Behavioural Brain Research, 37 (1990) 197–214
Elsevier
BBR 01024

Research Reports

Neural representation of sound amplitude in the auditory cortex: effects of noise masking

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(Received 30 August 1989)
(Revised version received 23 September 1989)
(Accepted 12 October 1989)

Key words: Auditory cortex; Neural representation; Signal amplitude; Masking

Single auditory cortical neurons express their sensitivity to the amplitude of a preferred-frequency tone pulse as either a monotonic, saturating intensity profile or as a non-monotonic, bell-shaped intensity function. In the presence of continuous, wideband noise masking, the tone intensity profile is displaced toward higher tone levels. The magnitude of the tone threshold adjustments brought about by increments in noise level very closely match the elevations in noise amplitude. The mechanisms underlying the threshold adjustments likely include neural adaptation. This is because the tone threshold shifts seen in the spike count data are paralleled by spike latency data, and because recovery of tonal sensitivity following noise offset proceeds in a negatively-accelerating fashion. In some instances, the slope of the masked tone intensity profile is greater than that for unmasked tones. For masked tone levels evoking submaximal responses, this has the consequence that cortical responses to masked tones are somewhat more salient than those for unmasked tones of comparable suprathreshold level. These observations bolster our understanding of the psychophysics of noise-masking in normal listeners, and they provide a partial explanation of the difficulty shown by patients with temporal lobe lesions in discriminating signals in noise.

INTRODUCTION

The primary auditory cortex is the target of a highly divergent and convergent afferent pathway arising from the two cochlear partitions. Architecturally, it is made up of strip-like assemblies of neurons in which each element derives its most sensitive inputs from topographically equivalent loci in the two cochleas[7,33,43,55]. These 'iso-frequency strips' (after Merzenich et al.[33]) are the cortical expression of the place code developed in the auditory periphery and preserved in the tonotopically constrained afferent pathway to the cortex. From the contralateral ear, each neuron receives a narrowly frequency-tuned excitatory input, and this frequency-intensity 'response area' may be flanked by inhibitory inputs originating from adjacent cochlear sites. These inputs jointly contribute to the sensitivity of some cortical cells to the spread of spectral energy close to their respective 'characteristic' (CF) or preferred tone frequencies[43,44,47]. Cortical neurons exhibit brisk responses to brief stimulus events, but show rather poorer spike discharge rates to maintained, invariant acoustic signals, especially in anesthetized animals[6,39,41,43].

In man, and in animals, attention to the consequences of primary auditory cortex lesions has recently focussed on studies using temporally complex stimuli, or studies of temporal process-

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ing per se[4,17,18,21,69]. There are at least 3 good reasons for this emphasis. One is the behavioral relevance of temporally-varying sounds, notably human speech and animal vocalizations. A second is the history of neuropsychological evidence implicating the temporal lobe in specifically temporal processing[27,66]. The third is the apparent survival of basic frequency[67] and amplitude[53,65] discriminations following cortical insults.

Notwithstanding these considerations, it remains true that temporal lobe injury in man and animals results in audiometrically detectable deficits. Data on behavioral audiograms following bilateral lesions of the auditory cortex suggest that in opossums[54] and ferrets[25], recovery can be almost complete, while in primates[17,18] and man[4,24,69] the recovery is less complete, and there may remain a permanent residual hearing loss. The nature of the cortical contribution to behavioral sensitivity is unclear. In alert, performing monkeys, behavioral reaction times to an acoustic signal may be inversely related to the instantaneous discharge rates of cortical neurons excited by the signal in the same animal[10]. To the extent that auditory cortical activity is required in the performance of auditory tasks, the absolute sensitivity of the cortical response might constrain that of the behavioral one. In addition, two independent studies have reported that human patients with temporal lobe damage display abnormal difficulty in the discrimination of signals presented against noise backgrounds at the ear contralateral to the lesion[20,37]. While more subtle than a simple threshold elevation for signals presented in silence, this deficit may be debilitating in any situation in which the listener must contend with acoustically complex environments.

Some recent studies in this laboratory have explored the responses of cat cortical neurons to combined tone–noise stimuli, with special emphasis on the neural mechanisms available to those cells for encoding the amplitude of a tonal signal which occurs against a continuous noise background[41,45,46]. The implications of these observations for the neural basis of the loudness recruitment seen in normal listeners with masked signals have been discussed elsewhere[42]. The present report represents a summary and extension of our previous analyses, and it has 3 purposes. The first is to present parametric data, obtained from a large neuron sample, on the fashion in which the amplitude of a tone pulse is represented in the auditory cortex. The second is to explore the more general question of the neural processes that may govern behavioral sensitivity to both masked and unmasked sounds. The third is to report those of our findings which may be relevant to understanding the neurophysiological basis of the sensitivity to noise-masking of listeners with temporal lobe damage.

MATERIALS AND METHODS

Animal preparation

All of the data come from studies which are reported in full elsewhere[41,44–48], and to which the reader is referred for detailed descriptions of the surgical, stimulating, recording and data analytic procedures used in the experiments.

Briefly, all of the data come from single neurons recorded using conventional extracellular recording techniques in adult cats with outer and middle ears free from otoscopically-detectable pathology. All of the surgical preparation, and the ensuing recording sessions, were performed with the cats under surgical anesthesia. At the end of the surgical preparation, general anesthesia was maintained either with artificially respired nitrous oxide and oxygen (70 : 30, in cats paralyzed with gallamine triethiodide, 10 mg/kg/h, i.v., supplemented with intravenous sodium pentobarbital: typically, 2–6 mg/kg/h), or with intravenous pentobarbital alone (in unparalyzed cats: initial dose, 40 mg/kg, i.p.). The anesthetic regimes were designed to maintain a slow-wave EEG (< 5–8 Hz; monitored using bilateral screw electrodes over the visual cortex) and a heart rate less than 180–200 beats per min (monitored using bilateral forepaw needle electrodes). In paralyzed cats, expired CO_2 was monitored spectrophotometrically, and maintained close to 4.0%. The core temperature of all animals was maintained at 37.5 °C, using a thermostatically controlled heating pad.

Stimulating and recording

The data on responses to tonal or to combined tone–noise stimuli are from 198 neurons in the left auditory cortex. Most of these cells were in the primary auditory cortex as defined by its tonotopic organization revealed by the spatial distribution of neural CFs in a few (usually less than 6) electrode penetrations per animal (after Merzenich et al.[33]). All stimuli were presented to the ear contralateral to the recording electrode using a sealed, calibrated stimulating system. This system consisted in two Stax SR44 electrostatic earphones whose signals were fed into a hollow acoustic coupler sealed into the surgically transected ear canal. The coupler speculum contained a probe microphone assembly for measurements of stimulus level close to the eardrum. In one animal (SD11, which contributed 7 neurons to the total sample), a persistent fluid condensation in the probe tube compromised the accuracy of these measurements. The data from this animal have been retained in the sample because our prime interest is in the relative effects of one stimulus on the response to another, and not in the absolute sensitivity of a neuron to either. Tonal and noise stimuli were mixed acoustically. The external auditory meatus ipsilateral to the recording electrode was deliberately collapsed to reduce acoustic cross-talk between the ears. Cross-talk mediated by bone conduction was probably insignificant, since most of the tonal signals had carrier frequencies in excess of 5 kHz, and were commonly in the range from 9.0 to 23.0 kHz.

