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Neuronal integration mechanisms have little effect on spike auto-correlations of cortical neurons

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Abstract

Cortical neurons of behaving animals generate irregular spike sequences, but the sequences generally differ from an entirely random sequence (Poisson process), and they have temporal correlations (spike auto-correlations). Temporally correlated spike sequences can be brought about because of incoming synaptic inputs to the neuron, or because of the neuronal integration mechanism. In this paper, we attempt to determine which is the origin of spike auto-correlations observed in the spiking data recorded from neurons in the prefrontal cortex of a monkey preserving a cue information in the delay response task experiment. Each incoming input is assumed to be independent from its own spike events, and the temporal integration in the neuron is assumed to be reset by every spike event. So, the process to spike is assumed to be divided into two processes: the process independent from its own spikes, which drives the process reset by its own spikes. Under these assumptions, it is found that the spike-independent process needs to have temporal correlations, through examinations of two kinds of correlation coefficient of consecutive inter-spike intervals. It is also found that the spike-reset process has little effect on the spike auto-correlations and the interval distributions. This suggests that the spike auto-correlation *does* originate in the temporal correlation of incoming synaptic inputs and the neuronal integration mechanism has little effect on the spike auto-correlation.

Keywords: Spiking statistics; Single neuron; Temporal coding; Temporally correlated inputs; Prefrontal cortex

1. Introduction

Irregular spike sequences are observed in large regions of cerebral cortex in vivo (Britten, Shadlen, Newsome & Movshon, 1993; Shinomoto, Sakai & Funahashi, 1999; Softky & Koch, 1993; Tomko & Crapper, 1974), while a cortical neuron under a constant current injection in vitro generates regular spike sequences (Kaneko, Kang & Mizuno, 1995; Thomson & Deuchars, 1997). This suggests that a neuron in vivo receives highly fluctuating synaptic inputs. When the fluctuation of incoming inputs is large relative to the mean, spikes are randomly discharged and the sequence exhibits high irregularity. In fact, a regular spiking neuron under a highly fluctuating current injection in vitro generates highly irregular spike sequences (Nowak, Sanchez-Vives & McCormick, 1997). Highly fluctuated inputs can be caused by inhibition balanced to excitation (Shadlen & Newsome, 1994, 1998). Balanced inputs can be brought about naturally in model networks (Amit & Brunel, 1997; Tsodyks & Sejnowski, 1995, Vreeswijk & Sompolinsky, 1996, 1998), but, taking higher statistics into account, many spike sequences are far different from entirely random one (Poisson process), even if the animal is behaviorally in a steady state (Sakai, Funahashi & Shinomoto, 1999). That is the spike sequences have temporal correlations.

Why can such a temporal correlation in a spike sequence be brought about? A cortical neuron has many presynaptic neurons; (Braitenberg & Schuz, 1991; Peters, 1987). If the presynaptic neurons emit spikes independently, then the incoming inputs have no temporal correlation, even though each of them generates a temporally correlated spike sequence. Can the neuron-received uncorrelated inputs generated temporally correlated spike sequences?

Shinomoto et al. (1999) and Sakai et al. (1999) attempted to determine whether the simple leaky integrate-and-fire mechanism statistically reproduces the spiking data recorded from a monkey prefrontal cortex. They found that the inputs need to have temporal correlation of the order of 100 ms so that the leaky integrate-and-fire mechanism might reproduce statistical coefficients of inter-spike intervals similar to the biological spiking data.

The leaky integrate-and-fire mechanism includes only

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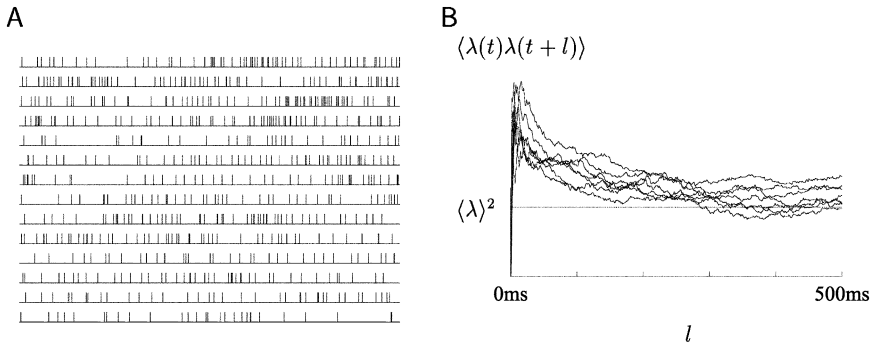

