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Research report

Factors shaping the response latencies of neurons in the cat's auditory cortex

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Abstract

This article addresses two issues. Firstly, the hypothesis that response latency might be a neural code for tone frequency was examined in single-neuron data from the primary auditory cortex of anesthetized cats. Minimal response latencies for characteristic frequency (CF) tones were independent of neural CF. Mean response latencies for a constant amplitude CF tone were also independent of CF. These data, and the fact that cortical neurons do not have an obvious independent referent for stimulus onset time, do not support the view that latency is a code for frequency. Secondly, to investigate a simple threshold model of spike initiation time, we describe the prolongations of response latency with increases in stimulus rise time and their dependence on the peak amplitude of the stimulus. These data show that in cortical neurons, it is not the peak stimulus intensity which determines first-spike latency, and second, that the response latencies are systematically not those expected on the basis of a simple threshold model.

Keywords: Auditory cortex; Single neuron; Response latency; Threshold model

1. Introduction

There has been a recent resurgence of interest in the timing of cortical responses evoked by peripheral sensory stimulation [6,21,25]. Part of this interest has been prompted by the general hypothesis that temporal synchrony among evoked responses might contribute to the mechanism underlying 'perceptual binding' [6,10,13,25,27]. In this regard, there has been the explicit suggestion that response latency may be a neuronal code for some stimulus features [5,6,25].

Roberts and Poeppel [25] reported that in the human primary auditory cortex, the latency of the M100 of the tone-evoked magnetoencephalographic response showed an apparently strong dependence on tone frequency; latencies were shortest (around 100 ms) for frequencies from 500 to 3000 Hz, and were 20–30 ms longer for frequencies above or below that range. Roberts and Poeppel [25] suggested that this latency shift might be a mechanism, in addition to the familiar tonotopic spatial one, by which the frequency of the stimulus is encoded. Eysel [5] suggested that the similarity of response latencies for frequencies in the speech range (500–2000 Hz) indicated a special role of latency in 'marking most important features'.

The finding of a frequency-dependent M100 response latency is of special interest because, unlike other basic properties of human auditory cortex (e.g. narrow frequency tuning [11]; tonotopic organization [26]), there has been no precedent for it in animal studies. If the M100 response to tones of any given frequency is generated largely by neurons tuned to the test frequency, then it follows that cortical neurons tuned to frequencies in the middle of the audiometric range must have shorter latent periods than those of neurons out-

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side that range. Systematic data on this issue have not previously been presented in animals. The first purpose of the present report is to describe the distribution of first-spike latencies of single neurons in the cat's primary auditory cortex as a function of those cells' characteristic frequencies (CF: the tone frequency for which the neuron has its lowest excitatory threshold). These data are directly relevant to the suggestion that response latency may be a code for tone frequency.

A second focus of interest on the response timing of auditory cortical neurons concerns the sensitivity of first spike latency to the rise-time of an acoustic signal [9,15,22]. In the primary field of auditory cortex, increases in the rise time of a CF tone prolong latent periods, suggesting that spike generation might be subject to a simple threshold phenomenon, i.e. that spike generation time depends on when during the rise-time of the signal some threshold sound pressure is exceeded. Heil and Irvine [9], however, point out that these prolongations of response latency for neurons in the primary field may not be those predicted by a simple threshold model. The generality of this observation across cortical fields has yet to be established. The second purpose of this report is to present detailed evidence on the sensitivity of latent periods to the rise time of a CF tone for neurons in the posterior field of the cat's auditory cortex.

2. Methods

The data come from an ongoing program of research [16–22]. Briefly, the data are quantitative records of the rates and timing of spike responses of single neurons recorded in the left auditory cortex of adult cats anesthetized with sodium pentobarbitol (35–40 mg/kg i.p. and supplemented intravenously to maintain a state of areflexia). A tracheotomy was performed, and the head was supported by a holder that left the ears free from obstruction. The pinnae were resected, and sealed around delivery systems, which incorporated calibrated probe microphone assemblies for in situ measurement of sound amplitude. A craniotomy was performed, and the electrode was advanced remotely through openings in the dura mater.

For the studies of the primary field, acoustic stimuli were tone pulses of 50 or 100 ms duration, including 5.0 ms (cosine) rise-fall times, presented at 1/800 ms. For the studies of neurons in the posterior field, stimuli were tone pulses of 50-ms duration including (cosine) rise times of 1.0, 5.0 or 20.0 ms. Plateau stimulus levels are expressed in dB SPL (sound pressure level: dB re 20 μPa). The responses of single neurons were recorded extracellularly using glass-coated tungsten or platinum-iridium electrodes. Spike times were digitized at either 0.1 or 1.0 MHz.

