

Rapid state switching in balanced cortical network models

M V Tsodyks and T Sejnowski

Howard Hughes Medical Institute, Computational Neurobiology Laboratory, Salk Institute, La Jolla, CA 92037, USA, and Department of Biology, University of California, San Diego, La Jolla, CA 92093, USA

Received 7 October 1994

Abstract. We have explored a network model of cortical microcircuits based on integrate-and-fire neurons in a regime where the reset following a spike is small, recurrent excitation is balanced by feedback inhibition, and the activity is highly irregular. This regime cannot be described by a mean-field theory based on average activity levels because essential features of the model depend on fluctuations from the average. We propose a new way of scaling the strength of synaptic interaction with the size of the network: rather than scale the amplitude of the synapse we scale the neurotransmitter release probabilities with the number of inputs to keep the average input constant. This is consistent with the low transmitter release probability observed in a majority of hippocampal synapses. Another prominent feature of this regime is the ability of the network to switch rapidly between different states, as demonstrated in a model based on an orientation columns in the mammalian visual cortex. Both network and intrinsic properties of neurons contribute to achieving the balance condition that allows rapid state switching.

1. Introduction

Cortical neurons *in vivo* are spontaneously active with a high degree of variability in the spike trains even in the absence of a driving input from the thalamus [1, 3]. As a consequence, most studies of single unit properties report average firing rates as a way to overcome this 'noise' and most models of cortical networks adopt the firing rate as the primary dynamical variable. Is noise a reflection of the inherent unreliability of single neurons or is it instead a fundamental feature of the dynamics of cortical networks?

In contrast to *in vivo* conditions, neurons recorded in cortical slice preparations have little spontaneous activity and are more hyperpolarized with respect to the firing threshold. The strengths of synaptic inputs to cortical cells are generally quite small, in the range 0.1–1 mV, which would require many tens of simultaneously activated excitatory inputs to reach threshold from the hyperpolarized levels found *in vitro*. This is consistent with the hypothesis that the function of a cortical neuron is to integrate many noisy inputs and to encode the average input in the average firing rate of a noisy spike train [2]. According to this view, only a time average of a spike train over a long interval (around 1 s), or population average across a large number of similar neurons (around 100) contains reliable information.

A different view has emerged from intracellular recordings of cortical neurons *in vivo*. In the awake state, cortical neurons are rarely hyperpolarized to the 'resting' levels observed *in vitro* but instead hover near threshold most of the time. In addition many cortical neurons *in vivo* have a small afterhyperpolarization following an action potential [4, 5].

These observations suggest that the 'operating point' of a cortical neuron may be just below spike threshold, in a state of readiness to respond to transient inputs [6–8]. If the average membrane potential *in vivo* were to be maintained just below threshold most of the time, the number of synaptic inputs required to reach threshold would be much smaller than in the case of cortical neurons *in vitro*.

In order to maintain the state of a neuron near threshold over a wide range of input conditions, excitatory inputs need to be balanced by inhibitory inputs, especially in cortical microcircuits where there are strong excitatory inputs from recurrent collaterals from neighboring pyramidal neurons. We have explored this *balance* hypothesis on the behaviour of a single neuron using a simple integrate-and-fire model and also a more realistic Hodgkin–Huxley-like model with active conductances [9]. We found that when the afterhyperpolarization in these models was small, and excitatory inputs were balanced by inhibitory ones, there was a high degree of fluctuation in the output spike train, similar to that observed from intracellular recordings of pyramidal neurons *in vivo* (see also [10, 2]).

A weak afterhyperpolarization implies that fewer simultaneous excitatory synaptic inputs are required to reach threshold; under these circumstances a single synaptic input could have a significant effect on spike firing. The amount of afterhyperpolarization in a neuron should therefore alter the dynamical behaviour of neurons in large networks. A central issue addressed in this paper is whether the balance condition, imposed artificially in models of single neurons, can be obtained in a network model that includes populations of inhibitory as well as excitatory neurons with small afterhyperpolarization. We find robust conditions under which a network of integrate-and-fire neurons with the basic architecture of a cortical column is balanced over a range of input conditions.

In contrast to conditions in which the output of a neuron is determined mainly by the average of its synaptic inputs, the so-called *mean-field* regime [11], the outputs of neurons in 'balanced' networks also depend on the fluctuations of the inputs. Strong fluctuations dramatically affect the time course of the transition of the network between different states of activity. We illustrate this in a network model of orientation columns in the visual cortex. The activity of the network switches rapidly to a new state when weak external input switches between two orientations.

