

NON-LINEAR DYNAMICAL SYSTEM THEORY AND PRIMARY VISUAL CORTICAL PROCESSING

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The vertebrate brain consists of a large number of neurons, each with highly complex non-linear dynamics. These neurons communicate with each other with a complex nexus of connecting nerve axons. The current work examines the dynamical properties of neurons located in cat primary visual cortex from the perspective of non-linear dynamical theory. The temporal patterns of activation of such neurons achieved with periodic stimuli suggest that the dynamics are relatively simple and may be modeled using a small set of coupled non-linear equations. Predictions are made based as to the patterns of activation to be found in populations of neurons.

1. Introduction

Visual perception is the activation of a selected set of visual neurons distributed among multiple visual cortical areas. Optical illusions also initially activate part of such a set of neurons. The illusion occurs when the neurons interact to complete the perception [1].

How does one discuss the properties of a set of neurons? How does one describe the dynamics of such large numbers of cells with many different patterns of connectivities and highly non-linear interactions? The thesis advanced here is that these interactions in a *real* neural network (1) result in simple dynamics, (2) are restricted to a limited repertoire and (3) are reflected in the temporal activation of single cells.

The origin of these ideas can be traced from the work of Hebb [2] who stated:

“...timing has its effect in the functioning of the cell assembly and the interrelation of assemblies: diffuse, anatomically irregular structures that function briefly as closed systems, and do so only by virtue of the time relations in the firing of constituent cells...”

It has been known for many years that there are spatial-temporal patterns in the electroencephalo-

gram (EEG). As early as 1954 the role of the alpha rhythm, for example, was thought to regulate the speed of transmission of information [3]. Hebb was unique for his time in that he attempted to describe how temporal patterns might be formed and what their role might be at *the single neuron level*. Experimental evidence for the temporal patterns in single cells has been accumulating over the past twenty years [4–14] although it is only recently that a theoretical description of the source of such patterns in the neural substrate has emerged.

In particular, Freeman and co-workers [15] have found evidence at the multi-neuronal level for oscillatory activity in the olfactory bulb. Freeman's major contribution is that he began to describe these temporal and spatial patterns in terms of non-linear distributed circuits. In this work they utilize the idea of a chaotic system as one from which many different possibilities can emerge.

However, a criticism of their work is that the basis of these oscillations at the single neuron level is still hidden. This viewpoint for studying the brain is important and has yielded many concrete and widely accepted results about processing in various regions of the brain during the past thirty years. In the brain the activity of the single unit encodes all the “messages” or “information” that can be passed onto another neuron due to the “all or none” nature of the conducted action

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potential in nerve axon. The information in population responses, such as those recorded with the local field potential method [15] or the EEG [16], are transmitted along many separate axons. Therefore the temporal code inherent in the single neuron's activity must be understood.

More recently two groups have demonstrated oscillatory activity in single neuron recordings [17, 18]. Both these groups see a strong 40 Hz frequency component in primary visual cortex in the cat. The appearance of these temporal patterns correlate nicely with the presentation of the visual stimulus. These two groups speak of these oscillations as reflecting "coherence" of the population of neurons, in much the same manner as the early EEG clinicians. Eckhorn et al. [18] have shown that two different cortical regions (V1 and V2) will oscillate together. These results are evidence for large numbers of cells working together.

Theoretical examination of the properties of net-like neuronal structures using excitatory connections showed that cortex was capable of sustaining waves of activity across its surface [19]. Later work added inhibition to this model showing that varied stable and unstable patterns could be found in a neural tissue [20]. The spatial distribution of neurons has been incorporated into these models and chaotic behavior has been formally described [21, 22].

Recently there has been a surge in interest in the dynamics of systems that are composed of large numbers of non-linear elements [23–25]. Many of these non-linear systems exhibit generic behaviors. One such well known behavior is period-doubling bifurcations as a function of some control parameter. As the parameter is varied, periodic oscillations occur which can turn chaotic. Such behavior is a universal property and particular universal constants can be derived [26].

The present work will explore the possibility that the cortical tissue, with its non-linearities and large number of cells will also exhibit such simple behavior. The strength of this non-linear approach, if successful, is that further quantitative predictions as to the temporal-spatial behavior of

real neural networks can be made without detailed examination of the underlying circuitry. And it is these same spatial-temporal patterns many neurophysiologists would agree underlie sensation, perception, motor control and other cognitive functions.

Therefore it is suggested here that the dynamics seen in the theoretical models [19, 20] and in the experimental measurements [15, 17, 18] all can be explained as particular instances of a non-linear dynamical system which may be modelled independent of the detailed underlying circuitry. These universal dynamics may be seen as the "lingua franca" of cortical tissue.

