Physiologically based predictions of psychophysical threshold, dynamic range and intensity difference limen in mammalian cochlear implant subjects

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ABSTRACT

In [Bruce et al., IEEE Trans. Biomed. Eng. 46, 1393–1404 (1999b)], a composite physiological/psychophysical model of cochlear implant stimulation was developed to investigate whether inclusion of physiologically-observed stochastic activity in the auditory nerve (AN) section of the model improves predictions of psychophysical data from human cochlear implant users. It was shown that the stochastic version of the model better predicts how psychophysical threshold and uncomfortable loudness vary with two stimulus parameters that directly affect single-fiber responses, stimulus phase duration and electrode configuration. In this paper, the hypothesis is investigated that the major properties of such psychophysical data are consistent across mammalian species and can be well understood from the basic physiological properties of the mammalian AN if stochastic activity is considered. In addition to the psychophysical measures of the previous study, the investigation is extended to intensity discrimination as function of stimulus intensity. Also examined are the effects of one feature of the AN population response, the number of surviving AN fibers, and of one feature of the psychophysical section of the model, temporal integration. Predictions of data from humans, monkeys, guinea pigs and cats provide substantial evidence for the perceptual importance of neural stochastic activity.

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INTRODUCTION

The ability to accurately relate perception of electrical stimuli in cochlear implant users to the auditory nerve (AN) fiber activity underlying that perception should help significantly in designing better speech processors for implants. However, neither theoretical analyses nor computational models of AN physiological data have historically been able to explain or predict a number of basic perceptual phenomena found in cochlear implant users. For example, studies of single AN fiber responses from a range of species where only an arbitrary, deterministic measure of threshold is recorded do not accurately predict behavioral threshold versus phase duration (strength-duration) curves for sinusoidal stimulation (Pfingst, 1988) or for pulsatile stimulation (Pfingst, 1990; Pfingst et al., 1991) in monkeys. Furthermore, strength-duration curves of cochlear implant users are not well predicted by deterministic models of AN response to electrical stimulation (Shannon, 1989; Pfingst, 1990).

One proposed explanation of such discrepancies in indirect comparisons is that they are due to physiological differences between the species in which the physiological and psychophysical data were collected. However, recent across-species comparisons of psychophysical strength-duration data (Miller et al., 1999a,b) suggest that the predominant effects observed are consistent across mammals. Therefore, for species differences to account for the apparent disparity between physiological and psychophysical data, different auditory nerve responses in the various species would have to give rise to the same perceptual characteristics. However, the physiological data used for the comparison in Pfingst (1988, 1990) and Pfingst et al. (1991) were from some of the same species as the psychophysical data of Miller et al. (1999a,b). Species differences appear unable then to explain the conundrum.

An alternative hypothesis is that the physiological estimates of threshold used in such comparisons were inaccurate, making the indirect comparisons invalid. In Bruce et al. (1999b) we showed that improved predictions of behavioral threshold and dynamic range in human cochlear implant users could be obtained by taking into consideration the stochastic component of the AN’s response to electrical stimulation (Bruce et al., 1999a,c, 2000). Importantly, the AN section of the model used to predict human psychophysics was derived solely from cat physiological data (Bruce
et al., 1999c).

In this paper we present further model predictions of psychophysical data from humans, monkeys, guinea pigs and cats. These comparisons provide further understanding of the model behavior, giving insight into the possible physiological basis of intensity perception in mammalian cochlear implant subjects. In addition to the psychophysical measures of the previous study, we extend our investigation to intensity discrimination as function of stimulus intensity. In the previous study we examined two stimulus parameters (pulse width and electrode configuration) that directly affect single AN fiber responses. In this paper we explore the effects of one model parameter that affects the population response of the auditory nerve, the number of surviving auditory nerve fibers, and of one feature of the psychophysical section of the model, temporal integration. The results support the hypothesis that the major properties of cochlear implant psychophysical data are consistent across mammalian species and can be well understood from the basic physiological properties of the mammalian auditory nerve if stochastic activity is considered (Bruce et al., 1999b). Given the predominant similarities, we then discuss how minor differences in auditory nerve physiology between species may result in secondary psychophysical effects. We also examine recently published physiological and psychophysical thresholds from the same experimental subjects, consider how neural field potentials may relate to single-unit and behavioral thresholds, describe physiological experiments that could be useful in improving the model, and discuss how neural survival may affect psychophysical measures and perception of speech.

I. METHODS

A. Psychophysical model

The purpose of our psychophysical model (Bruce et al., 1999b) is to take a given stimulus produced by a cochlear implant and from that predict whether that stimulus is audible (i.e., above the psychophysical threshold), whether its intensity is below the minimum level that produces an uncomfortably loud percept (i.e., is within the dynamic range), and how discriminable its intensity is.
from the intensity of a different stimulus. The model, illustrated in Fig. 1, has two major divisions, an auditory nerve section and a psychophysical section.

[Figure 1 about here.]

For the AN section of our model we utilize a description of AN response to electrical stimulation based on the model of White (1978, 1984a, 1987) and White et al. (1987) that we developed further in Bruce et al. (1999c). In summary, this model consists of (i) an input/output (I/O) function for each of an arbitrary number of AN fibers and (ii) a function approximating the attenuation of the excitatory current as it spreads from the active electrode to the site of action potential generation in each fiber. Unless otherwise stated, simulations were conducted with 10 000 model fibers.

The fiber I/O function describes the probability of discharge in response to a single pulse as a function of the stimulus intensity (expressed in absolute units) delivered to the fiber. For the deterministic model, this is a step function (Eq. 2 of Bruce et al., 1999c), where the intensity at which the discharge probability changes from zero to one is referred to as the fiber’s threshold. For the stochastic model, the I/O function is an integrated-Gaussian (Eq. 3 of Bruce et al., 1999c):

\[
p = \frac{1}{2} \left( 1 + \text{erf} \left( \frac{I_{\text{stim}} - I_{\text{thr}}}{\sqrt{2}\sigma} \right) \right),
\]

where the stimulus intensity \(I_{\text{stim}}\) corresponding to a discharge probability \(p\) of 0.5 is referred to as the fiber’s threshold \(I_{\text{thr}}\). The slope of the integrated-Gaussian is determined by the Relative Spread (RS), which is the standard deviation of the Gaussian noise \(\sigma\) divided by the threshold (Eq. 4 of Bruce et al., 1999c). The Gaussian noise is the underlying mechanism that generates the integrated-Gaussian I/O function.