2. Methods

2.1. The model

Consider a network of *integrate-and-fire neurons*, with separate excitatory and inhibitory populations. The dynamics of neuron i ($i = 1, \dots, N$) are governed by a leaky integrator model:

$$C \frac{dV_i}{dt} = -g(V_i - V_0) + I_i(t) + I_i^0 \quad (1)$$

where $V_i(t)$ represents the membrane potential of the i th neuron, V_0 is the resting level, assumed to be the same for all the neurons in the model, g is the input conductance, I_i^0 is an external input to the neuron i , and $I_i(t)$ is the synaptic current, generated by inputs from excitatory and inhibitory populations: $I_i(t) = I_i^{ex}(t) - I_i^{inh}(t)$. Each component is a sum of contributions from the spiking of individual neurons from the corresponding population. For simplicity, we neglect the driving forces for the synaptic currents and assume that these contributions do not depend on the postsynaptic membrane potential. This is a reasonable assumption if the membrane potential hovers around threshold most of the time. When a spike arrives at a presynaptic terminal, the postsynaptic current instantaneously increases

according to the strength of the synapse, and decays with the time constant τ^{ex} (τ^{in}). We neglect the rise time of the postsynaptic current, assuming that it is much smaller than the decay constant [14]. This dynamics of the synaptic currents are given by

$$\frac{dI_i^{ex,in}(t)}{dt} = -\frac{I_i^{ex,in}(t)}{\tau^{ex,in}} + s^{ex,in}(t) \sum_j K_{ij}^{ex,in} \delta(t - t_j^{ex,in}) \quad (2)$$

where K_{ij}^{ex} (K_{ij}^{in}) is the strength of the excitatory (inhibitory) synapse between neurons j and i . The synapses are probabilistic and the binary random variable $s^{ex}(t)$ ($s^{in}(t)$) equals 1 with the probability p_{ex} (p_{in}), and is zero otherwise. The spike in neuron j occurs at time t_j .

When the potential V_i reaches a threshold value V_{th} , neuron i emits a spike and its membrane potential is reset instantaneously to a fixed reset potential, V_{res} . The synaptic strengths are comparable in magnitude to the reset potential.

Rather than model the inputs to the network from the thalamus and other cortical areas with spike trains, the external input current I_i^0 is a continuous variable and provides only a weak bias to the network rather than a strong driving input.

2.2. Conditions for validity of the mean-field approximation

The amplitude of synaptic current fluctuations in a neuron can be estimated from the parameters in the model. Consider the excitatory component of the synaptic current for one of the model neurons. From equation (2), the average value of $I^{ex}(t)$ in the steady state is approximately $I \sim K\tau(p\nu n) \equiv K\tau^{ex}\nu_{tot}$, where K and p are average strength and reliability of an excitatory synapse, ν is an average spiking rate, n is the number of excitatory synapses, and $\nu_{tot} = p\nu n$ is the total frequency of incoming excitatory synaptic inputs. We can estimate the minimum amplitude of the fluctuations of the current when the presynaptic neurons are uncorrelated. Then the standard deviation of I is approximately

$$\sigma_I \sim K\sqrt{\tau^{ex}\nu_{tot}}. \quad (3)$$

In the static mean-field approximation the fluctuating current is replaced by the average current, as determined by the average spiking rates of the presynaptic neurons (for a more detailed discussion see [12, 13, 15]). This approximation is valid when the fluctuations are small compared to the mean current required to drive the neuron from the reset potential to the threshold potential, $I_{res} = g(V_{th} - V_{res})$. The reset current *rather* than the total excitatory current should be compared with the size of the fluctuations since a significant fraction of the total excitatory current must be compensated by the inhibition to prevent overloading the neuron with excitation. Thus, the mean-field approximation is only valid when the value of dimensionless parameter $k = \sigma_I/I_{res}$ is much less than one. Alternatively, one may consider the standard deviation of the potential, $\sigma_V \sim \sigma_I/g$, relative to the change of voltage following reset. Expressing parameter k in terms of the average excitatory postsynaptic potential, $E \sim K\tau^{ex}/g\tau_m$, the static mean-field regime is given by the condition

$$k \sim \frac{E}{\Delta V} \frac{\tau_m}{\tau^{ex}} \sqrt{\tau^{ex}\nu_{tot}} \ll 1 \quad (4)$$

where $\Delta V = V_{th} - V_{res}$, $\tau_m = C/g$ is the membrane time constant. Intuitively, k is large when the magnitude of reset is small compared to the expected fluctuation in the membrane potential within the time course of the synaptic current. A rough estimate for k for a neuron in visual cortex responding to its preferred stimulus based on $E \sim 0.1$ mV, $V_{th} - V_{res} \sim 5$ mV, $\tau_m \sim 20$ ms, $\tau^{ex} \sim 5$ ms, $p \sim 0.1$, $n \sim 1000$, $\nu \sim 50$ s⁻¹ in equation (4) [2] yields

$k \approx 0.5$. Under these conditions, the magnitude of current fluctuation within a synaptic time constant is comparable to the current needed to reach threshold. Hence, the dynamics is dominated by fluctuations and the mean-field approximation would not hold. Although some of these numbers are far from being reliably known, and our estimate could be an order of magnitude off, the above estimate for k indicates that there are reasonable values for the parameters under which the visual cortex may be operating in the non-mean field regime. In this paper, we use network simulations of integrate-and-fire neurons to explore this regime.

For convenience, the membrane potential was linearly transformed so that $V_0 = 0$, $V_{th} = 1$ and $C = g = 1$. With these choices the current was dimensionless.