In order to test this possibility, techniques from non-linear dynamical systems are used to study single unit activity of single cells in cat primary visual cortex. The phase plots and parameter sensitivities obtained suggest that the cortical mantle has interesting temporal patterns with features in common with known non-linear dynamical systems and has orderly transitions in temporal activity.

2. Methods

2.1. Data collection

Recordings were made from primary visual cortex (area 17) of the cat using standard methods [27]. In brief, the cat was anesthetized with sodium pentothal (20 mg/kg i.v. (intravenous), supplement as needed i.p. (intraperitoneal)) and paralyzed with succinylcholine (10 mg/kg/h i.v.). The EEG, heart rate, and expired CO₂ were monitored. The eyes of the cat were dilated with atropine, then refracted with contact lenses to focus on a tangent screen at 100 inch. A craniotomy was performed at Horsey–Clarke coordinates –5 mm AP. The dura was resected and standard tungsten electrodes were used to record from single neurons along the medial bank of area 17. The signal was ac amplified and bandpass filtered at 300–3000 kHz. Single action potentials were discriminated

on their amplitude and time course. The amplified signal was also sent to an audio monitor which permitted the experimenter to listen to the neuron.

The receptive fields of the cells were first plotted using a hand held projector. Then a bar of light was projected onto the screen and a high-speed shutter was used to turn the light on and off (fig. 1). Timing pulses for the shutter (Uniblitz Co.) were obtained from Wavetek 191 waveform generator.

The time of the onset of the shutter and the time of the occurrences of single spikes were collected using Heurokon M68020 VME board with a precision of 0.1 ms. This system was attached to a Sun 3/160, which transferred the data from the slave CPU system to a disk. Data were analyzed after the experiments using either the Sun 3/160 or an IBM PC with custom written software.

2.2. Analysis

Two standard neurophysiological and one novel analysis were performed on the interspike interval data. Interspike interval histograms, which are the distribution of times between spikes, and post-stimulus time histograms, which show the correlation between the stimulus and spike, were computed. "Return maps" of the i th and the $(i + 1)$ th interspike interval were plotted as described in further detail below.

3. Results

3.1. Single cell response

The effect of the flashed stimulus on one neuron is described using both the standard techniques of neurophysiology and techniques derived from non-linear dynamical system theory. The first cell to be described (fig. 2) was a complex cell; it was responsive to the onset of the stimulus.

The raw data from the first 40 s of the 20 min of recording is first shown. The plus sign indicates

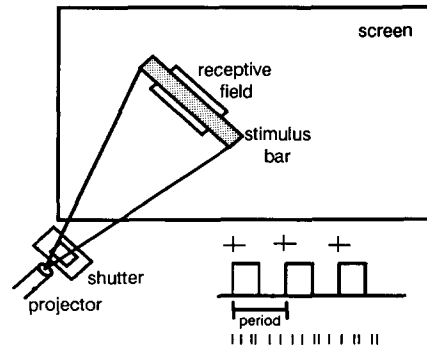


Fig. 1. Schematic representation of the recording situation. Stimuli were projected onto a tangent screen which was at a distance of 100 inch from the cat's eye. The receptive field was first mapped using a hand held projector and is marked by the open rectangle. Then an illuminated stimulus bar (shaded rectangle) was projected at the optimal angle for the cell. A shutter attached to a square wave generator was used to turn the stimulus on and off. The stimulus timing, which always had a 50% duty cycle, is illustrated at the lower right. The stimulus period was defined as the time between successive stimulus onsets. A few sample spikes are schematically displayed below the stimulus trace. The plus signs indicate the time of stimulus onset and are again seen in fig. 2.

the stimulus onset; the vertical bar indicates the time of spike occurrence. Stimuli were presented at 271.5 ms intervals. A burst of action potentials can be seen following each stimulus. By plotting the distribution of interspike intervals, it can be seen that there is a major periodicity at approximately 270 ms. There are also peaks at 175, 540, and 1080 ms. The latter two values are roughly integer multiples of the driving period. These slow frequencies can be attributed to the cell not being activated by one or more stimuli in a row.

The 175 ms peak is not directly attributable to any linear or non-linear processing thought to occur in single cells (e.g. a potassium conductance). Furthermore as will be seen below, these periodicities were dependent on the stimulus.

