An Interaction Model of a Poisson and a Renewal Process Related to Neuron Firing

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Received: April 5, 1974

Abstract

This paper discusses a neuronal model based on a model of Coleman and Gastwirth (1969). It is assumed that the excitatory input forms a Poisson process while the inhibitory input forms a stationary renewal process. The proposed interaction scheme is as follows: an inhibitor deletes at most \( N \) consecutive excitatory inputs and a response only occurs after the cumulative storage of \( M \) excitatory inputs. The Laplace transform of the probability density function (p.d.f.) of the inter-response intervals is derived together with results of the numerical inversions.

1. Introduction

Bishop, Levick, and William (1964) reported experimental data showing that the distribution of interspike intervals for lateral geniculate neurons is multimodal in nature. As an explanation of this phenomenon, Ten Hoopen and Reuver (1965) introduced the selective interaction of two independent recurrent point processes. They assumed that the primary (excitatory) process is a stationary renewal point process consisting of the arrival of stimuli and with a p.d.f. \( \phi(t) \), while the secondary (inhibitory) process is another stationary renewal process consisting of the arrival of inhibitors and with a p.d.f. \( \psi(t) \). An inhibitor deletes the following stimulus, always providing that this stimulus has occurred before the following inhibitor, otherwise no deletion is caused by the previous inhibitor. The sequence of undeleted stimuli forms the response process, corresponding to the firings of a neuron.

Basing on the model of Ten Hoopen and Reuver (1965), Coleman and Gastwirth (1969) proposed three other models for neuron firing. In this paper, we will generalize their Model IIIb. The mechanism of interaction in the original model of Coleman and Gastwirth is as follows:

(a) an inhibitor has a life time \( \tau \) (a period during which the inhibitor is effective) which is an arbitrary positive random variable with a p.d.f. \( g(t) \),
(b) the arrival of an inhibitor wipes out the remaining effect (if any) of the preceding one and it remains effective until it "dies off" or an inhibitor arrives during its life time,
(c) all the stimuli occurring during the life time of an inhibitor will be deleted, and
(d) any undeleted stimulus will give rise to a response.

Conditions (c) and (d) do not seem to be very realistic and in this paper we propose to change these respectively to

(c') an inhibitor will delete \( 1, 2, \ldots, N \) stimuli occurring during its life time and it will "die off" immediately after the \( N \)th deletion, and
(d') an undeleted stimulus will be stored and a response occurs only after the cumulative storage of a certain number \( M \) of stimuli. If, in the course of accumulation of stimuli, one inhibitor arrives, then the accumulated stimuli are lost, accumulation starting anew as soon as the inhibitor dies off.

A typical realization for the resulting model is depicted in Fig. 1.

2. P.d.f. of the Inter-Response Intervals

The Laplace transform \( p^*(s) \) of the p.d.f. \( p(t) \) of the inter-response intervals (where by definition \( p^*(s) = \int_0^\infty e^{-st}p(t)\,dt \)) will be derived for a Poisson process (parameter \( \lambda \)) deleted by a renewal process with inter-arrival interval p.d.f. \( \psi(t) \).

Let us take \( t = 0 \) as the beginning time at which a response has occurred. We observe that if \( p_1(t)dt \) and
denotes the probability that the next response after 
\( t = 0 \) occurs in \((t, t+dt)\) while \( k \) inhibitors have 
ocurred in between, given that a response has occurred 
at \( t = 0 \), then
\[
p(t) = \sum_{k=0}^{\infty} p_k(t) .
\]  
(1)

To find \( p_k(t) \), \( k \geq 0 \), we first of all define the following 
probabilities:

- \( R(t)dt \) – the probability that the next inhibitor 
after \( t = 0 \) occurs in \((t, t+dt)\) given that a response has 
occurred at \( t = 0 \),

- \( A(t)dt \) – the probability that the next inhibitor 
after \( t = 0 \) occurs in \((t, t+dt)\) with no response up 
to \( t \), given that a response has occurred at \( t = 0 \),

- \( B(t)dt \) – the probability that the next inhibitor 
after \( t = 0 \) occurs in \((t, t+dt)\) with no response up to 
\( t \), given that an inhibitor has occurred at \( t = 0 \),

- \( C(t)dt \) – the probability that the next response 
after \( t = 0 \) occurs in \((t, t+dt)\) while no inhibitors occur 
in \((0, t)\), given that an inhibitor has occurred at \( t = 0 \).

