DISTRIBUTION CODING IN THE VISUAL PATHWAY

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ABSTRACT Although a variety of types of spike interval histograms have been reported, little attention has been given to the spike interval distribution as a neural code and to how different distributions are transmitted through neural networks. In this paper we present experimental results showing spike interval histograms recorded from retinal ganglion cells of the cat. These results exhibit a clear correlation between spike interval distribution and stimulus condition at the retinal ganglion cell level. The averaged mean rates of the cells studied were nearly the same in light as in darkness whereas the spike interval histograms were much more regular in light than in darkness. We present theoretical models which illustrate how such a distribution coding at the retinal level could be "interpreted" or recorded at some higher level of the nervous system such as the lateral geniculate nucleus. Interpretation is an essential requirement of a neural code which has often been overlooked in modeling studies. Analytical expressions are derived describing the role of distribution coding in determining the transfer characteristics of a simple interaction model and of a lateral inhibition network. Our work suggests that distribution coding might be interpreted by simply interconnected neural networks such as relay cell networks, in general, and the primary thalamic sensory nuclei in particular.

INTRODUCTION

The detection and recording of neuronal spike trains by both intra- and extracellular microelectrode techniques has become an important branch of neurophysiological investigation. Although these methods have been used extensively for 20 years, the interpretation of spike trains as carriers of information is not at all clear. Even in the same organism many different types of spike trains can be found: very random or very regular, high mean rate or low mean rate, constant in time or time varying. It is apparent that as with the electrophysiological recording of gross potentials such as the electroencephalogram (EEG) or the electroretinogram (ERG), the interpretation of neuronal "signals" is a task not easily amenable to the classical methods of communication theory.

Both sensory perception and motor commands are represented internally in the nervous system of living creatures in the form of neural codes. Such codes are the vehicles for the processing of information and determine what we know as behavior. It is now recognized that both graded (continuous) activity and spike (all-or-none) activity are important to neural coding mechanisms. Perkel and Bullock (1968) described a large number of neural coding possibilities. They concluded that many different types of codes are present in the nervous system of different creatures and that many codes may be active simultaneously in a single neural system.

In this paper we discuss the role of regularity or randomness in the temporal firing pattern of retinal ganglion cells as an information coding parameter in the visual system. In order to quantitatively examine the regularity of firing of these cells, we computed the interspike interval histograms under uniform illumination and under uniform darkness. If we represent a spike train mathematically as a stochastic point process, then the interval histogram may be considered as the statistical estimator of the probability density function of interspike intervals. A regular process has a sharply peaked probability density function, while a random process has a nearly exponential probability density function. Our experimental results suggest that some retinal ganglion cells fire much more regularly in the light than in the dark, while the mean rates remain relatively constant. We refer to the different interval distributions in the light and in the dark as *distribution coding.*

Perkel and Bullock (1968) suggest four formal properties for a neural code: (*a*) the referent, (*b*) the transformation process, (*c*) the transmission process, and (*d*) the interpretation process. In the visual system, the pattern of light stimulation incident on the retina is the *referent* for retinal coding. The *transformation,* or encoding process, occurs in the retina itself. The retina is a complex receptor assembly with horizontal and vertical interconnections by means of the horizontal, bipolar, amacrine, and ganglion cells. The physical referent is transformed into an internal representation in the nervous system, in this case, a representation in the firing patterns of retinal ganglion cells. The *transmission* process carries the coded information toward the brain along the optic nerve and tract. Our experimental results suggest that the coherence of regular distributions may be maintained in transversing the optic nerve.

The neural code which has been transmitted must be interpreted at the next level. In more general terms, it must somehow alter the activity of the next level of neural structure. Perkel and Bullock (1968) speak of the "recoding" of information at the next level. In the visual system, the first recoding takes place at several diencephalic and mesencephalic nuclei. Prominent among these is the lateral geniculate nucleus, the primary thalamic sensory nucleus of the visual system, which serves as an interface between information from the retina and information going to the primary visual cortex. The primary thalamic sensory nuclei are structurally and functionally quite similar in the visual, auditory, and somatosensory systems. They prepare sensory information for the well-defined cortical displays outlined by Hubel and

Wiesel (1963) in the visual system, by Whitfield and Evans (1965) in the auditory system, and by Werner and Whitsel (1968) in the somatosensory system.

In this paper we pursue mechanisms for the "interpretation" of retinal distribution coding by examining models for the simple interaction of excitatory and inhibitory spike trains. In order for distribution coding to be interpreted or recoded, it must affect the activity of the network to which it is transmitted. The analysis of simple interaction models and the lateral inhibition network model in particular shows that this is indeed possible. The mean rate outputs of these network models depend explicitly on both the input mean rates and the input distributions. This result implies that distribution coding could be "recoded" in a network involving simple interactions of incoming spike trains. The theoretical analysis suggests that the distribution coding which we observe in the output of retinal ganglion cells might have functional importance for a relatively simply interconnected structure such as the lateral geniculate nucleus.

METHODS

Cats, weighing 2–5 kg, were used for this investigation. They were initially anesthetized with 5,5-diallylbarbituric acid (Dial), 40 mg/kg, for experiments on the retina and with sodium pentobarbitone (Sagatal [May & Baker Ltd., Dagenham, England]), 40 mg/kg, for the experiments on the lateral geniculate nucleus, injected intraperitoneally together with atropine sulfate, 1 mg/cat. After the operation, anaesthesia was maintained by means of a continuous intravenous infusion of sodium pentobarbitone at the rate of 5 mg/h. Initial paralysis of the the cat was accomplished by intravenous injection of 80 mg gallamine triethiodide (Flaxedil, May & Baker). Complete paralysis was essential in order to maintain the eyes in a fixed position. The intravenous infusion fluid contained, apart from anesthetic, also Flaxedil delivered at the rate of 28 mg/h. The amount of infusion fluid was 6.5 ml/h.

The cats were artificially ventilated by means of a respiration pump. Stroke volume was set at approximately 20 ml/kg body weight. The respiratory rate was 20/min. The temperature of the cat was maintained by means of an electric heating blanket wrapped around the cat's chest and belly. A thermistor connected to the electric blanket power supply circuit enabled the cat's temperature to be maintained at 38°C automatically. The cat's trunk was supported by a trough and the head was rigidly fixed in a Horsley-Clarke type stereotaxic apparatus specially constructed so as not to obstruct the visual field.

The pupils were fully dilated with 1% atropine sulfate and 2.5% phenylephrine (Neo-Synephrine, Winthrop Laboratories, New York). Phenylephrine obviates the need for mechanical retraction of the nictitating membrane and eyelids. A range of plastic contact lenses having inner radii of curvature 8.0, 8.5, and 9.0 mm with three optical powers (in diopters) (0 *D*, +1 *D*, +2 *D*) for each radius of curvature were used.

In order to record the activity of single retinal ganglion cells in conditions as close to normal as possible, a special micromanipulator was used (Kozak and Stephens, 1961). The general principles of recording are similar to those of Talbot and Kuffler (1952) and Brown and Wiesel (1958). The cat's eye remained unopened, with its dioptric organs intact. The cornea was protected by a plastic contact lens. A small opening was made in the sclera just behind the cilliary body. The micromanipulator tube containing a microelectrode was inserted through this opening into the vitreous body of the eye.

For the lateral geniculate experiments, a hole usually 12 × 10 mm was drilled in the skull

above the lateral geniculate nucleus. A bone wax prosthesis was used to prevent pulsation of the brain and leakage of cerebrospinal fluid (Bishop et al., 1962), enabling a stable recording from single lateral geniculate nucleus cells. Tungsten microelectrodes (Hubel, 1957) passed easily through the wax and their electrical properties remained good after the passage.

RESULTS

Using the methods described above, tungsten wire microelectrodes (Hubel, 1957) were inserted into the unopened cat's eye. Before detecting a single ganglion cell, mass activity was recorded in the dark which could be abolished by illumination of the retina. Due to the complete paralysis of the extraocular muscles achieved by a constant infusion of Flaxedil solution and due to the rigid construction of the micromanipulator assembly, it was possible to record from single units for many hours. The unit spikes were recorded extracellularly judging from their diphasic shape and amplitude of a few millivolts.