Although only minimal physiological data have been collected on the distributions of thresholds and RSs in a population of AN fibers at any cochlear location using biphasic current pulses, there exist just enough to enable us to set distributions of model parameters to approximate those seen in the physiological data from cat (Bruce et al., 1999c). We model the threshold and RS of each fiber as two independent random variables. Pseudo-random numbers are generated using the estimated distributions to obtain different thresholds and different RSs for each fiber. In order to
maintain the same set of I/O functions across simulations with the same number of fibers, the seeds of the pseudo-random number generators are two different fixed values, one for the generation of the thresholds and one for the RSs.

For both the deterministic and the stochastic model, fiber thresholds are uniformly distributed from $-5 \text{ dB}$ to $+5 \text{ dB}$ with respect to the mean threshold, which is determined by:

$$E[\text{Threshold}] = \text{Mean Threshold}$$

$$= 121.04 \times \text{PW}^{-0.18} \text{ (dB re. 1 } \mu\text{A)} \quad (2)$$

where PW is the pulse width (phase duration) in $\mu\text{s}$/phase. Fiber thresholds are uniformly distributed in units of dB re. 1 $\mu\text{A}$.

For the stochastic model, the RSs are normally distributed, with the mean determined by:

$$E[\text{RS}] = 0.12 + 9.51 \times 10^{-5} \times \text{PW} - 7.90 \times 10^{-9} \times \text{PW}^2 \quad (3)$$

and a standard deviation of 0.06. The effect of PW on RS is based on a quite small but consistent set of physiological data collected using biphasic pulses (Bruce et al., 1999c). However, this effect is known to be absent in the case of monophasic pulses (Verveen, 1960), and it is conceivable that under some physiological conditions it may not be present for biphasic pulses. The stochastic model results presented in Sections II.A. and II.B. are highly dependent on this behavior, and consequently we will discuss in those sections how these predictions would change if RS were independent of PW. In the Discussion we will consider a possible biophysical cause of PW effects on RS and why there is a difference for monophasic and biphasic current pulses.

To model the current spread, we assume that the turns of the cochlea can be unrolled and can be considered electrically uncoupled, such that the scala tympani acts more or less as a leaky transmission line through the cochlea and that the potential and current decay along the scala tympani as an exponential function (for a recent review see Briaire and Frijns, 2000). Fibers are assumed to be uniformly distributed along the length of the cochlea; the actual site of excitation (i.e., node of Ranvier) on a fiber is ignored. We assume that the stimulating electrode is placed 15 mm in-
side a 30 mm cochlea and that the stimulus is attenuated at the rate of 0.5 dB/mm for monopolar (MP) stimulation (Merzenich and White, 1977) and 4 dB/mm for bipolar (BP) stimulation—the latter value is appropriate for both radial-BP pairs (Merzenich and White, 1977) and closely-spaced longitudinal-BP pairs (O’Leary et al., 1985). Less closely-spaced BP configurations should produce a lower rate of attenuation, such that model responses and the resulting predictions will tend towards those given for MP configurations.

For the psychophysical section, following White (1984a) and O’Leary et al. (1995), intensity discrimination is based on the summed discharge rate of all AN fibers activated by the electrical stimulation. Following White (1984a), from psychophysical measurement of threshold versus stimulus duration (Shannon, 1993; White, 1984b), we use a 100 ms rectangular integration window. These two aspects, the summing of responses across all fibers and the long-term temporal integration, can be modeled by spatial summation and temporal integration of the output of the neural section.

The neural/psychophysical model can be used to predict psychophysical results using signal detection theory. A range of psychophysical paradigms were used in the collection of the behavioral thresholds and intensity difference limen (IDL) presented in Section II. For consistency and simplicity, we use a standard two-interval forced-choice paradigm (Levitt, 1971) to determine the model’s prediction of the psychophysical data. Two stimuli are perceived to be equal in intensity when the spike counts at the output of the temporal integration section of the model are identical for the two stimuli. If we assume an ideal observer, then the presentation interval eliciting the greater number of discharges is chosen as the one containing the stimulus, in the case of a threshold measurement. Similarly, in the case of an IDL measurement, the interval with the larger spike-count is chosen as the interval containing the higher intensity stimulus. In the rare case when the number of discharges is equal for the two intervals an “unbiased coin is flipped” to generate the decision. The equations used to perform these calculations are given in Bruce et al. (1999b).

Not all of the experimental methods used to collect the data presented in the Results were criterion-specific. In such cases we used 70.7% as the criterion. For a 70.7% criterion, a discriminable difference for the stochastic model corresponds to the intensity difference at which more
spikes are produced by the comparison interval than by the reference interval approximately 70% of the time.

We define dynamic range as the difference in dB between behavioral threshold and the lowest stimulus level to elicit an uncomfortably loud percept. One long standing and intensively studied hypothesis is that loudness is simply proportional to the summed AN response (e.g., Fletcher and Munson, 1933). While there is now direct evidence to suggest that loudness is not directly proportional to the summed AN response, loudness does appear to be monotonically related to the neural response (Relkin and Doucet, 1997). Therefore we assume here that uncomfortable loudness (UCL) corresponds to a fixed number of neural discharges for the whole AN within the period of temporal integration, i.e., the output of the model’s temporal integrator section. We will call the number of discharges required to reach uncomfortable loudness \( N_{ucl} \). We have no way of determining how many responses correspond to UCL in the model, so we therefore present predictions for three different values of \( N_{ucl} \), which appear to best account for the psychophysical data.

B. Psychophysical data

To test the predictive power of our model for mammalian cochlear implant subjects, we have selected published data from humans, monkeys, guinea pigs and cats, and further published data are cited in the text. Some previously unpublished data (collected by White) from a human subject are also included. The implant type, experimental setup and methods used were identical to those used in White (1984b). In summary, the subject was implanted with a scala tympani intracochlear electrode array of sixteen wires that were connected to a stimulus generator via a percutaneous link. BP electrode pairs were oriented approximately radially relative to the axis of the cochlea. Thresholds were measured with a modified Békésy tracking procedure using a minimum of six threshold crossings for each threshold estimate. The average of the stimulus minima and maxima was computed to determine the estimated threshold stimulus current.
II. RESULTS

A. Threshold versus phase duration (pulse width): Strength-duration

In Bruce et al. (1999b) we investigated predictions of threshold versus phase duration curves from two human subjects. Here we extend this study to data from monkeys, cats and guinea pigs, and examine how electrode configuration and neural survival may explain some of the inter-subject variability. Pfingst et al. (1991) have investigated how behavioral thresholds in adolescent or adult male macaques (M. mulatta and M. radiata) vary as a function of pulse duration for single biphasic pulses. Their data are plotted in Fig. 2A (solid lines), along with a line indicating a 6 dB reduction in threshold per doubling of the phase duration (dotted line), which corresponds to equal charge per pulse. Pfingst et al. noted that the slopes begin to steepen with phase durations greater than 500 µs/phase and that in some subjects they are steeper than $-6 \text{ dB/doubling}$ in the region from 1 000 to 2 000 µs/phase. This is more than would be expected if it were assumed that threshold corresponds to a certain level of charge delivered by an implant. Such behavior is also observed in some human subjects (e.g., see Fig. 6 of Bruce et al., 1999b, and Fig. 2 of Moon et al., 1993.).