Tonal stimuli were brief pulses, usually shaped to 50 or 100 ms duration, including 5 ms rise–decay times, and presented with interstimulus intervals from 500 to 800 ms. Masking noise was 100 kHz wide at its source, but was unavoidably low-pass filtered (cut-off near 25 kHz) by the frequency response of the earphones and the transmission properties of the stimulus delivery system. Sound amplitudes are expressed in dB sound pressure level (SPL: dB re 20 μPa) measured close to the tympanic membrane in situ. Note that thresholds for tone and noise stimuli cannot easily be compared within a neuron, first, because the neuron probably responds only to those spectral elements near its CF, and second, because the noise spectrum, even for frequencies less than 25 kHz, was far from flat ($\pm$ 6–10 dB, based on pure tone transmission properties of the stimulating systems). This means that for some neurons, CF was at a peak in the noise spectrum, while for others it was at a trough, or in between.

The data collected were typically input–output functions (spike count versus tone intensity functions: 'intensity profiles') under each of a variable number of masking conditions. Each intensity profile was based on 30–100 repetitions of each of 11–20 tone levels (separated by 5 or 6 dB), often in random order (157 of the 198 neurons). Unless otherwise specified, the carrier frequency of the pulse was always set to the neuron's CF. When a complete intensity profile had been obtained, a new masking condition was set, and the procedure was repeated. Stimulus and response event times were digitized and stored by an on-line computer (PDP 11/34 or IBM PC-XT).

RESULTS

Coding of signal level for sounds in quiet

Intensity profiles are the usual means with which the sensitivity to tone amplitude of central auditory neurons are depicted. These curves plot the spike discharge count (or rate) of a single neuron as a function of the tympanic SPL of the stimulus in question. The curves provide quantitative evidence on the threshold SPL and the dynamic range of the neuron for that stimulus.

In the cat's auditory cortex, most neurons display one of two broad types of intensity profiles. Fig. 1 presents data on 6 neurons, 3 representatives of each of the two classes. Panels A–C depict the most common form of intensity profile seen in the primary auditory cortex. These functions are termed 'monotonic' because their slopes show no systematic reversals in their signs. Each of the neurons has a clearly defined threshold SPL. At suprathreshold tone levels, spike counts increase toward a ceiling maximum, and that level of spike discharge is retained with further increments in stimulus amplitude. Panels D–F show examples of neurons displaying the

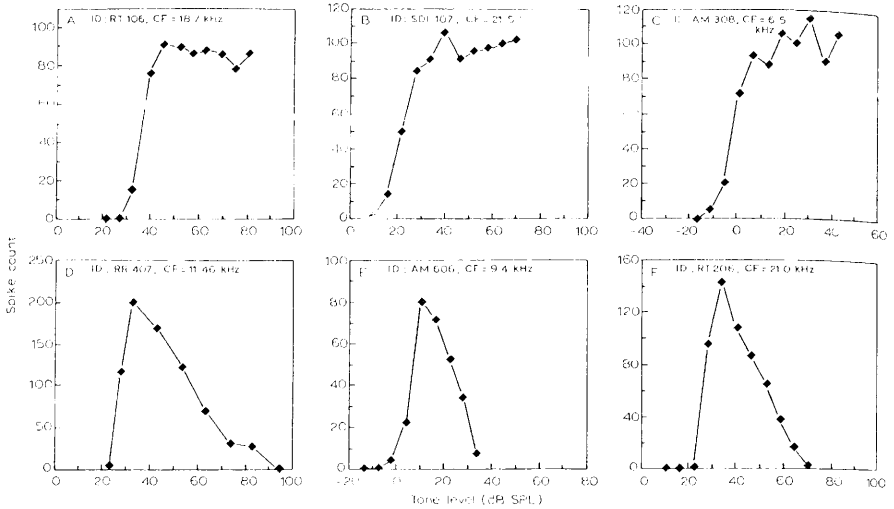

Fig. 1. Intensity profiles for 6 neurons of specified ID number and CFs. Each panel plots the total number of spikes evoked by tone onset as a function of tone SPL, summed across a fixed number of stimulus trials for each neuron. The functions in A–C are monotonic and saturating, while those in D–F are non-monotonic and bell-shaped.

second form of intensity profile: 'non-monotonic' functions. For these cells, there is both a clear threshold SPL and an optimum stimulus amplitude ('best SPL', after Brugge and Merzenich[6]). At stimulus levels above the best SPL, spike counts may fall precipitously, often to zero.

For neurons of any given CF, thresholds may vary over at least a 40-dB range. Fig. 2 presents a scattergram of neural threshold at CF plotted as a function of CF for 198 cells. All of the thresholds were measured from intensity profiles. It is clear that for neurons of any given CF, there is a wide range of thresholds. Some of this variance reflects differences in absolute sensitivity seen between cats. In other cases, we have seen threshold variations of over 40 dB across neurons of the same CF within a cat. Across CFs, minimum neural thresholds parallel the behavioral audiogram. The cat's audiogram is relatively flat between 3.0 kHz and 15 kHz[19], and it is therefore not surprising that the minimum unit thresholds for cortical cells with CFs in this range are also relatively similar.

It is difficult to interpret a neuron's threshold for noise stimuli, because one does not know in

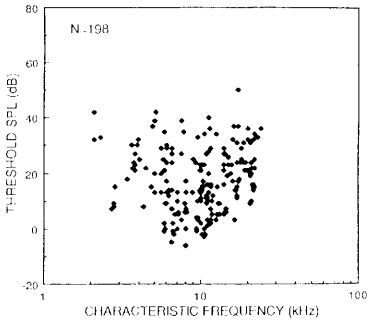

Fig. 2. CF tone thresholds, plotted as a function of CF, for 198 neurons. In all cases, thresholds are measured from intensity profiles. For neurons of any given CF, there is a 30–40 dB range of absolute sensitivities. The minimum CF thresholds for cells with CFs in the range from 2 to 22 kHz are in the range from −5 to +8 dB SPL, which roughly matches behavioral sensitivity across this range.

advance the spectral range in the noise that contributes to the noise response. Moreover, many neurons with obvious excitatory responses to CF tones are inhibited by noise pulses[45,51], and these

are difficult to detect in the absence of significant spontaneous activity. In neurons excited by both tones and noise, one can compare the CF tone threshold to the spectrum level at CF of a threshold noise stimulus (with the qualification that the noise spectrum at the eardrum is rarely flat). In a sample of 40 neurons for which this was possible (Phillips, Orman, Musicant and Wilson, unpublished), the two measures were linearly related, with the neurons showing the highest tone thresholds also showing the highest noise thresholds. The line relating the two thresholds had a slope of 0.81, and the correlation between them was 0.65, indicating that there was considerable variability in the relation. Threshold noise spectrum levels were usually about 30 dB lower than the CF tone threshold.