Post-stimulus time histograms were also constructed averaging the cell's response to each stimulus. Such histograms are often used to classify single visual neurons. For example, the present cell is called an "on-cell" because of the response to the onset of the stimulus. Other cells are called

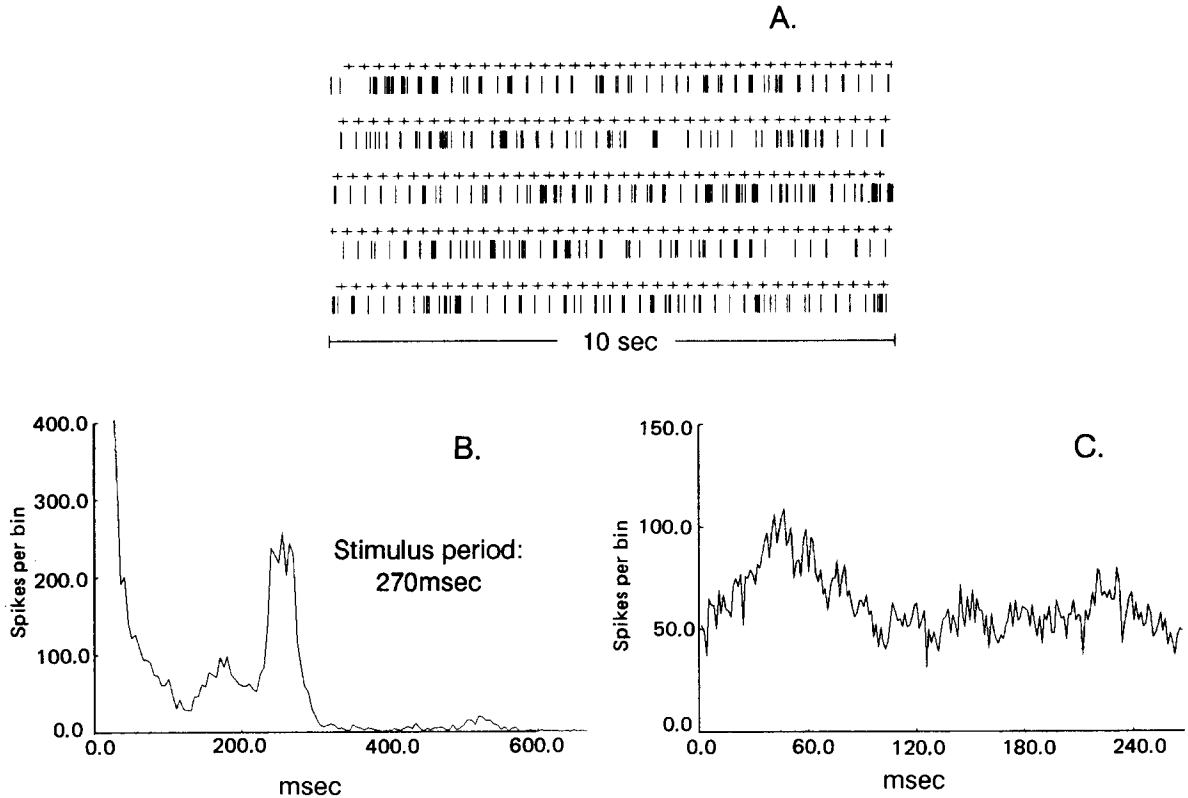


Fig. 2. Standard analysis of the interspike interval data. (A) The first 40 s of a 20 min recording session are shown. The plus sign indicates the stimulus onset; the vertical bar indicates the actual time of spike occurrence. Occasional bursts can be seen in this presentation of the data which tend to occur after the stimulus onset. (B) The interspike interval histogram is formed by counting the number of spikes that occur with different interspike intervals. (C) The post-stimulus time histogram is computed by computing a histogram of the time of spike occurrence relative to the stimulus onset. More formally it is a cross-correlogram of the point processes that constitute the stimulus and the spike train. The 270 ms peak shown correspond to the period of the stimulus.

“off cells”, which respond to the off-set of the stimulus or “tonic” cells which have sustained responses [27].

The third and final analysis to be performed was the generation of “return maps”. The motivation for these plots in the present work comes from non-linear dynamical theory where similar graphs from continuous time systems reveal temporal patterns which are not seen in the raw data. Such reconstructions in continuous time systems have been shown to embody the complete dynamics of the system under study [28]. Poincaré sections can be taken through these phase trajectories which can show fractal structure [26]. If the repeated stimulation of the cell was to lead to a

temporal pattern of activation, then the return maps of the interspike interval should show interesting structure.