[Figure 2 about here.]

Model predictions of the Pfingst et al. (1991) data are plotted in Fig. 2B. The deterministic model predicts (i) relatively high absolute values for threshold, (ii) a monotonic reduction in slope with increasing phase duration such that slopes are always less than $-6 \text{ dB/doubling}$ in the region from 1 000 to 2 000 µs/phase, and (iii) no effect of electrode configuration or neural survival. In contrast, the stochastic model predicts (i) absolute values of threshold that are significantly lower than those predicted by the deterministic model, (ii) slopes that begin to steepen with phase durations greater than 500 µs/phase and slopes that are steeper than $-6 \text{ dB/doubling}$ in the region from 1 000 to 2 000 µs/phase, and (iii) lower thresholds and steeper slopes for MP stimulation than BP and for greater neural survival.
Over the range of electrode configurations and numbers of fibers shown in Fig. 2B, the stochastic model always predicts slopes steeper than $-6\,\text{dB/doubling}$ in the region from 1 000 to 2 000 $\mu\text{s/phase}$ and the deterministic model always predicts slopes shallower than $-6\,\text{dB/doubling}$ in this region. This can be understood by studying how thresholds for the two models arise. Behavioral threshold for the deterministic model is at the threshold of the most sensitive model fiber. From Eq. (2), single-fiber thresholds decrease with increasing phase duration, but the rate of decrease diminishes, producing the deterministic model threshold curves seen in Fig. 2B. In contrast, for the stochastic model small discharge probabilities from individual fibers may sum up across many fibers to a sizeable mean spike count. This explains why the stochastic model’s behavioral threshold is well below that of the most sensitive fiber in the deterministic model. Furthermore, behavioral threshold for the stochastic model is very sensitive to the slope of the low-probability “tails” of the integrated-Gaussian I/O function. From Eq. (3), the slopes of the stochastic single-fiber I/O functions decrease with increasing phase duration. This causes steeper predicted behavioral threshold curves in Fig. 2B for phase durations above 500 $\mu\text{s/phase}$.

In contrast to the stochastic model predictions and some subjects’ psychophysical data, strength-duration curves for a number of subjects in Fig. 2 have slopes shallower than $-6\,\text{dB/doubling}$ in this region, consistent with results in some human subjects (e.g., Moon et al., 1993). The deterministic model appears to be more accurate in these cases but is unable to explain subjects with the steeper slopes. In contrast, it can be demonstrated that the behavior of the stochastic model could approach that of the deterministic model in some situations, such that it can explain all the data. Factors such as residual synapse-driven spontaneous activity and noisy central processing (e.g., see Durlach et al., 1986) may mean that a greater number of spikes than indicated by the model predictions may be required to reach behavioral threshold. For the deterministic model (see Fig. 3A), spike counts one or two orders of magnitude greater are reached with only a very small increase in intensity above threshold, and therefore the deterministic model predictions of strength-duration curves would not be greatly changed in such “non-ideal” cases. In contrast, the mean number of discharges changes only slowly with intensity for the stochastic model (see Fig. 3B), such that a larger increase in intensity would be required to reach threshold in non-ideal
situations. For the stochastic model, the standard deviation of the number of discharges grows less with an increase in intensity than does the mean (see Fig. 3B), such that the relative noise in the neural response decreases with intensity (see Fig. 3C). Consequently, the stochastic model would predict shallower strength-duration curves in the non-ideal cases described above. The higher the intensity required to reach threshold, the shallower the curve; in the extreme case, the stochastic model and deterministic model predictions would be identical. We would argue thus for the superiority of the stochastic model because of its ability to predict a range of slopes both steeper and shallower than \(-6 \text{ dB/doubling}\), while the deterministic model can only explain those curves shallower than \(-6 \text{ dB/doubling}\).

Another possible cause of slopes shallower than \(-6 \text{ dB/doubling}\) is that the effect of PW on RS described by Eq. (3) is not present in these subjects. The exact biophysical cause of this effect is not known (one likely candidate is described in the Discussion section), and therefore it is difficult to postulated here why the effect might be absent in some subjects.

Some of the variability in slopes in the psychophysical data may also be predicted by considering the various electrode configurations (narrow BP through to MP) used in the different subjects and the possible range of neural survival. The effects of electrode configuration on threshold strength-duration curves have been investigated directly in a number of species (Miller et al., 1995; Pfingst et al., 1995a,b; Smith and Finley, 1997; Miller et al., 1999b). In Fig. 4, psychophysical strength-duration curves from guinea pigs (Miller et al., 1995) and from cats (Smith and Finley, 1997) in response to single biphasic pulses are plotted for different electrode configurations. In both data sets, consistent with the stochastic model predictions, the mean thresholds are similar for BP and MP stimulation at short phase durations, but diverge as phase duration increases because of the MP curve’s steeper slope. This effect has also been observed in nonhuman primates (Pfingst et al., 1995a,b) and neomycin-deafened guinea pigs (Miller et al., 1999b).

[Figure 3 about here.]

[Figure 4 about here.]
Regarding the effects of the number of fibers, the stochastic model predicts that for short durations (100 to 1,000 µs/phase), increased neural survival leads to steeper strength-duration curves for both MP stimulation and BP, with a slightly bigger effect for MP than BP. For long durations (1,000 to 5,000 µs/phase), increased neural survival has little effect for MP but produces a steeper strength-duration curve for BP. The model predictions for MP stimulation are consistent with the data recently reported by (Miller, 2000). Our model predicts the trend observed in a linear regression of the data. The data show a mean increase of ~5% in slopes for phase durations between 25 and 400 µs/phase when the number of fibers is increased from 1,000 to 30,000 and ~18% for phase durations between 400 and 1,600 µs/phase, compared to the model predictions of ~10% and ~20% respectively. These are relatively good predictions given the weakness of the correlation in the data as noted by Miller (2000). Such a weak correlation suggests that factors other than the number of fibers are major contributors to inter-subject variability in psychophysical strength-duration slopes. For example, deafness is known to alter the I/O functions of individual AN fibers (Shepherd and Javel, 1997), which would have a strong affect on strength-duration slopes predicted by our model. The effects of neural survival on threshold, dynamic range and intensity discrimination are investigated further in Section II.D.