It is clear that
\[
p_0(t) = \phi M(t) \int_{\gamma=1}^\infty R(\gamma) d\gamma
\]  
(2)
and
\[
p_1(t) = A(t) \ast C(t),
\]  
(3)
where \( \ast \) denotes convolution, and
\[
\phi M(t) = \frac{(\lambda t)^{M-1}}{(M-1)!} e^{-\lambda t} .
\]  
(4)

To find \( p_k(t) \), \( k \geq 2 \), we note that if \( x_i \), where 
\( 1 \leq i \leq k \) and \( k \geq 2 \) denotes the time of occurrence of 
the \( i \)th inhibitor in \((0, t)\) during which no responses 
have occurred, then \((0, x_1), (x_1, x_2), \ldots, (x_k, t)\) form a sequence of \( k+1 \) independent intervals. 
In particular, \((x_i, x_{i+1})\), \( 1 \leq i \leq k-1 \) form a sequence of 
\( k-1 \) identically distributed renewal intervals 
characterized by the function \( B(t) \), while \((0, x_1) \) and 
\((x_k, t)\) will be characterized by \( A(t) \) and \( C(t) \) respectively.

Thus we obtain
\[
p_k(t) = A(t) \ast B^{(k-1)} \ast C(t), \quad k \geq 2,
\]  
(5)
where \( B^{(*)} \) for \( n \geq 1 \) is the \( n \)-fold convolution of \( B(t) \).

We have
\[
A(t) = R(t) J_{M-1}(t)
\]  
(6)
where
\[
J_{M-1}(t) = \sum_{k=0}^{M-1} \frac{(\lambda t)^k}{k!} e^{-\lambda t},
\]  
(7)
and \( B(t)dt \) equals the sum of

(A) the integral of the product of the probabilities that

(a) (i) the life time of the inhibitor at \( t = 0 \) lies in 
\((\tau, \tau+dt)\) with \( 0 < \tau < t \) and at most \( N-1 \) stimuli occur in \((0, \tau)\) 
with \( 0 < \tau < t \),

or (ii) the \( N \)th stimulus after \( t = 0 \) occurs in \((\tau, \tau+d\tau)\) 
with \( 0 < \tau < t \) while the inhibitor at \( t = 0 \) dies in \((\tau, \infty)\),

(b) at most \( M-1 \) stimuli occur in \((\tau, t)\),

(c) the next inhibitor after \( t = 0 \) occurs in \((t, t+dt)\) 
(see Fig. 2);

(B) the integral of the product of the probabilities that

(a) at most \( N-1 \) stimuli occur in \((0, t)\),

(b) the next inhibitor after \( t = 0 \) occurs in \((t, t+dt)\),

(c) the inhibitor at \( t = 0 \) dies off in \((t, \infty)\) (see Fig. 3).

It follows that
\[
B(t) = \psi(t) \left[ \int_{\tau=0}^{\infty} J_{N-1}(t) \cdot g(\tau) d\tau \right] \ast \left[ J_{M-1}(t) \right] + \int_{\tau=t}^{\infty} \psi(t) \cdot \int_{\gamma=0}^{\infty} g(\gamma) d\gamma \cdot J_{N-1}(t) .
\]  
(8)

To compute \( C(t) \), we note that \( C(t)dt \) equals the integral of the product of the probabilities that

(a) (i) the life time of the inhibitor at \( t = 0 \) lies in 
\((\tau, \tau+d\tau)\) with \( 0 < \tau < t \) and at most \( N-1 \) stimuli occur in \((0, \tau)\) 
with \( 0 < \tau < t \),

or (ii) the \( N \)th stimulus after \( t = 0 \) occurs in \((\tau, \tau+d\tau)\) 
with \( 0 < \tau < t \) while the inhibitor at \( t = 0 \) dies in \((\tau, \infty)\),

(b) the next inhibitor after \( t = 0 \) occurs in \((t, \infty)\),

(c) the next \( M \)th stimulus after \( \tau \) occurs in \((t, t+dt)\) 
(see Fig. 4).