It can be seen that the return map of the interspike interval has a number of features (fig. 3). No such features were seen when the cell was spontaneous active (c.f. fig. 5a, 5b). Furthermore these patterns are reminiscent of the spatial return maps from coupled map lattices [23]. The origin of some of these features is trivial; some require deeper explanations. First there is the density close to the origin. It is caused by the rapid firing of the neuron. In this particular cell, there is no apparent structure at the shorter intervals, however with other cells (fig. 4) there are some relationships

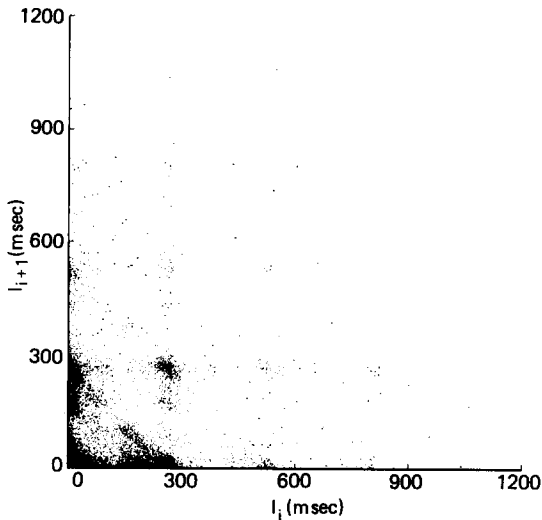


Fig. 3. "Return map" computed by plotting the i th interspike interval against the $(i + 1)$ th interval. A number of regions with concentrations of points can be seen. These correspond to attractors. See text for details.

between the interspike intervals. This pattern indicates that there are mechanisms which regulate the relationships between intervals at the onset of a burst as action potentials. There are two possible choices for such mechanisms – either an intrinsic property of the neuron (e.g. refractory period) or a network property.

There is also a clear repeating structure both vertically, horizontally and parallel to the lines:

$$I_i + I_{i+1} = kT,$$

where T is the period of stimulation, I_i is the i th interspike interval and k is 1, 2 or 3.

The vertically and horizontally oriented clusters of points are arranged at intervals equal to the base period. They arise from the neuron failing to fire from a given presentation of the stimulus. Some intervals approximately equal to the driving period are followed predominately by intervals at the same period. This cluster represents an 1:1 relationship between the stimulus and response respectively. In some cells, there are phase locking patterns of 2:1, 3:1 where one burst of action potentials occurs for every 2 or 3 stimuli.

A projection of this two-dimensional distribution onto one dimension results in the major peaks seen in the interspike interval histogram. The source of the peak in the interspike interval histogram at 175 ms can now be seen to be the result of a density of points clustering about a portion of the diagonal line given by eq. (1). Part of this diagonal line is stimulus driven and part is related to the temporal dynamics of the system.

The stimulus driven portion of the signal can be understood by example. Suppose every time the stimulus is turned on, a short burst of spikes occurs. Now imagine that a spike occurs at random in the interval between the two stimuli. This would result in points evenly distributed on the line given by eq. (1) with $k = 1$.

The basic diagonal structure is a result of the driving stimulus. What is interesting is that the spike data are not evenly distributed among the diagonals given by eq. (1). In this example, the points are clustered at part of the diagonal. Some process is permitting the neuron to fire with a particular clustering of interspike intervals. In the parlance of non-linear theory, the clusters seen in figs. 3, 4 and 5 are attractors.

Other neurons have other clusters of interspike interval sequences (figs. 4, 5). (These clusters can also be constructed in three-dimensional return maps.) These attractors are stable over long periods; some neurons show the same patterns for the full 1.5 h tested. These attractors do not appear to be linked to the slow electrical waves in EEG that arise when the animal is anesthetized. Furthermore, these attractors change very systematically with the stimulus period.

3.2. Sensitivity to period of stimulation

It was noted during the course of these experiments that these patterns could easily be heard on the audio monitor. Small changes in the stimulus period resulted in a change in the response of the cell from sounds like a "trotting" to a "galloping" horse. Did these patterns change in some orderly