It is possible that the mass dark activity which is suppressed by the light, originates from the smallest retinal ganglion cells. These small cells are particularly densely agglomerated in the region of area centralis (Ganser, 1882; Chievitz, 1889; Zurn, 1902) of the cat's retina. They seem to be segregated in the upper portion of the optic tract (Bishop et al., 1953; Bishop and Clare, 1955; Lennox, 1958). On the other hand, we know that the macular bundle of fibers constitutes the afferent pathway of the reflex pupillary constriction to light (Hess and Groethuysen, 1929). We can assume, then, that the unresolved mass activity is mainly the activity of the small cells and fibers and that it is influenced by light in a different manner than the activity of single large ganglion cells. Thus, the population of single units investigated in our present study is likely to be mainly the large cell population and we shall discuss the distribution of intervals between spikes recorded from these single retinal units.

In the present study we were interested in a quantitative analysis of the spike activity of single retinal ganglion cells in the steady state of darkness and moderate illumination (1–7 cd/m^2). The continually maintained activity of these cells could easily be altered by a sudden change in the level of general illumination or by an object moved across their receptive field. The spike potentials recorded from a retinal neuron by a microelectrode were first amplified and then fed into a Schmitt trigger circuit which generated a brief standard pulse for each spike potential. The standard pulses were recorded on a magnetic tape and after each experiment they were fed into an electronic analyzer (RCL 256 channel scaler [Radiation Counter Laboratories Inc., Skokie, Ill.]; Levick, 1962), which was used for analysis of the distribution of spike intervals in the steady state of light and darkness. The spike mean rate was continuously recorded during the experiment, and histograms were computed only for cases where the mean rate was constant.

When recording with a microelectrode extracellularly from the nervous tissue we can usually pick up high amplitude spike discharges from a single unit close to the microelectrode tip and, simultaneously, lower amplitude spikes from more remote units. For the

analysis of single unit activity it is important that only the large amplitude spikes are permitted to generate standard pulses and the small ones are prevented from doing so. Moreover, we must make sure that every large spike generates a pulse. The fulfillment of these two conditions is essential for the train of standard pulses to correspond exactly to the train of spike potentials of a single neuron. In order to secure a reliable triggering, the unit spike potential was continuously monitored on a cathode ray oscilloscope screen in the synchroscopic way. The spike potential itself, after a suitable amplification with a short (about 10 ms) decay time constant, was used to trigger the oscilloscope time base, so that every spike potential, small or large, could be seen on the screen. Simultaneously, the standard pulses from the Schmitt trigger were displayed on the second oscilloscope beam. The standard pulse-triggering level was adjusted so that the pulse appeared every time, and only when, the large spike potential occurred. The triggering level was watched continuously during the recording lasting many hours and as soon as the recording conditions changed, the level was readjusted. We found that for a reliable triggering the record must be free of mains frequency pickup, the large spike amplitude must exceed at least twice the amplitude of the next smaller spike and that the spike amplitude must exceed at least three times the background noise level. When these requirements were not fulfilled, the triggering became unreliable and the record was discarded. In the case of a reliable triggering, the standard pulses were fed into a mean rate device which recorded the mean frequency of standard pulses with the integration time constant of 0.5 s on a continuously moving paper tape. The interval analysis was made only during such periods of the cell activity when the mean rate of spike discharges remained constant or varied only slightly. This condition is essential, because the interval histograms are meaningful only during the steady state of cell activity (Rodieck et al., 1962).

Fig. 1 *a* shows typical interval histograms of spike trains from two ganglion cells in the dark. On the left-hand graph the most common (modal) interval length was 8 ms. The shorter and longer intervals were less common. The distribution of intervals longer than 8 ms was particularly interesting in that the tail of the interval histogram was to a good approximation exponential. A straight line could be easily fitted to the envelope of the histogram plotted on semilogarithmic paper. Small up and down deviations are statistically not significant. Their amplitudes are relatively greater for lower count numbers conforming with the law of great numbers, the deviation being equal to the square root of the number of counts.

Another example of an interval histogram of dark firing of a retinal ganglion cell is shown on the right in Fig. 1 *a*. Here, the modal interval has the length of about 32 ms. The dead time equals about 4 ms and the distribution of intervals shorter than 32 ms follows a curved line on the semilogarithmic plot. The statistically significant deficit of the shortest intervals visible on both graphs suggests that the common cause is a dead time associated with the refractory period after every impulse in the ganglion cell. The shapes of interval histograms of other ganglion cells in darkness were usually intermediate between the extreme cases shown in Fig. 1 *a*. Fig. 1 *b* shows typical interval histograms of spike trains of retinal ganglion cells in the light. They show the characteristic multimodal distributions discussed by Kozak et al. (1962, 1968) and Bishop et al. (1964). The characteristic feature of the ganglion cell interval distribution in light is its regularity, expressed by the presence of highly

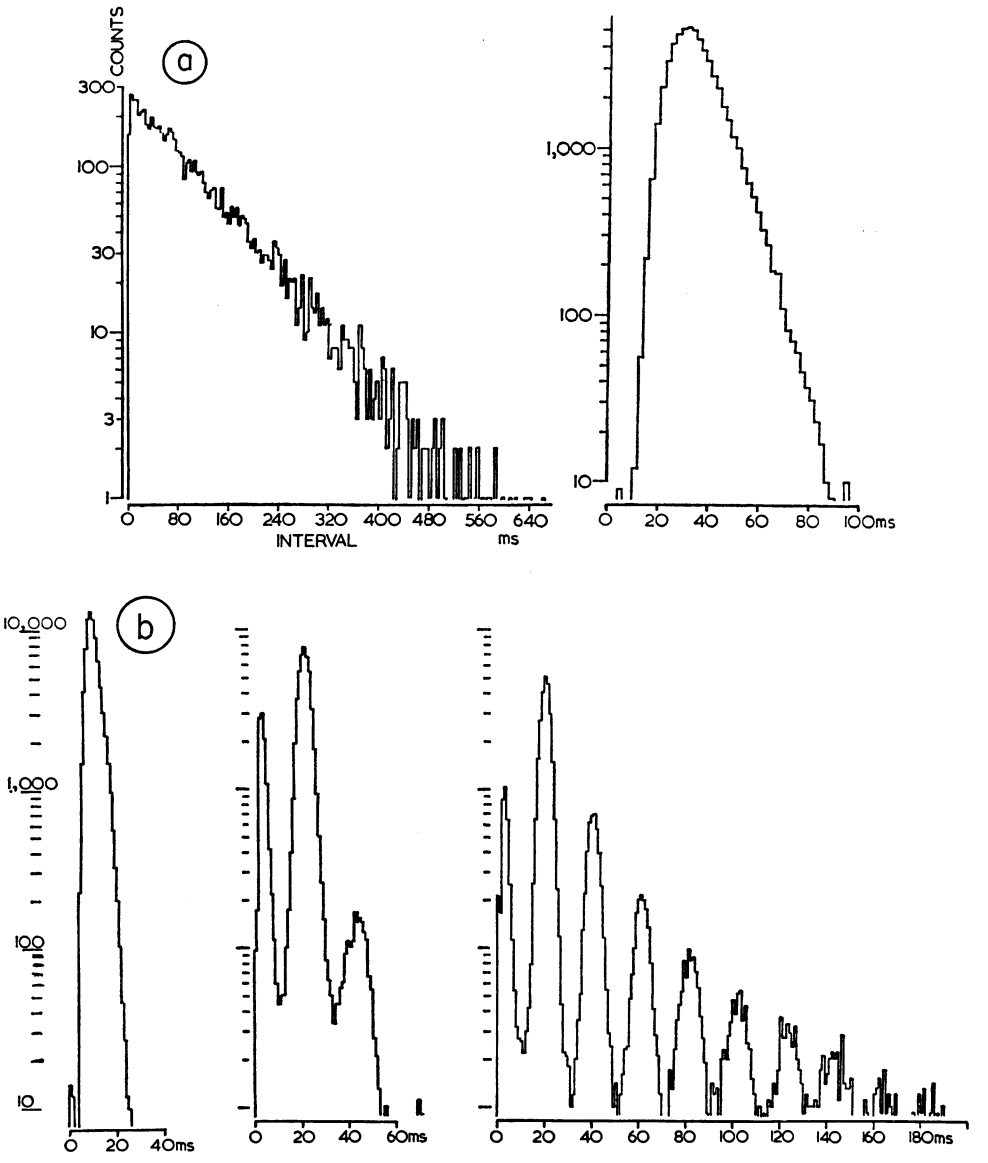

FIGURE 1 Spike interval histograms recorded from cat retinal ganglion cells. The spike interval histogram plots the number of times which an interval is observed vs. the length of that interval and, therefore, serves as a statistical estimator of the probability distribution of intervals. *a* shows histograms recorded from retinal ganglion cells in steady darkness In the left-hand figure the most common (modal) interval length is 8 ms. The form of the distribution is approximately exponential. The modal interval length of the distribution on the right is about 32 ms. *b* shows histograms recorded in steady illumination (1–7 cd/m^2). The distributions observed in the light are extremely regular or multimodal. The presence of multiple peaks in the interval histograms can be explained by a mechanism of random deletion from a regular train of impulses. Vertical scales are logarithmic.