In each animal, the primary auditory cortex (AI) or the posterior field (P) was defined by obtaining partial maps of the spatial distribution of neuron or neuron cluster CFs (after Reale and Imig [24]). When a single neuron was isolated, its threshold CF was determined using tones of the neuron's preferred laterality (usually contralateral alone, or binaural, equally-intense stimuli). A spike count vs. SPL function was then obtained using 5-ms rise-time CF tones (usually 40 blocked trials at each SPL, in 5 or 10 dB steps over the effective SPL range of the neuron). Neurons were classified as 'non-monotonic' if their spike counts fell by more than 50% at high stimulus SPLs. All other cells were classified as 'monotonic' [21,22]. If recording conditions were sufficiently stable, additional intensity profiles were obtained for CF tones with rise times of 1.0 and 20.0 ms.

All latency measures are mean latency to the first spike after stimulus onset. In practice, almost all of the neurons gave transient 'on' responses, which showed orderly shortenings of latent period and increased regularity with increases in stimulus SPL (e.g. [17]). This meant that it was usually possible to set an arbitrary temporal window (e.g. 8–70 ms post stimulus onset) within which a response fell and was counted as stimulus-driven. In order for a mean latency data point to be included for any neuron in the data set, it had to be based on at least 10 (first) spikes.

The CFs of the neurons in this sample were all in the range from 300 Hz to 28 kHz. This range includes the most sensitive audiometric range of the cat [8], missing only the highest and lowest two octaves of this species' audible range, and is in this sense comparable to the frequency range used by Roberts and Poeppel [25] in man.

3. Results

3.1. Dependence of response latency on CF

Fig. 1A shows representative latency-vs-intensity functions obtained from six neurons. Each curve plots mean first-spike latent period as a function of the intensity of a CF tone delivered to the preferred ear. For each neuron, the CF is indicated by the number (in kHz) labelling each curve. In each neuron, mean first-spike latency declined toward an asymptotic minimum with increasing stimulus level. The shapes of these functions varied between neurons, being steeper at low SPLs in some neurons than in others. As revealed by inspection of Fig. 1A, this was true in neurons of similar or identical CF (e.g. rightmost curves in Fig. 1A).

The minima towards which latency functions tended at suprathreshold SPLs also varied between neurons. Fig. 1B shows minimal latent period plotted as a function of CF for the complete sample of neurons. Data

