Heil, Peter. Auditory cortical onset responses revisited. II. Response strength. J. Neurophysiol. 77: 2642 ± 2660, 1997. Most neurons of the auditory pathway discharge spikes locked to the onset of an acoustic stimulus, but it is largely unknown in which way the acoustic parameters of sound onsets shape the neuronal responses. In this paper is analyzed the number of spikes discharged by single neurons in primary auditory cortex of barbiturate-anesthetized cats to the onsets of tones of characteristic frequency. The time course of the peak pressure (i.e., the envelope) was altered by parametrically varying sound pressure level (SPL), rise time, and rise function (linear or cosine-squared). For both rise functions, rise time had manifold, and in some cases dramatic, effects on conventional spike count–level functions. In general, threshold SPL, dynamic range, and the lowest SPL at which monotonic spike count functions saturated increased with prolongation of the rise time. In neurons with mostly nonmonotonic spike count–level functions, “best SPL” increased and the descending high-SPL arms flattened, so that functions obtained with long rise times were often monotonic whereas those obtained with shorter rise times were highly nonmonotonic. Consequently, the “tuning” to SPL was less sharp for longer rise time tones, and spike count versus rise time functions changed from “short-pass” to “long-pass” with an increase in SPL. Systematic effects of rise time persisted when spike counts were plotted against the rate of change of peak pressure or against the maximum acceleration of peak pressure. However, when spike counts were plotted as a function of the instantaneous peak pressure at the time of response initiation, the functions obtained with different rise times, and even with different rise functions, were in close register. This suggests that the stimulus-dependent component of first-spike latency can be viewed as an integration window, during which rate of change of peak pressure is integrated. The window commences with tone onset and its duration is inversely related to the maximum acceleration (or, for linear rise functions, the rate of change) of peak pressure and the neuron’s transient sensitivity. The present findings seriously question, for onset responses, the usefulness of the spike count–level function and measures derived from it, such as threshold SPL, dynamic range, best SPL, or degree of nonmonotonicity. They further cast doubt onto the validity of current concepts of intensity coding at cortical levels, because most neurons’ onset responses are not indicative of a signal’s steady-state SPL. However, they suggest a mechanism by which a neuronal population will sample a given transient in an orderly, sensitivity-dependent, temporal sequence. The sampling rate is automatically adjusted to, and adjusted by, the rapidity of the signal’s change. And the instantaneous properties of the transient could be represented by the ratios and spatial distribution of responses across the simultaneously active subpopulation. Such a mechanism could provide the basis for the demonstrated capability of discrimination of rapid transients.

INTRODUCTION

Most neurons of the auditory pathway respond briskly to transients in acoustic stimuli, such as their onsets, provided the stimuli have frequency spectra adequate to excite the neurons. The responses of neurons in the auditory cortex, particularly of anesthetized animals, are dominated by such transients: most, if not all, of their discharges are locked to the onset of a sound, such as a tone. This is seen as a vertical alignment of initial discharges in dot-raster displays and is reflected in a sharp initial peak of the poststimulus time histogram (e.g., Brugge et al. 1969; Calford and Semple 1995; Phillips 1993; Phillips and Sark 1991; Semple and Kitzes 1993a).

Despite the general recognition that the initial discharges of most auditory neurons are locked to, and are thus evoked by, stimulus onset, the question of which physical attributes of sound onsets are relevant for shaping onset responses of auditory neurons has received little attention (Condon et al. 1991; Gooler and Feng 1992; Hall and Feng 1988, 1991; Phillips 1988; Phillips et al. 1995; Suga 1971). On the contrary, it is a common and rather paradoxical practice to plot or analyze such responses as a function of a stimulus parameter that characterizes the steady-state portion of that stimulus. An important case in point is the sound pressure level (SPL), a parameter that quantifies a normalized (i.e., re 20 μPa) and averaged (i.e., root-mean-square) peak pressure of a stimulus. When the spike counts or discharge rates, e.g., of neurons in the auditory cortex, are plotted as a function of a tone’s SPL, the resultant level or “intensity” functions of auditory neurons are of diverse shape (see, e.g., Brugge and Merzenich 1973; Brugge et al. 1969; Heil et al. 1992a, 1994; Phillips 1988, 1990; Phillips and Irvine 1981; Phillips et al. 1995; Schreiner et al. 1992; Suga 1977; Sutter and Schreiner 1995). Above a particular SPL, the threshold SPL, spike counts can increase monotonically over some range of SPL, the dynamic range, and then saturate. In other instances, spike counts can change nonmonotonically with SPL: beyond an initial increase of spike counts with SPL to a maximum, defining the “best SPL” (Brugge and Merzenich 1973), spike counts decrease with further increases in SPL. The exact proportions of functions falling into the two categories vary somewhat in different studies, in part because of the criteria employed. Functions obtained from different neurons differ in threshold SPL, in dynamic range, in best SPL, and in aspects of their nonmonotonicity.

The general practice of plotting spike counts or discharge rates derived from neurons with clear onset response as a function of the SPL implies that such responses are in fact sensitive to the SPL, an implication that in turn has led to the notion that such responses might code or process SPL (for studies of auditory cortex, see, e.g., Heil et al. 1994; Phillips 1990; Phillips and Kelly 1989; Phillips et al. 1995; Schreiner et al. 1992; Sutter and Schreiner 1995). However, this need not be the case. Because of concern about spectral splatter at the onset of narrowband signals, such as tones,
signals are often shaped with rise functions of some duration to reduce such splatter. When the rise time is fixed, as is routinely done, any alteration of the SPL inevitably alters parameters of stimulus onset. Such parameters include the rate with which the peak pressure or envelope increases during the rise time, and the acceleration of peak pressure. In the companion paper (Heil 1997) it is shown that the latency of a cortical neuron’s response is in fact a function of these parameters. It is thus conceivable that the response strength of these neurons may be shaped by the same parameters rather than by the steady-state SPL. The practice of plotting spike counts or discharge rates of onset responses as a function of steady-state stimulus properties is also commonly encountered in studies concerned with the effects of differences in the SPL at each ear [interaural intensity differences or interaural level differences (ILDs)], which constitute the major cue to the azimuthal location of high-frequency narrowband sounds. Again, such practice (for auditory cortex, see, e.g., Brugge et al. 1969; Irvine et al. 1996; Orman and Phillips 1984; Phillips and Irvine 1981; Semple and Kitzes 1993a,b) implies that the neurons are sensitive to a particular ILD or combination of SPLs at the two ears. However, the steady-state SPLs or the ILD are achieved only at the end of the rise time, i.e., after stimulus onset, whereas during the rise time SPL and ILD are dynamic, i.e., they increase continuously with time.

To determine the features of tone onsets relevant for shaping neuronal onset responses, we have altered the onsets of tone bursts by varying SPL, rise time, and rise function. The present study focuses on neurons in the primary auditory cortex (AI), because their responses are dominated by sound onsets, but the results obtained might apply to onset responses of auditory neurons in general. Furthermore, the knowledge derived from the analysis of responses to isolated onsets may have implications for the coding of transients in more complex acoustic signals and environments.