Fig. 1. (A) A set of spike sequences of trials classified by a neuron and a cue, for statistical analyses in this paper. (B) Spike auto-correlograms estimated from a neuron for classified sets by cues.

one dimensional linear integration, but biological neurons have higher dimensional nonlinear integration mechanisms (Hodgkin & Huxley, 1952; Koch, 1999). So, there still remains the possibility that the neuron-received uncorrelated inputs can reproduce the temporal correlation observed in the biological spike sequence. In this paper, we examine the necessity of temporally correlated inputs under only the assumption that the integrated variables are reset by every spike event.

The existence of temporally correlated inputs seems to imply the existence of temporal codings at the single neuron level. However, if this is so, the neuron should generate a spike sequence due to the temporal pattern of inputs. Accordingly, the spike auto-correlations should exhibit the reflection of neuronal integration. In this paper, we attempt to determine whether spike auto-correlations observed in biological spike sequences exhibit the reflection of neuronal integration reset by its own spikes.

Now, we summarize the working assumptions. It is assumed that the spike event process can be described by two stochastic processes: a spike event occurs due to a stochastic process reset by every spike event and driven by another stochastic process independent of spike events. Under the working assumption, we attempt to determine which process is the main factor of the temporal correlations in the biological spike sequences, the 'spike independent process', or the 'spike reset process'.

This assumption implicitly includes the two further assumptions: the inputs incoming process is independent of its own spikes, and the neuronal integrated variables are reset by its own spikes. In the present formulation, however, all of the neuronal variables need not be reset by spike events. Some of the exceptions can be classified into the inputs, the spike independent part, in the formulation. We provisionally regard the spike independent process as the incoming inputs, and the spike reset process as the neuronal integration.

2. Biological spiking data and auto-correlations

In this paper, we analyze delay period activities of cortical neurons in a delay response task experiment by Funahashi and co-workers, whose task paradigm is identical to one of the varieties in Funahashi, Bruce and Goldman-Rakic (1989); Goldman-Rakic, Bruce and Funahashi (1990). The detail of the experiment is shown in Shinomoto et al. (1999).

In the experiment, a monkey is required to preserve a visual cue information presented in advance during a 3 s delay period. Iterating the experiment, the spiking data were obtained from a total of 233 neurons in the prefrontal cortices of three monkeys. We use only the middle 2 s in the delay period of 3 s in order to avoid the possible initial and final transient changes. The 2 s spike sequences are classified according to the cues and the neurons, and 1864 sets (233 neurons × eight cues) of spike sequences are obtained. For reliable statistical analyses, we adopt only the sets including more than 100 spikes. The data sets containing more than 100 spikes are 666 of 1864. A set of classified spike sequences corresponding to a cue and a neuron is shown in Fig. 1A. Every sequence length, L, is equal to 2 s. The number of sequences, N, varies by different cues, (six–25 sequences), because cues are selected randomly trial by trial. In this paper, we do the statistical operations for such a set of spike sequences.

We can see in Fig. 1A that the spike sequences are highly irregular. Most of the others also exhibit high irregularity, as well as this example (Fig. 1A), but many of them differ from an entirely random sequence (Poisson process) in that they have temporal correlation. Auto-correlation is often used to see the temporal correlation in time sequences. Spike auto-correlation is defined as a function of lag l, $\langle\lambda(t)\lambda(t+1)\rangle$, where $\lambda(t)$ is the firing rate defined as the probability of a spike event per infinitesimal unit time at time t, and the notation $\langle\cdots\rangle$ represents a temporal averaging operation: $\langle f(t)\rangle = \lim_{t\to\infty}(1/t)\int_0^t f(t)\mathrm{d}t$. It can be estimated from finite spiking data as the 'spike auto-correlogram', which is defined as the frequency histogram of events that spikes are simultaneously in two time windows separated by lag l. If the spike sequence has no temporal correlation (Poisson process), then the auto-correlation is equal to the square of the mean firing rate, $\langle\lambda(t)\lambda(t+l)\rangle = \langle\lambda\rangle^2$. We estimate the auto-correlograms from the biological spiking data. Some