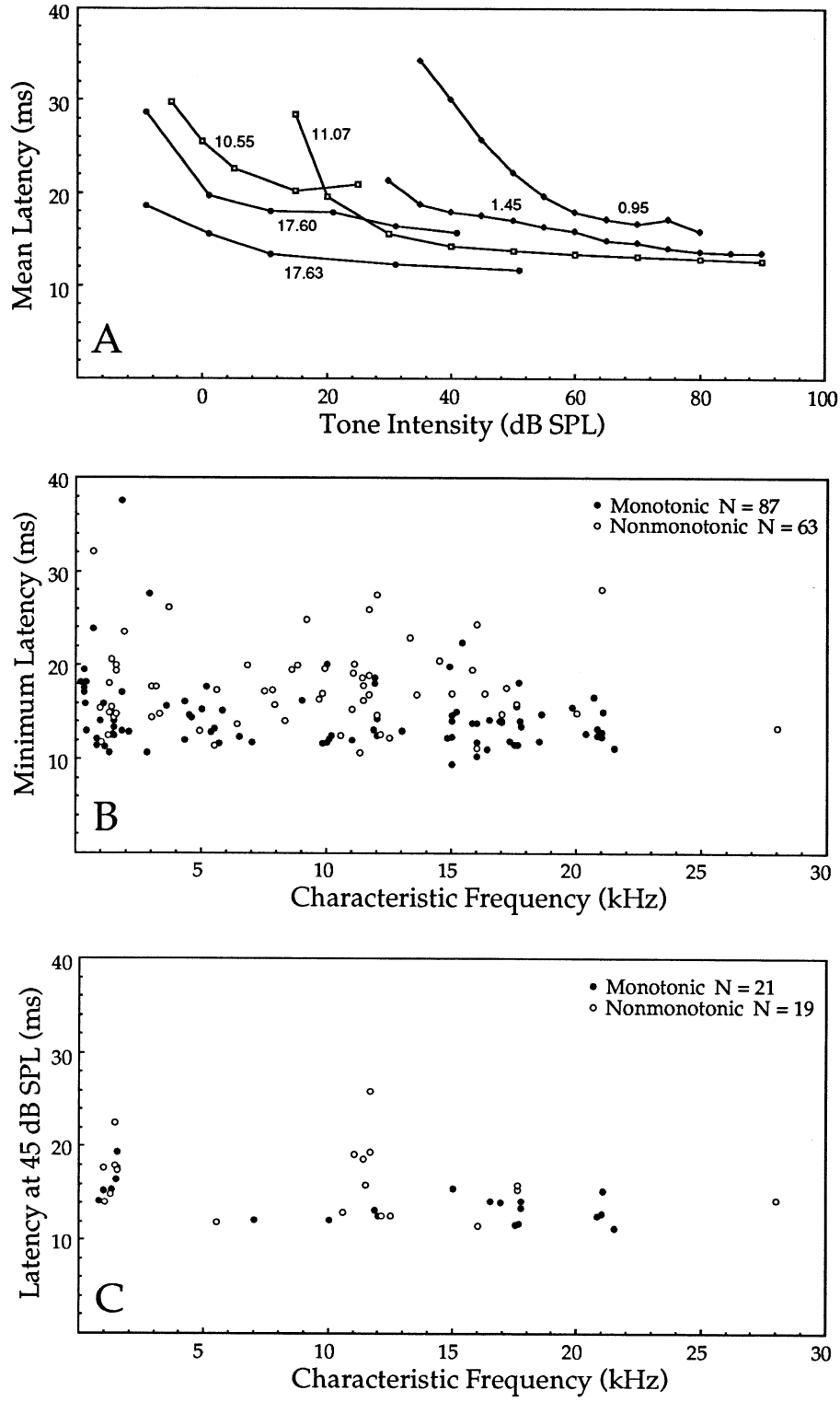

Fig. 1. A shows latency-vs-SPL functions obtained using CF tones in six neurons. CF (in kHz) of each neuron is indicated for each curve. B shows minimum response latency for CF tones, plotted as a function of CF. Data are shown separately for cells with monotonic and nonmonotonic spike count functions. Sample sizes are indicated. C shows mean response latency for CF tones presented at 45 dB SPL, plotted as a function of CF.

are shown separately for monotonic (filled symbols) and nonmonotonic cells (open symbols) because there is a modest tendency for the latter neurons to have longer minimal latencies [20]. An inspection of Fig. 1B reveals that minimal latencies were generally in the range from 12–22 ms, irrespective of CF, and irrespective of the monotonic or nonmonotonic shape of a neuron's spike count vs. SPL function.

