

CONTROL STRATEGIES FOR NERVES MODELED BY SELF-EXCITING POINT PROCESSES

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INTRODUCTION

Cochlear implants electrically stimulate the auditory nerve with the aim of generating a perception of sound via an evoked neural response pattern. An electrically stimulated auditory nerve responds differently to an acoustically stimulated auditory nerve, and the surviving nerves of patients with a hearing loss may exhibit characteristics different from those of normal-hearing people. Thus, the cochlear implant evoked response pattern differs greatly from that of the normal hearing situation. One method of understanding such response patterns is to employ a mathematic model. If possible, the model should permit the determination of neural response differences between closely related sounds, and facilitate the design of stimuli that evoke desired neural response patterns. How should such a model be chosen?

The auditory nerve's response to sound can be closely approximated by a series of stochastically distributed identical spikes, and has been successfully modeled via point process models.¹ The simplest such model, the Poisson process, has an intensity (average rate) that is independent of the realization of the process. However, the probability of auditory nerve action potential generation is affected by the history of the process via the refractory nature of neural response. Thus, a more accurate model should take into account the response history of the nerve, resulting in a lowered probability of an action potential within the nerve's refractory period. In the model analyzed in this paper,²⁻⁵ the intensity of the process is both a function of time (via a stimulation function) and of the history of the process (via a hazard function). If this model is applied to compare cochlear implant and normal hearing neural responses, then the response differences may be ascribed to differences in the stimulation and hazard functions for each case.

The stimulation function of the process model is defined to be the time-varying expected rate of neural discharge in the fictitious case in which there is no neural refractory period. It should not be confused with the applied electrical or acoustic signal level, as it is a function not only of the applied signal's level and type, but also of the neural characteristics. For instance, an unstimulated nerve (zero applied signal) will exhibit a spontaneous firing pattern, and thus the process model will have a nonzero stimulation function. Research is currently being undertaken to quantify the relationship between the applied electrical or acoustic signal, and the stimulation function of the stochastic process model. Conversely, the hazard function has been found to be largely independent of the intensity of the applied signal,² and thus may be treated as an invariant for a particular neural population stimulated via a particular method (electrical or acoustic).

The purpose of this paper is to demonstrate how stimuli may be designed to force the post-stimulus time histogram

(PSTH), and consequently the period histogram, of the neural response of cochlear implant patients to approximate that of normal-hearing patients. Here this is achieved for the case in which the (step-shaped) hazard functions of the auditory nerves stimulated via the cochlear implant differ from those of the normal hearing situation, and full control is assumed over the stimulating functions. Work to extend these results to a more general case is currently in progress.

NEURAL RESPONSE MODEL

The conditional probability for a nerve firing in the small time interval $(t, t + \Delta t)$ is modeled by^{3,6}

$$(1) \quad \Pr [\text{one spike in } (t, t + \Delta t) \mid \text{No previous spikes}] = s(t)\Delta t \\ \Pr [\text{one spike in } (t, t + \Delta t) \mid \text{the last spike was at time } \tau] = s(t)r(t - \tau)\Delta t$$

where $s(t)$ is the "stimulation function" and $r(t - \tau)$ is the "hazard function."

Equation 1 is a special case of a self-exciting point process.¹ For this case it is convenient to define the quantity $m(t)$,⁴ where