B. Threshold versus number of pulses: Temporal integration

While our psychophysical model includes a temporal integration section, the effects of this temporal integration were not examined in Bruce et al. (1999b). Temporal integration is important because most cochlear implant stimulation strategies produce trains of pulses, such that a number of pulses will occur within the temporal integration window, depending on the stimulation rate. Here we investigate the effects of the number of pulses in a pulse train on behavioral threshold. Plotted in Fig. 5 are data from human subjects and model predictions of thresholds versus number of pulses. Figure 5A shows psychophysical data (replotted from Fig. 5 of Moon et al., 1993) from five subjects for moderately wide (one separating electrode) longitudinal-BP stimulation, 100 pulses-per-second (pps) pulse trains with phase durations of 96 and 1,536 µs/phase. Moon
et al. (1993) found that the slopes of the curves are significantly different ($p = 0.017$) for the two phase durations. The previously unpublished data in Fig. 5B are from a human subject for radial BP, 100 pps pulse trains at four different phase durations. Figures 5C and D respectively show deterministic and stochastic model predictions of the data in Fig. 5B. Unlike the psychophysical data, the slopes of the temporal integration curves predicted by the deterministic model (Fig. 5C) do not increase with pulse duration, but rather remain at approximately zero. In contrast, the stochastic model (Fig. 5D) predicts temporal integration curves with non-zero slopes that increase with phase duration. These results are also consistent with data from monkeys (see Fig. 7 and Table V of Pfingst et al., 1991). Note that the increase in temporal-integration slope with phase duration is a consequence of the stochastic AN model’s behavior, i.e., RS increases with phase duration as described by Eq. (3); the increase in the temporal-integration slope would not occur if RS and PW were independent.

Again, these results can be best understood by comparison of the neural response curves plotted in Fig. 3. As the number of pulses in the stimulus train is increased, fewer discharges (i.e., lower discharge probabilities) are required per pulse to reach threshold, because discharges occurring with the 100 ms integration window will add to reach a threshold response. For the deterministic model, it is likely that threshold occurs on the steep section of the curve. As a consequence, only a very small decrease in stimulus current will be required to cause the requisite decrease in neural response per pulse. In contrast, the stochastic model has a shallow slope at low intensities, so a relatively large decrease in current will be required.

Preliminary simulations, where we include additional noise to model (i) residual synapse-driven spontaneous activity and/or (ii) central noise, indicate that the stochastic model predictions may be improved slightly in such cases, i.e., the curves for short pulse durations become flatter while those for the longer pulse durations remain steep. The additional noise also gives rise to a slight slope in the deterministic model curves, better matching the psychophysical data for the short pulse durations, but the steep slopes for the longer pulse durations still cannot be explained by the deterministic model.
For the longest pulse durations in the psychophysical data of Fig. 5B there is a sharp increase in the slope of the temporal integration curves for the larger pulse counts. This is not predicted by either model. This may be due to the inaccuracy of the stochastic AN model at very low discharge probabilities for long pulse durations. For these very long pulses, model responses for 4 and 16-pulse thresholds are reached at discharge probabilities that we hypothesize (Bruce et al., 1999c) are not fitted well by the integrated-Gaussian function used in the stochastic model.

C. Intensity difference limen

Figures 3 and 7 illustrate that the level of stochastic activity in the auditory nerve is dependent on stimulus intensity. It is therefore of interest to see how this changing variability in neural response affects intensity discrimination at different stimulus levels within the dynamic range. Nelson et al. (1996) have measured IDL in eight human subjects, for 200 µs/phase biphasic pulse trains delivered at 125 pps using a BP electrode configuration. The psychophysical paradigm used estimated the difference in current level \( \Delta I_{\mu A} \) from the reference current \( I_{\mu A} \) producing a 79.4% correct discrimination. IDL were collected and Weber fractions expressed in dB \( = 10 \log_{10} \left( \Delta I_{\mu A} / I_{\mu A} \right) \) were calculated for reference current levels spanning each subject’s dynamic range. Linear Weber functions \( = \alpha I_{\text{dB}} + b \) were fitted to each subject’s data collected over a range of electrode pairs. The reference intensity was expressed as a percentage of the dynamic range \( \%\text{DR} = I_{\text{dB}} / \text{DR}_{\text{dB}} \times 100 \).

The linear Weber function fits to the psychophysical data of Nelson et al. (1996) (dotted lines) are plotted in Fig. 6A, along with the deterministic model predictions (dot-dashed, dashed and solid lines) of these data. In contrast to the observed behavior, the deterministic model predicts very erratic Weber functions, with the magnitudes of the predicted Weber fractions almost all lower than the those seen in the psychophysical data. In agreement with the data, the deterministic model predicts roughly an 8 dB difference between the maximum Weber fraction at 5% of the dynamic range and the maximum Weber fraction at 95% of the dynamic range.
The linear Weber functions of Fig. 6A are replotted in Fig. 6B, along with the stochastic model predictions (dot-dashed, dashed and solid lines) of these data. In comparison to the deterministic model the magnitudes of the predicted Weber fractions are in better agreement with the psychophysical data. Furthermore, the stochastic model predicts smooth but nonlinear Weber functions, which may provide an even more accurate description of the psychophysical data than a linear Weber function (e.g., see Figures 2 and 6 of Nelson et al., 1996). Like the predictions of dynamic range versus pulse width and versus electrode configuration predictions in Bruce et al. (1999b), it appears that UCL for these data corresponds to an $N_{\text{ucl}}$ for the stochastic model approximately in the region of 100 to 1 000 spikes.

The stochastic model appears to overestimate somewhat the slopes of the Weber functions. The reduction in the Weber fraction over the dynamic range is between $\sim 12$ and $17$ dB, depending on the value of $N_{\text{ucl}}$. Nelson et al. (1996) found that the average reduction in the Weber fraction over the dynamic range was $\sim 8$ dB, with a maximum of $\sim 10$ dB. However, the true reduction in the Weber fraction in these subjects may actually be greater, since some shallow Weber functions that contributed to this mean value were caused by limitations in the intensity resolution of the implanted receiver/stimulators (Nelson et al., 1996). If so, this would provide a better agreement between the stochastic model and the data.

These predictions can be better understood by observing each model’s neural response growth function. In Fig. 7, neural responses predicted by the deterministic model (dashed line) and the stochastic model (solid line) are plotted as functions of stimulus intensity (percentage of dynamic range, with $N_{\text{ucl}} = 500$ spikes). It can be seen that:

1. The improvement in intensity discrimination (reduction in Weber fraction) over the behavioral dynamic range is due primarily to the increase in the slopes of the neural response growth curves at UCL when compared with their slopes at threshold. Although the stochastic model has slightly steeper neural response growth at UCL than the deterministic model does, the presence of variance in the neural response indicated by the error bars ($\pm 1$ std.) causes the discrimination to be worse than if no variance were present, resulting in an IDL only slightly greater than that predicted by the deterministic model.
2. The erratic Weber functions predicted by the deterministic model result from the discrete steps in neural response created by the step-function fits to single fiber I/O functions (Bruce et al., 1999c), i.e., the IDL is purely the increase in intensity required to reach one more fiber’s threshold. Therefore, the minimum IDL is practically zero if the reference intensity is immediately below the threshold of the next most sensitive fiber. The maximum IDL corresponds to the widest step in the neural response growth function, i.e., the greatest difference in consecutive neural thresholds. The smoothness of the Weber functions predicted by the stochastic model is due to the smoothness the stochastic model’s neural response growth function.