**METHODS**

The methods were identical to those used in the companion paper and are therefore only briefly described here. Adult cats with healthy ears were anesthetized with pentobarbitone sodium (40 mg/ml ip) and were given atropine sulphate (0.3 ml im) and a broadband antibiotic (Amoxil, 0.5 ml im). The trachea and a radial vein were cannulated and anesthesia was maintained by additional intravenous injections of pentobarbitone sodium when required. A round window electrode and a length of fine-bore polyethylene tubing to allow static pressure equalizations within the middle ear were implanted in each bulla. The left middle eustachian gyrus was exposed and a Perspex chamber was mounted to the skull around the opening. The chamber was filled with warm saline and was sealed with a glass-plate housing the glass-insulated tungsten microelectrode. The electrode was advanced near-normal to the cortical surface by a small hydraulic microdrive.

The responses of well-isolated single neurons in AI were recorded and event times were stored on disk for off-line analysis. Acoustic stimuli were digitally produced (Tucker Davis Technology) and presented to the cat’s tympani via precalibrated sealed sound delivery systems (STAX SRS-MK3). After an initial brief characterization of the neuron with regard to its characteristic frequency (CF) and preferred laterality of stimulation [i.e., monaural contralateral, monaural ipsilateral, or diotic (binaural with identical stimuli presented to each ear)], quantitative data were collected with the use of tone bursts 400 ms in duration including the symmetrical rise and fall times. SPL, rise and fall time, and rise and fall function were varied. Tone bursts were shaped with either cosine-squared or with linear rise and fall functions. With the cosine-squared rise function, the envelope, i.e., the peak pressure, changes as a function of rise time $t$ (in s) according to

$$PP = P_{plateau} \cos^2 \left( \frac{t}{CRT} \pi^2 + \pi/2 \right), \quad 0 \leq t \leq CRT$$

where $CRT$ is the rise time and $P_{plateau}$ is the plateau peak pressure at the end of the rise time.

First, 20 repetitions of CF tones of the preferred laterality and with a given rise function and rise time were presented (rate: 1 Hz) at each of a number of levels ranging from subthreshold to 90 dB SPL. Thereafter, spontaneous activity was determined in an analogous fashion, i.e., in 20 400-ms intervals at 1 Hz, before selection of another rise time and repetition of the same procedure. As many as seven different rise times, covering a range from 1 to 170 ms, were selected and presented in pseudorandom sequence. In 30 of 72 neurons, linear as well as cosine-squared rise functions were tested in this way.

Spikes in response to each stimulus were displayed on- and off-line as a poststimulus time histogram. This histogram was used to select an analysis window that would comprise only spikes locked to the tone’s onset and would discard late discharges, offset responses, or presumed spontaneous spikes. The selection of such a window was generally straightforward. In agreement with observations made by others (Brugge et al. 1969; Calford and Semple 1995; Phillips and Irvine 1981), most neurons discharged only one spike or a short burst of two or three spikes tightly locked to the onset, so that the poststimulus time histogram showed only a single sharp peak shortly after tone onset. A response to stimulus offset (i.e., a peak in the poststimulus time histogram at an interval of >400 ms after onset) was seen in rare instances with tones of high amplitudes and short rise times. Late discharges were also rare, although somewhat more common than offset responses, and appeared to be related to the depth of anesthesia. Late discharges were readily distinguished from onset responses by a marked interval of no activity. Spontaneous activity was generally very low. It never exceeded 3 spikes/s, and most neurons showed no spontaneous activity at all. The number of discharges to the 20 repetitions of each stimulus in the analysis window and the response probability were computed.

**RESULTS**

**Effects of rise time on conventional spike count–level functions**

In accordance with general practice, spike counts obtained from a given neuron with tones of a particular rise function and rise time were plotted as a function of SPL. These functions will be referred to as spike count–level functions. When, for a given neuron, spike count–level functions obtained with different rise times were compared, marked effects of rise time on multiple aspects or features of the functions were apparent.

**Threshold.** One effect of rise time is on the firing threshold of a neuron when threshold is expressed in the conventional way, viz., in terms of the plateau amplitude (in dB SPL) of a tone needed to meet some threshold criterion. I have selected a response probability of 0.1 as the threshold criterion rather than a particular spike count, because some neurons discharged more than one spike per stimulus, even for tones near threshold. Rise time effects on threshold SPL can be seen in the seven spike count–level functions
Fig. 1. Effects of rise time of characteristic frequency (CF) tone bursts on spike count functions for 2 neurons. Neuron 95-98/10 (a–d) was tested with contralateral tone bursts of 10.3 kHz and neuron 95-98/14 (e–h) was tested with diotic tone bursts of 5.5 kHz. All tone bursts were shaped with cosine-squared rise functions and the rise times are identified in the key in a. Key applies to all panels but b and f. Spike counts were accumulated in 20 repetitions of each stimulus. In a and e, spike counts are plotted as a function of the sound pressure level (SPL; in dB SPL). In b and f, data are replotted as a function of rise time for tones of different SPLs (identified in the keys). Spike counts are plotted as a function of the maximum rate of change of peak pressure in c and g, and as a function of the maximum acceleration of peak pressure in d and h.

of neuron 95-98/10 (Fig. 1a). For each rise time, the spike counts increase monotonically with SPL until a saturating response of 20 spikes (1 spike per trial) is reached. However, the functions “take off” at different SPLs, i.e., they differ in threshold SPL. Threshold SPL is low for short rise times (~10 dB SPL) and relatively unaffected for rise times up to 17 ms, but increases by >20 dB for rise times of ≥42 ms. In some other neurons, systematic increases in threshold SPL could be observed even for short rise times, whereas in others, such as neuron 95-98/
Fig. 2. Effects of rise time of cosine-squared rise function tones on threshold characteristics. a: plot of SPL (in dB SPL) of the CF tone burst eliciting a threshold response against the rise time (in ms/1.7). Data obtained from a given neuron and with a particular stimulus laterality are represented by the same symbol and are connected. b: plot of the maximum acceleration of peak pressure of the CF tone burst eliciting a threshold response against the rise time (in ms/1.7). Other conventions as in a.

14 (Fig. 1e), there were hardly any effects of rise time on threshold SPL. For this neuron, spike counts for most rise times are nonmonotonic functions of the SPL (see below).

Figure 2a provides an overview of the effects of rise time on threshold SPL. For each neuron for which level functions were obtained with cosine-squared rise function tones, the SPL of the threshold obtained with presentation of tones with a given laterality is plotted as a function of rise time. It is readily apparent from this figure that in some neurons threshold SPL is independent of the rise time, at least over the range tested, and that for most of these neurons threshold SPLs are quite low (<15 dB SPL). For the majority of neurons, however, the SPL of a threshold tone increases with rise time, and increases can be ≥60 dB. Some neurons did not respond to CF tones, even at 90 dB SPL, when the rise time was long. In some neurons increases in threshold SPL occurred only for the longest rise times tested; in others, such increases are evident for intermediate rise times, and in a few neurons even for very short rise times. The latter neurons tend to have relatively high-threshold SPLs, even for the shortest rise times. Consequently, the range of threshold SPLs covered by the neuronal sample also widens markedly with the rise time of the tone bursts. Note that rise time is plotted on a logarithmic scale to provide a higher resolution for the range of short rise times. Nevertheless, it is apparent that the slope with which the threshold SPL increases with rise time changes with rise time for a given neuron and, over a given rise time range, differs among neurons. In summary, Fig. 2a illustrates that in general the threshold SPL of a CF tone increases with rise time but that the details of this effect vary with rise time and vary among neurons. Similarly, the threshold SPL can increase with rise time also for tones of frequencies away from the CF, and the magnitude of this effect varies with frequency (see Fig. 6 in the companion paper).