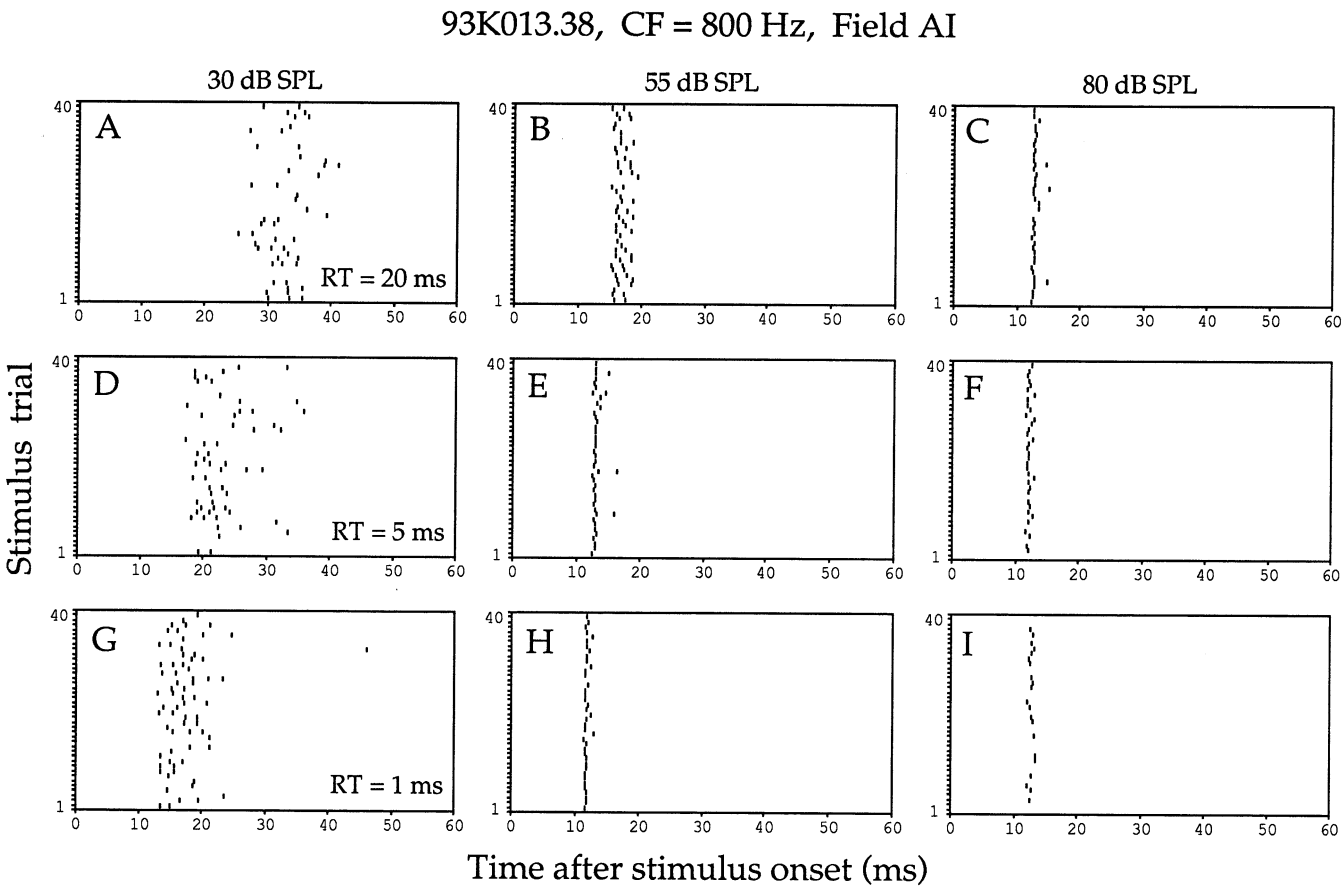

Fig. 2. (A–I) Dot rasters showing the spike event times of a single AI neuron in relation to stimulus onset time. Each raster represents the responses of the neuron to 40 presentations of a CF tone whose SPL and rise time (RT) are specified by the column and row, respectively, in which the raster is located.

In the Roberts and Poeppel [25] study, the human M100 response latency was plotted as a function of tone frequency for a constant amplitude stimulus, and it was this curve that showed the dependence of response latency on tone frequency. In the present study, such a relation might be seen in response to a constant-SPL signal, despite a homogeneous distribution of minimal latencies (e.g. Fig. 1B), if the latency functions for very high- and low-CF cells were steeper than those with middle CFs. In order more closely to mimic the Roberts and Poeppel [25] analysis, mean latency for a constant-SPL CF tone has been plotted as a function of CF in Fig. 1C.

Data are shown for responses to CF tones presented at 45 dB SPL, an intensity in the middle of the amplitude range typically studied in cats. There were fewer data points in this analysis, principally because 45 dB was below threshold for some cells, and above cut-off intensity for many nonmonotonic cells (e.g. [22]). Once again however, Fig. 1C provides no evidence for a relationship between response latency and CF. The data points have a nearly horizontal distribution in the plot.

3.2. Dependence of response latency on tone rise time

Sample responses of a single AI neuron to variations in the rise time and SPL of a CF tone are provided in Fig. 2. Each panel of Fig. 2 shows a dot raster, depicting the responses of this neuron to 40 successive presentations of a CF tone whose amplitude and rise time are specified by the column and row, respectively, in which the raster occurs. As with almost all neurons in the sample, this neuron usually discharged 1–2 spikes in response to stimulus onset. Note that the response latencies are relatively long and variable for threshold levels of stimulation (left column), and much shorter and more regular for intense tones (middle and right columns). Note also that variations in the rise time of the tones have a dramatic effect on response latency for the least-intense signals (30 dB: panels A,D,G), but only a small effect on latent period for intense tones (80 dB: panels C,F,I).

More detailed data on this issue are presented in Fig. 3. Fig. 3A–C plots, separately for three other neurons, mean latency as a function of the SPL of a CF tone; in each panel, the parameter is the rise time of the tonal signal. The ID numbers, CFs, and fields of origin are