preferred ranges of intervals. Within a preferred range of intervals, i.e. within one peak of the histogram, the standard deviation is very small and the distribution resembles a gaussian one. The presence of multiple peaks in the interval histograms in light can be explained by a process of random deletions from a regular train of impulses. Such a model does not, however, produce the first group of shortest intervals. It seems reasonable that the peak of shortest intervals (about 3 ms), corresponds to a multiple (burst) firing of the unit, and not to the main train, which is represented by the second peak of the histogram occurring around 20 ms.

Fig. 2 summarizes the properties of the distributions for histograms recorded in the light and in the dark from retinal ganglion cells. This summary emphasizes the result that more regular, multimodal distributions are associated with illumination,

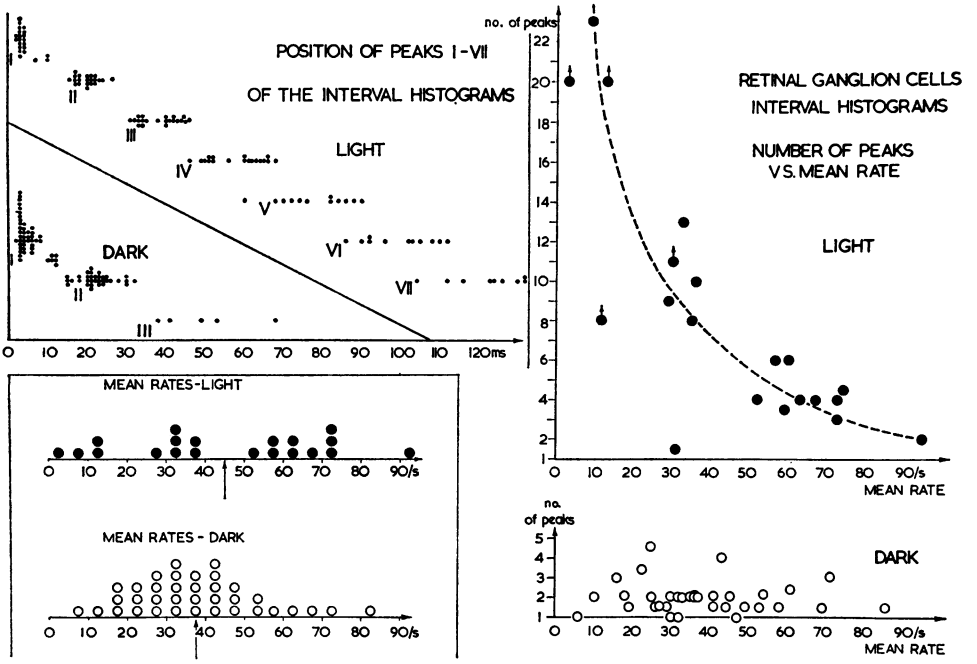

FIGURE 2 Summary of the properties of spike activity of the retinal ganglion cells studied Upper left: preferred intervals in the spike trains of different cells in light and in darkness. The occurrence of the first peak, second peak, etc. is marked with roman numerals. Every dot corresponds to a peak, or mode, in the interval histogram. Two graphs on the right: the number of peaks of interval histograms vs. the mean rate of spike firing. The black dots with an arrow indicate that there were probably more peaks, though they were not recorded for technical reasons. The dotted line is drawn to fit the black dots. There is a reciprocal relationship between the mean rate and the number of peaks in the light. The corresponding dark activity shows no such correlation. Two graphs on the lower left: distribution of the mean rates of spike firing of different cells in light and darkness. Note the clustering of rates in the light and the broad distribution of rates in the dark. The arrows indicate the average mean rates for the two conditions. The average mean rate in the light is only slightly higher than in the dark.

while more random exponential distributions are associated with dark activity. There is a distinct inverse correlation between the number of peaks of the distribution in the light and the mean rate. The decrease of the mean rate of the cell firing is associated with an increase in the number of groups of intervals (in the number of modes). The longer the intervals, the smaller their numbers and the lower the peaks of groups. We took 15 as an arbitrary number of intervals of the same length as a criterion for the existence of a peak, and plotted in Fig. 2 (upper right) the number of recorded peaks on the histogram vs. the mean rate of the spike discharges. The black dots with arrows indicate that there were probably more peaks though they were not recorded because of technical reasons. The dotted line is drawn to fit the black dots. We can see that there is a reciprocal relationship between the mean rate and the number of peaks of the histogram. The corresponding graph for dark activity (Fig. 2, lower right) shows no correlation between the number of peaks and the mean rate. (It is possible that where several peaks were observed in the dark case we were recording from an off-center ganglion cell, that is, one which responds normally to darkness. If this were true, then the fact that, as a rule, we observed more regular and multimodal distributions in response to light suggests that the majority of the cells which were studied were of the on-center type.)

Fig. 2 (lower left) shows the distribution of mean rates of spike activity in light and darkness. The distribution of mean rates in darkness seems to be monomodal with the mode around 37 ms. The mean rates in light seem to be grouped into four groups, around the values of 10, 32, 62, and 92 ms. In Fig. 2 (upper left) the position of peaks of the interval histograms in light and darkness is displayed. The length of intervals is plotted on the abscissa. The designation of a given peak as a first one, second and so on, is marked with roman figures. Above the oblique line the position of peaks in light is shown, and below in darkness. We can notice that the first peak in all units both in light and in darkness is around 3 ms. The second peak in darkness is around 23 ms, the third one about 50 ms, but the third peak is very variable and occurs rarely.

We could not find any significant differences between the mean rates of spike firing of the single retinal ganglion cells in light and in darkness (Fig. 2, lower left). Similarly, Kuffler et al. (1957) have found that the mean rate of spike firing tends to be similar during light and dark periods provided that sufficiently long periods of time are allowed for adaptation. Arduini and Pinneo (1962) summarize this state of affairs by saying that no clear pattern of discharge can be detected in a given unit during either dark or light adaptation. It is likely that the single unit recording, especially with metal microelectrodes, does not give a fair sample of all units present in the retina and the optic nerve. Possibly the microelectrode will pick up the large ganglion cells and the large optic axons rather than the small ones (Rushton, 1949; Wiesel, 1960). On the other hand, the recording of unresolved mass activity from the optic fiber layer of the retina or from the surface of the optic chiasma (Arduini and

Pinneo, 1962; Bornschein, 1958; Lennox, 1958) is likely to be influenced by the activity of the far more numerous small fibers.

Fig. 3 shows interval histograms of spike trains recorded from an optic tract axon in light (A) and in darkness (B). The tip of the recording microelectrode was situated at the posterolateral border of the lateral geniculate nucleus. The character of the distributions illustrate much the same pattern as recorded at the retinal ganglion cell level, multimodal in the light, and nearly random in the dark. This result shows that the marked coherence of the distributions which were recorded at the retinal ganglion cell level is maintained in traversing the optic axon to the lateral geniculate nucleus, rather than becoming dispersed.

