilsen (1977), provides another possibility. According to that model, the binaural system constructs a continuum of central power spectra parameterized by different internal interaural delays. The model allows one to decouple the BEP from the masking experiments of this paper, because detection of the BEP and detection of the signal in a masking experiment could be mediated by different interaural delay channels. Therefore, the existence of the BEP does not necessarily imply structure near the phase boundary.

Alternatively one may suppose that the equalization–cancellation central spectrum model for the BEP is correct, that central lateral inhibition does exist, but that our experimental search for it was done in the wrong way. The factors of noise spectrum level and bandwidth, noise and signal timing, envelope and overlap are known to have significant effects on thresholds in nonsimultaneous masking measurements of suppression (Verschuure *et al.*, 1976; Schreiner *et al.*, 1977; Moore, 1981; Yama, 1982). Indeed the problem is not one of finding an explanation for our negative result, but rather one of deciding which of several possible explanations is most plausible. Several scenarios follow:

(1) Central lateral inhibition exists but only at the onset of a stimulus. Pulsation thresholds, on the other hand, are determined by a noise excitation level averaged over the noise interval in some way which gives no special weight to the onset portion. Therefore, an experiment with noise intervals as long as ours (100 ms) does not reveal the central lateral inhibition. This view is consistent with the results of the central masking study of Zwislocki *et al.* (1968), which do appear to show central inhibitory effects (small secondary threshold peaks on either side of the central maximum). The inhibitory effects there are much reduced 40 ms after the onset of the masking tone. Such an explanation does not necessarily contradict the observation that the binaural edge pitch persists throughout the 500-ms interval used in the pitch-matching experiment. A narrow spectral region, initially made prominent by central inhibition, might retain its prominence well after the effect which caused it to be noticed has vanished.

(2) Central lateral inhibition exists but its effects persist beyond the termination of the masker, thus inhibiting the signal as well. Therefore, central lateral inhibition is not seen in a pulsation threshold experiment. This view is consistent with the physiological study by Klinke *et al.* (1968) of inhibition in the cochlear nucleus by a contralateral tone. The inhibitory effects persisted 50–100 ms beyond termination of the contralateral tone. Such an effect was actually proposed by Soderquist (1981), in a monaural context, to account for his observed dependence of suppression on signal duration. Such an explanation would work best if pulsation threshold measurements gave particular weight to the onset of a stimulus, a hypothesis which does not seem unreasonable. (We note that the further observation by Klinke *et al.* that inhibition was followed by activation would appear to predict an enhancement of the valley and peak structure which we supposed to arise from inhibition and release from inhibition in the masker itself.)

It seems possible that further temporal masking studies, with different values of the timing parameters, and possibly a higher masker level, could enable one to decide which of the two above possibilities, if either, is correct. They might even finally reveal central lateral inhibition.

ACKNOWLEDGMENT

B. K. Smith programmed the 4C synthesizer to run the experiments of this paper.

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