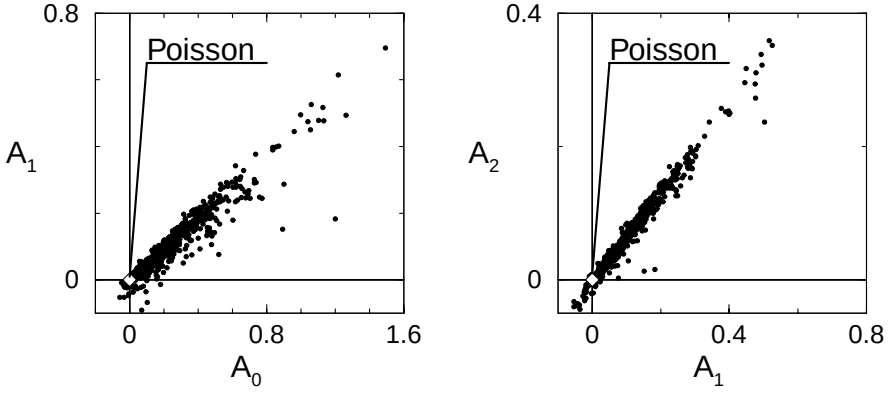

Fig. 2. Normalized moments of auto-correlations, (A_0, A_1, A_2), estimated from the biological spiking data. A Poisson process gives $(A_0, A_1, A_2) = (0, 0, 0)$. Auto-correlations of most of the data have positive moments.

examples are shown in Fig. 1B. We can see in Fig. 1B that the biological spike sequences differ from the Poisson process and that they exhibit positive correlations (larger than $\langle\lambda\rangle^2$).

To see about the other data, we define several coefficients to characterize spike auto-correlation. The auto-correlogram itself largely depends on the width of histogram time bin. Accordingly, the estimation of an auto-correlogram contains arbitrariness of the analyzer. So, we define cumulative quantities of auto-correlation with no need of time bin,

$$A_k \equiv \int_0^\infty l^k \left(\frac{\langle\lambda(t)\lambda(t+l)\rangle}{\langle\lambda\rangle^2} - 1 \right) \mathrm{d}l.$$

Call the A_k the normalized k-th moment of auto-correlation. The correlation is not considered to last infinitely, so each of the moments $\{A_k\}$ has finite value. When a set of N sequences with length L is given, the moments $\{A_k\}$ are estimated without use of the explicit auto-correlogram or histogram bin as follows,

$$A_k \approx \int_0^M l^k \left(\frac{\langle\lambda(t)\lambda(t+l)\rangle}{\langle\lambda\rangle(\langle\lambda\rangle - 1/L)} - 1 \right) \mathrm{d}l$$

$$= \sum_{0<l_{ij}<M} \frac{l_{ij}^k}{\langle\lambda\rangle(\langle\lambda\rangle - 1/L)(L - l_{ij})} - M,$$

where l_{ij} represents a time lag from the i-th spike to the j-th spike, $l_{ij} = t_j - t_i$. The M represents the integral range. Because the sequences have finite length L, the integral range is required to be also finite, $M \leq L$. The factor $\langle\lambda\rangle(\langle\lambda\rangle - 1/L)$ corresponds to the normalizing factor, which is revised from $\langle\lambda\rangle^2$ for the sake of unbiased estimation. The factor $(L - l_{ij})$ corresponds to the range of averaging time at lag l_{ij}. In the prepared data set, the sequence length L of each trial is equal to 2 s, and we set the integral length M as 1 s ($L = 2$, $M = 1$). The number of trials, N, varies with each data set.

The k-th moment A_k has the dimension of k-th power of time. If all of the $\{A_k\}$ are equal to zero, then the sequence has no correlation, which corresponds to a random spike sequence (Poisson process). If each A_k has positive value, then the ratio A_{k+1}/A_k characterizes the sustaining time scale of positive correlation. In the case of exponential correlation, $\langle\lambda(t)\lambda(t+l)\rangle = (\lambda)^2 \propto exp(-l/s)$, the ratio A_{k+1}/A_k is exactly equal to the correlation time scale, s.