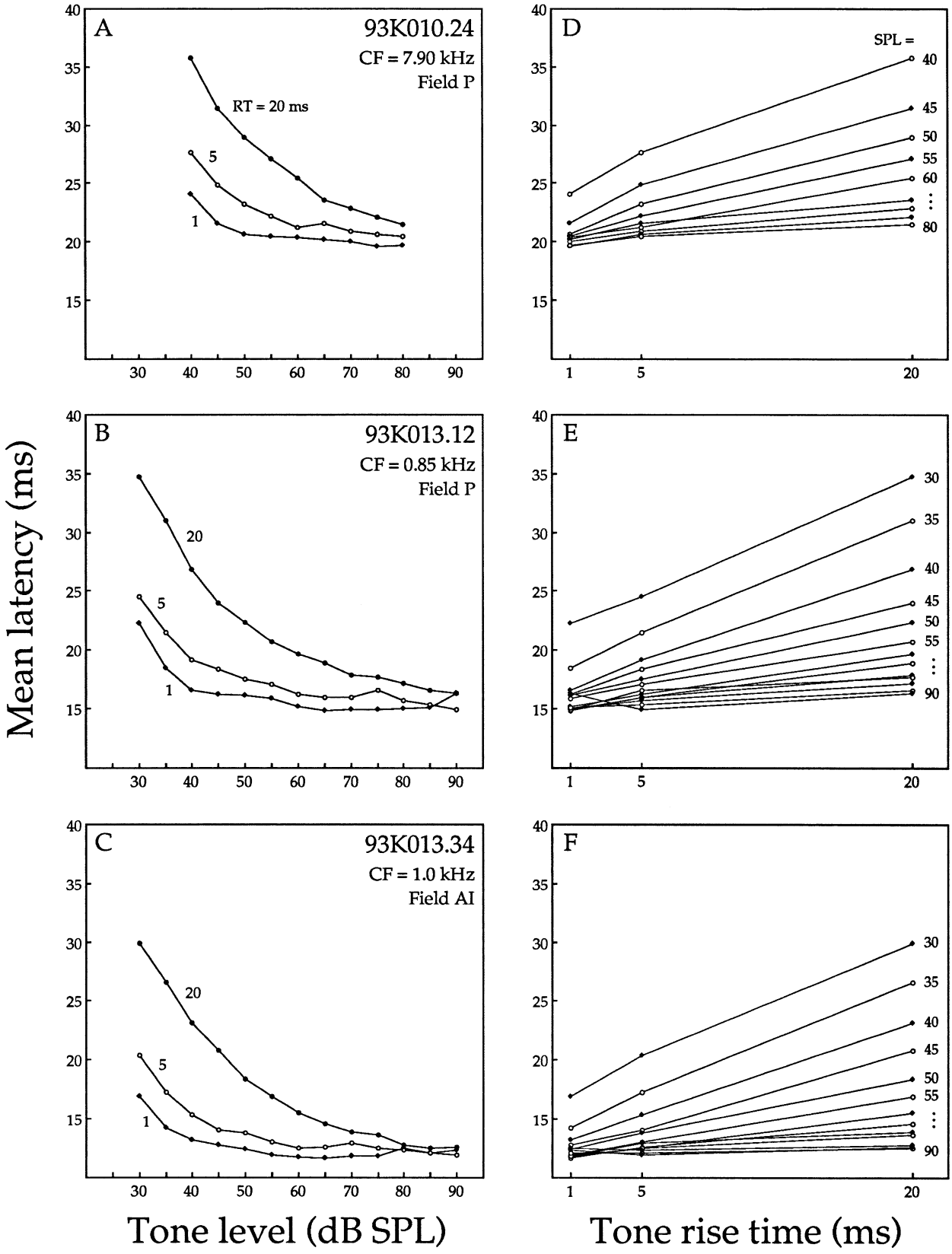

Fig. 3. Left panels: Latency-vs-SPL functions for three neurons, of indicated fields of origin and CF, obtained using CF tones. Parameter is tone rise time (RT). Right panels: same data, replotted so that latency is plotted as a function of tone rise time, with SPL as the parameter.

indicated for each neuron. First-spike latencies were long for stimuli at threshold levels, and declined toward asymptotic minima at high SPLs. Note that the slopes of these functions were steeper for long rise-time tones than for short ones, and that this had the consequence that the three curves for each neuron tended to converge at high SPLs.

Following Burkard [3] and Heil and Irvine [9], the data in panels A–C of Fig. 3 have been re-drawn in panels D–F of Fig. 3, so that mean first-spike latency is plotted as a function of tone rise time, with SPL as the parameter. These functions show a modest tendency toward saturation (i.e. the individual functions tend slightly towards the horizontal at high values of the independent variable), but for the present purposes were treated as roughly linear. What is of special interest is the slope of the individual functions. For low-amplitude tones, the functions are relatively steep, while for high-SPL tones, the functions are nearly horizontal, and there is a quite orderly trend in this behaviour between the two extremes.

According to a simple threshold model (see also Heil and Irvine [9]), the response latency should be a function of the threshold sound pressure required to evoke a spike (P_t), the peak pressure of the sound (P_p), the rise time of the stimulus (R), and a constant transmission time from the periphery (C):

$$L = (P_t/P_p)*R + C \quad (1)$$

i.e. the proportion of the rise time required for the stimulus to reach threshold for spike initiation, plus the constant transmission time. The slope of the latency-vs-rise time function is thus P_t/P_p. This means that for every 5 dB increase in SPL, the slope of that function should fall by a factor of 1.78. (This is because *adding* 5 dB to P_p is, following the dB scale, equivalent to *multiplying* the value of the denominator by a factor of 10 raised to the power of 5/20.)

