The Effect of Temporal Stimulus Characteristics in Maintenance of the Acoustic Reflex

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ABSTRACT

In normal listeners, acoustic reflex decay (ARD) typically occurs for high- but not for low-frequency tones. In patients with acoustic neuromas, decay can be obtained at all frequencies, presumably due to poor neural synchrony. These observations have led us to hypothesize that resistance to decay is due to robust encoding of temporal fine structure of the eliciting stimulus. For a 4-kHz stimulus, ARD is reduced by sinusoidal amplitude modulation (SAM), a result attributed to the low-frequency pattern of SAM providing the temporal characteristics necessary to maintain the reflex. If this interpretation is correct, then further reductions in ARD should be seen for stimuli having temporal characteristics that even more closely resemble the neural response to low-frequency stimulus fine structure. On the other hand, if other perceptual qualities of a SAM tone are responsible for the effect (e.g., rate pitch), then manipulations of perceived sound quality, rather than temporal characteristics *per se*, should produce similar effects. The experiment reported here included a reference condition, (1) 5-kHz pure tone, and three “temporal” manipulations, composed of a 5-kHz tone multiplied by (2) a raised 100-Hz sinusoid, (3) a noise sample, lowpass filtered at 100 Hz, and (4) a half-wave rectified 100-Hz sinusoid. Additional conditions manipulated perceived pitch. These stimuli spanned 4.5–8 kHz, including a reference condition, (5) Gaussian noise, and a stimulus associated with a 100-Hz pitch, (6) iterated rippled noise. Results show

the greatest reductions in ARD with the half-wave rectified stimulus, thought to most closely mimic the temporal characteristics of a low-frequency tone. Little or no reduction in ARD was associated with the iterated rippled noise, suggesting that perceived pitch does not play an important role in maintaining the acoustic reflex.

Keywords: acoustic reflex decay, temporal coding

INTRODUCTION

The acoustic reflex is a stapedial muscle contraction elicited by a high-intensity stimulus. It has been hypothesized that the acoustic reflex serves a protective role by limiting the sound energy transmitted through the middle ear (Kobrak et al. 1941; Takahashi 1954; Hilding 1960; Simmons 1960; Mills and Lilly 1971; Karlovich et al. 1977; Zakrisson 1975, 1979). Diminution of the reflex in response to a sustained stimulus is known as the acoustic reflex decay (ARD). The ARD is most often quantified by eliciting the reflex with a continuous stimulus and measuring the associated change in middle ear admittance over time.

The extent of ARD in normal subjects depends on the frequency of the eliciting stimulus. While low-frequency tones are associated with little or no decay, frequencies of 2 kHz and higher are typically associated with substantial decay (Johansson et al. 1967; Habener and Snyder 1974; Wilson et al. 1978; Givens and Seidemann 1979). The degree of reflex decay may also be affected by the bandwidth of the eliciting

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stimulus. Octave band and wide band noise samples typically result in less decay than high-frequency pure tones (e.g., Wiley and Karlovich 1975; Klodd and Nearhoff 1977; Wilson et al. 1978). It is not clear at this point whether this effect is driven more by bandwidth *per se* or by the amplitude fluctuation characteristics of noise stimuli at the output of the auditory filters.

Acoustic reflex decay in patients with acoustic neuroma can be quite different from that observed in the normal auditory system. Patients with acoustic neuromas often demonstrate substantial decay, even when the eliciting stimulus is a low-frequency tone (Anderson et al. 1969, 1970). While this result is somewhat variable across patients, decay in response to low-frequency pure tone stimuli in conjunction with other symptoms is often followed up with MRI to rule out neuroma. Another clinical test result typical of acoustic neuroma is abnormal or absent auditory brain-stem response. This is generally thought to result from poor neural synchrony secondary to a mass impinging upon the auditory nerve (Sheehy and Inzer 1976; Selters and Brackmann 1977). Such observations may suggest that the resistance to decay observed for low-frequency tones in normal subjects is due to the robust encoding of temporal fine structure of the eliciting stimulus (Cook et al. 1999). In the case of high-frequency tones (Kiang et al. 1965; Rose et al. 1967) or in the case of acoustic neuroma (Selters and Brackmann 1977), encoding of temporal fine structure is presumed to be less precise, which may account for the observed decay.