The values of (A_0, A_1, A_2) estimated from the 666 sets of biological spiking data are plotted in Fig. 2. The (A_0, A_1, A_2) values are positive at most data sets (92.2%: 614 out of 666). It shows that the biological spike sequences have positive temporal correlations. It is found by simulations of the Poisson process that the differences of the negative A_0, A_1 or A_2 values from 0 of the Poisson process are not statistically significant ($P > 13\%$). So, we can say that all of the data have non-negative auto-correlations. In this paper, we attempt to determine where the positive auto-correlations are generated.

3. Process to a spike event

The process to a spike event is described by two processes: the incoming of input signals to the cell body, and the temporal integration of the inputs in the neuron. The incoming inputs can be described by a stochastic process, which reflects the ensemble activity of the presynaptic neurons. The inputs are integrated in the neuron as the membrane potential, membrane properties, ion densities, and so on. An action potential (spike) is generated at a condition of the integrated quantities. Accordingly, a spike event is described by a stochastic process. Since Gerstein and Mandelbrot (1964), there have been many studies concerning stochastic spiking processes (Inoue, Sato & Ricciardi, 1995; Lánský & Radil, 1987; Ricciardi & Sato, 1988; Tuckwell, 1988;).

A cortical neuron has thousands of synaptic contacts (Braitenberg & Schuz, 1991; Peters, 1987). So, its own spike has little influence on the incoming inputs, even if

the network has recurrent connections. Here, the input incoming process is assumed to be independent from a spike event. On the other hand, the intra-cellular quantities are largely influenced by a spike event. An action potential resets most of the quantities integrated in the neuron. In the present paper, we assume that the integrated quantities are all reset at a spike event. It may be possible that some quantities are not reset by spikes. For example, the dynamics of $[Ca^{2+}]$ and Ca-dependent channels can have long time scales and not be reset by an action potential. However, if the quantities have only little dependencies on its own spikes, such a type of dynamics can be classified into the part of 'incoming inputs' in the present formulation. Now, the stochastic spike event process is described by two processes: a spike event occurs due to a stochastic process reset by every spike event and driven by another stochastic process independent from spike events. In this paper, we attempt to determine which process is the main factor of the temporal correlations in the biological spike sequences.

We provisionally regard the spike independent process as the incoming inputs, and the spike reset process as the neuronal integration.

Assumption 1. The input incoming process is independent from its own spike events.

Assumption 2. The integrated quantities in the neuron are reset at every spike event.

In the case of uncorrelated inputs, temporal correlation in a spike sequence is generated by only the neuronal integration mechanism. The integrated quantities in the neuron are reset at a spike event, so an event after a spike is uncorrelated to any event before the spike. This type of stochastic process is called a 'renewal process'. A renewal process is determined by an interval distribution.

In the case that the instantaneous firing rate does not depend on the integrated quantities in the neuron, and depends on only the inputs incoming at the moment, the spike auto-correlation is proportional to the auto-correlation of incoming inputs. This type of stochastic process is called a 'double stochastic Poisson process'. When the inputs are also uncorrelated, the spike event process corresponds to a Poisson process.

4. Rejection of uncorrelated inputs

The assumption of uncorrelated inputs corresponds to the assumption of renewal processes in the present formulation. In this section, we test whether the renewal processes can reproduce the spiking statistics of the biological data.

Serial correlations of interval sequence are often used to test the hypothesis of renewal process (e.g. Tuckwell, 1988). The correlation coefficient of consecutive intervals, Cor[T], the serial correlation at lag 1, is defined as,

$$\mathrm{Cor}[T] = \frac{\sum_{i}^{n-1} (T_i - \bar{T})(T_{i+1} - \bar{T})}{\sum_{i}^{n-1} (T_i - \bar{T})^2},$$

where T represents an inter-spike interval, and $\{T_1, T_2, \ldots\}$ is the interval sequence. The notation $\bar{\cdots}$ represents an averaging operation through the interval sequence: $\bar{T} \equiv (1/n) \times \sum_{i=1}^{n} T_i$. Renewal processes give $\mathrm{Cor}[T] = 0$.