The dynamic ranges of cortical neurons for CF tone pulses are almost always less than 40 dB wide, and there is some tendency for neurons with monotonic intensity profiles to have wider dynamic ranges than cells with non-monotonic intensity functions. Fig. 3 presents 80% dynamic ranges (i.e. tone level ranges over which spikes counts increased from 10% to 90% of maximum) for 197 neurons. Histograms, with bin widths of

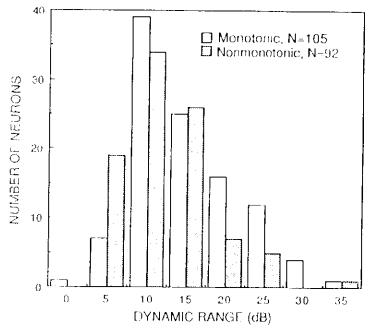

Fig. 3. Distribution of CF tone dynamic ranges for 197 cortical neurons, plotted separately for monotonic (unshaded histogram) and non-monotonic (shaded histogram) cells. Note that although the distributions are similar, most of the neurons with dynamic ranges of less than 10 dB had non-monotonic intensity profiles, while most of the cells with dynamic ranges of 20 dB or more were those with monotonic profiles.

5 dB, depict separately for the two neuron groups the frequency of occurrence of each dynamic range. The two distributions are broadly similar, except that narrow dynamic ranges (less than 10 dB) are more common among non-monotonic cells, while wide dynamic ranges (20 dB or more) are more common among monotonic cells. The two distributions are significantly different (χ^2 (df = 7) = 16.52, $P < 0.025$). Since all auditory nerve fibers have monotonic intensity profiles, we suspect that the inhibitory processes shaping the descending slope of the non-monotonic intensity profile may also foreshorten the dynamic range[42]. Note that the data in Figs. 2 and 3 taken together indicate that the population of neurons of any given CF has a dynamic range that spans at least 60 dB (i.e. 40 dB threshold spread, plus 20 dB dynamic range within neurons). Comparable data have been presented for the rat[58] and monkey[6,40].

Effect of continuous noise on tone thresholds

The most obvious effect of a continuous wideband noise masker on the tonal sensitivity of cortical neurons is a displacement of the tone intensity profile toward higher SPLs. Data for 3 neurons are shown in Fig. 4. Each of the panels presents data for a single neuron. In each case, the solid curve (TA) represents the intensity profile for tones alone. Each of the remaining curves represents the intensity profile of the same neuron, studied with the same set of tone levels, but in the presence of continuous noise of SPLs indicated by the number labeling each curve. For each of the 3 neurons, it is apparent that as the level of the background noise is increased, the tone intensity profile shifts further rightwards. In neurons which are excited by both tone and noise pulses, the minimum masker level effecting a tone threshold shift is the same as the noise level which, when presented alone, produces a threshold excitatory response. These observations indicate that in some sense, the neuron's tone sensitivity is tracking the level of background stimulation.

The broadly preserved shapes of tone intensity profiles seen in the presence of continuous noise reflect the fact that the noise itself does not significantly elevate the rate of ongoing spike dis-

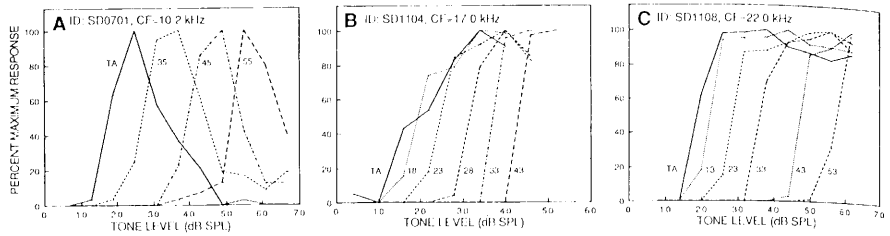

Fig. 4. For 3 neurons of specified ID numbers and CFs, normalized intensity profiles for CF tones presented alone (TA) and in the presence of continuous wideband noise of SPLs specified by the number (in dB) adjacent to each curve. With successive increments in the level of the masking noise, the tone intensity profile is displaced further rightwards toward higher SPLs.

charges. Cortical neurons may be either excited (monotonic cells) or inhibited (non-monotonic cells) by wideband noise[45,51]. As in the case of tonal stimuli, however, these responses are typically transient and locked in time to signal onset. In turn, this means that the full range of a cell's spike discharge rates is potentially available to encode the occurrence of a signal presented against a continuous background. The peak spike rates evoked by masked and unmasked tones vary within and between neurons. In some cells, responses to masked tones are significantly more vigorous than responses to tones alone, while in other cells, the reverse is true. The difficulty in sorting out this issue is that it can take 2–4 h to collect the relevant data from any single neuron, and over these periods, cortical neurons can vary in their spike counts to a given signal by as much as 30% (although such changes need not be accompanied by sensitivity drifts[41]). It is for this reason that the intensity profiles in Fig. 4 (and in most of the Figures to follow) have each been normalized to their respective maxima. Nevertheless, the absence of significant ongoing spike activity in response to continuous maskers has the consequence that the responses to a superimposed tonal or other signal can be as salient as the response to a signal in quiet.

A further question concerns the accuracy with which a cortical neuron's threshold adjustments match the increments in the level of masking noise. A partial answer to this question is provided in Fig. 5. In practice, once the noise level was high enough to produce a tone threshold shift,

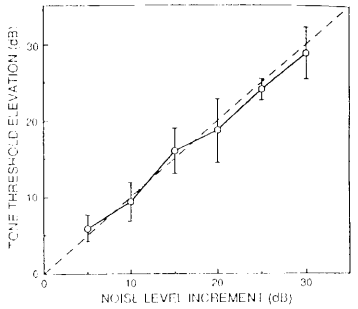

Fig. 5. Data for 34 neurons, showing the effect of increments in masking noise level on CF tone thresholds. Partially overlapping subsets of the 34 neurons contributed to measurements for each noise level increment. Dashed line, y = x, is shown for purposes of comparison. Error bars on the data curve are standard deviations.

then further increments in noise level were quite closely matched by elevations in tone threshold. For 34 neurons studied in some detail, tone threshold elevations (in dB) were compared to the noise level increments (in dB) bringing about those elevations. This was done as follows. Tone thresholds were defined as the SPL associated with the beginning of the steeply rising segment of the intensity profile. The minimum masker level evoking a tone threshold shift was identified. Further elevations in tone threshold were then plotted as a function of the further increment in noise level. Fig. 5 plots the mean threshold elevation as a function of the noise level increment. The error bars on the data curve are standard deviations.

The dashed line has a slope of one, and it is shown for purposes of comparison. An inspection of these data reveals that the neurons' threshold adjustments closely paralleled elevations in the level of masking noise, and that they did so over a suprathreshold noise level range of at least 30 dB.