Cook et al. (1999) provided some initial support for the hypothesis that maintenance of the acoustic reflex is associated with the fidelity with which temporal features of the eliciting stimulus are encoded. In that study, the acoustic reflex was elicited using stimuli having a relatively high carrier frequency (4 kHz). Decay was substantially ameliorated by the introduction of sinusoidal amplitude modulation (SAM), suggesting that the amplitude fluctuation of the 4-kHz SAM tone restored some of the temporal information necessary to maintain the reflex. These results were discussed in terms of the similarities between the neural response to the fine structure of a low-frequency pure tone and the neural response to the envelope of a high-frequency SAM tone. By this interpretation, decay to the high-frequency stimulus was reduced by AM because of the periodic neural synchrony elicited by low-frequency modulation.

The conditions included in the present study were chosen to address issues following from the Cook et al. (1999) study. In that study, it was noted that although SAM reduced the ARD associated with a high-frequency carrier, the decay seen for the SAM stimulus was still greater than that typically found for a low-frequency tone. If acoustic reflex decay is related

to the temporal information encoded by the auditory system, then one possible explanation is that the quality of that temporal information is in some sense poorer for the high-frequency SAM stimulus than for the low-frequency tone. Interestingly, recent results of the binaural masking-level difference (van de Par and Kohlrausch 1997) and interaural time discrimination (Bernstein and Trahiotis 2000) paradigms suggest that this interpretation is reasonable. In those studies a high-frequency carrier was multiplied by a half-wave rectified modulator waveform, producing a stimulus that was intended to mimic the phasic inner hair cell response to a single polarity of stimulation. It is argued that timing information is encoded in essentially the same way in low- and high-frequency auditory nerve fibers (Colburn and Esquissaud 1976), and so the half-wave AM stimulus was hypothesized to result in an VIIIth nerve firing pattern similar to that associated with a low-frequency tone. Using such stimuli, van de Par and Kohlrausch (1997) and Bernstein and Trahiotis (2000) were able to obtain binaural performance based on a high-frequency envelope cue that was very similar to binaural performance associated with low-frequency fine-structure cues. A major goal of the present experiment is to apply the half-wave rectified modulation stimulus to the acoustic reflex decay paradigm. If temporal coding is the same in low- and high-frequency fibers (Colburn and Esquissaud 1976), and if acoustic reflex decay is truly related to the quality of the coded timing information, then the half-wave rectified modulation stimulus should result in less decay than the SAM stimulus.

While the AM manipulation tested by Cook et al. (1999) is consistent with the parallel between encoding of low-frequency fine structure and encoding of a slowly varying sinusoidal envelope, other possible explanations exist to account for the observed reductions in decay. For example, it is not clear from those results whether the periodicity of the AM envelope was a factor, or if some other nonperiodic envelope pattern would have produced comparable results. A high-frequency carrier modulated by a low-pass noise was used to assess this possibility. Periodic AM of high-frequency stimuli is often associated with a percept of low pitch corresponding to the envelope rate. In some cases the basis of this pitch is a low-frequency distortion present at the periphery, but in other cases it is thought to be extracted at a fairly high level of auditory processing (Ritsma 1962; Burns and Viemeister 1976). This factor raises the possibility that the virtual pitch quality of the SAM stimulus could have played a role in reducing reflex decay. To examine whether reflex decay is affected by a low perceived pitch, two further stimuli were employed in the present experiment. Both of the stimuli were

bandpass noises (filtered between 4 and 8 kHz). Whereas one of the bandpass noise stimuli was simply a Gaussian noise, the other was an iterated rippled noise (IRN; e.g., Yost 1982, 1996), which is associated with a perceived low pitch. If reflex decay is related to the low pitch associated with the stimulus, then decay should be less for the IRN stimulus than for the Gaussian noise stimulus. Gaussian noise of equal bandwidth was used as a baseline to compare decay for the IRN stimulus because increased bandwidth has been shown to be associated with some degree of resistance to decay compared to tonal stimuli (Wiley and Karlovich 1975; Klodd and Nearhoff 1977; Wilson et al. 1978).

In summary, the present experiment was meant to assess the stimulus features important for acoustic reflex decay. The features examined included the presumed temporal pattern of neural responses to the stimulus envelope (AM versus half-wave rectified AM), the presence of a low pitch related to the stimulus periodicity (Gaussian versus iterated rippled noise), and the regularity of periodicity (SAM versus lowpass noise AM).