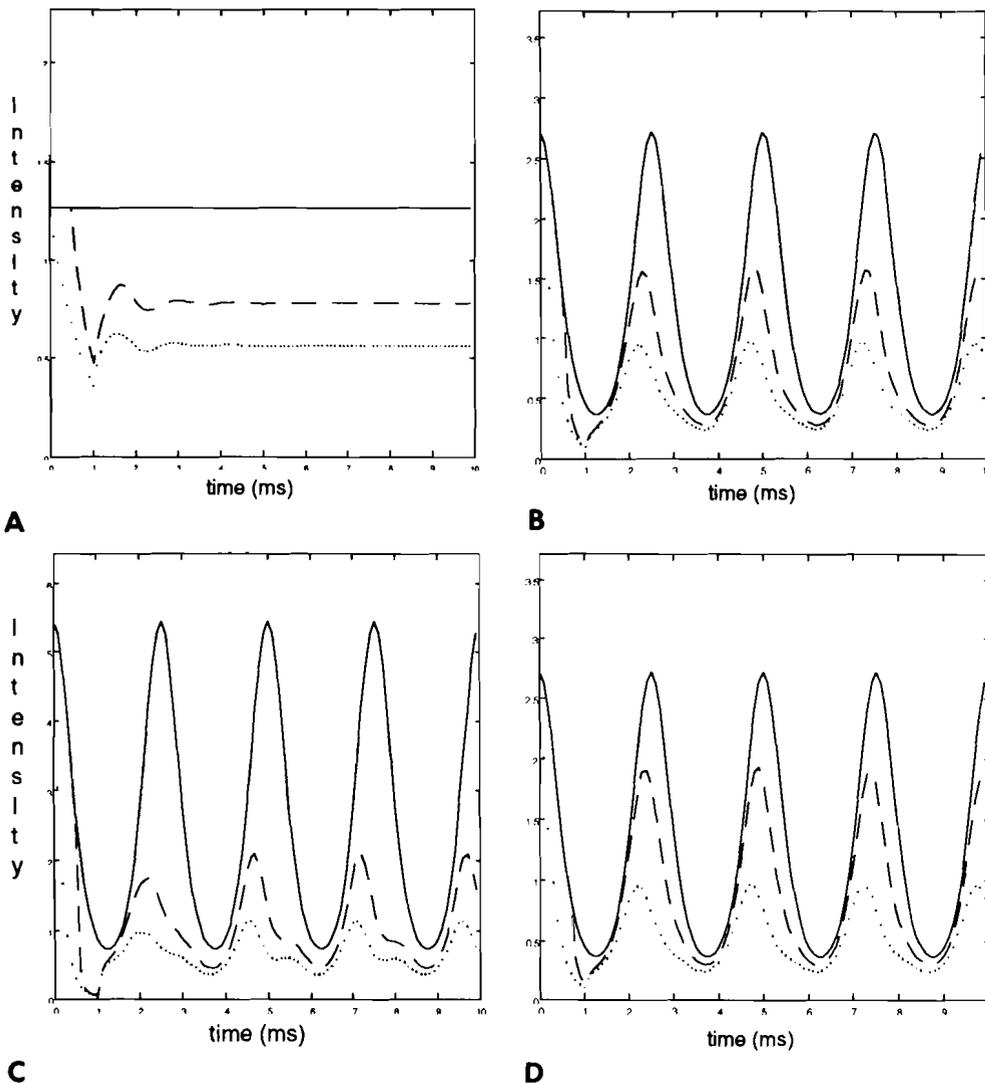
$$(2) \quad m(t) = s(t)\Pr[\text{no previous firings}] + E[s(t)r(t - \tau) \mid \text{nerve has fired}] \Pr[\text{nerve has fired}]$$

and $m(t)$ equals a scaled version of the average post-stimulus time response for either a number of identical nerves, or for one nerve under repeated stimuli. The function $s(t)$ depends on the characteristics of the specific nerve, and on the stimulating signal's level and type. Inherent in this model is the assumption that the function $r(t - \tau)$ is a hazard function that lowers the probability of spike generation shortly after earlier spike generation. The shape of $r(t - \tau)$ may vary as a function of neural population or signal type (electrical or acoustic), but not signal level.

NEURAL CONTROL FOR STEP-SHAPED REFRACTORY FUNCTION

In this section we address two key issues. Given that the shape of the hazard function is different in the cochlear implant case and the normal hearing case, it is important to determine how this will affect the neural response pattern in either case. Furthermore, it would also be useful to be able to design stimuli for the cochlear implant that take into account these shape differences, and produce neural response patterns identical to those of the normal hearing situation. Both issues are addressed in the following lemma.

For reasons of clarity and brevity we specialize to the case in which there is an absolute refractory period and no relative refractory period (modeled via a step-shaped hazard function), and demonstrate how stimuli can be generated such that a system with a lower absolute refractory period will exhibit the same PSTH as a system with a higher absolute refractory



Applied stimulation function s_1 (solid line); neural response post-stimulus time histogram (PSTH), m_1 (dotted line); and stimulation function s_2 required to cause another nerve to exhibit same PSTH (dashed line). See text for details. A) Step stimulus function. B) Strongly phase-locked 1. C) Strongly phase-locked 2. D) Strongly phase-locked 3.

period. Some of the results of the lemma are also found elsewhere.^{4,5} A more general version of this lemma will also be reported in a lengthier version of this paper.

Lemma 1. Consider the case of two nerves stimulated via any means, such that the firing rate of each nerve is described via a self-exciting point process, with intensity equal to $s(t)r(t - \tau)$ where $s(t)$ is a stimulation function of time, and $r(t - \tau)$ is a hazard function of time since the last action potential. Let nerve 1 have a stimulation function s_1 , and hazard function $r_1 = u(t - a)$, and nerve 2 have a hazard function $r_2 = u(t - b)$. Here $u(t)$ denotes the step function. Define $m_1(t)$ via equation 2, for $s_{1\cdot}(\cdot)$ and $r_{1\cdot}(\cdot)$.