The presence of a high peak of preferred intervals close to 20 ms in light (A) arouses natural suspicions, because the mains frequency was 50 Hz which corresponds to a cycle length of 20 ms. It would be sufficient that the power supply of our light source was not properly filtered, in order to produce a 50 or 100 Hz ripple in our light. The mains frequency could affect our recording and analysis in many ways, such as oscillations of the level of triggering of the standard pulses and so on. In order to check the hypothesis of a mains interference, we applied our electronic analyzer to find any possible correlation between the mains frequency and the occurrence of the standard pulses triggered by the neuron's spike potentials. We set the analyzer for the poststimulus time (P.S.T.) mode, so that the analyzer was reset (recycled) by the mains frequency. Such analyses invariably indicated that there

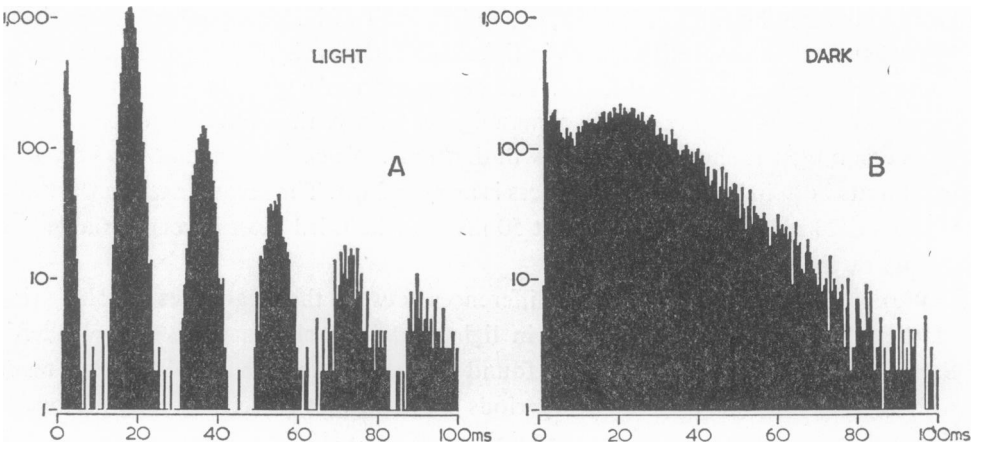

FIGURE 3 Spike interval histograms (15,000 spikes) of spike activity recorded from an optic axon. The site of recording was at the posterolateral border of the lateral geniculate nucleus. (A) Interval histogram of spike train in light. The multimodal character of the distribution is clearly seen. (B) Interval histogram of spike train in the dark. The distribution approaches a random, or exponential, type. A and B show that the character of the interval distributions observed at the retinal ganglion cell level is preserved in traversing the optic axons. In particular, A shows that the discrete nature of the multimodal distribution is not dispersed significantly.

was no correlation between the mains frequency and the frequency of the standard pulses generated by the optic axon in light.

THEORY

Distribution Coding

A single nerve spike is a complex wave form which is initiated at the nerve cell body and which propagates in an all-or-none fashion along the axon of that cell and terminates at a synapse either with another nerve cell or with an effector muscle. A series of such nerve spikes are shown in Fig. 4 *a*. There is considerable neurophysiological evidence to suggest that the information in the spike train is embodied in the time of occurrence of each spike rather than in any properties of the spike wave form. Making this assumption we can abstract the relevant properties of the spike train shown in Fig. 4 *a* by considering it mathematically as a point process as shown in Fig. 4 *b*. The spike train is represented as a series of events in time. In view of the random properties of spike trains we will consider, in general, the neuronal spike train as a stochastic point process.

Perkel et al. (1967 *a*, *b*), among others, have considered in some detail the properties of stochastic point processes relevant to neural coding. The most commonly used neural coding parameter is the mean rate of the process expressed in spikes per second. The mean rate has been used extensively to interpret neurophysiological experiments. Other parameters which have been studied are the serial correlation, the autocorrelation or expectation density, and the probability distribution of interspike intervals (Cox and Lewis, 1966). The use of the spike interval histogram as a statistical estimator of the probability density function of interspike intervals was introduced by Gerstein and Kiang (1960).

Our experimental results have shown that the distribution function for interspike intervals depends on illumination. In fact, as the level of illumination is increased the

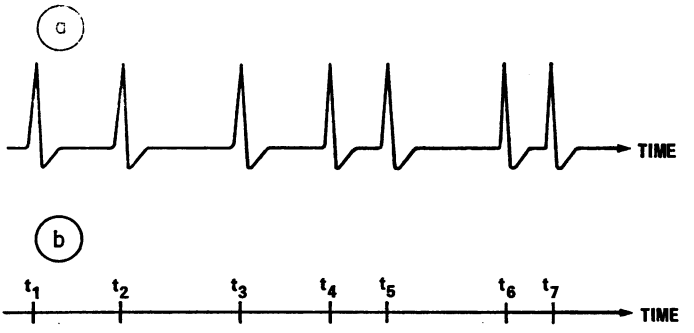

FIGURE 4 (*a*) Series of nerve spikes. (*b*) Mathematical representation of the spike train as a stochastic point process, where t_i indicates the time of occurrence of a spike. The information transmitted by a spike train is thought to be contained in the time of occurrence of a spike rather than in any properties of the complex wave form.

distribution becomes more regular. The theoretical models which are developed in this section will show how inhibitory and excitatory inputs with random or regular distribution interact in single cells and in lateral inhibition networks. We will characterize the regular process with a gamma distribution which is monomodal. These results can be extended in straight-forward manner to handle multimodal regular distributions. While our experimental results indicate that for the cases reported the regularity of the distribution depended on the level of illumination, we have no reason to believe that all cells will behave in this way and would expect the distributions of others to depend on other parameters of the visual stimulus.

Fig. 5 illustrates the use of the probability density function to describe two stochastic point processes considered as mathematical representations of spike trains. The probability density function (pdf) of interspike intervals plots the probability per unit time of an interval occurring vs. the length of that interval. The events of the process in Fig. 5 *a* occur quite randomly in time, approaching a Poisson process. The pdf of that process is nearly exponential. The events of the process in Fig. 5 *b* occur quite regularly, all intervals being nearly equal, and the pdf is sharply peaked around the mean interval. The vertical scales are linear.

These distributions are similar to those recorded from retinal ganglion cells as shown in Fig. 1, with logarithmic vertical scales. As we have described, the distribution of such retinal ganglion cell spike firing changes from exponential (Fig. 1 *a*, left side) to regular (Fig. 1 *b*) as the intensity of uniform illumination of the visual field is increased.

If we propose to consider this quality of randomness or regularity as a specific coding property of the spike train, then it is convenient to describe the pdf's of the

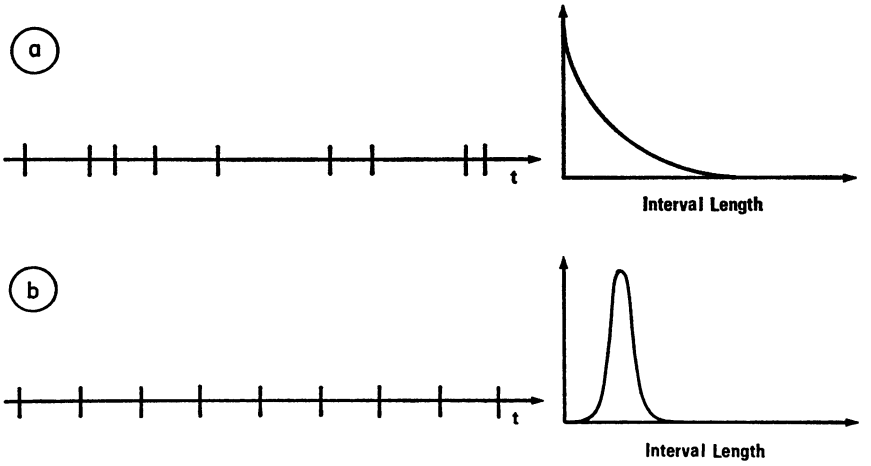

FIGURE 5 The probability density functions of interspike intervals corresponding to (*a*) a random process and (*b*) a narrow gaussian, i.e. fairly regular, process. Vertical scales are linear.