Dynamic range and best SPL. Figure 1a shows that spike count–level functions are not simply shifted in a parallel fashion along the SPL axis with increments in rise time. Rather, the rise time also affects the dynamic range of such functions, i.e., the range of tone SPLs over which the neuron’s response increases from threshold or near threshold to maximum or near maximum. For neuron 95-98/10 (Fig. 1a) the dynamic range of the spike count–level functions is rather narrow for short rise times. The precise limits of this range depend on the criterion used, but the dynamic range extends approximately from 10 to 20 dB SPL, a 10-dB range. For longer rise times, the dynamic range becomes systematically wider, that obtained with 170-ms rise time tones extending from ~30 to 70 dB SPL, a 40-dB range. Similar observations hold true for neuron 95-98/14 in Fig. 1e. In fact, an overview over the effects of rise time on dynamic...
range allows observations similar to those made for threshold SPL, i.e., the dynamic range generally increases with rise time, but different neurons are affected to a different degree (not shown).

It necessarily follows from combination of the effects of rise time on threshold SPL and on dynamic range that for monotonic spike count–level functions the lowest SPL that elicits the maximum (i.e., saturation) response from a given neuron increases with rise time (e.g., from 20 to 70 dB SPL for neuron 95-98/10, Fig. 1a). For nonmonotonic functions it is the best SPL, i.e., the position of the peak response, that increases with rise time (e.g., from 20 to 60 dB SPL for neuron 95-98/14, Fig. 1e).

NONMONOTONICITY AND ‘‘TUNING’’ TO SPL. Other prominent effects of rise time on features of spike count–level functions are also illustrated by neuron 95-98/14 (Fig. 1e). With longer rise times, the descending, high-SPL arms of the spike count–level functions become shallower. In other words, the functions become increasingly less nonmonotonic. For example, the spike count–level function of neuron 95-98/14 obtained with 170-ms rise time tones would be classified as monotonic. The functions obtained with rise times of 85 and 42 ms would likely also be classified as monotonic (depending on criterion), whereas those obtained with shorter rise times are clearly all nonmonotonic because the neuron ceased to fire at high SPLs. If one were to measure the SPL at which the spike counts dropped by 20% from maximum on the descending high-SPL slope of the spike count–level function (defined as an ‘‘inhibitory threshold’’ by Phillips 1988), these values would also increase with rise time. Also note that such an inhibitory threshold increases more rapidly with rise time than the threshold SPL. Similarly, if one were to measure the (high) SPL at which the neuron ceases to discharge (defined as an ‘‘upper threshold’’ by Suga 1971), these values would also increase with rise time, and would do so more rapidly than the threshold SPL. Another consequence of the decreasing nonmonotonicity with prolonged rise time is that the bandwidths of spike count–level functions become wider. The width may be measured at some convenient level (e.g., 50%; Phillips 1988) below the maximum response, which in this neuron scatters around 40 spikes independent of the rise time. In other words, the sharpness of the apparent tuning to SPL decreases with rise time. Similar findings were made in other neurons (see, e.g., Figs. 5, 7, 9, and 12).

TUNING TO RISE TIME? The rise time effects on spike count–level functions, as described above, can be viewed from another perspective. When the data are replotted as spike count–rise time functions, these functions are severely effected by SPL (Fig. 1, b and f). For neurons with largely monotonic spike count–level functions, spike counts decrease with prolongation of the rise time when the SPL is low (e.g., between >10 and 30 dB SPL for 95-98/14; Fig. 1f), but increase with increasing rise time for higher SPLs (e.g., for levels >40 dB SPL for neuron 95-98/14; Fig. 1f). Thus, for nonmonotonic neurons, spike count–rise time functions change from ‘‘short-pass’’ to ‘‘long-pass.’’

Basically identical effects of rise time on spike count–level functions were seen with linear rise function tones, corroborating previous observations by Phillips (1988). Data from two neurons (95-87/12 and 95-92/20) are illustrated in Fig. 3, top.

Effects of rise time on functions relating spike counts to peak pressure changes

Any alteration of the rise time or of the SPL of a tone burst also alters the rate of change and the acceleration of peak pressure at the tone burst’s onset. It is thus conceivable that the spike counts of cortical neurons might be functions of these parameters rather than of a tone burst’s SPL or its rise time. These possibilities are particularly intriguing in this context, because a neuron’s first-spike latency is an unambiguous function of the rate of change of peak pressure (for cosine-squared rise function tones) and of the acceleration of peak pressure (for cosine-squared rise function tones; see companion paper).

In Fig. 1, the spike counts of neurons 95-98/10 and 95-98/14 obtained with tones of different cosine-squared rise time and SPL are plotted against the maximum rate of change of peak pressure (c and g) and against the maximum acceleration of peak pressure (d and h). For each neuron, the spike count versus rate of change of peak pressure functions obtained with different rise times appear more similar to one another than do the corresponding spike count–level functions (compare Fig. 1, c with a and g with e). This apparently greater similarity is in part due to the differences in the scales of the abscissas. The 100-dB range of SPLs is equivalent to a range of plateau peak pressure of 5 orders of magnitude. But with the range of rise times, which covers 2 orders of magnitude, the maximum rate of change of peak pressure varies over a range of 7 orders of magnitude. Despite this compressive scaling effect, spike count versus rate of change of peak pressure functions obtained with different rise times are displaced and exhibit shape differences (Fig. 1, c and g).

Similar observations can be made when inspecting the spike count versus maximum acceleration of peak pressure functions (Fig. 1, d and h). Partly because of the compressive scaling of the abscissas (9 orders of magnitude), spike count–acceleration functions appear more similar in shape than the corresponding spike count–level functions (compare Fig. 1, d with a and h with e). Inspection of the predominantly nonmonotonic spike count–acceleration functions of neuron 95-98/14 (Fig. 1h), but also of other neurons, revealed that the descending slopes of the functions obtained with different rise times align reasonably closely. Overall, however, functions obtained with different rise times are not congruent. Thus, although for cosine-squared rise function tones first-spike latency is an unambiguous function of maximum acceleration of peak pressure, irrespective of rise time and SPL, the number of spikes, the firing probability, and the firing threshold are not.
FIG. 3. Effects of rise time of CF tone bursts shaped with linear rise functions on spike count functions for 2 neurons.

Neurons 95-87/12 (left) and 95-92/20 (right) were tested with diotic tone bursts of 21 and 12.5 kHz, respectively. Spike counts are plotted as a function of SPL (top) and of the rate of change of peak pressure during the rise time (bottom). Note that features of spike count functions are affected by the rise time of linear rise function tones in much the same way as they are by the rise time of cosine-squared rise function tones (cf. Figs. 1 and 2).

The general validity of the latter point is substantiated by the summary of the maximum acceleration threshold data for all neurons in Fig. 2b. This figure plots the maximum acceleration of peak pressure at the onset of a threshold tone (i.e., a tone meeting the threshold criterion of a response probability of 0.1) as a function of rise time. For many neurons, the acceleration of peak pressure of a threshold tone decreases with rise time, as is the case for the neurons illustrated in Fig. 1, d and h. A slope of −2 in this log-log plot, as exhibited by most of the lower lines throughout the entire range of rise times and by most neurons for short rise times, is equivalent to a horizontal course of the corresponding threshold SPL versus rise time function in Fig. 2a. Only for very few neurons, or over some narrow rise time range, does a neuron’s threshold (in terms of acceleration of peak pressure) appear to be independent of rise time. In some neurons, the acceleration of peak pressure of a threshold tone can even increase with rise time, most notably for long rise times, leading to U-shaped threshold acceleration versus rise time functions.