Fig. 3D–F revealed that the slopes of latency-vs-rise time functions did indeed fall with increases in tone SPL. The generality of these observations is shown in Fig. 4. For 34 neurons (6 in AI and 28 in field P; 20 of the 34 cells had nonmonotonic intensity profiles), data were available on response latencies for wide ranges of tone SPLs, and for each of the three rise time conditions (1, 5, 20 ms). The slopes of the latency-vs-rise time functions were measured from a least-squares linear regression analysis, and this was done for responses to tones of each effective SPL (e.g. for each curve in each panel of Fig. 3D–F). In Fig. 4, the value of this slope is plotted as a function of tone SPL, separately for each of the 34 neurons in the sample.

With very few exceptions, these curves are disposed in a highly orderly swathe from the upper left to the lower right of the plot. The data in this plot come from neurons that differed in field of origin (AI or P), CF, absolute sensitivity, and monotonicity of their intensity profiles. Despite this, the slopes of their latency-vs-rise time functions show a strikingly similar dependence on the peak SPL of the stimulus. For low-SPL tones (upper left), the slopes tend toward 0.8. Recall that the present analysis excluded mean latencies that were based on fewer than 10 spikes over 40 trials because of the very high variability in spike response times at low sound pressures. With this caveat, the data in Fig. 4 suggest that at low SPLs, cortical neuron latencies follow the rise time of the tonal signal relatively well, since a slope near unity here would indicate a good match between prolongation of stimulus rise time, and the prolongation of response latency. This is what one would expect at threshold levels of stimulation. At high SPLs, however, the slopes of the latency-vs-rise time functions tended toward low values (bottom right of Fig. 4: slopes between 0.0 and 0.3). This means that latency prolongations were systematically shorter than the rise-time lengthenings for CF tones of high SPL.

Now, according to the simple threshold model, for every 5 dB increment in stimulus intensity the slope of the latency-vs-rise time function should fall by a factor of 1.78. To test this model, the slopes of the functions in Fig. 4 were measured at 5 dB intervals (the data acquisition resolution), and a ratio was taken of the slopes at the two ends of each interval. Those ratios were then plotted as a function of the SPL at which the ratio was taken. These data area provided in Fig. 5. (Data for one neuron were excluded, because they were obtained with only 10 dB resolution for that cell.) The

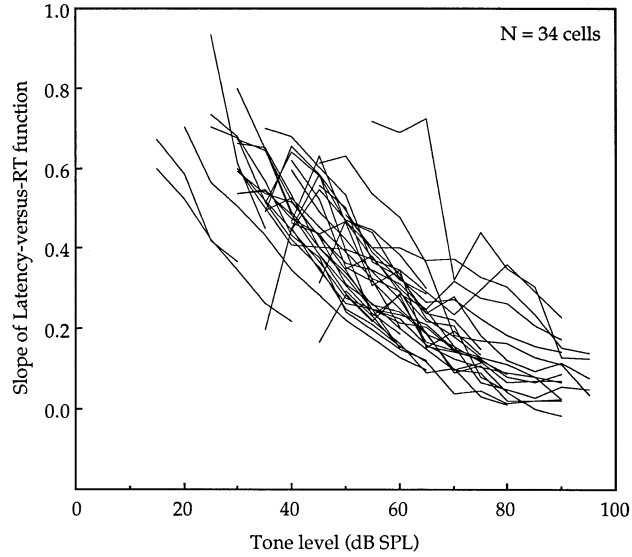

Fig. 4. 3Slope of the latency-vs-rise time function, plotted as a function of SPL, separately for the 34 neurons in the sample. For each neuron, the slope of the latency-vs-rise time function was calculated by least-squares regression, separately for tones of each SPL. That slope was then plotted as a function of SPL, separately for each neuron.