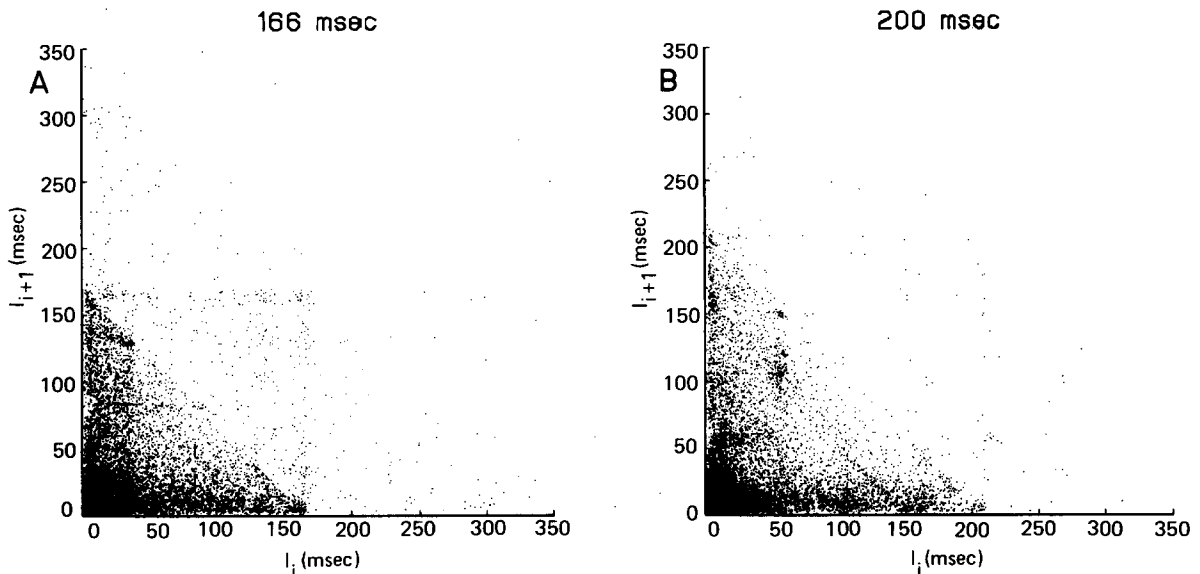


Fig. 4. Sensitivity of the return map to a 36 ms change in stimulation period. (A) The return map for a stimulation period of 166 ms. Note the multiple densities which represent attractors. Close to the origin (< 50 ms), finer patterns can be seen. The main diagonal intercepts the two axis at the driving period. There is a second weak diagonal band that intercept the axis at about twice the driving period. Other complex patterns of point densities can be seen. (B) Increasing the stimulus period by 36 ms leads to a large change in the return map patterns even though there only is a small change in the overall firing rate. Note the asymmetries in the return map.

way as function of stimulus period? The dependence of the neural response on the stimulus period was therefore examined. Fig. 4 shows the phase plots for a neuron different from that in figs. 2 and 3 with the stimulus periods of 166 and 200 ms. This neuron was of the complex type; it had almost no spontaneous activity. These particular stimulus periods were chosen due to the complexity of the auditory patterns. The "return map" also showed numerous complex periodicities. (This cell had the most complex patterns observed in the 20 cells in 5 cats studied.)

This sensitivity to stimulus period was then studied in greater detail by varying the stimulus period over a wide range. An example of such an experiment for a third cell is depicted in fig. 5. Spontaneous firing, and three stimulus periods are shown. No patterns are seen when the cell is spontaneously firing. Decreasing the stimulus period lead to additional periodicities and changes in the return map pattern.

To further quantify these effects, a finer exploration of the dependence of the stimulus period

was performed. The stimulus period was set at some value for 2–3 min duration and then changed to a new value. The data is displayed by plotting the interspike interval against the stimulus interval (fig. 6A). In order to get a better representation of the interspike interval density, a small random value was added to the stimulus period prior to plotting. Such a plot reveals that at critical values, new multiples of the stimulus period are added to the interspike intervals. These data were also plotted using a normalized ordinate (fig. 6B) by dividing the interspike interval by the stimulus interval. In this representation, a value of "1" on the ordinate means that the cell was firing at an exactly the same interval as the stimulus period, "2" is twice the stimulus period, etc. The interspike interval is now seen to be phase locked to the stimulus with patterns of 2:1, 3:1, 4:1, ..., 8:1. (Some cells also show 1:2, 1:3, 1:4, etc.) It appears from the qualitative picture of fig. 6 that there is a systematic increase in interspike intervals at multiples of the stimulus period. Similar effects were seen in five other cells suggesting that