Comparable observations were made in neurons studied with linear rise function signals. When spike counts were plotted against the rate of change of peak pressure, the functions obtained with different rise times were more similar in shape and were often more closely aligned than the corresponding spike count–level functions, but were not entirely congruent (Fig. 3). However, the descending arms of non-monotonic functions were often in rather close register (e.g., neuron 95-92/20; Fig. 3, bottom right).

Thus, although the initial spikes of a cortical neuron are triggered by tone onsets, the number of these spikes and the firing probability, in contrast to first-spike latency, are not unambiguous functions of the onset parameter maximum acceleration or, in case of linear rise functions, the rate of change of peak pressure.

Concept of an integration window

As shown in the companion paper, the latency $L_{\text{CRF}}$ of a neuron to cosine-squared rise function tones is a function of the maximum acceleration of peak pressure $\text{APP}_{\text{max}}$, and can be described by

$$L_{\text{CRF}} = L_{\text{max}} + \hat{A}_{\text{CRF}} \ast (\log \text{APP}_{\text{max}} + S)^{-1}$$

where the subscript indicates that the measures were derived from responses to cosine-squared rise function tones. $S$ is the measure of a neuron’s transient sensitivity and refers to the logarithm of an acceleration of peak pressure (in Pa/s$^2$). $\hat{A}_{\text{CRF}}$ is the best average scaling factor of the cortical latency functions and is $\sim 12.8$ s. $L_{\text{min}}$ is the minimum against which the latency asymptotically converges. It is a neuron-specific constant and is independent of the magnitude of the stimulus’ acceleration of peak pressure. It includes acoustic delays, traveling wave delays in the cochlea, the total axonal travel time, and some synaptic delays, and is thus expected to increase with the synaptic distance of the neuron from the cochlea. The right-side term of the above equation, viz., $[\hat{A}_{\text{CRF}} \ast (\log \text{APP}_{\text{max}} + S)^{-1}]$, however, depends on the magnitude of $\text{APP}_{\text{max}}$ and on the neuron’s $S$. It identifies the time of response initiation rela-
Fig. 4. Time courses of peak pressure (top), rate of change of peak pressure (middle), and acceleration of peak pressure (bottom) during the onsets of 3 signals shaped with cosine-squared rise functions (left) and 3 signals shaped with linear rise functions (right). Signals at left have in common the maximum acceleration of peak pressure at signal onset, but differ in the time course of acceleration, of rate of change of peak pressure, and of peak pressure, and in rise time and plateau peak pressure of SPL. Signals at right have in common the (constant) rate of change of peak pressure during the rise time, but differ in rise time and plateau peak pressure. If the 3 signals in each column were tone bursts of a given frequency, they would elicit a response from a given neuron with the same latency.

...tive to tone onset, and I therefore propose that this term may specify the duration of a window $\tau$, which might be used by the neuron to integrate aspects of the signal. This window commences with tone onset. For a given neuron and for tones of a given frequency, the window’s duration depends only on $APP_{\text{max}}$. Across different neurons, the integration window for a signal of a given $APP_{\text{max}}$ and frequency depends on the neuron’s $S$. The higher $APP_{\text{max}}$ or $S$, the shorter the window.

Figure 4, left, shows the time courses of peak pressure (top), rate of change of peak pressure (middle), and acceleration of peak pressure (bottom) for three cosine-squared rise function signals that share the same maximum acceleration of peak pressure. The signals differ in rise time and in the plateau peak pressure or SPL. If these signals are tone bursts of the same frequency, then for a given neuron all three signals will result in an integration window with the same duration. If the neuron responds to these signals it will do so with the same latency, i.e., at a delay of $L_{\min}$ after the end of the integration window.

Let us assume that a neuron simply integrates one of the three signal parameters over its integration window, and that the number of spikes it then discharges is a function of the size of the integral. If this is the case, it is readily apparent that the number of spikes discharged by the neuron in response to the three stimuli would be different. Furthermore, the shorter the integration window, i.e., the higher $APP_{\text{max}}$ or the neuron’s $S$, the smaller would be the differences in the integrals for the three stimuli, and the smaller would be the expected differences in spike counts. Analogous arguments apply to the responses to linear rise function tones. This suggestion is compatible with the observations made...
on the spike count versus acceleration of peak pressure functions (or rate of change of peak pressure for linear rise functions) obtained with different rise times: the effect of rise time diminishes with higher acceleration (or rate of change) of peak pressure (Figs. 1, d and h, and 3, bottom).

Test of the integrator concept

This integrator idea can be examined more quantitatively. If a neuron integrated the acceleration of peak pressure during the proposed integration window \( \tau \), its spike counts should be a function of the instantaneous rate of change of peak pressure at the end of the integration window. Likewise, if a neuron integrated the rate of change of peak pressure, its spike counts should be a function of the instantaneous peak pressure at the end of the integration window. Let us first consider responses to cosine-squared rise function tones.

For each neuron and stimulus condition the integration window \( \tau \) was calculated by applying the formula

\[
\tau = \frac{L_{\	ext{end}} - L_{\text{min}}}{\dot{A}_{\text{app}}} = \frac{\log APP_{\text{max}} + S}{A_{\text{app}}} \tag{3}
\]

Note that the calculation of \( \tau \) is based on the best average fit of the shape of latency-acceleration functions measured in AI neurons as represented by Eq. 2. The alternative approach, viz., subtraction of \( L_{\text{min}} \) from the first-spike latency actually measured in response to a given stimulus, would have precluded the estimation of \( \tau \) for stimuli ineffective in eliciting a response. Otherwise, no appreciable difference between the two procedures is expected, because Eq. 2 describes the latency data well (see companion paper).

Next, the instantaneous rate of change of peak pressure and the instantaneous peak pressure at the end of \( \tau \) were calculated for each stimulus, and the spike counts of a given neuron were plotted against these parameters. It soon became apparent that spike count functions obtained from a given neuron with different rise times did not align with the instantaneous rate of change of peak pressure at the end of the integration time (see also results with linear rise function tones below). In particular, a range of spike counts could be obtained from a given neuron when the instantaneous rate of change of peak pressure at the end of the integration time was zero, i.e., in instances when \( \tau \) was longer than the rise time. These observations suggest that the neurons did not integrate acceleration of peak pressure during the proposed integration window.