METHODS

Subjects

Twenty-nine healthy adults (17 males and 12 females, 20–43 years, mean 25.6 years), with no history of middle or inner ear disease, served as paid subjects. All subjects had pure tone thresholds between –5 and 20 dB HL at octave intervals from 0.25 to 8 kHz and normal, single-peaked 226-Hz type A tympanograms. Subjects in whom reflexes were absent or of insufficient amplitude using a 5-kHz stimulus at a level of 115 dB SPL and subjects who demonstrated no ARD in response to a 5-kHz tone were excluded from the study. Fourteen of the 29 subjects were excluded based on these criteria. The use of adults as paid subjects was approved by the Human Subjects Institutional Review Board.

Stimulus generation

Stimuli were generated digitally and played out of a digital-to-analog converter (DAC) at 40 kHz. Each waveform was 2^{15} points long and was generated as an integer number of stimulus periods, such that continuous cycling through the waveform buffer did not introduce artifacts or discontinuities. All stimuli were highpass filtered at 4 kHz and lowpass filtered at 8 kHz (Kemo VBF8, Kemo Inc., Landrum, SC), after which the level was adjusted by means of a programmable attenuator. Stimuli were then routed to a headphone buffer and played out of a single circum-

aural earphone (TDH-50P, Telephonics Corp., Farmingdale, NY). Six different stimuli were utilized. Of these, the first 4 were spectrally narrow and varied primarily in terms of their temporal envelope. The final 2 types were spectrally broader and varied in perceived pitch quality. The spectrally narrow stimuli were (1) a pure tone centered on 5 kHz (*5-kHz*), (2) a pure tone centered on 5 kHz, amplitude modulated via multiplication with a raised sinusoid at 100 Hz (*SAM*), (3) a pure tone centered on 5 kHz, amplitude modulated via multiplication with a half-wave rectified cosine at 100 Hz (*HWR-AM*), and (4) a pure tone centered on 5 kHz, amplitude modulated via multiplication with a Gaussian noise sample lowpass filtered at 100 Hz (*LPN-AM*). These AM stimuli are illustrated in Figure 1 in both the time domain (left column) and the frequency domain (right column). The spectrally broader stimuli were (1) a Gaussian noise sample bandpass filtered between 4.5 and 8 kHz (*GN*), and (2) a sample of iterated rippled noise with 8 iterations at 10 ms delay intervals and 10 dB attenuation, digitally bandpass filtered between 4.5 and 8 kHz (*IRN*). These stimuli are depicted in Figure 2. All stimuli were ramped on and off with $5 \text{ ms } \cos^2$ ramps, imposed via programmable switches.

Procedures

Contralateral acoustic reflex measurements were obtained using a GSI-33 Middle Ear Analyzer (Nicolet Biomedical Inc., Madison, WI) with a probe frequency of 226 Hz. Reflexes were measured as a change in acoustic admittance in units of millimhos (mmho). The subject's measurement ear was fitted with the probe of the GSI-33, and one earphone of the TDK was fitted over the other ear. In order to control for a possible effect of reflex magnitude on decay measurements (Anderson et al. 1970; Wilson et al. 1978), stimulus intensity was adjusted up to a maximum of 115 dB SPL¹ such that the reflex amplitudes from a given subject were all within 0.02 mmho of one another and fell within a range of 0.05–0.10 mmho. For one subject reflex amplitudes between 0.05 and 0.10 mmho could not be obtained. However, all reflexes from this subject did fall within 0.02 mmho of one another and were relatively free of artifact. Given the stability of these traces they were included within the subsequent analysis of ARD. Reflex data were discarded from analysis if obvious artifact (e.g., swallowing, sneezing, movement) was apparent.

Acoustic reflex thresholds were obtained using the aforementioned criteria with a stimulus on-time of 2 s. Once threshold was established for a given stimulus

¹See Hunter et al. 1999 for a discussion of safe levels of stimulation.

FIG. 1. Schematics for each of the three spectrally narrow AM stimuli represented in the time domain (left column: amplitude vs. time) and frequency domain (right column; level, 10 dB/div, vs. frequency). Stimulus conditions are indicated at the far left of each row.