Then for a positive, bounded, and differentiable $s_1(t)$, $m_1(t)$ can be calculated via the relationship:

$$(3) \quad \begin{aligned} \frac{dm_1(t)}{dt} &= m_1(t) \left(\frac{ds_1(t)}{dt} / s_1(t) - s_1(t) \right), \\ &= m_1(t) \left(\frac{ds_1(t)}{dt} / s_1(t) - s_1(t) \right) + s_1(t)m_1(t - a) \end{aligned}$$

$$m_1(0) = s_1(0) \quad \begin{aligned} &\text{for } t \leq a \\ &\text{for } t > a \end{aligned}$$

Define:

$$(4) \quad \begin{aligned} s_2(t) &= m_1(t) / [1 - \int_{t-b}^t m_1(u) du] && \text{for } t > b \\ &= m_1(t) / [1 - \int_0^t m_1(u) du] && \text{for } t \leq b \end{aligned}$$

If $b \leq a$, then $s_2(t)$ is positive and bounded. If $b \leq a$, and nerve 2 is stimulated so that the stimulation function equals s_2 , then $m_2(t) = m_1(t) \forall t \geq 0$, or equivalently, the two nerves will expect the same PSTH.

Remarks:

1. Clearly, if $b > a$, then it is not generally possible for m_2 to equal m_1 , since the maximum steady state value m_1 can attain is $1/a$, greater than the maximal steady state value of m_2 , which is $1/b$.

2. High-characteristic frequency nerves stimulated via a pure tone at their characteristic frequency often exhibit a constant rate of firing, modeled here by a step function-shaped stimulation function. In this case, $s_2(t)$ of equation 4 will approach the steady state value of $s_2(t) = s_1 / [1 + (a - b)s_1]$. See the Figure, A, for an example.

SIMULATION EXAMPLE

In the Figure, the solid line denotes the stimulation function s_1 , and the dotted line denotes the shape of the PSTH for such a stimulation function incident on a nerve with an absolute refractory period of 1 millisecond (ms), and no relative re-

factory period. The dashed line denotes the stimulation function that would cause a nerve with an absolute refractory period of 0.5 ms (see Figure, A-C) or 0.7 ms (see Figure, D) to respond with the same PSTH.

In the Figure, A, the stimulation function is chosen to be a step function (remark 2). Here it can be seen that the response asymptotes to the constant value of $s1/(1 + a s1) = s2/(1 + b s2)$ as in equation 4.

In the Figure, B,D, the stimulation function $s1 = \exp[\cos(2512t)]$, simulating a strongly phase-locked neural response to a 400-Hz signal. In the Figure, C, $s1 = 2 \exp[\cos(2512t)]$, simulating a stronger signal, also at 400 Hz.

In the case of a nonstationary stimulation function, such as a strongly phase-locked nerve (see Figure, B-D), the shapes of the stimulation functions required to produce the same PSTH in nerves with different hazard functions are considerably different (especially the Figure, C). A larger stimulation (compare the Figure, B and C), or a larger difference between the hazard functions of the two types of stimulation (compare the Figure, D and B), results in greater differences between the shapes of stimulation functions that result in the same PSTH. This is why simply applying an electrical signal that is the same shape (even possibly resized) as an acoustic signal does not evoke the same PSTH as a nerve directly stimulated via the acoustic signal.

CONCLUSION

Improvements to the simulation of acoustically generated neural firing patterns by artificially evoked neural firing patterns should lead to improved speech perception of cochlear implant patients. To the knowledge of the authors, this paper is the first to present the idea of utilizing the point process models of auditory nerve response to design stimuli that cause this improvement. Work is under way to extend these ideas to a practical algorithm.

REFERENCES

1. Snyder D, Miller M. Random point processes in time and space. Berlin, Germany: Springer-Verlag, 1991.
2. Gaumont R, Kim D, Molnar C. Stimulus and recovery dependence of cat cochlear nerve spike discharge probability. *J Neurophysiol* 1982;48:856-73.
3. Johnson D, Swami A. The transmission of signals by auditory-nerve fiber discharge patterns. *J Acoust Soc Am* 1983;74:493-501.
4. Bi Q. A closed-form solution for removing the dead time effects from the post-stimulus time histograms. *J Acoust Soc Am* 1989;85:2504-13.
5. Jones K, Tubis A. On the extraction of the signal-excitation function from a non-Poisson cochlear neural spike train. *J Acoust Soc Am* 1985;78:90-4.
6. Gaumont R. Studies of the stimulus and recovery dependence of cat cochlear nerve fiber discharge probability [Thesis]. St Louis, Mo: Washington University, 1980.



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