A least-squares linear regression was performed on the data contributing to Fig. 5, and provided the following equation for a line of best fit:

$$y = 0.921x + 0.641$$

where 'y' refers to the magnitude of the threshold adjustment, and 'x' refers to the magnitude of the noise level increment. This relationship accounted for 82.5% of the data variance. Note that the slope of the fitted line (0.92) is very close to one, and that the y-intercept is very small, less than 1 dB. These observations confirm that once the masker level is high enough to bring about a threshold adjustment, then further increments in masker level are closely matched by further signal threshold increments. In one respect, the data in Fig. 5 may understate the precision of these adjustments. This is because noise levels were, for technical reasons, set in integral multiples of 5 dB, while tone intensity profiles were usually obtained in 6-dB steps. The mismatch in the grain of these variables may have introduced jitter into the measurements.

Mechanisms underlying threshold adjustment: forward masking studies

Studies using a forward masking paradigm provided evidence suggesting that one mechanism underlying noise-induced threshold adjustments was adaptation[41]. These experiments obtained intensity profiles for CF tones delivered at fixed intervals after the onset or the offset of an 800-ms noise masker. The amplitude of the noise was selected to cause a 15–30 dB tone threshold shift for the same tones presented against continuous noise. Each stimulus trial thus consisted in a single noise signal and a single tone pulse; the amplitude of the noise was constant, and the amplitude of the tone was varied randomly over a 60-dB range. The resulting tone intensity profile was based on 40 repetitions of each stimulus condition. It was the comparison between the tone-alone data, the tone-in-continuous-noise data, and the forward masking data which provided clues to the identity of the mechanism underlying the threshold adjustments.

Fig. 6 presents data on one neuron, cell AS0403. Studies with tones alone (TA in Fig. 6A), this neuron's intensity profile was non-monotonic in form. In the presence of 55 dB SPL continuous white noise, this cell's intensity profile was displaced by about 24 dB (T + N in Fig. 6A). When the same neuron was studied with the same range of tone levels, presented as pulses 250, 500 or 800 ms after the onset of the noise-masker, the intensity profiles closely matched that for tones in continuous noise. This means that whatever process was responsible for the threshold adjustment was fully activated by 250 ms after the mask's onset. In other neurons, it is possible for a tone pulse to evoke spike discharges as early as 50 ms after the onset of a noise-masker, and in those instances also, the threshold shift (otherwise seen with a continuous masker) is already fully developed[41].

Fig. 6B presents latent period data for the responses depicted in Fig. 6A. Latencies were defined as the period between tone onset and the peak bin of the resulting peristimulus-time histogram. The tone-alone curve (TA) depicts the familiar, asymptoting latency–intensity relation. In the presence of 55 dB SPL continuous noise (T + N), the latency curve is displaced to the right, and by about the same amount as seen in the spike count data. Again, the curves for responses to tones delivered 250, 500 and 800 ms after masker onset are grouped around the function for tones in continuous noise.

Since the effect of noise masking is expressed similarly in the spike timing and spike count data, the tone threshold adjustments are not a simple obfuscation of the tone-alone response, as might, for example, be brought about by tonic inhibition. This conclusion is bolstered by the independent observation that when these neurons are studied with *simultaneously-gated* excitatory and inhibitory signals, the resulting excitatory responses

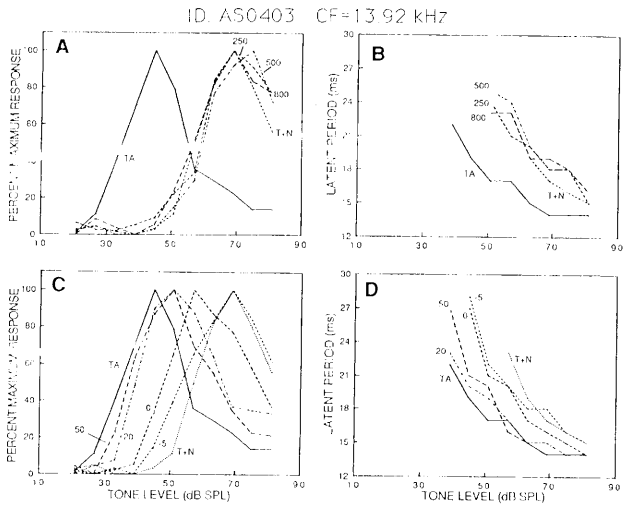

Fig. 6. Detailed data from forward masking studies of neuron AS0403. A: normalized intensity profiles for CF tones delivered alone (TA), in the presence of continuous, 55-dB masking noise (T + N), and presented 200, 500, and 800 ms after the onset of an ongoing 55-dB noise-masker. Note that the curves for masked tones are almost completely overlapping. B: latent period data for the responses depicted in (A). All of the latency functions for masked tones are clumped around that for tones in continuous noise. C: normalized intensity profiles for CF tones presented in the absence (TA) and after the offset of an 800 ms, 55 dB, noise-masker. Numbers labeling each curve indicate delay (in ms) between noise offset and tone onset. '−5' indicates that the rise-time of the tone pulse was synchronous with the decay-time of the noise-masker. Responses to tones in continuous noise (T + N) are shown for comparison. As the delay between noise offset and tone onset is increased, the tone intensity profile resets towards unmasked levels. D: latent period data for the responses in (C).

have reduced strength but normal latencies[45]. These observations constitute one line of evidence favoring adaptation as a mechanism underlying the threshold adjustments: the response latencies for masked tones are appropriate to those tones' suprathreshold level.

Fig. 6C presents intensity profiles of the same neuron, this time studied with tone pulses presented at various intervals after the offset of the 55 dB noise-masker. As in the previous panels, the TA curve represents responses to tones in silence, and the T + N curve represents responses to tones in continuous noise (of 55 dB SPL). Two features of these data are noteworthy. The first is that the tone sensitivity shift brought about by the noise-masker outlasts the masking stimulus. The second is that the neuron reset its sensitivity gradually, over a period of about 50 ms, and that this recovery was negatively accelerating. Fig. 6D shows the latent period data for the responses in Fig. 6C. It reveals that the behavior seen in the spike counts extended to response timing. We see that following the offset of the noise masker, the neuron's latency functions readjusted to unmasked levels, and over a time course comparable to that seen in the spike count data.

Fig. 7 shows the generality of some of these phenomena. For each of 17 neurons, threshold elevation, as measured from intensity profiles, has been plotted as a function of time after masker offset. Different symbols represent data from different neurons. For any given offset time, the data points are quite widely dispersed. This is because the noise levels used did not generate equivalent tone sensitivity shifts in all of the cells, although for 0 ms delay between noise offset and tone onset, all of the threshold elevations were in the range from 10 to 25 dB. Nevertheless, it is apparent that the data points are disposed as a single distribution and that threshold recovery proceeds in a negatively accelerating fashion.

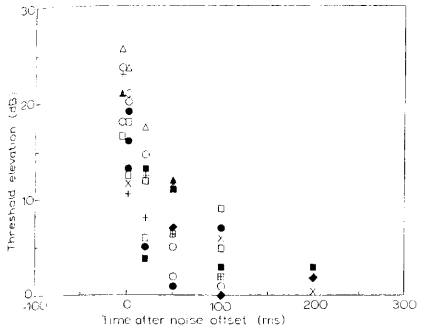

Fig. 7. Population data on recovery of tone thresholds following the offset of an 800 ms noise-masker. The level of the masker was always set to produce a 10–25 dB tone threshold elevation for 0 ms offset time. Different symbols represent data from different neurons. All data were measured from intensity profiles for CF tones presented at offset times specified by abscissa.