processes functionally as gamma distributions of the form:

$$g(\tau; n, \mu) = \frac{\mu^n \tau^{n-1} e^{-\mu\tau}}{\Gamma(n)}, \qquad \tau \geq 0, \mu > 0, n > 0,$$
$$0 \quad , \qquad \tau < 0 \quad , \tag{1}$$

where $\Gamma(n)$ is the gamma function defined by

$$\Gamma(n) = \int_0^\infty r^{n-1} e^{-r}\, dr, \tag{2}$$

and if n is an integer:

$$\Gamma(n) = (n - 1)! \tag{3}$$

Some typical gamma distributions are plotted in Fig. 6. The mean of the gamma distribution is

$$\bar{\tau} = n/\mu, \tag{4}$$

the variance is

$$v = n/\mu^2, \tag{5}$$

and the mean rate of the process is

$$m = 1/\bar{\tau} = \mu/n \text{ spikes/s}. \tag{6}$$

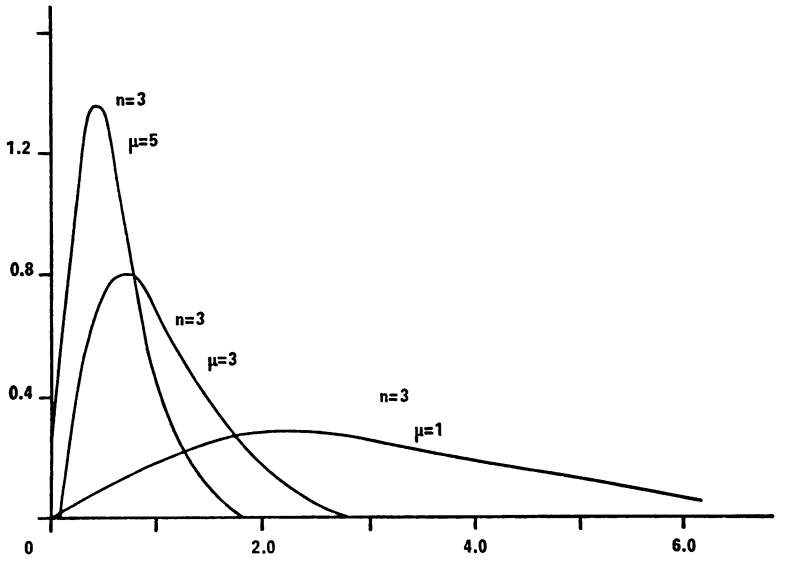

FIGURE 6 Some examples of gamma distributions.

The gamma distribution is a useful representation of regularity or randomness of the process since for $n = 1$:

$$g(\tau; 1, \mu) = \mu e^{-\mu\tau}, \tag{7}$$

the exponential pdf corresponding to a Poisson process of events, a completely *random* process. As $n \to \infty$ at a given mean rate, the gamma distribution approaches a gaussian distribution corresponding to a very *regular* process. Thus, using the gamma distribution as a model for the pdf of neuronal spike trains, we can characterize a given process by two parameters, m, the mean rate parameter, and n, the distribution parameter. The representation in the nervous system of information in terms of mean rate m, we will refer to as *mean rate coding*, and the representation in terms of distribution n, we will refer to as distribution coding.

It should be emphasized that the choice of the gamma distribution to characterize distribution coding (Kuffler et al., 1957) is somewhat arbitrary and not based directly on elucidation of the physiological mechanisms generating spike trains or on optimized curve fitting of recorded histograms. We justify the use of the gamma distribution in a modeling context by noting that variation of a single parameter n describes, at least approximately, a complete range of random to regular processes. Other modes of representation of distribution coding, for example, the expansion of the distribution in terms of gaussian distributions, are certainly of interest, though they will not be discussed here.

Models of Spike Train Interactions

In this section we will review the mathematical description of models of superposition of spike trains, models of inhibitory interaction, or deletion of spike trains, and some modifications of the deletion processes which make them physiologically more reasonable. The goal of the calculations which we will discuss is to determine the output probability density function $p(\tau)$ generated by the interaction of two or more primary input processes. Such models are directly relevant to our discussion of distribution coding in the nervous system since they consider specifically the probability density functions of input and output processes.

The spike trains recorded by Fatt and Katz (1952) were among the earliest electrophysiological experiments to be subjected to careful statistical analysis. In two papers, Cox and Smith (1953, 1954) treated the spontaneous activity at many nerve endings on one muscle fiber as a superposition process. The problem was to infer the spontaneous activity at individual endings from spike activity in the muscle fiber. Cox and Smith computed the variance-time curve for the data and compared the result with models for the superposition of various renewal processes considered as independent, identically distributed random variables. (A renewal process is characterized by a series of events in which the times between events are independently and identically distributed.) The interval distribution of the resulting process is

known to approach exponential for a sufficient number of sources, and the variance-time curve can be invoked to distinguish the component processes.

A more recent group of experiments has suggested the incorporation of impulse deletion to account for observed interval distribution curves. Poggio and Viernstein (1964) measured impulse trains in a thalamic somatic sensory neuron of unanesthetized monkeys. They observed spike interval histograms which were characterized by being distinctly monomodal or multimodal rather than exponential. Bishop et al. (1964) computed interval histograms for spike trains recorded from the dark discharge of lateral geniculate neurons in cats. They described a variety of results ranging from nearly exponential to monomodal, to multimodal distributions, including various combinations of these types. They were able to explain three characteristics of their results by three different mechanisms. They first observed that by the superposition process described by Cox and Smith they could generate approximately exponential distributions from the pooling of many regular processes. Secondly, they suggested that multiple-firing (burst) events could account for the groups of short intervals which appeared. The third process which they described was a random deletion of the monomodally distributed train, yielding a multimodal distribution similar to those measured. They simulated this procedure by first generating a gamma distribution by frequency division (retaining every nth event and deleting the rest) of the output of a Geiger counter and then making random deletions. The resulting distributions appeared quite similar to the experimental observations.

Ten Hoopen and Reuver (1965, 1967, 1968) have considered the types of interactions described by Bishop et al. from a statistical point of view. They derived analytical expressions for the output probability distributions to be expected from specific models of superposition and deletion of interacting stochastic point processes.

Ten Hoopen (1966) considered the superposition of N stationary, ergodic, and independent sequences of events denoted by

$$f_i(t) = \sum_j \delta(t - t_{i,j}), \tag{8}$$

originating from the ith source with probability density function $p_i(\tau)$, and distribution function $P_i(\tau)$. The mean rate m_i of the ith source is given by

$$m_i = (\theta_i)^{-1} = \left[\int_0^\infty \tau' p_i(\tau')\, d\tau'\right]^{-1}. \tag{9}$$

The problem is to calculate the probability density function of the superposition of the N sources, $p(\tau)$, where

$$f(t) = \sum_{i=1}^{N} f_i(t), \tag{10}$$

$$f(t) = \sum_s \delta(t - t_s). \tag{11}$$

Such a superposition of spike trains is shown schematically in Fig. 7.

While, as Ten Hoopen shows, the superposition of spike trains tends to obscure the form of the distributions of independent input processes, approaching a Poisson process in the limit, the deletion of events from an excitatory spike train by an inhibitory spike train shows very specific dependence on input distributions. Ten Hoopen and Reuver (1965) described a model for the interaction of an excitatory spike train and an inhibitory spike train converging on an output neuron (Fig. 8). In their model, an inhibitory spike would always delete the next succeeding excitatory spike. The output of the deletion process was a spike train consisting of the undeleted excitatory events. An illustration of this deletion process is shown in Fig. 8, where $x(t)$ is the excitatory input, $y(t)$ is the inhibitory input, and $z(t)$ is the output.

If we consider that the excitatory process has a pdf of the form $\phi(\tau)$, and the inhibitory process has a pdf of the form $\psi(\tau)$, where both are renewal processes, then the output pdf $p(\tau)$ may be calculated by the methods of renewal theory (Cox, 1962). Ten Hoopen and Reuver (1967) obtained a general expression for the pdf $p(\tau)$ of the output process, while Srinivasan and Rajamannar (1970 *b*) considered the calculation of the higher order moments of the process. The general form of the result for $p(\tau)$ is very complex, and two specific cases were solved first (Ten Hoopen and Reuver, 1965): (*a*) a Poisson process inhibiting a renewal process, and (*b*) a renewal process inhibiting a Poisson process.