3. The increased magnitude of the Weber fractions predicted by the stochastic model, when compared with those predicted by the deterministic model, is due to the variance in the number of discharges in the stochastic model, indicated by the error bars (± 1 std.).

[Figure 7 about here.]

In Fig. 7, both the deterministic model and the stochastic model have a lower growth of response near threshold than at higher intensities. If loudness is approximately a linear function of neural response, then both models predict that loudness growth will be lower at low intensities than at high intensities. This matches the psychophysical data reviewed in Zeng and Shannon (1992).

D. Effect of number of fibers on threshold, dynamic range and intensity difference limen

In Section II.A. we have investigated how varying degrees of neural survival may account for inter-subject variability in threshold strength-duration curves. Here we extend this study to thresholds, dynamic ranges, intensity difference limen, and the correlations between these psychophysical measures. In Fig. 7 of Nelson et al. (1996), dynamic range and Weber fraction were plotted against threshold for eight human implant subjects using either a BP (adjacent electrodes) or a
wider BP (one separating electrode) configuration. In Fig. 8A and B we replot these data. Linear fits to these data showed a wide, *systematic* inter-subject variance: subjects with lower thresholds tended to have larger dynamic ranges and IDLs.

[Figure 8 about here.]

There are a number of inter-subject differences, e.g., differences in current spread, differences in fiber I/O functions, differences in nerve survival, that could create such variability. The effects of differences in nerve survival have been studied directly in monkeys: Pfingst et al. (1981, 1983) collected thresholds, dynamic ranges and IDLs and afterwards conducted histological evaluation of neural degeneration. They found that subjects with *less* neural damage had (i) lower thresholds, (ii) larger dynamic ranges and (iii) larger IDLs. Additionally, Kawano et al. (1998) found that dynamic range was correlated with neural survival in four human subjects. These two sets of results are consistent with the linear fits plotted in Fig. 8A and B if the lower thresholds in some human subjects are a result of better neural survival.

Plotted in Figures 8C–F are deterministic and stochastic model predictions of dynamic range (left panes) and Weber fraction (right panes) versus threshold, for BP (top panes) and MP (bottom panes) stimulation, from models of 1 000 to 30 000 fibers, in response to a 125 pps pulse train. These plots show that both the deterministic and the stochastic model predict changes in dynamic range and Weber fraction with respect to threshold as the number of fibers is varied. However, (i) the deterministic model predicts practically no change in threshold with the number of fibers, and (ii) the deterministic model predicts decreases in dynamic range and Weber fraction with increasing neural survival, whereas the stochastic model predicts the opposite (except for small numbers of surviving fibers in BP mode with a high $N_{uct}$). For the deterministic model, the intensity required to reach UCL decreases with the number of surviving fibers and threshold remains unchanged, producing a net decrease in the dynamic range. In contrast, both the threshold and the UCL of the stochastic model decrease with increasing neural survival and the rate of decrease of threshold is *greater* than that of UCL, producing a net increase in the dynamic range. Comparison of the model predictions with the psychophysical data of Fig. 8A and B suggests that the stochastic model, in contrast to the deterministic model, can account for the slopes of the psychophysical
data of Nelson et al. (1996) reasonably well. These results are also consistent with the correlations of thresholds, dynamic ranges and IDLs with neural survival found in monkeys by Pfingst et al. (1981, 1983). As shown in Fig. 8C–F, such correlations are predicted by the stochastic model, but the opposite behavior is predicted by the deterministic model.

The explanation of this behavior is similar to that of the effects of electrode configuration as described in Bruce et al. (1999b). For the deterministic model the major factor in determining the dynamic range and IDL is the rate at which fibers are excited with increasing intensity, and this will be less for a sparse population of fibers than for a dense one, such that dynamic range and Weber fractions are higher for a model with fewer fibers. In contrast, for the stochastic model the number of low-threshold/shallow-slope fibers is the major factor in determining behavioral threshold. With greater numbers of fibers, there is a greater “pool” of these sensitive fibers, such that thresholds decrease and dynamic ranges and IDL increase.

III. DISCUSSION

A. Interspecies differences

Despite the predominant agreement of cochlear implant psychophysical data across mammalian species, some interspecies differences have been noted. For example, Miller et al. (1999a) found that human threshold strength-duration functions for sinusoidal stimuli were steeper than all animal functions in the range of 100–300 Hz. In addition, differences were noted in the frequency at which slope decreased, with slopes for nonhuman subjects showing the decrease at higher frequencies than did those for human subjects. For pulsatile stimuli, the same authors found that statistically significant differences in threshold level were observed between human subjects and all other species (Miller et al., 1999b).

Some of these effects may be due to the different electrodes and methods of deafening used for each (species) group of experimental subjects. However, they may also be caused by species variations in cochlear and neural anatomy and physiology. For example, Frijns and Briaire (1999)
have shown with a cochlear model that differences in the anatomy of the human and guinea pig cochleae could potentially result in different populations of AN fibers and/or different nodes of Ranvier being excited by the same stimulus conditions in the two species. Furthermore, unlike most other mammals, human spiral ganglion cells have unmyelinated cell bodies (Nadol, 1988). Using a neural model, Rattay et al. (2001) showed that this may significantly affect the generation and propagation of action potentials, particularly if the initial site of excitation is on the peripheral dendrite (see also Cartee et al., 2000, for a discussion of this issue).

It would be interesting to include such factors in our composite neural/psychophysical model to see if such interspecies differences in anatomy and physiology are the cause of some or all of the psychophysical differences. Across-species comparisons of psychophysical data in which the electrode system and method of deafening are identical would also help in answering this question. Additionally, the model could be extended to take into account the significant effects that etiology and duration of deafness have on the stochastic response properties of AN fibers to electrical stimulation (Shepherd and Javel, 1997), and the predicted consequences of these on psychophysical performance could be evaluated.