The coefficient Cor[T] is enhanced by consecutive long intervals relative to the mean interval, and cannot well detect whether short intervals are consecutive. Therefore, we also define another correlation coefficient of inverses, Cor[$1/T$],

$$\mathrm{Cor}[1/T] = \frac{\sum_{i}^{n-1} (1/T_i - \overline{1/T})(1/T_{i+1} - \overline{1/T})}{\sum_{i}^{n-1} (1/T_i - \overline{1/T})^2}.$$

Renewal processes give $\mathrm{Cor}[1/T] = 0$, as well as $\mathrm{Cor}[T] = 0$. The coefficient Cor[$1/T$] is enhanced by consecutive short intervals, so it is available to detect burst-like spike patterns.

The two correlation coefficients estimated from the 666 data sets of spike sequences are plotted in the (Cor[T], Cor[$1/T$]) planes in Fig. 3A. Each dot corresponds to a set of spike sequences classified by a particular neuron and a particular cue position. The coefficient values are widely scattered and some of them exhibit anomalously large positive correlations by means of Cor[T] or Cor[$1/T$], while both correlation coefficients are expected to be zero with the assumption of uncorrelated inputs. Practical experiments, however, do not provide us with infinite length of spike sequence, so we must test whether these serial connections are statistically significant or not.

The standard t-test is often used for the correlation coefficient, but it is based on the assumption of independent normal distributions. The inter-spike interval distributions and its inverse distributions are, however, both far different from a normal distribution. Therefore, the standard t-test is not suitable in the present case. For this reason, we use a random sampling simulation to calculate the sample distributions of Cor[T] and Cor[$1/T$]. About each data set, we obtain a set of sample intervals, $\{T_1, T_2, \ldots, T_n\}$, from a long spike sequence serially linked with 2 s sequences from trial to trial. We select an interval randomly from the interval set $\{T_n\}$, and arrange this sample interval serially. Iterating this operation, a sample spike sequence is obtained. Then, we divide the sequence into trials of 2 s sequences. A sample pair of (Cor[T], Cor[$1/T$]) is obtained by calculating in the same way from a sample set containing the same number of trials. Iterating these operations 10,000 times, we obtained the sample distributions of Cor[T] and Cor[$1/T$]. The significance (or P-value) of a Cor[T] value is defined as the ratio of Cor[T] samples outside of the

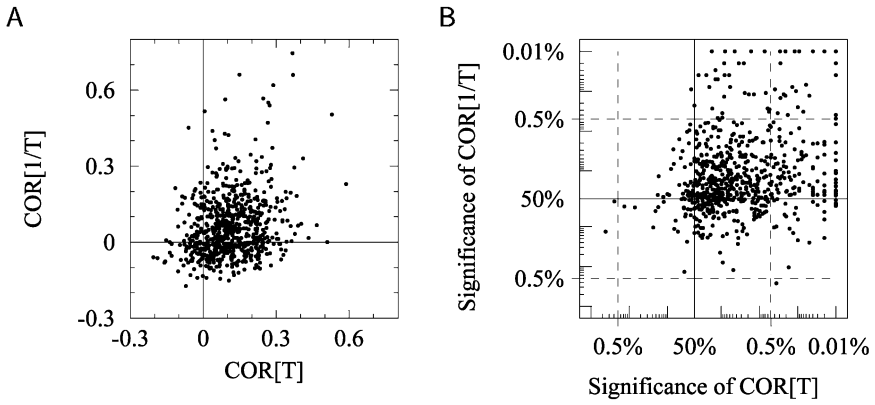

Fig. 3. Rejection of renewal process by two serial correlation coefficients. (A) The two serial correlation coefficients (Cor[T], Cor[1/T]) estimated from the 666 data sets of spike sequences. (B) The significances of Cor[T] and Cor[1/T] calculated by random sampling simulations under the null hypothesis of renewal process. Dots corresponding to the 666 data sets are plotted on log-scale plane. The upper side corresponds to Cor[1/T] > 0, the lower side to Cor[T] < 0, the right hand to Cor[T] > 0, and the left hand to Cor[T] < 0. The four dashed lines represent 0.5% one-sided significance lines on both sides of the two coefficients.