In contrast, close alignments of the spike count functions obtained with different rise times were seen when spike counts were plotted against the instantaneous peak pressure at the end of the proposed integration time \( \tau \). This finding is illustrated in Fig. 5 for four neurons with largely nonmonotonic spike count functions. At left, spike counts are plotted in the conventional fashion, i.e., against the SPL of the tone burst. For ease of comparison, however, SPL has been converted into the plateau peak pressure and is expressed in Pa. The 5 orders of magnitude of this parameter correspond to a 100-dB range of SPL from about \(-10 \) to \( 90 \) dB SPL. The five spike count–level functions of neuron 95-92/03 (top left) show the range of rise time effects described above, viz., threshold SPL, dynamic range, and best SPL increase with prolongation of the rise time. In addition, the functions become less nonmonotonic and the sharpness of tuning to SPL declines. The same data are plotted against the instantaneous peak pressure at the end of the proposed integration time \( \tau \) in Fig. 5, top right. Note that the abscissas of both plots span the same range of peak pressure. Many of the rise time effects seen in the conventional spike count–level functions are considerably reduced when spike counts are plotted against the instantaneous peak pressure at the end of the integration window. Thresholds are much closer together, the dynamic ranges are more similar, the peaks coincide more closely, and the descending arms of the nonmonotonic functions have very similar slopes so that the sharpness of tuning to the instantaneous peak pressure at the end of the integration time is relatively unaffected by rise time. A scrutiny of the five functions shows that the remaining differences between them are no longer systematically related to rise time. Note that the apparently monotonic spike count functions obtained with rise times of 170 and 85 ms cover only the ascending portions of the bell-shaped functions obtained with shorter rise times.

Similar results can be seen in the other three neurons illustrated in Fig. 5. Neuron 95-92/04 (2nd row) was recorded in the same penetration and in close proximity to neuron 95-92/03, but could discharge more than one spike per stimulus. Furthermore, the functions relating the spike counts to the instantaneous peak pressure at the end of the integration window were slightly broader than those of neuron 95-92/03 and appeared somewhat skewed. The maximum number of spikes was not systematically related to rise time. The spike count functions of neuron 95-98/14 (Fig. 5, 3rd row), the same neuron for which binaural data are illustrated in Fig. 1, were obtained with contralateral tone bursts. When plotted over the instantaneous peak pressure at the end of the integration window, spike count functions are in very close register and reveal common details in their shape, such as a prominent shoulder on the descending arm, which would not have been easily anticipated from inspection of the conventional spike count–level functions. Neuron 95-100/01 (Fig. 5, bottom row) could discharge bursts of four to five spikes to optimal stimuli. Spike count functions of the instantaneous peak pressure at the end of the integration time have very similar peak positions and have very steep ascending and descending slopes. Note that the maximum number of spikes decreases, if anything, with prolongation of the rise time.

Figure 6 illustrates analogous findings for two neurons with exclusively monotonic spike count–level functions. For neuron 95-98/01 (Fig. 6, top 3 rows), data were obtained with stimulation of the contralateral ear alone, the ipsilateral ear alone, and with diotic stimulation (identical signals to each ear). For each stimulus condition the conventional spike count–level functions (left) show increases in threshold SPL and in dynamic range with prolongation of the rise time. As a consequence, the maximum number of discharges decreases with increasing rise time. At right, the same data are plotted against the instantaneous peak pressure at the end of the integration window. The considerably closer match of these functions compared with the conventional spike count–level functions can be readily appreciated. Comparison of the functions obtained with the three different stimulation conditions reveals similar thresholds for contralateral, ipsilateral, and diotic stimulation, but the steepest increase of
FIG. 5. Onset responses of primary auditory cortex (AI) neurons are functions of the instantaneous peak pressure at the end of a proposed integration window. Data for 4 neurons with largely nonmonotonic spike count functions are shown. Neurons were stimulated with contralateral CF tone bursts, all shaped with cosine-squared rise functions of rise times as identified in the keys, and frequencies of 19.3 kHz (neuron 95-92/03), 19.5 kHz (neuron 95-92/04), 5.5 kHz (neuron 95-98/14), and 8.7 kHz (neuron 95-100/01). Left: conventional spike count–level functions. For ease of comparison with the plots at right, SPL or plateau peak pressure is expressed in Pa, rather than in dB SPL. Note the rise time effects on threshold SPL, dynamic range, best SPL, and aspects of nonmonotonicity described in detail in the text. Right: spike counts plotted against the instantaneous peak pressure at the end of the proposed integration window \( \tau \). Note the close alignment of the spike count functions obtained with different rise times and the absence of systematic effects of rise time.

Spike counts with increasing instantaneous peak pressure with diotic stimulation and the shallowest increase with ipsilateral stimulation. Thus nonlinear facilitatory binaural interactions are most pronounced at low levels of the instantaneous peak pressure.

Neuron 95-98/11 (Fig. 6, bottom row) represents an ex-
FIG. 6. Onset responses of AI neurons are functions of the instantaneous peak pressure at the end of an integration window. Data for 2 neurons with monotonic spike count functions are shown. Neuron 95-98/01 was tested with monaural contralateral (top row), monaural ipsilateral (2nd row), and diotic (3rd row) CF tones of 20.3 kHz. Other conventions as in Fig. 5. Note the close match of the spike counts obtained with different rise times when spike counts are plotted as a function of the instantaneous peak pressure at the end of the integration window. Only in neuron 95-98/11 (bottom right), stimulated with diotic CF tone bursts of 10.3 kHz, seemingly systematic effects of rise time on spike counts remain. Note lateral displacement of intermediate sections of spike count functions with increasing rise time.
traordinary case in that apparently systematic effects of rise time remain after spike counts are related to the instantaneous peak pressure at the end of the integration time. Over some intermediate range along the abscissa, spike counts decrease systematically with prolongation of the rise time (bottom right). This phenomenon persisted when the integration window was calculated by subtracting the estimate of $L_{\text{min}}$ (8.77 ms) from each measured mean first-spike latency rather than by applying Eq. 3. No such effects of rise time were seen in this neuron’s spike count functions obtained with monaural contralateral or ipsilateral stimulation (not shown).

Equivalent results were obtained with linear rise function tones. The integration window was calculated in an analogous fashion as described above for cosine-squared rise function tones, viz., by subtracting $L_{\text{min}}$ from the best average fit of the shape of latency versus rate of change of peak pressure functions measured in AI neurons (see companion paper)

$$\tau = L_{\text{REF}} - L_{\text{min}} = \hat{A}_{\text{REF}} \times (\log \text{RCP} + S)^{-4}$$  (4)

where $\hat{A}_{\text{REF}} = 1.28$ s. RCPP is the rate of change of peak pressure and is constant during the rise time of linear rise function tones (see Fig. 4). As with cosine-squared rise function tones, the neurons did not integrate the quasi-instantaneous acceleration of peak pressure. If this had been the case, the spike count functions obtained with different rise times should have been closely aligned throughout when plotted as a function of the rate of change of peak pressure. Clearly, this was not the case (Fig. 3, bottom row). Spike count functions were in register, however, when plotted against the instantaneous peak pressure at the end of the integration window. Two examples are illustrated in Fig. 7.

These findings also provide an explanation of the close alignment of spike count versus acceleration or rate of change of peak pressure functions seen for high magnitudes of these parameters (see, e.g., Figs. 1 f and 3, bottom right). For such stimuli, integration windows of a given neuron are very short so that the peak pressures obtained with tones of different rise times are still in very close register or, with linear rise functions, even identical (see Fig. 4).

Considerations of the time course of peak pressure during the integration window

It is noteworthy that across the range of rise times and SPLs used here a given instantaneous peak pressure can be reached at different times after a tone’s onset. Consequently, a particular instantaneous peak pressure can occur at the end of a range of proposed integration times of a neuron. This is illustrated for neuron 95-87/03 in Fig. 8. In Fig. 8, bottom, the integration times to all the cosine-squared rise function tones tested are plotted against the instantaneous peak pressures reached at the ends of these integration times. The range of integration times at the end of which a particular instantaneous peak pressure is reached is wider for lower values of peak pressure. It can cover $>1$ order of magnitude. However, these differences in the effective stimuli do not seem to be reflected in the number of spikes discharged by the neuron. This is evident from inspection of Fig. 8, top, which shows the spike counts of this neuron plotted against the instantaneous peak pressure at the end of the integration time. Note that, as for the neurons illustrated in Figs. 5–7, spike count functions obtained with different rise times are in close register. There is no indication that the duration of the integration window preceding the response systematically affects the number of spikes discharged.