The fact that the threshold adjustment, seen concurrently in the spike count and spike timing data, outlasts the noise-masker again favors adaptation as the mechanism of the adjustment. This view is strengthened by two other observations cited here. One is that the extent of the threshold shift is linearly related to the amplitude of the noise (Fig. 5) and the second is that recovery from the effect of the masker is negatively accelerating (Fig. 7). These are classical signs of neural adaptation (see Discussion), but they are difficult to reconcile with a tonic inhibition hypothesis.

Strength of responses to suprathreshold masked tones

As mentioned above, responses to masked tones may be as salient as those to tones in quiet because the continuous noise-masker has little effect on the base rate of spike discharges. In some neurons, the intensity profile for masked tones was steeper than that for tones in quiet. This has the consequence that responses to comparably suprathreshold tones were stronger for masked signals than for unmasked ones. This effect was most marked in neurons whose unmasked tone intensity profiles had broad dynamic ranges, and then only for signals within about 15–20 dB of masked threshold.

Fig. 8 shows data for 3 neurons. Each panel shows the intensity profile for responses to CF tones presented alone (TA) or against a continuous noise background of specified SPL (T + N). None of these intensity profiles has been normalized. Each of these neurons had a relatively broad dynamic range for tones alone. The effect of continuous noise-masking was, in each case, to increase the tone threshold and to steepen the ascending slope of the intensity profile while being inconsequential to the peak response rates evoked by the tones. Because of the steepening, responses to masked tones of suprathreshold but submaximal level were more vigorous than were responses to unmasked tones of the same suprathreshold level. At still higher tone levels, masking

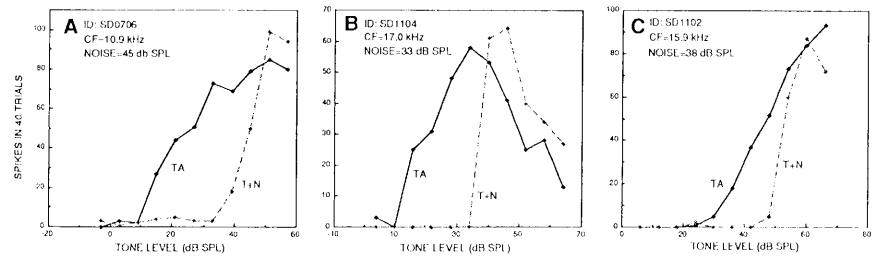

Fig. 8. For 3 neurons, intensity profiles for CF tones presented alone (TA) and in the presence of continuous noise (T + N). Each of these neurons had a broad dynamic range for unmasked tones, but a narrow one for masked tones.

provided little or no response enhancement, at least over the tone amplitude ranges tested. This follows from the finite maximal firing rates of the neurons. Note that the dynamic range compression in these functions was too great to be accounted for by the relatively coarse grain (6 dB) [illegible] were measured.

The [illegible] dynamic range compression (whether expressed as direct measurements of the dynamic range, or in the form of absolute slopes, in spikes/trial/dB[46]) was dependent on both the size of the tone sensitivity shift incurred by the masker, and the breadth of the unmasked tone dynamic range. Fig. 9 presents two scattergrams that illustrate these points. 80% dynamic ranges (i.e. intensity ranges over which spike counts increased from 10% to 90% of maximum) were measured from intensity profiles for masked and unmasked tones, and the dynamic range compression brought about by the masker was plotted as a function of unmasked dynamic range. Positive values on the ordinate indicate a compression of the dynamic range; negative values, an expansion. Points falling on the horizontal line through the middle of the plot (compression score of zero) indicate that the noise background was without effect on the tone dynamic range. Panel A in Fig. 9 shows these data for neurons whose intensity profiles were displaced by 15 dB or less. Each of 46 neurons contributed one data point to this plot. For neurons with unmasked dynamic ranges of less than 20 dB, compression scores show a wide distribution, with as many neurons showing dynamic range expansion (points below the line) as those showing compression. In contrast, all of the neurons with broader dynamic ranges showed compression. Fig. 9B presents comparable data for 37 neurons studied with noise-masking levels sufficiently high to cause an intensity profile displacement in excess of 15 dB. Some of the cells contributing to Fig. 9B also contributed to 9A. In this case, most of the data points lie above the horizontal line, indicating that most of the neurons showed dynamic range compression. Again, the greatest effect was seen in neurons with the broadest dynamic ranges for unmasked tones.

Differential effect of noise-masking across the frequency tuning curve

Within any given neuron, the threshold elevations produced by a specified level of noise-masking is typically greatest at the neuron's CF, and least at the frequencies making up the skirts of the neuron's frequency tuning curve. Fig. 10 presents data on 3 neurons. For each of these cells, intensity profiles were obtained for masked and unmasked tones of carrier frequencies distributed across those cells' effective frequency domains. Thresholds for tones alone (solid lines) and thresholds for tones masked by a single level of continuous noise (dashed lines) were measured from these profiles, and were plotted as a function of tone frequency in Fig. 10A. For each of these cells, it is apparent that thresholds for tones of almost all frequencies are elevated by the presence of a continuous noise masker. A closer inspection of the two curves for each neuron reveals that the threshold elevations for tones at or near CF are greater than those for frequencies at the limits of the tuning curve.

This effect is more clearly seen in Fig. 10B in which, for the same 3 neurons, the magnitude of the threshold adjustment has been plotted as a function of tone frequency. Note that the ordinate in B is expanded with respect to that in A. Each of the curves in Fig. 10B has an inverted V-shape, and the tip of the V occurs on the abscissa at the neuron's unmasked threshold CF (Fig. 10A). This analysis shows more clearly that the masking effect of a given background noise level is greatest at a cell's CF, and declines at the skirts of the tuning curve.

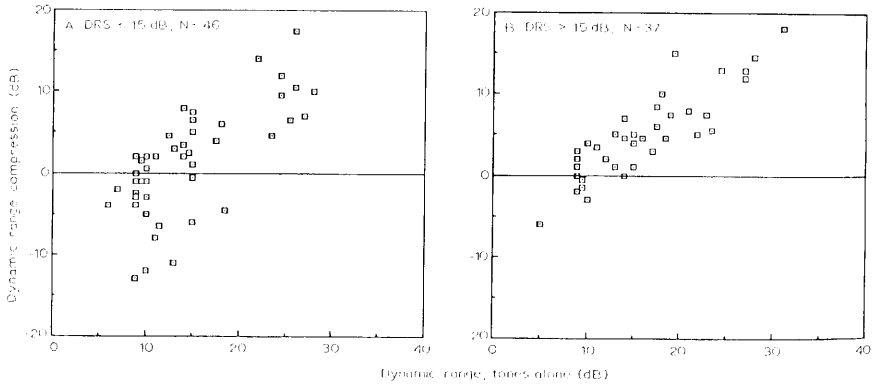

Fig. 9. Scattergrams showing the extent to which the CF tone dynamic range was compressed by the presence of noise-masking. Each data point represents the dynamic range compression, plotted as a function of unmasked tone dynamic range. Data in (A) are for neurons whose tone dynamic ranges were shifted by 15 dB or less by the noise-masker. Data in (B) are for a partially overlapping group of neurons for which the noise-masker caused a tone dynamic range shift (DRS) in excess of 15 dB. Note that within (A) or (B), compression was most marked for cells with broad unmasked tone dynamic ranges.