Cor[T] value. The pairs of Cor[T]-significance and Cor[1/T]-significance are plotted as dots on log-scale plane in Fig. 3B. Four dashed lines represent 0.5% one-sided significance lines on both sides of the two coefficients. Many dots are outside of the dashed lines on the positive sides. Dots outside of the dashed line are rejected at the 0.5% significance level. The ratio of data sets rejected at the 0.5% one-sided significance level is, respectively, 22.8% (152/666) on the side of positive Cor[1/T] (ratio of dots on the right side of the vertical dashed line), 9.16% (61/666) on the side of positive Cor[1/T] (ratio of dots on the upper side of the horizontal dashed line), 0.30% (2/666) in the side of negative Cor[T] (ratio of dots on the left side of the vertical dashed line), and 0.15% (1/666) on the side of negative Cor[1/T] (ratio of dots on the lower side of the horizontal dashed line). The ratio of rejected data sets on the side of negative correlation is always below any significance level in the range 0–5%. Thus, negative values are not significant in the range of statistical fluctuation. Positive sides are clearly significant, and the ratio of data sets rejected at the 0.5% level by Cor[T] or Cor[1/T] is 27.4% (183/666) (ratio of dots on the region of left or upper side of the dashed lines).

The results lead to the conclusion that the assumption of renewal process is rejected. The spike independent part of spike event processes needs to have positive temporal correlation to reproduce the biological spiking data statistically. It suggests that the incoming inputs need to have temporal correlation.

The inconsistency is mainly due not to Cor[1/T], but to Cor[T]. It suggests the necessity of long time scale correlation relative to the mean interval, and it also suggests that the inconsistency does not originate in bursting neurons.

5. Effects of neuronal integration

It is found that the incoming inputs need to have temporal correlation in order to reproduce the temporal correlations observed in the biological spike sequences. In the present section, we examine whether the neuronal integrations have an effect on the temporal correlations observed in the biological spike sequences.

When the spike auto-correlation is generated by only the neuronal integration, namely in the case of a renewal process, there is one-to-one correspondence between the inter-spike interval distribution and the spike auto-correlation. The auto-correlation is described as an infinite series of convolutions of the interval distribution. The exponential distribution leads to an uncorrelated spike sequence (Poisson process, dashed lines in Fig. 4), a distribution biased to short and long intervals leads to a positive correlation decaying to zero (Fig. 4A), a distribution biased to a little shorter than the mean interval leads to a negative correlation decaying to zero (Fig. 4B), and a normal-like distribution leads to an oscillatory auto-correlation. Some examples of the correspondence are shown in Fig. 4. Renewal processes can generate any type of auto-correlations, but there is a restriction in the relationship with the interval distribution. Therefore, the characteristic time scales of auto-correlations are proportional to the mean intervals, as long as the shapes of the interval distributions are the same.

Property (by neuronal integration). The characteristic time scales of auto-correlation, A_k/A_{k-1}, are proportional to the mean inter-spike intervals, $\bar{T}$

$$A_k/A_{k+1} \propto \bar{T}.$$

With the temporally correlated inputs, the spike auto-correlation exhibits a compound reflection of the input correlation and the integration mechanism. It is not easy to separate them from each other, but the time scales of auto-correlation must have a positive correlation to the mean intervals, as long as the shapes of interval distributions are similar. To see properties of distribution shapes, we estimate statistical