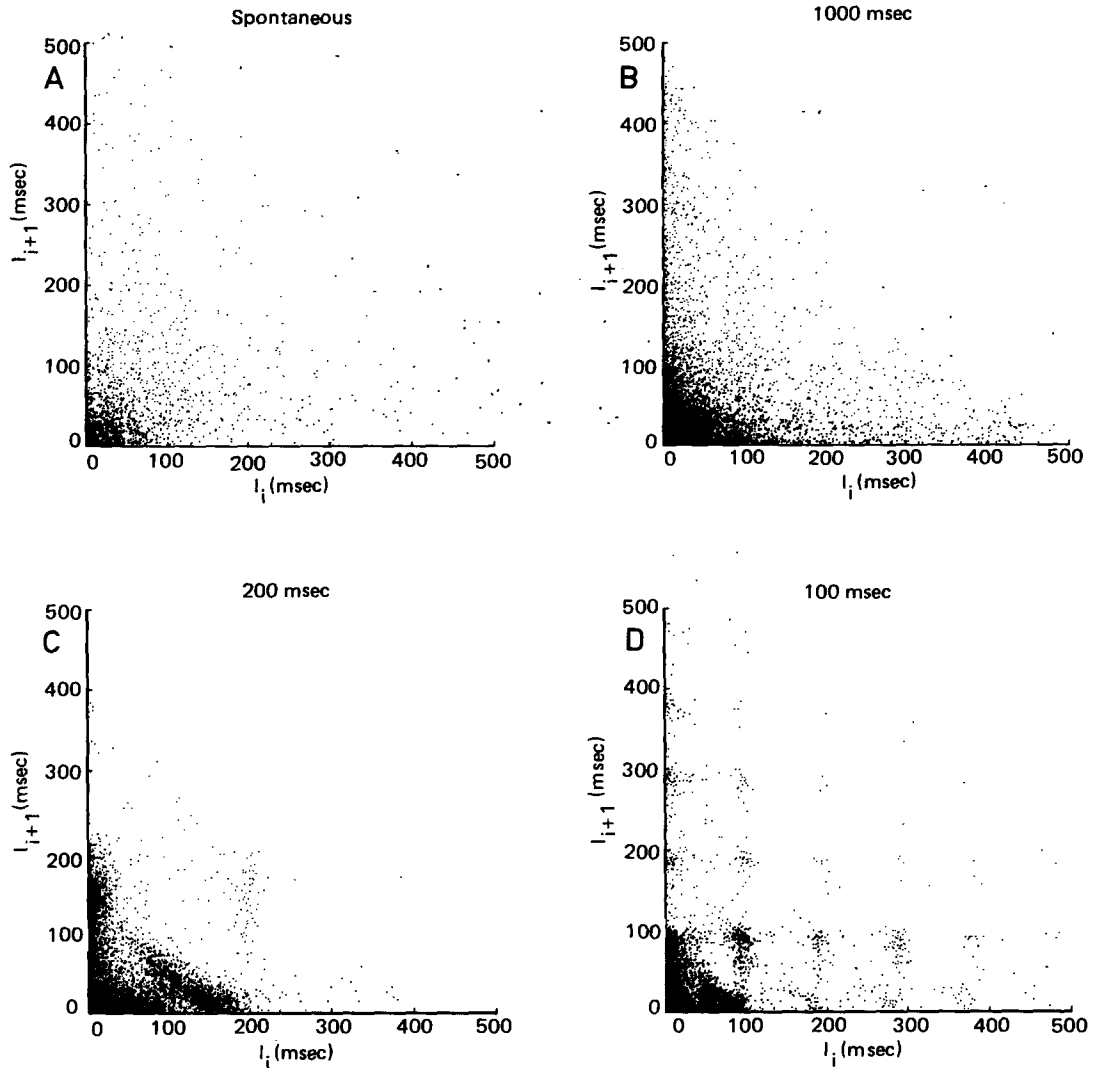


Fig. 5. Return maps for spontaneous and three different stimulus periods of a cat visual neuron. (A) Spontaneous activity. The spikes occur in bursts, but there is no pattern to the sequence of bursts that occur. (B) 1000 ms stimulus period. At this stimulus period, there begins to be some structure in the return map. Most of the points fall below the line given by eq. (1) with $k = 1/2$. (C) 200 ms stimulus period. The temporal pattern becomes more complex; an attracting cluster at around (175, 25 ms) emerges. As well, the cell begins to have longer interspike intervals at multiples of the stimulus period. (D) 100 ms stimulus period. More complex patterns are seen. The attracting cluster stays at the same relative position in the graph relative to the driving period. Longer multiples of the driving period are seen.

the change in temporal dynamics for these neurons is restricted to a particular repertoire.

In order to quantify these effects, normalized interspike interval histograms were created for each of the stimulus periods. In these plots the spike count was divided by the total stimulus duration since different durations were used for

each stimulus period. This spike count per stimulus duration, defined here as the “density”, was then plotted as a function of stimulus period (fig. 7A). Multiple peaks can be seen that correspond to the cluster of points seen in fig. 6B.

The relationship of the peaks to the normalized interspike intervals was next investigated. A geo-