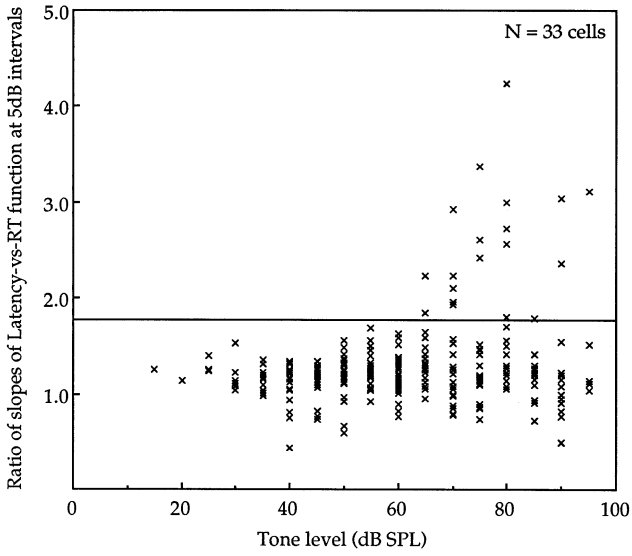

Fig. 5. Ratio of the slopes of latency-vs-rise time function, measured at 5 dB intervals, and plotted separately for the 33 neurons studied in 5-dB steps. The horizontal line represents the slope ratio predicted by the simple threshold model. Note that the vast majority of data points fall below this line.

horizontal line represents the ratio of 1.78 predicted by the threshold model. Almost all of the data points (245/265, or 92.5%) fall below this line, and most of them are in the range from 1.0 to 1.4. The few data points above the line probably reflected noise in the data: at high SPLs, where the absolute values of the slopes of the latency-vs-rise time functions were very low (Fig. 4), this noise had dramatic effects on the slope ratios. In any event, the fact that the slope ratios fell below the line means that the neurons were not initiating spikes at the time that any putatively constant P_t (from Eq. (1), above) had been reached.

4. Discussion

4.1. Dependence of response latency on neural CF

This study has presented evidence that the mean first-spike latency of cortical neurons in the primary field is independent of neural CF. We found that most cat AI neurons had minimal latencies in the range from 12–22 ms, irrespective of CF. When response latencies were studied for constant-SPL stimuli, comparable findings were seen. In the cochlea, there is an inverse relation between the latency of response and neural CF, but this is a small effect (a few ms) attributable to travelling wave delays [7,12]; it may be masked in the present observations by the greater variability of latencies introduced by differences in synaptic path length to the neurons studied.

The failure to observe a relationship between response latency and tone frequency in the present data raises intriguing questions about the origin of the effect in the magnetoencephalographic data reported by Roberts and Poeppel [25]. One possibility is that the phenomenon is unique to man, which would be surprising since other basic response features of the human primary auditory cortex are also seen in animal studies [11,26]. A second possibility is that the magnetoencephalographic technique is measuring a different response to that seen in traditional electrophysiology. In this regard, direct electrical recordings from the primary auditory cortex of both animals (present study; [17,22]) and man [4,14] reveal evoked responses with latencies systematically well under the 100 ms or so that defines the M100. This difference suggests that the two recording techniques are recording fundamentally different processes, and given this, it is unsurprising that two different neural processes have different sensitivities to variations in stimulus parameters. This difference does not dispute the veracity of either response measure; it simply serves to indicate that they reveal the activity of mechanisms at different stages or streams in the processing of the sensory signal. The short latencies of the responses recorded electrically suggests that those responses are generated by activity close to the thalamocortical input. The longer latency of the M100 suggests that it has its origins later in the processing stream.

The further question concerns the suggestion that response latency might serve as a neuronal code for any stimulus parameter [5,6,25]. There is no doubt that, within a neuron, response latency varies with changes in stimulus properties, notably tone intensity (present study; [2,18,22]) and tone frequency [2,18]. This does not mean that latency serves as a neuronal 'code'. By definition, latency is the time between stimulus onset and response onset, and since cortical neurons probably have no independent information about the timing of stimulus onset, isolated response event times have no referent and so do not in themselves provide information about the stimulus. Moreover, response latencies are multiply determined, and thus, like spike counts, inherently ambiguous. The present study confirmed that response latency is determined by both tone intensity and tone rise time. Other studies have shown that latency is also determined by tone frequency or binaural stimulus parameters [2,18]. This multideterminism means that an absolute latency value in itself cannot uniquely specify the stimulus conditions that generated the response.

None of the foregoing disputes that the timing of afferent events in cortical cells has a dramatic influence on shaping response rates in individual neurons (indeed, in determining which neurons are activated by the stimulus), and in shaping the timing of those responses.

Through those mechanisms, afferent latencies can shape both the spatial distribution of the activated cells across the cortical mantle, and the relative timing of spike activity in those populations; but that is not the same thing as an assertion that a response latency in its own right is a neural code. For a neural event time to have information content, that event time must have a referent, whether it be a response event time from the opposite ear in the case of binaural processing [2], or quite possibly from the same ear, as in the instance of echo-location where the delay between neural event times (representing pulse and echo event times) carries information about target distance [29]. Note that in both of these instances, it is not the latent period of a response per se which constitutes a code; it is the temporal relation between event times.