Because of the wide range of integration windows that can precede a particular instantaneous peak pressure, and thus a particular response from a given neuron, it necessarily follows that the time courses taken by the peak pressure during such integration windows are also very different. Again, such differences are not reflected in the number of spikes discharged by the neuron. This notion is further substantiated by a comparison of the functions relating spike counts to the instantaneous peak pressure at the end of the integration window that were obtained with different rise functions. Data from four neurons are illustrated in Figs. 9–12. In each figure, the conventional spike count-level functions are shown at left, and spike count versus instantaneous peak pressure functions are shown at right. The functions at top and bottom of each figure were obtained with cosine-squared and linear rise functions, respectively. For each neuron, the spike count versus instantaneous peak pressure functions obtained with either rise function were strikingly similar. Note that for each neuron the ordinates have the same scaling. The similarity is particularly evident in neurons with predominantly nonmonotonic spike count functions. Neurons 95-95/03 and 95-92/02 (Figs. 9 and 12) are two examples. The similarity is also evident in largely monotonic functions with conspicuous features, like the hump in those of neuron 95-95/04 (Fig. 10). In neuron 95-92/01 (Fig. 11), the functions obtained with cosine-squared rise function tones (top right) seem to be somewhat shallower than those obtained with linear rise function tones (bottom right). This difference, however, may be due to some drop in excitability during recording rather than reflecting a specific effect of rise function. Changes in excitability might also account for some mismatches in spike count versus instantaneous peak pressure functions such as those shown by neuron 95-92/02 (Fig. 12). The spike counts to tones of 1.7-ms cosine-squared rise times are generally lower than those obtained with longer rise times (Fig. 12, top right). But this is unlikely to be a specific rise time effect because it is not seen with linear rise function tones (Fig. 12, bottom right).

The general validity of the correspondence of spike count versus instantaneous peak pressure functions for the two rise functions is emphasized by Fig. 13. The figure shows, for three selected instantaneous peak pressures, a scatterplot of the spike counts to 20 repetitions of tones shaped with cosine-squared and with linear rise functions. Each data point represents the mean across all rise times tested. Note that the data points scatter around the line of unity slope.

Taken together, the data suggest that the number of spikes discharged by a given neuron in response to a tone of a given frequency is a function of the instantaneous peak pressure at the end of the integration window, and is independent of the duration of the integration window and thus independent of the time course with which the particular peak pressure is reached. This in turn suggests that adaptation during the integration window is not a major factor influencing the response.
FIG. 7. Onset responses of AI neurons are functions of the instantaneous peak pressure at the end of an integration window. Data for 2 neurons tested with linear rise function tones presented to the contralateral ear are shown. The (monotonic) spike count functions of neuron 95-95/21 were obtained with CF tones of 34 kHz, and the (nonmonotonic) functions of neuron 95-95/05 were obtained with CF tones of 24 kHz. Other conventions as in Figs. 5 and 6.

DISCUSSION

In the present study the responses of neurons in AI to various onsets of CF tones are analyzed. The data suggest that onset responses are a function neither of SPL, a parameter characterizing the steady-state portion of such stimuli, nor of the rate of change of peak pressure or acceleration of peak pressure, because spike counts to tones that share the same value of one of these parameters are systematically affected by rise time. Instead, the number of spikes discharged by a neuron is better described as a function of the instantaneous peak pressure at the time the response is initiated. As described in the companion paper (Heil 1997), this time is a function of the maximum acceleration (for cosine-squared rise functions) or of the rate of change of peak pressure (for linear rise functions) and the neuron’s transient sensitivity. It is suggested here that this time may be viewed as an integration window, so that the finding that a neuron’s onset response is a function of the instantaneous peak pressure at the time of response initiation is equivalent to stating that the response is a function of the integral of rate of change of peak pressure within that integration window. This suggests that, with respect to underlying mechanism, onset responses are distinct from sustained discharges, which are, almost by definition, evoked and sensitive to static aspects of sounds, such as the plateau peak pressure of a tone burst. In this respect it is interesting that neurons whose responses to prolonged tone bursts are either dominated by phasic onset responses or by sustained discharges, suitable for representation of steady-state stimuli, are spatially separated in different cortical fields. This was found in the Mongolian gerbil (Thomas et al. 1993), but may also hold for other species. For central neurons, the distinction between onset and sustained discharges has been recognized early (e.g., Geisler et al. 1969). If that distinction applies also to auditory nerve fibers, it would suggest that the rapid drop in a fiber’s firing rate from an initial peak to a more steady-state level may not be adaptation due to fatigue of the synapse between the inner hair cell and the auditory nerve fiber (e.g., Russell and Sellick 1978), but instead would reflect mechanistically different response components.

Comparison with previous studies

The rise time of cosine-squared and of linear rise function tones has systematic and profound effects on features of conventional spike count–level functions. Threshold SPL, dynamic range, the lowest SPL at which responses saturated (for monotonic functions), and the best SPL (for nonmonotonic functions) all increased with prolongation of the rise time. The degree of nonmonotonicity, the sharpness of tuning to SPL, inhibitory thresholds, and upper thresholds were also affected in neurons showing predominantly nonmonotonic spike count–level functions. These results corroborate and extend previous observations on AI neurons studied with linear rise function tones by Phillips (1988). Strikingly similar results were obtained from onset neurons in the posterior field of the cat’s auditory cortex (Phillips et al. 1995). Com-
would result if the tone’s SPL was of a level corresponding increasingly invaded by the broadening of the short-term effect was found. However, note that such a decrease in the possess sensitive inhibitory frequency domains, flanking neurons. In a few neurons in which Suga kept the SPL the sensitivity of these neurons to the short-term spectrum in the threshold SPL with prolongation of the rise time, and 1995) have argued that the effects of rise time on spike and Feng 1992), as well as from onset neurons in bat inferior prolongation of the rise time (e.g., see Figs. 1, 3, 5, and 9).

from the VIIIth nerve, the superior olivary complex (Condon how this model could work for the many neurons that show anuran auditory system, namely the dorsal medullary nucleus opposite constellation of thresholds and latencies for excitatory response can differ in duration by different rise times, as in the cases illustrated in Figs. 5-7.

Suga (1971) has suggested that the effects of rise time of the signal. Those researchers suggested that the neurons constant and altered the rise time, the allegedly opposite input. The neuron would fail to respond because the excitatory input would exert its effect those obtained with the same rise time are connected. Note that for a given low instantaneous peak pressure the integration windows preceding the response can differ in duration by >1 order of magnitude.

patible results were obtained from phasic neurons in the anuran auditory system, namely the dorsal medullary nucleus (Hall and Feng 1988, 1991), which receives direct input from the VIIIth nerve, the superior olivary complex (Condon et al. 1991), and the midbrain torus semicircularis (Gooler and Feng 1992), as well as from onset neurons in bat inferior colliculus (Suga 1971). Suga generally observed increases in the threshold SPL with prolongation of the rise time, and noted that the amount of increase could differ greatly among neurons. In a few neurons in which Suga kept the SPL constant and altered the rise time, the allegedly opposite effect was found. However, note that such a decrease in the apparent threshold SPL with prolongation of the rise time would result if the tone’s SPL was of a level corresponding to the descending slope of most spike count–level functions of that neuron (e.g., neuron 95-98/14, spike count–rise time functions for SPLs above ~50 dB; Fig. 1f).