Since the work of Ten Hoopen and Reuver (1965, 1967, 1968), a series of papers has appeared describing the statistical analysis of models of inhibitory interaction of stationary point processes in relation to their probability density functions

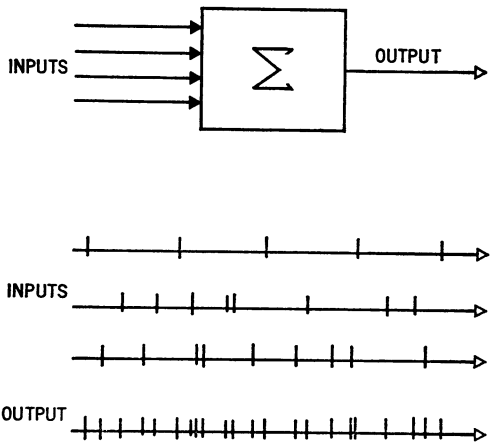

FIGURE 7 A model for the superposition of spike trains.

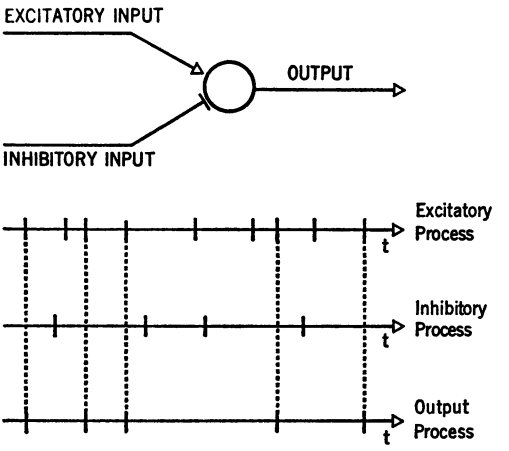

FIGURE 8 A model for the simple interaction of two spike trains in which each inhibitory event deletes the next succeeding excitatory event. The remaining excitatory events form the output process.

(Coleman and Gastwirth, 1969; Lawrence, 1970 *a*, *b*, 1971; Srinivasan and Rajamannar, 1970 *a*, *b*; Srinivasan et al., 1971). Many of these papers presented modifications of the simple interaction model to make it correspond more closely to physiological cases. Srinivasan and Rajamannar (1970 *b*) analyzed the case of dependent interference between excitatory and inhibitory processes as first proposed by Ten Hoopen and Reuver (1968). Coleman and Gastwirth (1969) described a model where the effectiveness of the inhibition decays with time. These modified interaction models show a similar marked dependence on both the mean rate and the distribution of the input process.

The Mean Rate Transfer Function

It is of considerable physiological interest to consider the role of spike train interaction models in relation to neural coding and in the function of neural networks. In order to characterize the processing of mean rate coding by a simple interaction model we consider mean rate coding of the excitatory input m_ϕ and mean rate coding of the output m_p. We can then define the *mean rate transfer function* T of the network as

$$T = m_p/m_\phi , \tag{12}$$

which will depend on both the mean rates and the distributions of the excitatory and inhibitory input processes. In order to specify the effective inhibitory mean rate, we define the *inhibition strength:*

$$\rho = m_\psi/m_\phi . \tag{13}$$

We wish to use the mean rate transfer function $T(\rho)$ to describe functionally the behavior of a network in response to various input mean rates and distributions. We are particularly interested in analyzing how input distribution coding effects the output mean rate of the network and thus inferring the extent to which input distribution coding can be "decoded" by the network. Our use of the gamma distribution to describe randomness or regularity of process will be useful for specifying this distribution coding dependence.

Although it would be possible to calculate the mean rate transfer function relationships for interaction models by using Ten Hoopen and Reuver's (1967) calculations of $p(\tau)$, such an approach yields useful results only for the cases where one of the processes is Poisson. Instead of first calculating the probability density function of the output to obtain the mean rates, we will proceed by calculating the renewal density of the output process and obtain the mean rate transfer function. Cox (1962) defined the random variable N_t of a renewal process as the number of renewals which occur in (0, t), and in general N_{t_1,t_2} is the number of renewals in the interval (t_1, t_2). The "renewal function" is defined as:

$$H(t) = E(N_t) \tag{14}$$

and more generally

$$H(t_1) - H(t_2) = E(N_{t_1,t_2}). \tag{15}$$

The "renewal density" is defined as

$$h(t) = \lim_{\Delta t \to 0+} \frac{E(N_{t,t+\Delta t})}{\Delta t}, \tag{16}$$

$$= H'(t). \tag{17}$$

The renewal density describes the probability of one or more renewal in an interval of length Δt. When the process is not strictly renewal, the more general term "product density" is used for $h(t)$.

Srinivasan and Rajamannar (1970 *a*) have described the product densities of degree one and two for the simple interaction model of Ten Hoopen and Reuver (1965). They observed that the set of registered events can be divided into two mutually exclusive classes depending on whether an inhibitory event occurs between 0 and t or not. Their expression for the product density of degree one is:

$$h(t) = g(t) \int_t^\infty p^i(t')\, dt' + \int_0^t F_1(u)\, du \int_u^t g(v)\phi(t - v\chi)\, dv(t - u), \tag{18}$$

where $g(t)$ is the renewal density of degree one of primary events:

$$g(t) = \phi(t) + \int_0^t g(u)\phi(t - u)\, du, \tag{19}$$

$p^i(\tau)$ is the probability density function of the interval between a registered event and the next inhibitory event:

$$p^i(\tau) = \frac{\int_0^\infty \phi(x)V(t+x)\,dx}{\int_0^\infty \phi(x)\int_x^\infty V(x')\,dx'\,dx}, \tag{20}$$

where $V(\tau)$ is the probability density function of the forward recurrence time of inhibitory events:

$$V(\tau) = \chi(\tau)/E(\tau). \tag{21}$$

where $\chi(\tau)$ is the survivor function of inhibitory events defined by

$$\chi(\tau) = \int_\tau^\infty \psi(u)\,du. \tag{22}$$

$F_1(u)$ is the renewal density of degree one of inhibitory events at u starting with a registered primary event at $u = 0$. $F_1(u)$ is given by the following renewal equation:

$$F_1(u) = p^i(u) + \int_0^u p^i(t')f(u-t')\,dt', \tag{23}$$

where $f(t)$ is the renewal density of degree one of inhibitory events given an inhibitory event at $t = 0$.

In the case of a renewal process, we can calculate the mean rate of the stationary process by taking the limit

$$\lim_{t\to\infty} g(t) = m \tag{24}$$

We can show that this is the same mean rate one would calculate from the probability density function in the following manner. From Eq. 24 we find

$$m = \lim_{s\to 0} [sG(s)], \tag{25}$$

where $G(s)$ is the Laplace transform of $g(t)$. From the renewal equation for $g(t)$ we know that:

$$G(s) = \frac{\Phi(s)}{1-\Phi(s)}. \tag{26}$$

Therefore,

$$m = \lim_{s\to 0}\left[\frac{s\Phi(s)}{1-\Phi(s)}\right], \tag{27}$$

by L'Hospital's rule

$$m = \lim_{s \to 0} \left[\frac{\Phi(s) + s\dot{\Phi}(s)}{-\dot{\Phi}(s)} \right], \tag{28}$$

$$= \lim_{s \to 0} [-\dot{\Phi}(s)]^{-1}, \tag{29}$$

$$= \left[\int_0^\infty \tau' \phi(\tau') \, d\tau' \right]^{-1}, \tag{30}$$

$$= 1/\bar{\tau}. \tag{31}$$

For a nonrenewal process we know that in general

$$G(s) \neq \frac{\Phi(s)}{1 - \Phi(s)}, \tag{32}$$

and we cannot directly obtain the pdf from the product density. With certain restrictions, however, we can still obtain the mean rate from the product density. These restrictions involve the range of serial order present in the nonrenewal process. For short range serial order, the average product density:

$$\overline{h(t)} = \frac{1}{T} \int_0^T h(t') \, dt', \tag{33}$$

approaches a constant k as T becomes large. In the limit we say that

$$\lim \overline{h(t)} = m, \tag{34}$$

the mean rate. In the following analysis of simple interaction between renewal processes we will assume that only short range order is present in the output process and that Eq. 34 holds.

We now wish to compute the mean rate transfer function for the simple interaction of two renewal processes with arbitrary gamma distributions. We designate the excitatory process as "1" and the inhibitory process as "2" and define:

$f_i(\tau)$	Probability density function.
$F_i(s)$	Laplace transform of pdf.
$\mathcal{F}_i(\tau)$	Distribution function.
$R_i(\tau)$	Survivor function $[= 1 - \mathcal{F}_i(\tau)]$.
$w_i(\tau)$	pdf of forward recurrence time $[= R_i(\tau)/E(\tau)]$.
$g_i(t)$	Renewal density.
$E_i(\tau)$	Mean interval.
m_i	Mean rate.
n_i	Order of the gamma distribution, $i = 1, 2$.