4.2. Dependence of response latency on tone rise time

The present study has provided detailed evidence on the sensitivity of the mean first-spike latencies of neurons in two auditory cortical fields to variations in the rise time of CF tones. In both AI and P, at low sound pressures, mean latent period tracked stimulus rise time relatively well, as indicated by the fact that the slope of the latency-vs-rise time functions were tending toward values near unity (Fig. 4). This is what would be predicted by a simple threshold model of spike initiation, because the stimulus would have to reach peak amplitude in order to reach spike threshold. For high-amplitude signals, tone rise time had smaller effects on first-spike latency (Fig. 2, Fig. 3 and Fig. 4). This direction of effect is again predicted by the threshold model. However, that model predicted a factor of 1.78 change in the slope of the latency-vs-rise time functions for every 5 dB increment in SPL, a value which was almost never reached (Fig. 5). These data confirm the preliminary observations of Heil and Irvine [9] for AI, and extend them to a second cortical field. The present data also show that the fashion in which the latency behavior of cortical cells fails to match that predicted by the model is, for the most part, highly regular.

We might reasonably ask whether the use of cosine rise-fall envelopes in the present study is responsible for our failure to support the simple threshold model. Recall that the cosine envelope begins with a gradual slope which later accelerates and then flattens. Accordingly, during the first half of the cosine rise time, the stimulus amplitude lags behind that of the linear rise-time stimulus of the same peak amplitude. For a series of constant rise-time tones, the time taken to reach a hypothesized threshold would decrease with increasing SPL, but would do so more slowly for the tones with the cosine envelopes. This means that any threshold level for spike triggering mechanisms would occur relatively *later* than it would have, had we used linear rise times (because the relevant threshold level would occur early in the rise time). The present analyses, in practice assuming a linear rise time, suggested that cortical neurons responded *before* any such threshold level had been reached. That this was true, despite a cosine envelope that would prolong response latencies for the threshold-level stimulus, testifies to the robustness of the present findings.

These data have important implications. The first is that the simple threshold model of first-spike timing in cortical cells, which has been implicit in discussions of central auditory wiring and response timing (e.g. [12,15,22,28]) is probably in error. It is possible that the hypothesized stimulus trigger level required for spike initiation is not a constant value, as is implicit in Eq. (1). In this regard, studies of the cortical spike rate-coding of tones presented against noise backgrounds have shown that spike thresholds track very closely the signal to noise ratio of the stimulus rather than the absolute value of the tone amplitude [16]. This observation does not explain the presently demonstrated failure of the threshold model, but it does provide a precedent for suspecting that the responses of cortical cells are driven by relative stimulus amplitudes rather than absolute ones.

A second implication of the present data is that it is not the tone's peak amplitude per se which determines first-spike latency, an assumption that has otherwise always been implicit in plots of response 'latency vs. SPL' (e.g. Fig. 3). Recall from Eq. (1) that there is a transmission time of sensory information from the auditory periphery to the cortex. A good estimate of the minimal transmission time is provided by cochlear implant studies in which the cochlear nerve array is stimulated directly. In this regard, Raggio and Schreiner [23] reported that minimal cortical response latencies to such electrical stimuli were on the order of 11 ms. The present study showed that minimal latencies to acoustical stimulation (see especially Fig. 1B, Fig. 2C,F,I) were in the order of 12–22 ms (see also Raggio and Schreiner [23]). This means that the stimulus events responsible for the timing of the first spike may be those in the first few ms of the stimulus, despite the fact that the rise time of the stimulus may be 20 ms long (e.g. Fig. 2C).

This general finding is not new. Suzuki and Horiuchi [30] drew the same conclusion from studies of the human brainstem auditory evoked response. Burkard and his colleagues [1,3] reported strikingly similar behavior to that seen in the present study in the latency of wave V of the gerbil and human brainstem auditory evoked response. This means that the latency/rise-time relationship seen in the responses of cortical neurons (present study; Heil and Irvine [9]) is not a uniquely cortical phenomenon. Indeed, the fact that this relationship is at least qualitatively similar to that seen in *wave*

I of the gerbil brainstem auditory evoked response [3] suggests that the phenomenon might have its origins in the cochlea. Detailed, quantitative comparisons of the rise time effects at the level of the cochlea and above have yet to be reported. They will, however, be an important source of information about the origins of the disparity between the predictions of the threshold model and the present neurophysiological data.

The further question is that of what property of the stimulus determines first spike latent period if it is not the peak amplitude? One possibility is the rate of change of stimulus pressure [9], for the obvious reason that this can be expressed prior to the stimulus reaching peak amplitude. The answer to this question is not recoverable from the present data because cosine-envelope rise times were used in most of the experiments, but it offers a useful direction for future research.

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