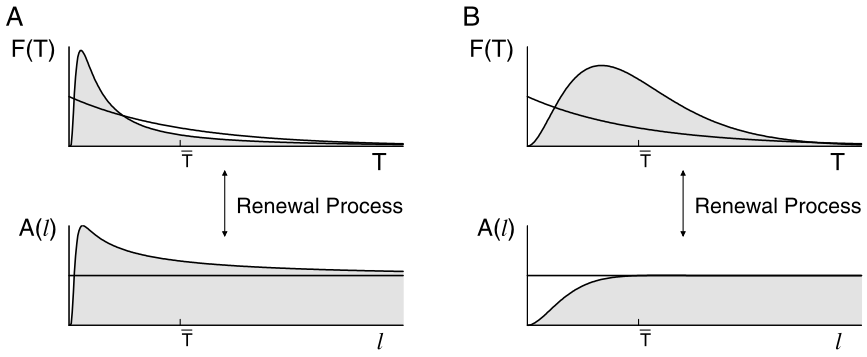

Fig. 4. One-to-one correspondence between the interval distribution $F(T)$ (top plots) and the auto-correlation $A(l)$ (bottom plots) of renewal process. Dashed lines represent uncorrelated case: Poisson process. (A) A distribution biased to short and long interval (inverse Gaussian distribution) leads to a positive correlation at short lag, and decaying to zero as the lag l get larger. (B) A distribution biased to slightly shorter than the mean interval (Gamma distribution of degree 2) leads to a negative correlation at short lag, and decaying to zero.

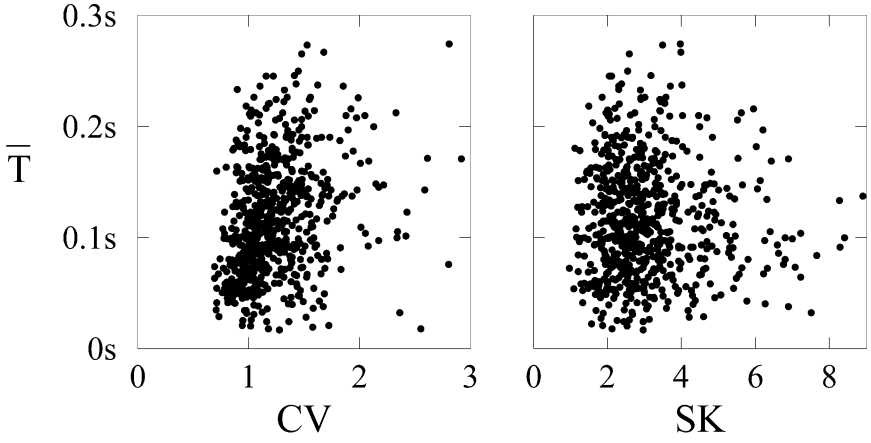

Fig. 5. Relationships between the mean interval and the shape of interval distribution. Statistical coefficients of intervals characterize the shape of interval distribution. The coefficient of variation (CV), and the skewness coefficient (SK) have no systematic relationships to the mean interval ($\bar{T}$).

coefficients of inter-spike intervals: the coefficient of variation (CV), the skewness coefficient (SK), defined as:

$$\mathrm{CV} = \frac{\sqrt{\overline{(T-\bar{T})^2}}}{\bar{T}},$$

$$\mathrm{SK} = \frac{\overline{(T-\bar{T})^3}}{\sqrt{\overline{(T-\bar{T})^2}^3}}.$$

The coefficients are dimensionless and represent properties of the distribution shape independently from its time scale. Fig. 5 shows the relations to the mean intervals, (CV, $\bar{T}$) and (SK, $\bar{T}$), estimated from the biological spiking data. We can see no systematic relationships between the mean interval and CV or SK. It suggests that the shape of the interval distribution is independent from the mean interval. Therefore, if the neuronal integration mechanisms have effects on the spike auto-correlations in the majority of the neurons, then the auto-correlation time scales must have a positive correlation to the mean intervals.

The auto-correlation time scale is estimated as the ratio of the normalized moments of auto-correlation, A_{k+1}/A_k, if A_{k+1} and A_k are positive. Most of the data sets have positive A_0, A_1, A_2 values (92.2%: 614/666, in Section 2). We examine only the 614 data sets. Then, the sustaining time scales of these correlations can be estimated by A_1/A_0 or A_2/A_1 from the 614 data sets. The relation between the auto-correlogram time scales and the mean intervals is shown in Fig. 6. We can see no positive correlation in either: $(\bar{T}, A_1/A_0)$ or $(\bar{T}, A_2/A_1)$. It is found that the biological spike auto-correlations do not exhibit the reflection of the integration reset by spikes, at least in the majority of the neurons. It suggests that the neuronal integration mechanism has little effect on the output spike sequences.