The probability density functions of the excitatory and inhibitory processes are

$$f_i(\tau) = \frac{\mu_i^{n_i} \tau^{n_i - 1} e^{-\mu_i \tau}}{(n_i - 1)!}, \tag{35}$$

with mean rate

$$m_i = \mu_i / n_i . \tag{36}$$

If we rewrite Eq. 14 using this notation and proceed to the limit

$$m_p = \lim_{t \to \infty} h(t), \tag{37}$$

we obtain the following result:

$$m_p = \frac{\int_0^\infty R_2(u) \int_0^u f_1(v)\, dv\, du}{\int_0^\infty \tau' f_1(\tau')\, d\tau' \cdot \int_0^\infty \tau' f_2(\tau')\, d\tau'}, \tag{38}$$

$$m_p = m_1 \cdot m_2 \cdot \int_0^\infty R_2(u) \mathfrak{F}_1(u)\, du. \tag{39}$$

Our general expression for the mean rate transfer function is then

$$T = m_p / m_1, \tag{40}$$

$$= m_2 \int_0^\infty R_2(u) \mathfrak{F}_1(u)\, du. \tag{41}$$

We can simplify Eq. 41 by noting that

$$\mathfrak{F}_1(u) = 1 - R_1(u), \tag{42}$$

and we obtain

$$T = 1 - m_2 \int_0^\infty R_1(u) \cdot R_2(u)\, du. \tag{43}$$

If we now write out the function $R_i(\tau)$ in terms of the gamma distribution, then

$$R_i(\tau) = 1 - \int_0^\tau f_i(x)\, dx, \qquad i = 1, 2 \tag{44}$$

$$= 1 - P(n_i, x), \qquad x = \mu_i \tau, \tag{45}$$

where $P(a, x)$ is a form of the incomplete gamma function such that

$$P(a, x) = \frac{1}{\Gamma(a)} \int_0^x e^{-r} r^{a-1}\, dr. \tag{46}$$

When a takes integer values, $a = n$, we can expand $P(n, x)$ in the following manner:

$$P(n, x) = 1 - \left(1 + x + \frac{x^2}{2!} + \cdots + \frac{x^{n-1}}{(n-1)!}\right) e^{-x}. \tag{47}$$

We can therefore rewrite our expression for the survivor function as

$$R_i(x) = \sum_{k=0}^{n_i-1} \left[\frac{x^k}{k!}\right] e^{-x}. \tag{48}$$

Substituting into Eq. 43 gives:

$$T = 1 - m_2 \lim_{s\to 0} \sum_{k=0}^{n_i-1} \frac{(-1)^k}{k!} (\mu_i)^k \cdot \frac{d^{(k)}}{ds^{(k)}} [R_j(s + \mu_i)],$$
$$i = 1, 2; j = 1, 2; i \neq j. \tag{49}$$

where

$$R_j(s) = \frac{1}{s}\left[1 - \left(\frac{\mu_j}{\mu_j + s}\right)^{n_j}\right]. \tag{50}$$

The derivative in Eq. 49 is

$$\frac{d^{(k)}}{ds^{(k)}} [R_j(s + \mu_i)] = (-1)^k \cdot k! \cdot [s + \mu_i]^{-(k+1)} (-1)^k \mu_j{}^{n_j}$$
$$\cdot \sum_{l=1}^{k} \left[\frac{(k-l)!}{(s + \mu_i)^{k-l+1}}\right] \left[\frac{(n_j + l - 1)(n_j + l - 2) \cdots (n_j)}{(s + \mu_j + \mu_i)n_j + l}\right],$$
$$k = 1 \cdots n_i - 1. \tag{51}$$

Substituting Eq. 51 into Eq. 49 yields

$$T = 1 - \frac{n_i m_2}{m_i}\left[1 + (1 + \mathcal{R})^{-n_j} - \sum_{k=1}^{n_i-1} \left\{1 - (1 + \mathcal{R})^{-n_j}\right.\right.$$
$$\left.\left. \cdot \sum_{l=1}^{k} (k-l)![(n_j + l - 1)(n_j + l - 2) \cdots n_j] \left[\frac{\mathcal{R}}{1 + \mathcal{R}}\right]\right\}\right]. \tag{52}$$

where $R = \mu_i/\mu_j$.

Eq. 52 is the general form of the mean rate transfer function for the simple interaction of two arbitrary gamma distributions, $f_1(\tau)$, the excitatory input, and $f_2(\tau)$, the inhibitory input. From the symmetry of Eq. 43 we know that either i or j in Eq. 52 can equal 1, and the other equal 2. The choice of which process should be i and which should be j is made such that the simplest form of the output is obtained. In this case we let n_i be the smaller of n_1 and n_2, so as to minimize the extent of the summation.

Table I shows the explicit functional forms of $T(\rho)$ for some specific choices of n_1

TABLE I

FUNCTIONAL RELATIONSHIPS FOR AN ELEMENTARY DELETION PROCESS

$T(\rho)$ $m_p(\rho)/m_\phi$ = mean rate transfer function.
ρ m_ψ/m_ϕ = inhibition strength.
P Exponentially distributed (Poisson process).
R Regularly distributed.
Γ_n Gamma distribution of order n.

Interaction	$T(\rho)$
$P \rightarrow R$	$e^{-\rho}$
$P \rightarrow \Gamma_n$	$\left(1+\frac{\rho}{n}\right)^{-n}$
$P \rightarrow P$	$1/(1+\rho)$
$\Gamma_n \rightarrow P$	$1-\rho\left[1-\left(1+\frac{1}{n\rho}\right)^{-n}\right]$
$R \rightarrow P$	$1-\rho(1-e^{-1/\rho})$
$R \rightarrow R$	$1-\rho$

The table shows the functional relationships between the mean rate transfer function T and the inhibition strength ρ for several different deletion processes. These results were derived from the statistical model of spike train interaction proposed by Ten Hoopen and Reuver (1965).

and n_2. These are the simplest analytical expressions obtainable from Eq. 52 since in each case, except for the last, $n_i = 1$. $T(\rho)$ is the mean rate transfer function which, as can be seen in the figure, depends explicitly on ρ, the inhibition strength, and n, the distribution coding parameter. These results are plotted in Fig. 9, for four particular combinations of input distributions: Poisson-Poisson, Poisson-Regular, Regular-Poisson, and Regular-Regular. Fig. 9 demonstrates that the simple interaction network has distinctly different mean rate transfer characteristics for different input distributions. This result suggests that distribution coding could play a significant role as a neural coding mechanism in particular network configurations.

Distribution Coding in a Lateral Inhibition Network

By assuming a simple network model of a relay cell network, we can examine the functional role of distribution coding in the context of sensory information processing. Burke and Sefton (1966) suggested that the lateral geniculate nucleus could be considered as a lateral inhibition network with primary optic nerve inputs to principal cells, primary outputs to the visual cortex, and inhibitory interneurons mediating the lateral inhibition. Fig. 10 shows a schematic diagram of a feed-forward lateral inhibition network where interneurons have been omitted for simplicity. It is apparent that if we consider each principal cell of the network as an interaction point for our statistical model of interaction, then we can calculate the mean rate transfer function for the array, given the input mean rate *and* distributions. First we

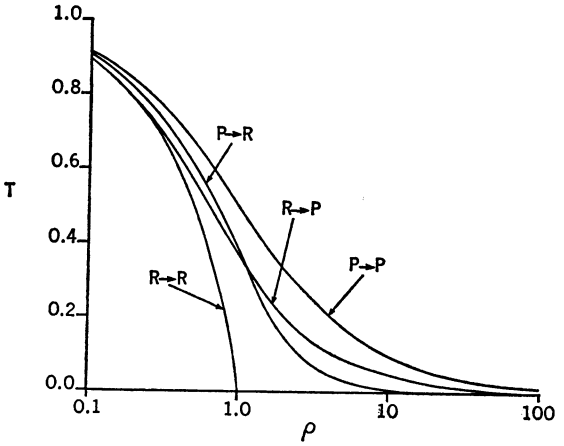

FIGURE 9 Plot showing the theoretically calculated relationship between rate and distribution parameters for the case where an inhibitory spike train (mean rate m_ψ) inhibits an excitatory spike train (mean rate m_ϕ) with a resulting spike train of mean rate m_p. The input spike trains have distributions of intervals P = Poisson, or random, or R = regular. $P \rightarrow R$, for example, symbolizes a random process inhibiting a regular process. The graph plots the mean rate transfer function T ($= m_p(\rho)/m_\phi$) vs. the inhibition strength ρ ($= m_\psi/m_\phi$). The graph was computed based on the statistical model of spike train interaction proposed by Ten Hoopen and Reuver (1965). The graph shows that the output mean rate from these model interactions depends on both the rates and distributions of the input processes.