FIG. 2. Schematics for the two spectrally broad bandpass stimuli represented in the time domain (left column: amplitude vs. time) and frequency domain (right column: level, 10 dB/div, vs. frequency). Stimulus conditions are indicated at the far left of each row.

condition, acoustic admittance was then measured over a 30 s duration (10 s prestimulus baseline recording, 10 s stimulus on-time, 10 s poststimulus baseline recording). At least two measurements for each stimulus condition were obtained before changing stimulus conditions. The resulting reflex traces were recorded and saved to disk. This process was repeated for each stimulus condition in the following order: 5-kHz, SAM, LPN-AM, HWR-AM, GN, and IRN conditions. All testing was conducted in a single-walled sound booth with subjects sitting in an upright position.

Quantification of reflex decay

Data saved to disk from the GSI-33 were uploaded to MATLAB (MathWorks, Inc., Natick, MA) for further analysis. Each 30 s sample was represented with 217 points, such that the 10 s portion of the trace during which the stimulus played was represented by 72 points. Traces with salient motion artifacts were omitted based on visual inspection. Because stimulus onset was dependent on keyboard input and was not synchronously triggered with the recording hardware, the exact point of reflex onset was defined based on

characteristics of the trace. The reflex portion of a data trace was defined as the 10 s following the first point in the array for which sequential points deviated by 0.01 mmho or more, a criterion that was consistent with informal identification of the reflex portion of the trace. Baseline drift was evident in a small proportion of the traces and corrected for prior to the double exponential fit described below. Drift was quantified by comparing the average admittance during the 0–5 s interval of the prestimulus period with the average admittance during the 20–30 s interval of the poststimulus period. Data points comprising the reflex were adjusted linearly for this drift and fitted with a double exponential using a least squares method (Tietze 1969a, b), of the form:

$$z = 100 \frac{(e^{(-x/\tau_b)} - e^{(-x/\tau_a)})}{1 - (\tau_a/\tau_b)}$$

where τ is time, τ_a is the time constant associated with decay, and τ_b is the time constant associated with the initial impedance change at onset of the stimulus. Percent decay was then calculated based on the admittance change at stimulus onset and offset as determined by the double exponential fit.

RESULTS

Figure 3 shows an example of a reflex trace obtained in the *HWR-AM* condition. These data are relatively free of noise (e.g., artifact caused by breathing) and are typical. The dark line shows the best-fitting exponential.

Table 1 reports the average reflex decay as a percentage of the maximum admittance change calculated from the double exponential curve fit in each of the six stimulus conditions. Also presented in Table 1 are the values of the curve fit parameters, τ_a and τ_b , and the percent of variance in the reflex portion of the data that is accounted for by these fits. For the spectrally narrow stimuli, the 5-kHz condition was associated with the greatest amount of decay at 66.5%. Reduced decay was observed in the *LPN-AM*, *SAM*, and *HWR-AM* conditions, each with an ARD of 40.3%, 35.9%, and 24.5%, respectively. The ARD obtained for the two spectrally broad stimuli was quite similar, the *GN* condition being associated with 44.1% decay, while the *IRN* condition was associated with 45.9% decay. The ARD for the 5-kHz stimulus was greater than that for the *GN* stimulus, as expected based on the previously documented stimulus bandwidth effect. As indicated by the standard deviations of these estimates of ARD reported in Table 1, there was substantial variability across estimates. Figure 4 illustrates the extent to which that variability can be at-

FIG. 3. An example of discrete acoustic admittance data recorded in *HWR-AM* condition is plotted as a function of time (grey dots). The exponential fitted to these data is represented by the dark line.

tributed to consistent individual differences across subjects. Estimated ARD is plotted for each condition, with symbols representing mean data from individual subjects. Symbols within a condition are ordered according to ARD for the 5-kHz condition, from high to low. Data in the *SAM* and *HWR-AM* conditions fall roughly along the descending diagonal, reflecting the fact that much of the variance in these conditions is due to a consistent pattern of individual differences. No such pattern is evident in the *LPN-AM* condition, suggesting that more of the variance in this condition is due to other sources of variance. One possible factor could be the fact that a unique lowpass noise sample was used to generate each stimulus, such that differences across samples could contribute variance to the measurements.