B. Physiological and psychophysical thresholds from the same experimental subjects

In this paper we have used a mathematical model to make comparisons between physiological and psychophysical data collected in different experimental subjects. In many cases the species, electrode type and deafening procedures also varied. We have argued that the apparent discrepancies between physiological and psychophysical data pointed out in previous comparisons (Pfingst, 1988, 1990; Shannon, 1989; Pfingst et al., 1991) are not predominantly due to such differences but rather to incomplete measurement and analysis of auditory nerve responses, i.e., the effects of stochastic activity were ignored. This hypothesis could be confirmed or refuted by an investigation in which auditory nerve and behavioral thresholds are collected in the same experimental subjects; no such experimental study has been conducted to date. However, recently a number of
studies have been carried out in which psychophysical thresholds and thresholds of neurons in the central auditory system have been collected in the same experimental subjects (Beitel et al., 1995, 2000a,b; Montney et al., 1998; Pfingst et al., 1998). As discussed below, the results of these studies are consistent with our hypothesis.

For bipolar intracochlear electrodes, the best neural thresholds in the inferior colliculus (IC) or primary auditory cortex (A1) of an experimental subject match (or in some cases are lower than) the psychophysical threshold of the same subject (Beitel et al., 1995, 2000a,b; Montney et al., 1998; Pfingst et al., 1998). This appears to hold over a large range of pulse phase durations, such that the slopes of neural strength-duration curves match those of the psychophysical strength-duration curves (Montney et al., 1998; Pfingst et al., 1998; Beitel, pers. comm.). These results suggest that some of the spatial summation across fibers that is included in our psychophysical model is occurring before or at the IC, which is quite feasible given the significant convergence of ascending axons\(^4\) within a relatively narrow frequency region onto many principal cells of both the cochlear nucleus (Voigt and Young, 1988; Liberman, 1991) and the IC (Oliver and Morest, 1984). Our modeling results show that behavioral thresholds could be attained by relatively low AN discharge probabilities \((\ll 0.5)\) if they are summed across all activated fibers. In the case of bipolar intracochlear stimulation, the spread of excitation is fairly restricted at threshold, such that individual IC neurons that sum responses within a narrow frequency region could provide, after summation, sufficiently high discharge probabilities for detection of the stimulus. Indeed, thresholds collected in the IC and A1 of cats using a range of threshold criteria (“threshold” defined from 0.5 to 1.0 discharge probability; Beitel et al. 2000a,b) well match behavioral thresholds in the same animals. Note that the discharge probabilities of the many AN fibers within a frequency region contributing to the excitation of such IC and A1 neurons are likely to be significantly lower than 0.5.

For monopolar extracochlear electrodes, behavioral thresholds are significantly lower than IC and A1 neural thresholds (Beitel et al., 2000a), suggesting that further spatial summation is occurring after the IC and A1 in anesthetized animals. This is consistent with our modeling results and known anatomy. Extracochlear, monopolar electrodes produce a wide spread of current, such that

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auditory nerve fibers beyond a single frequency region are likely to be activated even at threshold. Therefore, more IC and A1 neurons would be activated than for intracochlear electrodes. The activity of A1 neurons could be summed in some other cortical area to achieve a perceptible response. Alternatively, such summation could be occurring in or before A1 but was not observed by Beitel et al. (2000a) due to the anesthetic used in the physiological experiments. Lateral connections that span frequency regions and could perform summation do exist in A1 (Kadia et al., 1999a,b), but their physiological effects have only been observed in an unanesthetized animal (Kadia et al., 1999a,b, 2000). It would therefore be of interest to see if A1 neural thresholds for extracochlear electrodes in unanesthetized animals are closer to behavioral thresholds.

Beitel et al. (2000a) found that the cumulative number of action potentials for a sample of IC neurons stimulated by intracochlear electrodes increased monotonically as a function of the number of pulses in a pulses train. This suggests that the temporal integration section of our model occurs after the IC. Beitel et al. (2000a) did not report on the cumulative number of action potentials in A1, so we are unable to say whether this integration occurs between the IC and A1, or if it is a result of further processing by the central nervous system. However, current theories of temporal processing in normal hearing that attempt to resolve long-term temporal integration with short-term temporal discrimination (Viemeister and Wakefield, 1991) may suggest the latter.

C. Neural field potentials

Because single-fiber responses cannot be measured in human cochlear implant users, there has been increasing interest in using neural field potentials to measure responses to electrical stimulation and to see how these relate to behavioral responses (e.g., Finley et al., 1997; Brown et al., 2000; Hughes et al., 2000). For the task of setting threshold and uncomfortable loudness levels in human cochlear implant users, it would be most desirable for field potential thresholds to be as low or lower than behavioral thresholds. However, field potential thresholds are consistently higher than behavioral thresholds in both human (e.g., Brown et al., 2000; Hughes et al., 2000) and non-human (e.g., Beitel et al., 2000a,b) subjects. Indeed, field potentials typically fall in the
upper half of the behavioral dynamic range and in many cases are around uncomfortable loudness levels (e.g., Brown et al., 2000; Hughes et al., 2000). More recent data (Cafarelli Dees et al., 2000) indicate that such field potential thresholds are even higher when the pulse rate is increased to rates used in some multi-channel cochlear implants (i.e., 1 250 pps/channel). Note that many comparisons between behavioral and evoked-potential thresholds, including the studies listed above, are confounded by temporal integration effects, i.e., the behavioral threshold are obtained using pulse trains whereas evoked potential measures are obtain from single-pulse stimuli. However, even in Brown et al. (1996) where only a single-pulse stimulus was used in the psychophysical threshold task, electrically-evoked whole nerve action potential thresholds were systematically higher than behavioral thresholds, many on the order of 3 to 4 dB, which is likely to be a consequential proportion of these subjects’ dynamic ranges. Thus, it is not surprising then that field potential threshold strength-duration curves measured in the IC (Parkins and Li, 1994) are similar to behavioral data and model predictions for uncomfortable loudness strength-duration curves (Bruce et al., 1999b). Furthermore, Miller et al. (1999d) found that the accuracy of model predictions of the electrically evoked compound action potential (CAP) in cats was dependent mainly on the distribution of fiber thresholds in the AN, with stochastic activity being a secondary factor. This is consistent with our model, if the measured CAP thresholds are systematically above behavioral thresholds, at which only the most sensitive AN fibers are excited. Indeed, Beitel et al. (2000a,b) found that brainstem field potentials in guinea pigs were approximately 6 dB above both the behavioral thresholds and the lowest IC and A1 single-unit thresholds in the same animals. Taken together, these results provide considerable evidence that behavioral threshold corresponds to a small number of relatively stochastic neural discharges (likely originating from a larger sub-population of similar, very sensitive fibers) that are often not discernible in measured field potentials, at least from the AN up to the IC. Consistent with this interpretation, Finley et al. (1997) found that CAPs were quite weak in response to stimulation from a single electrode even when using stimulus amplitudes at the upper limit of levels employed in typical multi-channel continuous interleaved sampling (CIS) processors.
D. Further model development

In Bruce et al. (1999a,b,c) we have discussed physiological data that would assist in further refinement of the neural section of our model. In particular, physiological data characterizing the stochastic activity of single fibers are required at least over the whole range of stimulation rates currently used in implants and preferably up to higher stimulation rates that are anticipated in future implants and at stimulus intensities that are within the normal operating range for (i) the output of a multi-channel speech processor in normal listening conditions and (ii) psychophysical tasks where simpler stimulus patterns are typically presented.