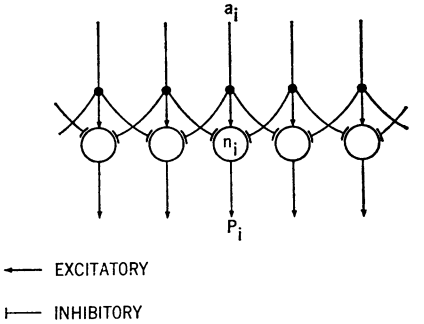

FIGURE 10 Schematic drawings of a feed-forward lateral inhibition network. The neurons are represented as a spatial array n_i, $i = 0 \rightarrow N$, where i denotes the ith neuron. An array of inputs $a_i(\tau)$ are shown.

calculate the interval distribution of the superposition of the two inhibitory processes using the model of Ten Hoopen (1966). Then we use a gamma distribution approximation in order to calculate the mean rate transfer function for each principal cell of the network using the general expression for the interaction of two gamma distributions (Eq. 52).

The apparent functions which a lateral inhibition network may have in the context of the visual system have been discussed extensively. The existence of a neurophysio-

logical correlate of contrast enhancement and Mach bands (Ratliff, 1965) has been verified in *Limulus* eye (Ratliff and Hartline, 1957) by studying the mean rate output from single visual receptors due to stimulation of the eye at various points. A similar phenomenon has been found in cats in the identification of "on-center" and "off-center" retinal ganglion cells by Barlow et al. (1957). Each type of cell has a lateral inhibitory effect, the first in response to on-center illumination, the second in response to off-center illumination. In such experiments it has usually been assumed that mean rate of firing alone is the significant parameter.

Fig. 11 shows the results of calculations to determine the mean rate transfer function for four different patterns of input stimuli to the lateral inhibition network. Each graph shows the spatial distribution of output mean rates for a particular spatial pattern of input mean rate and interval distribution. The spatial coordinate represents the spacing of the principal neurons in the network. The pattern of inputs in each of the four cases is represented at the top of each graph by a horizontal line divided into two segments. *P* indicates a Poisson process, and *R* indicates a regular

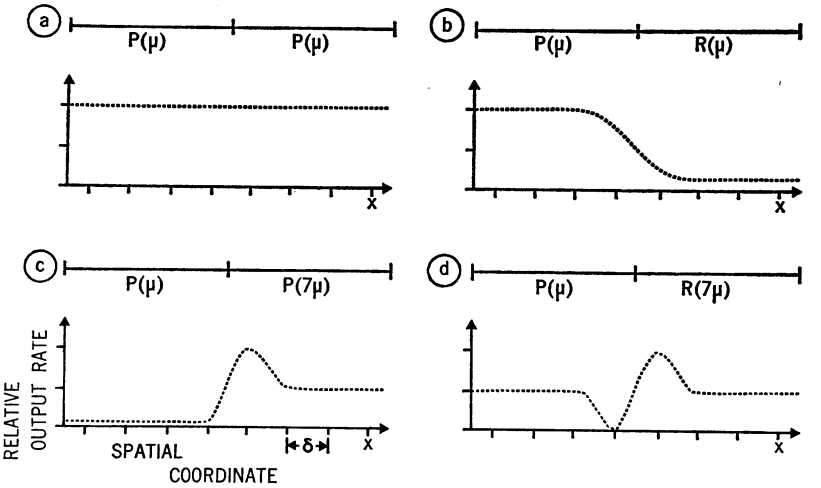

FIGURE 11 The spatial distribution of output mean rates from a model feed-forward lateral inhibition network with four different input spatial patterns of mean rate and interval distribution. The spatial coordinate δ represents the spacing of the neurons in the feed-forward network. The pattern of inputs to the network in each case is represented at the top of each graph by a horizontal line divided into two segments. Again, *P* indicates a Poisson process, and *R* indicates a regular process. The inputs to the left side of all four graphs is a Poisson process of mean rate μ. The inputs to the right halves of the graphs are:

(*a*) Poisson process of mean rate μ, (*b*) regular process of mean rate μ, (*c*) Poisson process of mean rate 7μ, and (*d*) regular process of mean rate 7μ. The dashed curves in each case represent the normalized mean rates of the output processes plotted vs. distance. These curves show that the spatial distribution of output mean rates from the network will depend on the mean rate and the spike interval distribution of the inputs. Thus distributions which we have observed experimentally at the retinal ganglion cell level could significantly affect the nature of spatial processing of information at the lateral geniculate nucleus level.

process. For the calculations we use a gamma distribution of order 10 ($n = 10$) as the regular process. The input to the left side of all four graphs is a Poisson process of mean rate μ. The input to the right halves are: (*a*) a Poisson process of mean rate μ, (*b*) a regular process of mean rate μ, (*c*) a Poisson process of mean rate 7μ, and (*d*) a regular process of mean rate 7μ. The dashed curves in each case represent the normalized mean rates of the output processes plotted vs. distance.

Fig. 11 shows that the spatial distribution of output mean rates from the network will depend on the mean rate *and* the spike interval distribution of the inputs. The model predicts that spatial information processing in the relay cell network will depend on both mean rate and distribution coding of input spike trains. This result is important for the study of neural coding mechanisms since, although many different parameters of spike trains have been suggested as having coding significance, few except the mean rate have been shown to have functional importance in the context of the neural network. Thus we have shown that distribution coding changes the mean rate transfer function characteristics of a simple interaction model and is consequently effective in changing the spatial processing characteristics of a lateral inhibition network. The results from the lateral inhibition model suggest that physiological evidence of distribution coding might be found in simply interconnected neural networks where the integrity of the input distributions is maintained. The class of relay cell nuclei, in general, and the primary thalamic sensory nuclei in particular seem to satisfy these criteria.

CONCLUSIONS

Our experiments have shown that the interval distributions of spike trains recorded from retinal ganglion cells when the visual field is uniformly illuminated systematically differ from those recorded when the visual field is dark. The distributions with illumination tend to be regular or multimodal, whole those in the dark approach exponential distributions. Such a systematic variation leads us to associate regular and multimodal distributions with the lateral inhibitory processess in the retina which accompany light illumination and enhance contrast, and associate random distributions with dark-adapted response where summation of input signals takes place to enhance brightness.

It is not our purpose in this paper to discuss the mechanisms by which distribution coding could be generated at the retinal level. The visual stimulus incident on the retina undergoes a complex "transformation" of information before being represented as a spike train code at the retinal ganglion cell level (Kozak, 1971; Sanderson, 1972). The theoretical models we have presented in this paper are models of the interpretation of a neural code rather than of the generation of a neural code. We have shown how mean rate coding and distribution coding affect the activity of a idealized neural network model (the feed-forward lateral inhibition network). The lateral inhibition network is intended to resemble the relay cell networks of the

primary thalamic sensory nuclei. The lateral geniculate nucleus is the primary thalamic sensory nucleus of the visual system and the principal network structure between the retina and the visual cortex. The lateral geniculate nucleus must "interpret" the information coding from the retina (e.g., Levick et al., 1969) and the visual cortex must interpret information coded by the lateral geniculate nucleus.

In analyzing experimental results related to neural coding, we feel that it is important to consider the neural code as having both an origin and a destination in the context of the nervous system. We observed experimentally that spike interval distributions in the retinal ganglion cell may be correlated with particular illumination conditions of the retina. Our theoretical model indicates that the transfer function of a neural network may depend on distribution coding as well as mean rate coding of the inputs. These results suggest that parameters other than mean rate could fruitfully be analyzed at many levels of neuronal systems and should be considered as clues to the information processing capabilities of a neural structure.

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