A repeated measures analysis of variance (ANOVA) comparing the spectrally narrow stimuli indicated a statistically significant effect of stimulus condition ($p < 0.0001$). The resulting contrasts and associated p -values are presented in Table 2. In an attempt to reproduce the results of Cook et al. (1999) and demonstrate an effect of AM on ARD, a planned contrast was performed comparing the 5-kHz condition and the AM tones collectively with equal weighting. This contrast produced a statistically significant effect for AM ($p < 0.0001$). In order to test our original hypothesis of reduced decay for the half-wave rectified AM as compared to the *SAM* stimulus, a planned contrast was performed comparing the *SAM* condition with the *HWR-AM* condition. This contrast revealed that the *HWR-AM* condition was associated with less ARD than the *SAM* condition ($p = 0.03$). To explore the possible role of periodicity in the maintenance of the acoustic reflex, a contrast was performed comparing the *SAM* with the *LPN-AM* condition, comprised of an effectively nonperiodic pattern of AM. This comparison indicated no statistically significant difference in associated ARD

TABLE 1

ARD (%) and associated curve fit parameters for 15 subjects

	Narrow stimuli				Broad stimuli	
	5-kHz	SAM	HWR-AM	LPN-AM	GN	IRN
Mean ARD	66.5	35.9	24.5	40.3	44.1	45.9
(std)	(20.1)	(17.2)	(11.0)	(22.9)	(20.2)	(21.1)
τ_a	3.91	4.30	6.76	5.60	4.22	4.23
τ_b	97	205	429	231	151	152
% variance	63	54	69	54	63	62
(std)	(28.1)	(22.1)	(19.7)	(28.7)	(15.8)	(20.1)

FIG. 4. Mean ARD for each subject (symbols) is plotted for each of six stimulus conditions (x-axis). The order of symbols within each condition is determined by the rank order of ARD measured in the 5-kHz condition.

($p = 0.39$). An ANOVA comparing the spectrally broad conditions was performed to determine the significance of virtual pitch in ARD. This analysis revealed no statistically significant difference between the GN and the IRN conditions ($p = 0.68$). A paired t -test comparing the 5-kHz and GN conditions showed a significant difference in ARD ($p < 0.0005$).

DISCUSSION AND CONCLUSIONS

The results of Cook et al. (1999) suggest that maintenance of the acoustic reflex may depend upon the envelope features of the eliciting stimulus. This is based on the observation that high-frequency tones, which are typically associated with extensive ARD, become relatively resistant to decay with the introduction of SAM. If similarity between response to envelope modulations and response to low-frequency tones underlies the enhanced resistance to decay, then further envelope manipulations to enhance this similarity might be expected to confer additional resistance. In the experiment described here, the half-wave rectified AM tone produced less decay than the SAM tone having the same rate of modulation. The envelope of the half-wave rectified AM stimulus has a steeper slope with longer periods of zero voltage in comparison with the SAM tone, hypothesized to produce a neural response more closely resem-

bling response to the fine structure of a low-frequency tone.

The reduced decay produced by the half-wave rectified AM tone relative to the SAM tone is consistent with data previously obtained in studies of binaural processing (Bernstein and Trahiotis 2000; van de Par and Kohlrausch 1997). While those studies showed that binaural processing based on envelope temporal information is more sensitive for a half-wave rectified AM stimulus compared with an analogous SAM tone stimulus, performance in those studies did not attain the level obtained with low-frequency stimuli in most cases. Because of the low envelope rate used in the experiment described here, no attempt was made to measure ARD to a 100-Hz low-frequency stimulus; however, the ARD data of Wilson et al. (1984) suggest that low-frequency pure tones as low as 250 and 500 Hz are associated with smaller ARD than those found for the half-wave rectified AM stimuli used here (5–10% in the first 10 s compared with 24.5%). These findings are consistent with the hypothesis that processes dependent upon fine temporal coding of an acoustic stimulus can be simulated to some degree at high carrier frequencies, where fine structure is no longer available, by envelope manipulations such as half-wave rectified AM. The fact that results with the half-wave rectified AM tended to fall just short of those obtained with low-frequency tones in studies of binaural processing as

TABLE 2

<i>p</i> -values derived from planned contrasts	
Comparison	<i>p</i> < 0.05 (value)
5-kHz vs. [SAM, HWR-AM, LPN-AM]	Yes (<i>p</i> < 0.0001)
SAM vs. HWR-AM	Yes (<i>p</i> = 0.03)
SAM vs. LPN-AM	No (<i>p</i> = 0.39)

well as ARD could be due to peripheral processing factors, such as the effects of filtering and the phase response across auditory filters.