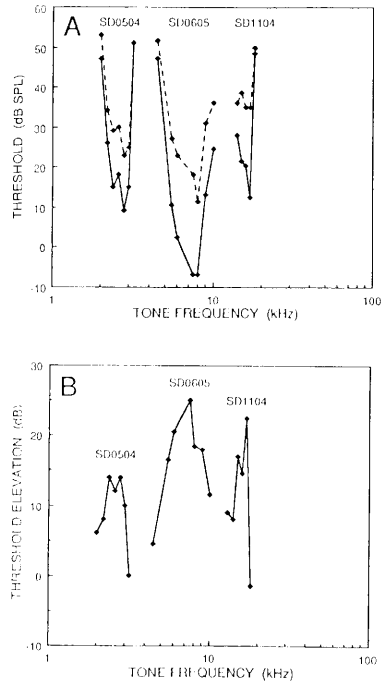

Fig. 10. A: solid lines show threshold tuning curves for 3 neurons of CFs 2.8, 7.7 and 17.0 kHz. Dashed lines show tuning curves for the same neurons obtained in the presence of continuous noise-masking. For neurons SD0504, SD0605 and SD1104, noise levels were 34 dB, 35 dB and 33 dB, respectively. B: for the same 3 neurons, threshold elevation incurred by the same noise masks has been plotted as a function of tone frequency. Note that each of these curves has an inverted V-shape, centered on the neuron's unmasked CF.

DISCUSSION

General anesthesia, and the functional significance of the results

An important qualification to what follows is that all of the data come from anesthetized animals. Our studies found little difference in the responses of cortical neurons in barbiturate-anesthetized and nitrous oxide-anesthetized cats[45,51], but this in itself does not mean that general anesthesia per se did not shape the response types seen. In this respect, all of the cell types distinguished on the basis of frequency or amplitude tuning properties, or binaural interactions, in anesthetized cats have also been described in awake animals[5,6]. This suggests that the neuronal properties in anesthetized animals are reliable indicators of how some cells might behave in the absence of anesthesia. On the other hand, comparison of the spike discharge patterns in alert and anesthetized animals typically reveals that responses in the unanesthetized animal are often more vigorous or sustained than those in the anesthetized cat[5,6]. This suggests that general anesthesia might modify the time course of some neuronal responses, even if it does not modify those cells' stimulus selectivities. It is also possi-

ble that general anesthesia might silence some neuronal cell types, so that the full complement of cortical circuitry may not be expressed in our studies. Nevertheless, to the extent that the experiments described in this report use as dependent variables response properties that are also seen in the awake animal, we are confident that our characterization of the anesthetized cat's cortex is relevant to the intact animal.

A second possible qualification is that almost all cortical neurons receive input from both ears, while the studies described here used (in most cases) monaural stimulation [illegible] contralateral [illegible]. The reasons for this strategy are two-fold. The first is the practical one, namely that many of the neurons in these studies were tested with 30–100 presentations of over 100 stimulus conditions, which, in our hands, tests the duration of extracellular recording techniques to its limits (up to 4–6 h, in some cases). To have introduced binaural stimulus parameters into these experiments would have been difficult or impossible. Second, there is no evidence to date indicating that the frequency-amplitude tuning properties of cortical neurons are correlated with binaural interaction pattern. Indeed, there is the contrary evidence that frequency-amplitude tuning properties can be remarkably different in two cortical auditory fields containing neurons with similar binaural interactions[39].

The cortical representation of signal level in quiet

The primary auditory cortex contains two broad groups of neurons distinguished by their tone amplitude coding properties. Monotonic neurons (Fig. 1A–C) have saturating, sigmoidal intensity profiles. Monotonic cells of any given CF, even within a single cortex, vary widely in their absolute sensitivities (Fig. 2; Refs. 46,49). For such neurons, the pattern of cortical activity evoked by tones of a single frequency but different amplitudes is thus likely to consist of spike discharges from all of those neurons with CFs at or near the stimulating frequency, whose thresholds are exceeded by the signal level. Presumably, that population of neurons is somewhat larger for more intense signals, and the rate of spike discharge within neural elements will be higher. Non-monotonic neurons (Fig. 1D–F) have bell-shaped intensity profiles, so that they respond to tonal signals within only narrow domains of both frequency and amplitude. Since these neurons vary widely in their best SPLs[6,39,50,62], the population of cortical neurons activated by a tone of any given level will be restricted to those neurons whose best SPLs are at, or close to, the stimulating SPL. Among these cells, the cortical representation of tone level thus resides in the identity of the discharging elements and in their rates of discharge. In general, then, the cortical representation of tone level incorporates the number of active elements (monotonic cells), which elements are active (non-monotonic cells) and their rates of spike discharge[42,50,64].

A quite different, but compatible, analysis has been presented by Ehret and Merzenich[10]. They obtained neuronal intensity profile data from the cat inferior colliculus which, like the cortex, contains both monotonic and non-monotonic elements. They averaged their spike rate data across neurons of different absolute threshold, and intensity profile shape, and plotted mean spike rate as a function of signal SPL. The resulting curve was flat over a range of suprathreshold tone levels at least 60 dB wide. They therefore concluded that average response rate was an unsuitable code for sound level at the inferior colliculus. It is not unreasonable to expect that a similar finding could be derived from the observations made in our laboratory. As those authors recognized, the difficulty with this approach is that it takes no account of which, or of how many, neurons are activated by the stimuli. Across a neural ensemble like the colliculus or the cortex, stimuli of different amplitude will activate only partially overlapping populations of neurons. Since all of these neurons have finite maximal discharge rates, a mean firing rate averaged across neurons is likely to be relatively constant, except at very low absolute signal amplitudes, where few neurons are responding, and then with low rates. Ehret and Merzenich's[10] analysis could equally be applied to the code for tone frequency: as the frequency of a tone pulse is varied, the number of neurons discharging might remain constant, and so too might the mean firing rates of the activated cells. This misses the point that the code for tone frequency resides as much in which elements are active as in the firing rates of the cells that are active. In the bat's cortex, we know that there is a topographic arrangement, or 'mapping', of neurons according to their best SPLs[64]. The prerequisite for such a mapping, i.e. neurons of the same CF but widely different best SPLs, exists in the cat[50] and monkey[6] cortex, but no neural map for stimulus amplitude has yet been found in these species.