These data, from nuclei as peripheral as the dorsal medullary nucleus to structures as high up the auditory pathway as the posterior auditory field, and from taxa as diverse as amphibians and mammals, suggest that onset responses in general might be governed by very similar mechanisms.

I propose that with respect to onset responses these findings, even when taken on their own, seriously question the usefulness of the conventional spike count–level function and measures derived from it, such as a threshold SPL, a dynamic range, a best SPL, a degree of nonmonotonicity, etc. Even the classification of monotonic versus nonmonotonic neurons on the basis of spike count–level functions may be effected, particularly when long rise times are used, or when data obtained with different rise times or rise functions are compared, because this could influence the proportions of functions assigned to either category, independent of the categorizing criteria. The data constitute a similar challenge to the classical tuning curves and response areas in frequency and SPL coordinates, as well as measures derived from it (e.g., various Q values), because the effects of rise time described above are not restricted to tones of CF. Consequently, tuning curves and response areas must also vary with changes in rise time, which indeed they do (Phillips et al. 1995; Suga 1971) (Fig. 6 in the companion paper). Because the onset response of a neuron to a tone with a particular SPL and the shapes of its spike count–level functions or tuning curves or response areas depend so critically on the tone’s rise time, it is incomplete, if not inappropriate, to characterize the neuron’s properties by SPL-associated measures.

Some mechanistical considerations

Suga (1971) has suggested that the effects of rise time on threshold SPL and on the upper threshold could be explained by the interaction of excitatory and inhibitory inputs with different thresholds and different latencies on a postsynaptic target neuron. For example, if the inhibitory input has a lower threshold but a longer latency than the excitatory one, the target neuron may respond to a tone with a short rise time, because the excitatory input would exert its effect before the inhibitory input. The neuron would fail to respond if the rise time was long, because the inhibitory input would be effective before the excitatory one. To explain the shift of the upper threshold with rise time, Suga suggested the opposite constellation of thresholds and latencies for excitatory and inhibitory inputs. However, it is difficult to see how this model could work for the many neurons that show increases in both threshold SPL and upper threshold with prolongation of the rise time (e.g., see Figs. 1, 3, 5, and 9).

Phillips and colleagues (Phillips 1988; Phillips et al. 1995) have argued that the effects of rise time on spike count–level functions of auditory cortical neurons reflect the sensitivity of these neurons to the short-term spectrum of the signal. Those researchers suggested that the neurons possess sensitive inhibitory frequency domains, flanking the excitatory ones (lateral inhibitory domains), which are increasingly invaded by the broadening of the short-term
spectrum with shortening of the rise time. This invasion of the presumed inhibitory domains was thought to underlie the transition of monotonic to nonmonotonic spike count–level functions, and the increasing degree of nonmonotonicity of these functions with shortening of the rise time. Although this idea is intuitively plausible (but see Hall and Feng 1988), lateral inhibitory domains were not demonstrated in these studies, but were rather inferred from the responses of neurons to noise bursts, and these responses in turn were largely recorded in different neurons and even in different animals. Furthermore, it is not clear how the spectral splatter could account for the shifts in threshold SPL and the widening of the dynamic range.

The present study suggests that neither excitatory and inhibitory inputs with different threshold SPLs and latencies, nor accommodation of the neural membrane (Suga 1971), nor spectral splatter into inhibitory frequency domains is necessary to explain the alleged rise time effects. Nevertheless, the generation of nonmonotonic spike count functions likely requires the combination of excitatory and inhibitory inputs. In fact, a variety of function shapes, including nonmonotonic ones, could easily be generated by excitatory or excitatory and inhibitory convergence of neurons with spike count versus instantaneous peak pressure functions of similar shapes, although somewhat different thresholds. The integration times of the input neurons could be identical, i.e., they could have the same sensitivity, and no delay of either input to the target neuron is required.

**Some functional implications**

**FEATURE DETECTION.** The present data challenge the notion that onset responses may exhibit selectivities for rise time. The responses of phasic neurons at various levels of the auditory pathway of the leopard frog (*Rana pipiens pipiens*) have been interpreted as disclosing filters selective for short rise times (Condon et al. 1991; Gooler and Feng 1992; Hall and Feng 1988, 1991), a possibly attractive idea, because short rise times are characteristic features of the mating and release calls of leopard frogs, but not of sympatric species (Hall and Feng 1988). However, the short-pass spike count–rise time functions reported in these studies were all obtained at low SPLs (relative to the neurons’ firing thresholds). And indeed, when the responses (of neurons in the dorsal medullary nucleus) were probed at higher SPLs, the rise time functions were essentially flat (Hall and Feng 1991), much like the results obtained from monotonic AI neurons in the present study (Fig. 1b). For nonmonotonic neurons, spike count–rise time functions at moderate and higher SPLs

**FIG. 9.** Comparison of spike count functions to contralateral CF tone bursts shaped with cosine-squared and with linear rise functions. Data are shown for neuron 95-95/03 with a CF of 21.7 kHz. *Left column:* conventional spike count–level functions. Note that, as in Figs. 5–7, SPL was converted into the plateau peak pressure. *Right column:* same data plotted as a function of the instantaneous peak pressure at the end of the proposed integration window. Note the close match of the spike count functions obtained with different rise times. Data in the top and bottom row were recorded with cosine-squared and with linear rise functions, respectively. Note the similarity in the shape of the spike count vs. instantaneous peak pressure functions recorded with the two rise functions.
may be expected to even show long-pass characteristics (Fig. 1f).

**REPRESENTATION OF STEADY-STATE ASPECTS OF SOUNDS.** Onset responses are not an unambiguous function of the steady-state SPL of the tone burst. Rather, as shown here for AI neurons, spike counts are much better described as a function of the instantaneous peak pressure at the end of a proposed integration time, largely irrespective of the steady-state SPL. The SPL of a tone characterizes the instantaneous peak pressure only when the proposed integration window is longer than the rise time, because then the instantaneous peak pressure at the end of the integration time is identical with the plateau peak pressure or SPL. For CF tones, this will be the case only when the SPL is low and the rise time relatively short. Thus the conventional spike count–level functions are distorted versions of the functions relating the response to the instantaneous peak pressure at the end of an integration time. For a given neuron, the degree of distortion increases with rise time but, to complicate matters, the distortion is not uniform across the neuronal pool. In other words, the combinations of SPL and rise time for which the instantaneous peak pressure at the end of the proposed integration time is equivalent to the SPL varies from neuron to neuron, even for CF tones. This is because the duration of the integration window depends on a combination of the maximum acceleration of peak pressure (or, with linear rise functions, its rate of change) of the stimulus and the neuron’s sensitivity to it (Eq. 3 and 4; Fig. 13b). The latter varies across the neuronal population, even for CF tones. Furthermore, for a given neuron the transient sensitivity is a function of frequency (see Fig. 6 in the companion paper), and therefore the sensitivity for a given frequency will vary across the neuronal pool. Consequently, the number of onset spikes discharged by most neurons activated by a particular tone is not indicative of its SPL, and, almost counterintuitively, this holds true particularly for neurons with a CF corresponding to the tone’s frequency. These findings cast doubt as to whether the SPL may be represented by the overall spatial pattern of activity (e.g., Heil et al. 1994; Phillips et al. 1994) resulting from the nonrandom spatial organization of various conventional response measures in auditory cortex (Heil et al. 1992b, 1994; Phillips et al. 1985; Schreiner et al. 1992; Suga 1977; Sutter and Schreiner 1995). However, the steady-state SPL could be indicated in the spike counts of those neurons whose integration windows, because of low transient sensitivities, are longer than most conventional rise times, i.e., of neurons with CFs relatively far away from the tone’s frequency. Information about the steady-state SPL may also be derived from the temporal change in the active neuronal population (see below).