In addition, our model’s distributions of thresholds and RSs in a population of fibers were based on relatively little data. More substantial data have been collected recently by Miller et al. (1999c,d) using monophasic pulses; it would be interesting to see how the model’s predictions would change if we used the Miller et al. distributions of thresholds and RSs. For example, Miller et al. showed a negative correlation between RSs and thresholds (not seen in our data), i.e., only a small population of fibers with low thresholds had RS values as high as the highest values in our data. This distribution of thresholds and RSs would produce psychophysical model predictions that are less dependent on stochastic AN activity except at very low stimulus intensities, i.e., right at behavioral threshold. We hypothesize that the distributions of thresholds and RSs may be quite sensitive to the experimental paradigm. The threshold of any single fiber is affected by both the attenuation of the stimulating current as it spreads from the stimulating electrode to the fiber and the amount of current (as measured at the fiber) required to depolarize that fiber. In our model we have separated out these two factors by using the Merzenich and White (1977) and O'Leary et al. (1995) data for the rate of current spread for different electrode configurations and the van den Honert and Stypulkowski (1987) data to estimate the distribution of thresholds for a local population of fibers. In the Miller et al. studies, only one electrode configuration was used, a monopolar electrode placed just inside the round window of the cochlea, and the cochlear origin of the recorded AN fibers was not determined. It is therefore not possible to separate out the effects of current spread and local threshold variation in these data.

The distribution of RSs may also be affected indirectly by current spread. RS is a property of
the node of Ranvier at which an action potential is generated, not the stimulating current. However, which nodes are excited by a given stimulus is dependent on current spread, thereby affecting the distribution of RSs in the stimulated nerve. RS is known to be negatively correlated to the node diameter (Verveen, 1962; Rubinstein, 1995). Node diameters vary amongst fibers and are correlated to the spontaneous rate of the fiber and/or the point at which the fiber innervates the cochlea in some species (see the introduction of Gleich and Wilson, 1993, for a review). In some species such as cat, fibers emanating from the basal turn of the cochlea tend to be smaller than apical-turn fibers (Arnesen and Osen, 1978) and consequently the depth of insertion of the stimulating electrode may affect RS values. The diameter of fibers is also known to increase along their length (again, see the introduction of Gleich and Wilson, 1993, for a review). Consequently, stimulating currents that excite the fiber’s axon peripherally (i.e., on nodes between the organ of Corti and the cell body) should produce larger RS values than currents exciting the axon distally (i.e., on nodes between the cell body and the cochlear nucleus). Physiological data from van den Honert and Stypulkowski (1984) and Javel and Shepherd (2000) indicate that both modes of excitation are possible, depending on the electrode configuration, the stimulus waveform and intensity, and the condition of the nerve. Leake and Hradek (1988) have shown that degeneration of the nerve caused by ototoxic drugs begins with demyelination and degeneration of the peripheral dendrites. Therefore, the status of the nerve also should affect the values of RS (as well as thresholds) observed in a particular experimental paradigm. The multiplicity of factors contributing to thresholds and RSs motivates further studies in which both the electrode placement and the status of the nerve are varied (e.g., Shepherd and Javel, 1997; Javel and Viemeister, 2000), in order to determine the range of distributions of thresholds and RSs possible in different subjects.

Another model behavior requiring further physiological investigation is the dependence of a fiber’s RS on the pulse width (phase duration) of a biphasic pulse, as described by Eq. (3). As stated in Section I.A., this effect is based on a relatively small but consistent set of data (Bruce et al., 1999c) and the physiological cause is as yet unknown. Interestingly, dependence of RS on pulse width is absent in the case of monophasic pulses (Verveen, 1960), suggesting that the hyperpolarizing phase of biphasic pulses may be what is causing the increase in membrane noise in the
former case. Experimental support for this hypothesis is the increase in membrane noise observed by Verveen and colleagues when they hyperpolarized neural membranes in vitro (e.g., see Fig. 17 of Verveen and Derksen, 1968). Verveen and colleagues did not determine the cause of this behavior; sodium and potassium channels are both inactivated by hyperpolarization and consequently should contribute to a reduction in membrane noise. However, recent studies have determined the existence of a hyperpolarization-activated cationic channel ($I_h$) in many neurons, including spiral ganglion cells from both neonatal rats (Mo and Davis, 1997) and adult guinea pigs (Chen, 1997). The time constant of this channel is on the order of tens to hundreds of milliseconds (very long compared to sodium and potassium channels; Santos-Sacchi, 1993), which could explain why membrane noise increases with pulse width for biphasic pulses. Depolarizing monophasic pulses will not activate the $I_h$ current, explaining why there is no effect of pulse width on RS in that case. Further experiments are required to characterize the location and kinetics of single $I_h$-channels in auditory nerve fibers. When such properties are known, they could be included in a model such as that of Rubinstein (1995), and the effects of the $I_h$ channel on membrane dynamics could be further studied. For instance, this channel could provide a depolarizing current during the membrane hyperpolarization following each firing of an auditory nerve fiber (Chen, 1997; Mo and Davis, 1997), altering the temporal characteristic of action potentials (which are not well described by present models; e.g., Phan et al., 1994; Cartee, 2000). The $I_h$ channel may also be important in the case of high-rate, subthreshold stimulation, which may lead to accumulated activation of this channel, in addition to its effect on sodium and potassium channels (Rubinstein et al., 1999b).