Effect of continuous noise-masking on neural and behavioral thresholds

Central auditory neurons are typically described as having 'threshold SPLs' and, in the case of non-monotonic neurons, 'best SPLs'. Even within a single neuron, both of these response properties usually vary with the stimulating frequency[2,5,50,51]. The persistence of these terms probably reflects the assumption that neuronal responses are dominated by stimulus events at, or very close to, CF. The present studies reveal that cortical neurons dynamically adjust their CF thresholds (and suprathreshold responses) to the level of background noise (Fig. 4). This suggests that even at CF, neither thresholds nor optimal stimulus levels should be assigned a single value in units of SPL. If there is any single feature of the level coding properties of cortical cells that survives variations in background noise amplitude, it is the relation between tone threshold and masker level. This follows from the fact that thresholds increase linearly, and with a slope close to unity, with increments in noise level (Fig. 5), though only for masker amplitudes high enough to effect a threshold adjustment. Neurons of the same CF but different absolute sensitivities show the same relation, but the absolute masker levels required to cause threshold adjustments reflect those cells' tonal sensitivities.

These findings parallel those in human psychophysics[15]. Background noise of very low amplitude is inconsequential to tone or speech detection thresholds, but once the masker level is above threshold for effecting a signal threshold shift, then further increments in noise level bring about signal threshold elevations that perfectly match the increments in masker level. Now, the behavioral tone audiogram likely represents the envelope of the lowest thresholds of the independent neural channels serving each cochlear place. In the presence of masking noise of any given level, only those channels sufficiently sensitive to have their thresholds exceeded by the masker will have their thresholds elevated, with the result that the audiogram becomes flattened in the middle where it is normally most sensitive (see also below). It is thus the functional independence of the neural channels serving each cochlear place that permits this differential sensitivity to masking, and it is the absolute sensitivity of the channels that determines which of them will be affected by a given noise level.

Mechanisms underlying threshold adjustment

The data presented in this report provide evidence that one mechanism mediating the tone threshold adjustments is neural adaptation. Adaptation as a sensory mechanism is well recognized at both psychological and neural levels[30,31,35,36,41,60,61]. The neural response is an adjustment of threshold sensitivity to the level of the adapting stimulus, and recovery from it is negatively accelerating. The threshold adjustments described in this report meet these criteria (Figs. 5 and 7). In addition, we have shown that the adapted responses have latencies that are appropriate to their suprathreshold levels (Fig. 6). It is very unlikely that this adaptation is a uniquely cortical phenomenon in the auditory system. Adaptation expressed in similar fashions occurs as far peripherally as the cochlear nerve[31,60], where it has been attributed to the metabolic properties of the neurotransmitter reservoir at the hair cell–afferent fiber synapse[31,61]. The adaptation seen in cortical cells therefore likely represents the cumulative effect of that at each serial synapse in the neural pathway transmitting the signal; it is expressed in the transient character of neural responses to the onset of a maintained signal.

The *magnitude* of the threshold adjustment is possibly set in large part by events at the auditory periphery. Smith[60], Costalupes et al.[8] and Gibson et al.[13] have previously performed experiments, broadly similar to those described here, on single

cochlear nerve fibers. Cochlear nerve fibers respond continuously to a noise-masker, and this sets a base rate of response upon which the response to a CF tone must be superimposed. The extent to which the tone threshold is elevated reflects at least 3 factors. One is that tones, which alone would evoke a spike rate less than that evoked by the masker, are largely ineffective in modulating the noise response. The second is that the widespread mechanical (basilar membrane) response to the noise physically interferes with the response to the tone at the site innervated by the fiber. The third is neural adaptation. The net result of these processes is that for many auditory nerve fibers, the response to a suprathreshold tone in noise is smaller than that to a tone in quiet, and it is expressed as a modulation of the background firing rate evoked by the noise-masker. The size of the tone response imposed on the noise response depends on the firing rate evoked by the noise, and therefore, on the noise level. Tonal signals may be without significant effect in those fibers whose firing rates are at saturation because of high noise levels. Interestingly, the slope of the line relating tone sensitivity shift to noise level increment is close to 0.7 for cochlear nerve fibers[8], nearer to 0.9 for cells in the dorsal cochlear nucleus[13] and close to 1.0 for cells in the auditory midbrain[56] and cortex (Fig. 5).

However, there exists a subgroup of auditory nerve fibers which have high thresholds, low spontaneous rates, and which are resistant to firing rate saturation since they possess broad, more gently sloped intensity profiles[8,28,29,57]. These afferent auditory neurons retain the ability to give a quite salient response to tones even when those tones are imposed on relatively high levels of background noise. This is because a significant portion of the spike rate dynamic range remains available after the baseline elevation incurred by the masker. The same group of neurons send rather densely ramifying axonal projections into the cochlear nuclear complex[11], so that their sensitivity to masked tones may well be conferred on many brainstem cells. Presumably, the more central auditory nuclei capitalize on this sensitivity to produce the responses seen in our studies of the cortex.

A related question concerns which spectral ranges in the noise-masker are responsible for tone threshold elevations. At the level of the cochlear nerve, there is good evidence that the suppressive effect of a masker on a CF tone response should be, and is, greatest for masker components near the cell's CF[14,22,23], at least for low-level maskers of the kind used here (cf. refs. 30,31 for the effects of intense sound exposure). The frequency range of effective masking elements probably extends beyond the width of the tuning curve because of the longitudinal mechanical interactions between tone and masker. It is difficult to imagine that the magnitude of the noise-induced tone threshold adjustments seen in the cochlear nerve are not preserved in the input to the cortex. Unlike auditory nerve fibers, many cortical neurons also possess sideband (lateral) inhibitory response areas[45,57,63]. The extent to which prolonged stimulation of these produces a specifically adaptation (cf. inhibitory) response in the more central excitatory response area is unknown. However, since the threshold adjustments described in this report extend to cortical neurons which probably lack inhibitory response areas (monotonic cells which are excited by noise[41,48,51]), it seems that inhibitory response areas are not required for the sensitivity adjustments to noise level.

Strength of cortical responses to suprathreshold masked tones, and effects of cortical lesions

Auditory nerve fibers show no steepening of their CF tone intensity profiles when the tones are masked by wideband noise[8]. Any foreshortening of the tone dynamic range by masking is due simply to the abbreviated spike rate range available for encoding tone level when the base rate of spike discharge is elevated by the noise. Moreover, the threshold tuning curve of an auditory nerve fiber is elevated as a whole, i.e. equivalently for all tone frequencies, for a given masker level[12,26]. Both of these findings stand in contrast to those for cortical cells (Figs. 8–10).

The findings in cortical neurons are understandable in terms of a quite simple model[46,47]. The fact that a given noise-masker affects responses to different tone frequencies non-equivalently *within a neuron* (Figure 10) necessarily means that the neuron receives partially independent inputs at those frequencies, and, in turn, that the unmasked tuning curve of a cortical neuron actually represents the envelope of the best sensitivities of a number of inputs with roughly similar CFs. We proposed that these inputs might have different absolute sensitivities, and that they may be independently susceptible to noise-induced adaptation. The circuitry required by this account is compatible with two independent lines of evidence. First, the afferent pathway feeding onto the cortex is tonotopically constrained throughout, but there is remarkable convergence of input from numerous, spatially separated brainstem nuclei differing in their neuronal response properties[1,3]. Second, recent neuropharmacological studies have revealed that different temporal components of a cortical neuron's response[32] and responses to different tone frequencies within the response area[34] are differentially affected by iontophoretic application of acetylcholine. These studies point not only to the convergence of afferent input to cortical neurons, but also to the possibility that those inputs have different physiologies.