6. Discussion

We analyze the spiking data recorded from neurons in the prefrontal cortex of a monkey preserving a cue information in the delay response task experiment. The spike sequences exhibit temporal correlations. If spike events occur due to a stochastic process reset by every spike event and driven by

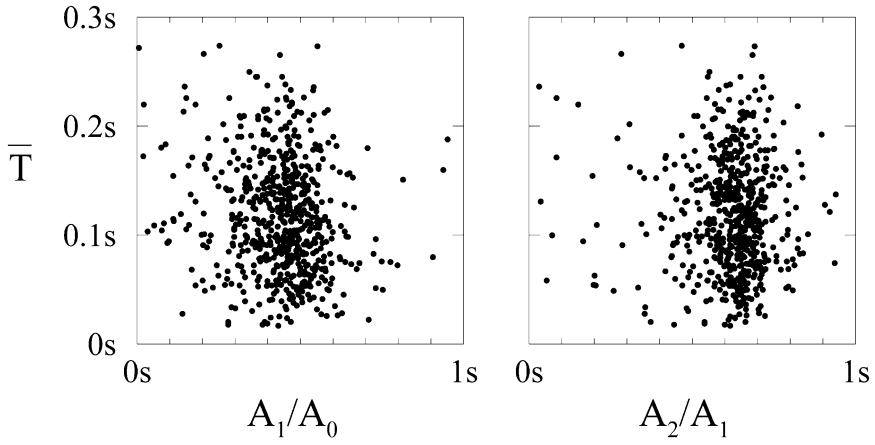

Fig. 6. Relationships between the auto-correlation time scale and the mean interval. Ratios of normalized moments of auto-correlation, A_{k+1}/A_k, characterize the time scale of the auto-correlation. The ratios A_1/A_0 and A_2/A_1 have no positive correlations to the mean intervals.

another stochastic process independent from spike events, the spike independent process is found to be temporally correlated, and the spike reset process is found to have little effect on the spike auto-correlation. We can see in Fig. 6 that the correlation time scales are of the order of 100 ms, so the spike independent process needs to have temporal correlations of the order of 100 ms. In the present paper, we let the two processes correspond, respectively, to the incoming inputs and the neuronal integration mechanism, because its own spikes are considered to have little effect on the incoming inputs, and reset most of the integrated quantities in the neuron. So, it is suggested that the incoming inputs need to have temporal correlations of the order of 100 ms, and that the neuronal integration mechanisms have little effect on the spike sequences.

However, there can be an exception. For example, the dynamics of $[Ca^{2+}]$ or Ca-dependent channels can have long time scales and not be reset by an action potential. Metabolic phenomena also have long time scale and are not reset by an action potential. In the present formulation, however, they can be classified into the part of 'incoming inputs'. The present analysis gives a suggestion that temporal correlation of the order of 100 ms must be in quantities that are not reset by a spike. If there are dynamics influenced, but not reset, by an action potential, it is not easy to divide the effects. The effects of this type of dynamics on the present analysis can be partly examined by simulations of realistic neuron models with multiple channels. We have to examine the consistency of the present analysis by the realistic neuron model simulations in the future.

Temporal correlation in a spike sequence is suggested to reflect the temporal correlation in the incoming inputs. It suggests that no temporal operations due to temporal signal patterns are performed at least in the single neuron level, while a single neuron has the ability of performing complex temporal operations (Matsumoto, Aihara, Hanyu, Takahashi, Yoshizawa & Nagumo, 1987). If the temporal integration in a neuron really has little functional means, it is possible to regard a cortical neuron as a unit to perform statistical operations through the presynaptic neurons at each instance rather than temporal operations on the history of synaptic inputs. This type of unit shares a common mechanism with the 'coincidence detector' advanced by Abeles (1982, 1991), and others (Fujii, Ito, Aihara, Ichinose & Tsukada, 1996; Koch, 1997; Softky & Koch, 1993; Watanabe, Aihara & Kondo, 1998). The temporal integration is neglected in both, but the term 'coincidence detector' has the additional means of the role to detect a specific temporal pattern of coincidently incoming inputs. It implies the temporal coding represented by the ensemble of neurons. It is an open problem whether such a type of temporal coding is used in a cortex.

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