E. Effects of neural survival

Our modeling results and the data of Pfingst et al. (1981) indicate that loss of spiral ganglion cells (SGCs) leads to a reduced dynamic range. However, model predictions and the data of Pfingst et al. (1983) suggests that there is a corresponding decrease in IDLs. Given our interpretation of the human data of Nelson et al. (1996), Fig. 9 of Nelson et al. (1996) would imply that the net effect of loss of SGCs is an increase in the number of discriminable intensity steps within the
dynamic range. It would be expected that a greater number of discriminable steps would improve speech perception (e.g., Loizou et al., 1999), which may partially explain why implant users with quite poor neural survival can still obtain good speech perception scores (e.g., Fayad et al., 1991; Linthicum et al., 1991). This is particularly relevant to ongoing efforts to find methods of promoting neural survival (Leake et al., 1999; Marzella et al., 1999): Is a reduced number of SGCs actually beneficial for implant users? To answer this question, factors other than intensity coding must be taken into consideration. Firstly, in experiments simulating cochlear implant stimulation in normal hearing subjects, Loizou et al. (1999) found that only a small number of intensity steps is required for good speech perception if the speech is represented by a large number of spectral channels. An individual with good neural survival may have a larger number of effective channels because each electrode stimulates a discrete population of fibers. Conversely, patchy neural survival may produce better intensity discrimination but may also reduce the number of effective channels. Secondly, our modeling results indicate that for stimulus levels within the behavioral dynamic range, discharge probabilities in individual fibers will be higher when there are fewer SGCs. Consequently, refractory effects will be more predominant and may distort the temporal representation of the stimulating pulse train. Thirdly, central processing factors may outweigh and obscure peripheral factors (Blamey et al., 1996; Pyman et al., 2000). Taken together, these four considerations support the hypothesis that neural survival is not a dominant factor in speech perception performance with present cochlear implant electrode arrays and stimulation schemes (Blamey et al., 1996; Blamey, 1997; Rubinstein et al., 1999a) because it interacts with other factors in a complex manner. With a deeper understanding and consequent appropriate design of electrodes and stimulations systems, nerve survival may ultimately be determined to be an important factor in speech perception.

IV. CONCLUSIONS

In this paper we have demonstrated via the use of a composite neural/psychophysical model that behavioral thresholds and suprathreshold intensity-discrimination for mammalian cochlear
Implant subjects can be explained, for the most part, by the response properties of the auditory nerve if stochastic activity is considered (Bruce et al., 1999c), without having to alter the auditory nerve model for each individual species. These results support the hypothesis that interspecies differences produce only relatively minor difference in psychophysical data (Miller et al., 1999b), validating across-species comparisons of physiological and psychophysical data. Such comparisons are particularly useful in understanding psychophysical results for human cochlear implant users, from whom only limited (i.e., noninvasive) physiological data can be collected.

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NOTES

1If the entire presentation interval is < 100 ms, as is the case for almost all the data presented in this paper, the integrator’s output is simply the sum of all spikes that occur during the interval. For the cases where the stimulus is > 100 ms (Sections II.B., II.C. and II.D.), we sum the responses elicited by the maximum number of pulses that can appear within the temporal integration window. For a uniform pulse train this is equal to the elicited response to \( n \) pulses, where \( n \) is the pulse rate times 100 ms.
70.7% is the point on a psychometric function that is estimated by a two-down, one-up procedure (Levitt, 1971). We performed sensitivity testing and found that comparisons between the deterministic and the stochastic model were relatively insensitive to the value of the criterion.

We cannot expect the model developed in this paper to predict absolute threshold with any degree of precision, because we do not have data on single-fiber I/O functions for each subject. For example, a change in electrode-to-electrode contact distance or electrode-to-nerve distance will lead to a shift in single-fiber thresholds, i.e., a change in Eq. (2). Such a shift in the threshold of single fibers will result in a horizontal shift of the curves in Fig. 8C–F.

Efferent and lateral inputs may also play a part in the neural responses in the awake, behaving animal. However, their effects will be greatly modulated by the anesthetic used during the physiological recordings.

REFERENCES


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FIGURE CAPTIONS

1 Composite physiological auditory nerve model and psychophysical intensity-discrimination/loudness model. Reprinted from Fig. 1 of Bruce et al. (1999b), © 1999 IEEE. .................................... 40

2 A: Threshold versus phase duration (pulse width) data from 12 subjects (each line indicates a separate subject), replotted from Fig. 2 of Pfingst et al. (1991) with permission from the Acoustical Society of America © 1991; B: Deterministic and stochastic model predictions of these data for BP and MP stimulation and for different numbers of surviving fibers, N. ........................................ 41

3 Number of discharges vs stimulus intensity from a model of 10 000 fibers in response to a single biphasic pulse of duration 1 000 µs/phase with a bipolar electrode configuration. A: Number of discharges for the deterministic model. B: Mean (solid line) and standard deviation (dashed line) of the number of discharges for the stochastic model. C: The standard deviation divided by the mean of the number of discharges for the stochastic model. Vertical dotted lines indicate stimulus intensities corresponding to threshold and uncomfortable loudness (at three different values of $N_{ucd}$), as labeled. ........................................ 42

4 Effect of electrode configuration on psychophysical strength-duration curves. A: Guinea pig psychophysical data, replotted from Fig. 6 of Miller et al. (1995) with permission from Elsevier Science © 1995. Symbols represent individual data points and solid lines represent least-squares power-function fits to the BP and MP data. B: Cat psychophysical data replotted from Fig. 4 of Smith and Finley (1997) with permission from the Acoustical Society of America © 1997. Symbols and lines represent means across a maximum of five subjects at each phase duration (less than five subjects contribute to the means at the longer phase durations). .... 43

5 Effect of pulse width on temporal-integration curves (threshold versus number of pulses). A: Psychophysical data, replotted from Fig. 5 of Moon et al. (1993) with permission from Elsevier Science © 1993, for 5 subjects, each subject indicated by a different symbol, for wide BP (one separating electrode), 100 pps pulse trains at two different phase durations, as given in the legend; B: Psychophysical data (previously unpublished) from one subject for radial BP, 100 pps pulse trains at four different phase durations, as given in the legend of (C). C,D: Deterministic model (C) and Stochastic model (D) predictions for the same stimulus conditions as B. ........................................ 44

6 IDL (Weber fractions) versus stimulus intensity (percentage of dynamic range): linear Weber function fits (dotted lines) to psychophysical data from 8 subjects, replotted from Fig. 6 of Nelson et al. (1996) with permission from the Acoustical Society of America © 1996, and deterministic model (A) and stochastic model (B) predictions for $N_{ucd} = 100$ spikes (dot-dashed line), 500 spikes (dashed line) and 1 000 spikes (solid line). ........................................ 45

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Number of neural discharges versus stimulus intensity (percentage of dynamic range, with $N_{\text{ucl}} = 500$ spikes) predicted by the deterministic model (dashed line) and by the stochastic model (solid line) for a pulse rate of 125 pps. Error bars indicate ±1 std.

Psychophysical data reprinted from Fig. 7 of Nelson et al. (1996) with permission from the Acoustical Society of America © 1996: corresponding dynamic range (A) and Weber fraction at 75% of dynamic range (B) functions for a 125 pps pulse train. Deterministic and stochastic model predictions of dynamic range (C,E) and Weber fraction at 75% of dynamic range (D,F) versus threshold, for BP (C,D) and MP (E,F) stimulation, from models of 1,000 to 30,000 fibers, in response to a 125 pps pulse train. The number of fibers used in the model to generate each point are as labeled. Curves are plotted for two different values of $N_{\text{ucl}}$, as indicated in the figure legend.
FIG. 1.
FIG. 3.
FIG. 4.
FIG. 5.
FIG. 6.

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FIG. 7.
FIG. 8.