The noise-masking studies described here revealed that tone threshold elevations incurred by a given noise-masker were greatest at the frequencies to which the neuron was most sensitive. This phenomenon would tend to force (unmasked) normally disparate thresholds of inputs at CF into closer register (masked state) by adaptation of the most sensitive inputs. On the assumption that the cortical neuron's CF intensity profile reflects the number, sign and rate–response of the inputs activated, the slope of the cortical cell's intensity profile should be steepened by noise-masking (Fig. 8). By the same token, the greater the threshold adjustment, the greater should be the steepening effect (Fig. 9). Neither the differential effect of noise across the frequency tuning curve, nor the steepening of the intensity profile should occur in cochlear nerve fibers by this line of argument, since those fibers receive a unitary input, namely from the single cochlear hair cell that each contacts. Direct studies of cochlear nerve fibers confirm that neither of these effects occurs at that locus[8,13,26,30].

By comparison with cochlear nerve fibers then, the response of some cortical neurons to suprathreshold, but submaximal masked tone levels are more salient. The salience of the responses is expressed in two fashions: first, in the relatively low level of background activity evoked by the continuous masker, and second, in the enhanced responses of some neurons to masked tones. Whether and how much *perceptual* salience might result from these phenomena is a matter for speculation. In normal listeners, thresholds for the detection of signals are elevated by noise-masks, and these threshold elevations closely match increments in noise level[15]. In addition, the rate of loudness growth for masked signals (i.e. the slope of the loudness function) is greater than that for unmasked signals, though only for signal levels within about 20 dB of masked threshold[62]. To the extent that perceived loudness may be associated with neural firing rates[42,68], the cortical data on responses to masked tones provide evidence of one mechanism that might underlie the steepened psychophysical function. It is not known whether this psychophysical response to noise-masking survives cortical lesions.

There is increasing evidence from studies of ferrets[25], primates[16,18] and man[4,69] that bilateral lesion to the primary auditory cortex does not result in complete deafness, and that behavioral sensitivity across the audiometric range can recover, at least to levels within about 20–30 dB of normal. These observations do not specify the nature of the contribution of the primary cortical field to behavioral sensitivity, but they suggest that, at least for simple signals, some auditory detection tasks may be mediated by subcortical structures or other cortical regions. Similarly, there is evidence that difference limens for stimulus amplitude are relatively unaffected by cortical damage[65]. Now, since the lesioned subject can detect both a tonal and a noise signal, the further question concerns the extent to which the loss of the cortex impairs the detection (and thereby the discrimination) of one in the presence of the other.

We know from studies of patients with temporal lobe lesions that the loss of the temporal

lobe results in impaired discrimination of speech signals presented against noise backgrounds. Olsen et al.[37] reported that, by comparison with normal listeners, some listeners with temporal lobe damage showed speech discrimination deficits in noise for a signal-to-noise ratio of 0 dB. Heilman et al.[20] reported that the speech discrimination deficit for a given signal level (40 dB SL) was present for signal-to-noise ratios over a 30-dB range (+5 to −25 dB). Normative data were not presented, and the disadvantage following from the pathology was measured from a comparison of the response to stimuli at the contralateral ear with performance for stimuli delivered to the ear ipsilateral to the lesion. The comparison revealed that the deficit was most marked at signal/noise ratios between +5 and −15 dB.

The quantitative details of the clinical findings are difficult to relate to the present data, first, because the task required of the listeners was a discrimination one rather than a detection, and second, because the *effective* signal/noise ratio for a person is probably quite different to that for a cortical neuron. We can only indicate that the auditory cortex has mechanisms available to it for extracting signals from noise, and that lesion of the temporal lobe in man results in discrimination deficits for signals in noise. If there is a link between these findings, then it may lie in the possibility that the increased salience of cortical responses for masked signals (by comparison with lower brainstem nuclei) provides a superior neural substrate for perceptual discriminations. In this respect, Heilman et al.[20] attributed their findings to a deficit in selective attention in brain-damaged listeners. Following from the modern 'spotlight' metaphor of selective attention[9,52], it is perhaps more likely that any deficit in selective attention is secondary to the deprivation of attentional processes from their normal neural representations of the signal.

ACKNOWLEDGEMENTS

Special thanks are due to S.R. Shaw, S.E. Hall and M.S. Cynader for helpful discussions of some of the issues examined here. Two anonymous reviewers provided excellent comments on a previous version of this manuscript. Some of the research described here was supported by NSERC Grants U0442, URF0035035 and E2745, and Dalhousie University Research Development Awards to the author.

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Behavioural Brain Research, 37 (1990) 215–225
Elsevier
BBR 01032

Early sensory determinants of locomotor speed in adult cats: I. Visual compensation after bilabyrinthectomy in cats and kittens

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(Received 28 September 1988)
(Revised version received 6 October 1989)
(Accepted 1 November 1989)

Key words: Labyrinthectomy; Locomotion; Vision; Cat

After a study of locomotion in cats deprived of their vestibular receptors early in life[31], the average speed of locomotion was measured in 3 adult cats, first before and then several months after bilateral labyrinthectomy, in a task involving free locomotion across platforms, rails or ladders and under normal illumination versus stroboscopic illumination or darkness. In the absence of visual cues, cats which received lesions as adults showed, on both wide and narrow supports, the same speed deficits as those of subjects which underwent lesions soon after birth. Visual cues were, however, used by some cats with early lesions to recover near normal performances on wide platforms. Evidence was found that the vestibular deficit comprised at least 3 components: a severe loss of dynamic balance occurred in all subjects with lesions and was mostly not compensated for by vision; a loss of the ability to keep to a straight course in darkness was observed on wide platforms, but vision considerably improved this function, even under stroboscopic illumination; a slight paw adjustment deficit was also observed on irregular supports, due to the fact that vision played a decreased role after the vestibular lesion. These effects point to the specificity of the vestibular control of balance, the availability of several sensorimotor strategies for orientation, and the complementarity of vestibular and visual inputs in guiding the subjects' step. We confirm here that vestibular inputs do not play a critical role during locomotor development, and stress the fact that peripheral visual cues about position or orientation (rather than motion), play a leading role in compensatory strategies.

INTRODUCTION

Postural and locomotor activities rely upon several regulatory as well as anticipatory sensory influences, including those from the vestibular and visual channels which have often been emphasized. Most studies in this field have been performed on adult subjects, so that the processes occurring during development remain largely unknown. It has been clearly established that the vestibular system plays a prominent role as an orientational reference in adult organisms[37]. However, although a disorganization of posture and gait occurs after total vestibular loss, these activities are mostly recovered within two months in many species.

In chronic labyrinthectomized animals, persistent balance deficits[14,23,42], as well as an impaired orientation in darkness[31], have been reported. Bilabyrinthectomy might also be expected to produce other types of deficits, due to effects such as the instability of the visually perceived world[10,43]. These could affect sensorimotor coordination, yet very little is known

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