One question not addressed by Cook et al. (1999) was whether envelope periodicity or some other property of SAM is responsible for reductions in decay. The spectrally narrow AM stimuli tested here collectively did indeed reduce reflex decay compared with the unmodulated 5-kHz tone. The SAM tone was associated with reduced ARD relative to the lowpass noise AM (35.9% vs. 40.3%). This difference, however, was not found to be statistically significant. It is not clear whether the SAM tone and the lowpass noise AM are equally effective at maintaining the acoustic reflex, or whether subtle differences went undetected due to the limited sample size and the variance associated with selecting a new lowpass noise sample on each stimulus presentation.

Previous studies (e.g., Wiley and Karlovich 1975; Klodd and Nearhoff 1977; Wilson et al. 1978) have shown that stimulus bandwidth can play a role in the maintenance of the acoustic reflex; octave band and wide band noise stimuli result in less decay than do high-frequency pure tones. This is consistent with the results presented here, with greater ARD for 5-kHz as compared with the GN conditions, with 66.5% and 44.1% decay, respectively. Of greater interest here was the comparison among the spectrally narrow stimuli. Inspection of the frequency components of these stimuli presented in Figure 1 reveals subtle differences in their associated power spectra. However, such differences in bandwidth likely play little, if any, role in maintenance of the acoustic reflex since the spectral components are spread across a subcritical band, and so would likely result in similar excitation patterns across the basilar membrane (Moore and Glasberg 1987).

Another question left unanswered by Cook et al. (1999) was whether other factors, such as derived pitch, play a role in mitigating ARD. This issue was explored by comparing reflex decay elicited by a Gaussian noise sample and an iterated rippled noise, a stimulus associated with a pitch percept corresponding to the reciprocal of the delay used to produce the noise (in this case 100 Hz; Yost et al. 1998). Because these two stimuli were passed through the

same bandpass filter, their major difference in the context of this experiment stems from a difference in perceived pitch. A comparison of these spectrally broad noise samples revealed no difference in associated ARD. These results may suggest that perceived pitch alone plays little, if any, role in the maintenance of the acoustic reflex.

Evidence that acoustic reflex decay may be related to the coding of temporal information arises from several sources. One is that acoustic reflex decay increases with increasing stimulus frequency, a finding that is consistent with the reduction in neural phase-locking that occurs with increasing stimulus frequency (Kiang et al. 1965). Acoustic reflex decay to low-frequency tones is relatively common in patients with VIIIth nerve lesions, again consistent with interpretations that some abnormalities in auditory brainstem-evoked responses (e.g., Selters and Brackmann 1977) and reduced masking-level differences (e.g., Olsen et al. 1976) are due to poor neural synchrony in these patients. Finally, the present research initiative suggests that reflex decay for high-frequency carriers can be reduced by applying an amplitude modulation that is intended to result in temporal information similar to that associated with a low-frequency tone. These results suggest that measures of acoustic reflex decay may be a useful indicator of the quality of temporal coding. Tests of acoustic reflex decay that are currently used clinically are relatively coarse, the most common procedure being a determination of whether or not the amplitude of the reflex decays by half of its initial magnitude within 10 s of continuous stimulation. It is possible that more fine-grained analyses could be used to gain insights into processes related to hearing loss, neural dysfunction, or normal processes related to aging.

One possible mechanism that could underlie the acoustic reflex and its continuity is a synchronous discharge of a number of frequency-specific neurons. At low frequencies, where the neural response is phase-locked to the acoustic stimulus, synchronous discharge would be supported by the response associated with a preferred portion of the fine-structure half-cycle. Synchronous discharge would become less likely as stimulus frequency increases, because of the increasingly poor relation between stimulus phase and neural discharge. However, modulation of the high-frequency carrier reintroduces a relatively high probability of a synchronous discharge related to the temporal envelope. This formulation requires a neural mechanism that would be sensitive to the synchronous discharge of a number of frequency-matched auditory nerve fibers. It is possible that this mechanism could be served by cochlear nucleus neurons such as the globular bushy cells, which receive converging input from a number of frequency-

specific fibers (Joris et al. 1994; Spirou et al. 1990; Rothman et al. 1993).

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