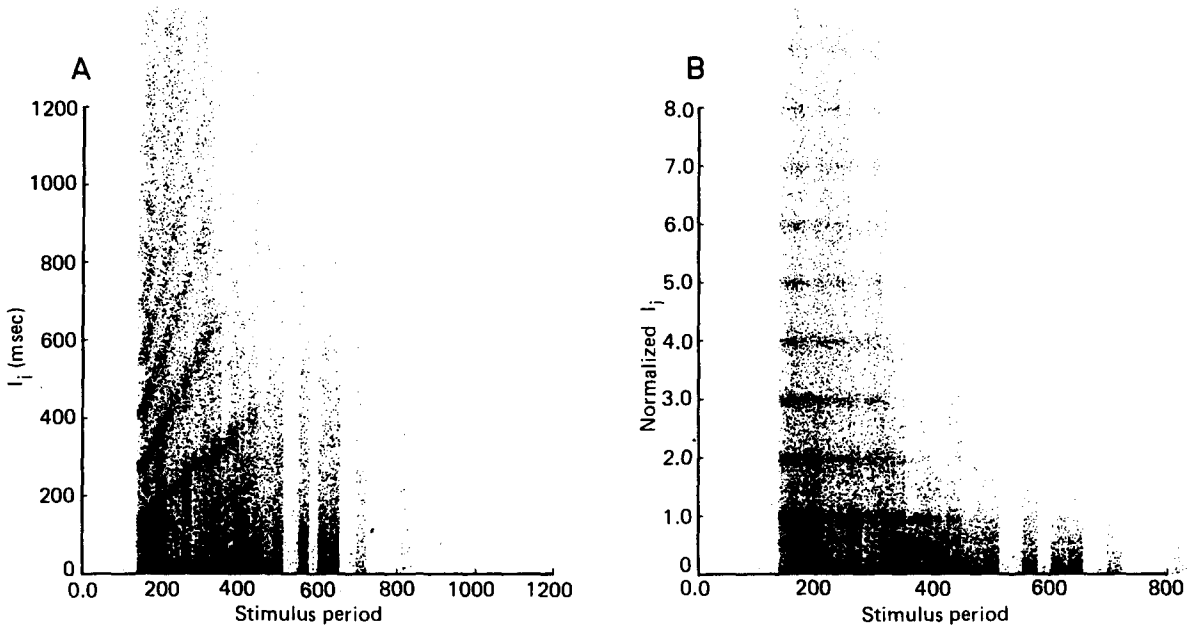


Fig. 6. The effect of the stimulus period on the interspike intervals. The cell presented here is the same as in figs. 2 and 3. The ordinate is the stimulus period; the abscissa is the interspike interval (A) or the interspike interval divided by the stimulus period (B). In order to better present the number of spikes for each stimulus condition, a random number was added to each of the stimulus periods. In both presentations, the decreases in stimulus period lead to longer interspike intervals at integer values of the stimulus period.

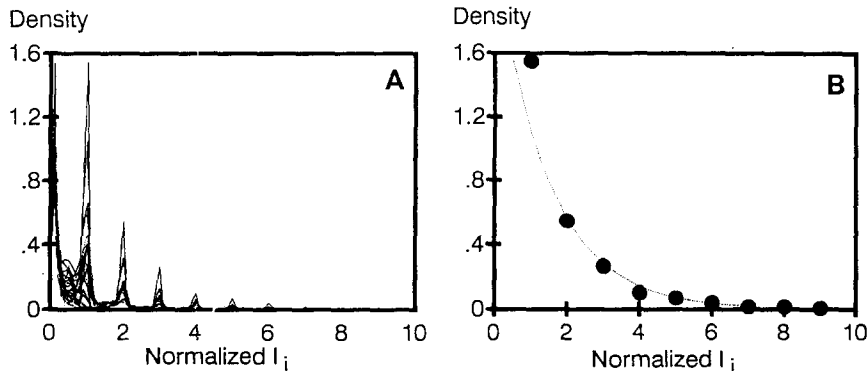


Fig. 7. Quantitative measures of the spike density as a function of the stimulus period and integer multiple of the stimulus period. (A) A series of overlaid interspike interval density plots for the 26 different stimulus periods used are shown. The abscissa is the interspike interval divided by the stimulus period; the ordinate is the spike density (see text). Successive peaks at up to seven times the base stimulus period can be seen. It appears that the peaks of the interspike interval densities decays roughly geometrically with the stimulus period. (B) This can be demonstrated by examining the data collected for one of the stimulus periods (135 ms). Again the abscissa is the interstimulus interval divided by the stimulus period; the ordinate is the spike density. Only the values of the density at the integer multiples of the interspike interval are plotted. A geometric relationship given by: $Y = 2.2 \times 0.51^X$ can be obtained (r^2 of 0.98). Similar plots are found for other stimulation periods.

metric function $Y = AB^X$ was a good fit for all the different stimulus periods used. Similar relationships were seen in other cells. The significance of these relationships with respect to underlying dynamics however remains an open question as there is little dynamical theory at present that addresses the dynamics of systems of this degree of complexity (see section 4).

4. Summary and discussions

The present work seeks to apply the theory and methodology from the study of non-linear dynamical systems to single unit activity in the brain. The underlying principle of this approach is that the dynamics of a large number of neurons (presumably determined by as many as 10^4 non-linear coupled differential equations) is not highly complex but can be simple. (The value of 10^4 is a conservative estimate of the number of cells in a local column that are interconnected and excludes all the equations that arise from the cable and membrane properties of excitable tissue.)