A similar reasoning would apply to the representation of interaural differences in SPL (ILDs). Although those neurons in our sample that were stimulated binaurally were tested only with zero ILDs, the present results suggest that onset responses may not be sensitive to the steady-state ILD or to the combination of SPLs at each ear, as may have been implied in previous studies (e.g., Brugge et al. 1969; Irvine et al. 1995; Orman and Phillips 1984; Phillips and Irvine
1981; Semple and Kitzes 1993a,b). Rather, onset responses may be a function of the instantaneous ILD, which is continuously increasing during the rise time until the steady-state value is reached.

**TEMPORAL TRACKING OF DYNAMIC ASPECTS OF SOUNDS.** However, the onset responses of the activated neurons could temporally track the course of a tone’s onset and represent its instantaneous properties via their discharges and their spatial patterns of activity. This is schematically illustrated in Fig. 14. Neurons with the highest $S$ to a particular onset have the shortest integration windows and will discharge first ($S$ high, Fig. 14c). Neurons with successively lower sensitivities will have longer integration windows and will discharge during successively later portions of the transient ($S$ low). To a first approximation the sensitivities to a tone with a particular frequency will decrease with the difference between the neuron’s CF and the tone’s frequency (Fig. 14a), although the details will depend on the diversity of transient sensitivity versus frequency functions (see Fig. 6 in the companion paper). Importantly, the temporal dispersion of response initiation within a neuronal population with a given range of transient sensitivities depends on the signal’s acceleration of peak pressure: for a low acceleration the dispersion is high and for a high acceleration it is low (Fig. 14, b and c).

The neurons that share the same sensitivity to the transient, and that thus share the same duration of their integration windows and therefore respond at the same instantaneous peak pressure, are likely to differ with respect to their spike count versus instantaneous peak pressure functions. Three such functions, which differ in threshold, dynamic range, optimum, and degree of nonmonotonicity, are schematically illustrated in Fig. 14d. A particular value of the instantaneous peak pressure (e.g., that indicated by the vertical dashed line in Fig. 14d) is thus unambiguously represented by the ratios of the responses of the neurons in the subpopulation with the same integration window. A subsequently achieved instantaneous peak pressure would be represented in an analogous fashion in a subpopulation of neurons with a longer integration window, and so forth. Thus the transient, and initial steady-state portions of the signal, will be fully characterized by the temporospatial pattern of the population’s response.

As mentioned above, the temporal dispersion of response initiation within a neuronal population decreases with the signal’s acceleration of peak pressure (Fig. 14b). In other words, the rate at which a transient is sampled by the different neurons of the responsive population is not fixed, but increases with maximum acceleration of peak pressure (Fig. 14c). Thus transients with rapidly changing properties will be sampled at higher rates than transients with slowly changing properties, a mechanism with indisputable advantages for the analysis of information in time-varying signals.

The brevity of the onset response of individual neurons (most neurons discharge only 1 or 2 spikes with a short interspike interval) and the high temporal precision of the spikes (see companion paper) (Phillips and Sark 1991) are also favorable for such a representation. The minimum
FIG. 12. Comparison of spike count functions to contralateral CF tone bursts shaped with cosine-squared and with linear rise functions. Neuron 95-92/02 with a CF of 18.5 kHz; conventions as in Fig. 9.

equipment required for the determination of a given value of instantaneous peak pressure by such a subpopulation would be three neurons with different yet partly overlapping spike count versus instantaneous peak pressure functions. The observed large diversity of the shapes of the functions (Figs. 5–12), as well as their likely topographic organization, at least within AI, and as suggested by extrapolation from conventional spike count–level functions (e.g., Heil et al. 1992b, 1994; Schreiner et al. 1992; Sutter and Schreiner 1995), might be of further advantage, particularly in noisy conditions (see, e.g., Tanaka and Nakayama 1995).

Conclusions

It is demonstrated here for tone burst stimuli that the phasic onset response of an auditory neuron can be well described as a function of the instantaneous peak pressure at the time of response generation, i.e., as a function of the integral of rate of change of peak pressure within a proposed integration window. The duration of the window is not fixed, but jointly depends on the acceleration (or, for linear rise functions, the rate of change) of peak pressure and the neuron’s sensitivity to that acceleration (or rate of change), so that any stimulus manipulation that affects either component is expected to affect the window’s duration. Such manipulations likely include the signal’s amplitude spectrum, peak amplitude, rise time, or spatial location. Similarly, the effectiveness of a particular integral of rate of change of peak pressure within a fixed integration window with respect to eliciting spikes will depend on stimulus properties, such as the spectral composition. Nevertheless it is conceivable that the onset response might generally be a function of the in-
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FIG. 14. Hypothesis of temporal tracking of the instantaneous properties of a tone’s onset. a: neuron’s sensitivity $S$ to acceleration or rate of change of peak pressure varies with frequency. Different neurons (--- and --- ---) may therefore display different sensitivity to the onset of a tone of a given frequency. b: a neuron’s integration time increases with decreasing transient sensitivity. Across a neuronal population with a range of sensitivities, the range of integration times, and therefore the temporal dispersion of 1st spikes, depends on the acceleration of peak pressure. Dispersion is high when the acceleration is low (---) and vice versa (--- ---). c: integration windows of neurons to a particular transient (2 different envelopes are shown by the curved lines) commence simultaneously, but neurons with successively lower transient sensitivity to that stimulus will have successively longer integration windows, and therefore respond at different instantaneous peak pressures. To another transient (differing for example in rise time, peak amplitude, or rise function) a given neuron will respond at a different instantaneous peak pressure. d: any neuron’s response is a function of the instantaneous peak pressure at the end of its integration window. Neurons that share the same transient sensitivity to a given stimulus, and thus respond at the same instantaneous peak pressure, will differ with respect to their spike count functions (--- --- ---, and --- --- ---) so that that instantaneous peak pressure (for example, that identified by the vertical dashed line) can be encoded in the ratio of the responses of that subpopulation of neurons.

instantaneous property of the stimulus at the end of the integration window. Then, the onset response of an individual neuron could constitute a general-purpose element for real-time coding (disregarding the inevitable minimum delays, summarized by $L_{\text{min}}$ in Eq. 2) of the dynamic changes in a signal’s envelope. Different neurons sample a given transient at different times. The sampling rate will automatically be adjusted to the rapidity of such changes, because the range of the duration of the proposed integration windows depends on the acceleration of peak pressure at onset. And the temporospatial pattern of the onset responses evoked by the stimulus could provide a basis for the discrimination of rapid transients (Cutting and Rosner 1974; Stevens 1980; van Heuven and van den Broecke 1979).

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