The present data are supportive of these claims. Interesting patterns are obtained by plotting adjacent interspike intervals. These patterns are sensitive to the period of the stimulus. These patterns exhibit multiple steady states which are not explicable based on standard membrane phenomena. Single neurons, when driven by periodic stimuli, do not show patterns of this type [29]. Preliminary work also indicates a sensitivity to the orientation of the bar, which is thought to be a result of interactions amongst neurons. These patterns are robust, repeatable and can be maintained over long periods of recording. It is suggested that the patterns seen in the return maps result from the network properties of the visual cortex.

It is remarkable that these patterns are quite similar to spatial return maps in coupled map lattices [23]. In these models, 100–1000 quadratic maps, given by $X_{i+1} = aX_i(1 - X_i)$, are coupled together. When the dynamics are explored by altering the coupling and the map parameter a ,

orderly changes in the dynamics are seen. These models have multiple attractors where the attractors are distributed across the lattice. It will be interesting to determine if periodic forcing of these coupled lattices can lead to patterns similar to those presented here from cerebral cortex. Also some of these analytical techniques developed for these lattices can be applied to neural data.

The sensitivity of these neural patterns to the stimulus period are qualitatively similar to period-adding bifurcations seen in electronic systems [30, 31]. Period-adding bifurcations are seen when a change in a parameter leads to an adding of a new periodicity rather than the splitting of periodicity into two periods (a pitchfork bifurcation). Again, it will be necessary to further explore these qualitative similarities with both models and experiments.

The implication of this work is that the temporal activity of a single neuron that is embedded in the matrix of approximately 10^4 neurons is relatively simple. Is it possible to reflect this temporal simplicity in a small set of equations? If so, it may not be necessary to model the visual cortex in complete detail in order to describe its activity. Rather, an emergent property of temporal dynamics can be used to describe this highly complex system. This is not to state that detailed physiological and anatomical analyses are not needed. Even with a high-level description such as that delimited here, it is still necessary to understand how the cortical circuit is formed from the constituent elements.

It is of course possible to numerically determine the relative simplicity or complexity of the neural system using dimensional analysis [32] or by computing the Lyapunov exponents [33]. Technically there are some problems since the reconstruction of the attractor requires data evenly spaced in time and the spike data are inter-event data. The one published example of a computation of the dimension using interspike interval data for somatosensory neurons [34] does not show good convergence of the computed dimension. (It is possible to perform temporal averaging to obtain

a continuous time series and more properly fulfill the requirements of dimensional or Lyapunov exponent analyses [35].)

However, there is a more important conceptual problem to be surmounted even if these sorts of analyses can be performed and low dimensionality and positive Lyapunov exponents can be determined. Neither of these two quantities are sufficient to establish that non-linear dynamical theory is applicable to brain tissue [36], nor that the tissue is truly chaotic. Rather it will take a concerted effort to first demonstrate that the dynamics of brain tissue exhibit *features* of non-linear dynamical systems (e.g. universality). Following that, it is then necessary to physiologically demonstrate that these dynamics are not epiphenomena but are actually used by the brain in its processing of the world about us.

It was assumed at the onset of this work that this temporal pattern of activity is indicative of the more distributed nature of the cortical mantle. It is predicted here that similar patterns should be observable in the spatial dimension (i.e. across the cortical surface and/or through the cortical layers) by plotting the interspike intervals of one cell against the interspike intervals of a second cell. Such data have been collected by a number of researchers [37–43] and can be easily examined to test this hypothesis. One supportive result for this prediction can be found in the work of Aertsen and Gerstein [14], who have demonstrated temporally varying relationships for two cells that have significant cross-correlation.

If these results are correct, then questions as to the underlying nature of these non-linear processes arise. Are these responses caused by a completely deterministic process or by a stochastic process? The visual system is a biological system and as such will have “real” noise. Two biological systems where non-linear dynamical approaches have been applied have demonstrated that the presence of noise results in a removal of more complex phase locking patterns [44,45]. Theoretical approaches to this problem have been initiated [25, 46, 47]; also ch. IV in ref. [24].

Finally, regardless of the underlying theoretical correctness of the above approach, it is also important to note that these patterns may prove to be useful indicators for the classification (e.g. morphology, laminar distribution, simple, complex, hyper-complex and end-stopped properties) of neurons with different connectivities in the nervous system. In the past it has been quite difficult to classify a neuron’s morphology based purely upon its response to light [48]. The current analysis may simplify this classification based upon the additional information that can be obtained from the “return map” representation of neural activity.

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