3. Results

3.1. Homogeneous network

The homogeneous model is meant to roughly approximate a small population of neurons in layer 4 of the cortex. Inputs from the thalamus contact both excitatory and inhibitory neurons in layer 4. Around 20% of cortical neurons are inhibitory and 20% of the synapses on a typical cortical pyramidal neuron from neighboring neurons are inhibitory [4]. In the network model there were 400 excitatory and 100 inhibitory neurons and the external input to each neuron, $I_0 = 1.001$, was chosen to bring each neuron just above the firing threshold. The reset was close to threshold, $V_{res} = 0.9$. The excitatory neurons in cortex have recurrent collaterals that make synapses with other excitatory neurons within the same column as well as with inhibitory neurons. This intracolumnar network was modelled with complete connectivity between all the excitatory neurons, and between all the excitatory and inhibitory neurons in the network. All the connections had the same strength $K^{ex} = K^{in} = 0.02$. With this choice of excitatory synaptic strength, five simultaneous excitatory inputs would be sufficient to elicit a spike starting from the reset level. Activity in the larger excitatory population was balanced by choosing a higher probability for transmitter release at inhibitory synapses $p_{in} = 0.7$ compared with that at excitatory synapses $p_{ex} = 0.2$. This choice of parameters leads to $k \sim 0.5$, similar to the estimate made for cortical neurons discussed above.

The unusual dynamics of the network are apparent in figure 1, which shows raster plots for selected neurons. All of the neurons in the network showed spike firing with high variations on a typical time scale of few tens of ms, with an average rate that varied from from 0 to 200 spikes per second. Although the pattern of firing varied dramatically with time (upper trace), all the neurons in the network, including the inhibitory neurons, had about the same average firing rate during any given interval. Despite strong correlations on the time scale of 20–50 ms, the spikes were not synchronous on the 1 ms time-scale, as shown in the cross-correlogram shown in figure 2. The broad peak in the cross-correlogram with a narrow peak near a time delay of zero is typical of spike trains recorded *in vivo* [4].

The total synaptic current in a single neuron fluctuated around zero even though there were massive swings of the excitatory inputs on a 20–50 ms time-scale, as illustrated in figure 3. This is consistent with a rough balance of excitation and inhibition as a consequence of feedback inhibition that closely matched the excitation. The fluctuations in the net synaptic current were nonetheless much higher than the superthreshold part of the external input I^0 . Thus, the positive feedback from other excitatory neurons in the same network served as a high-gain amplifier. Stability was insured by strong inhibition that throttled the chain reactions triggered by chance firing in some excitatory neurons.

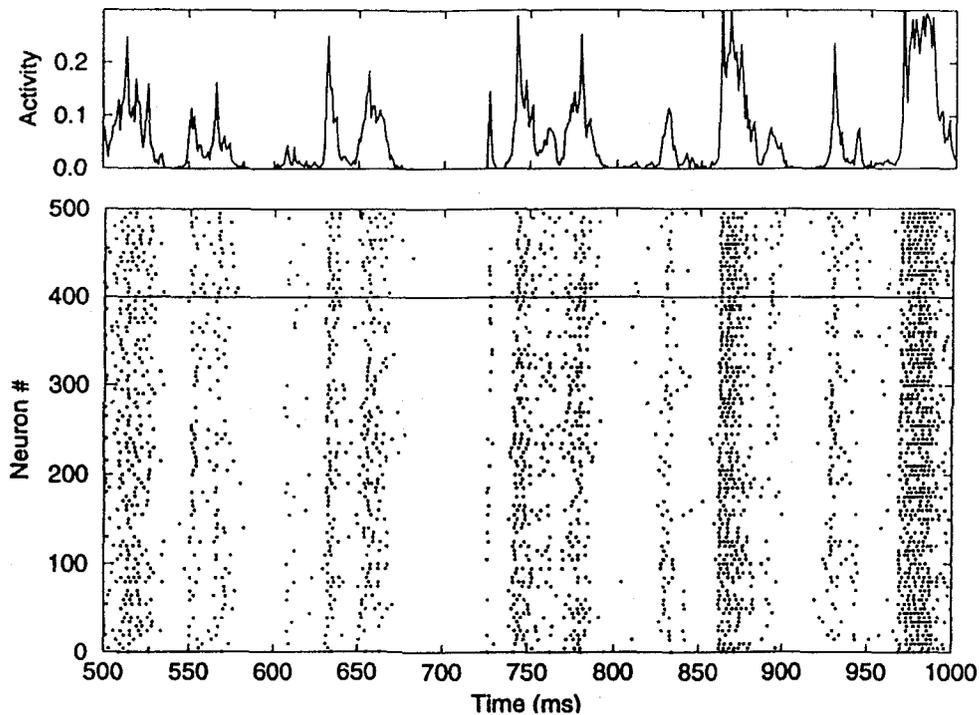


Figure 1. Dynamics in a network of integrate-and-fire neurons with complete connectivity. The full curve on top is the fraction of neurons spiking within a 1 ms time bin. Individual spike trains from a network of 400 excitatory neurons (1–400) and 100 inhibitory neurons (401–500) are shown below. Each dot is a single spike and each row is a single neuron. The spikes for every fifth neuron are plotted. Parameters of the network are: $\tau_m = 10$ ms, $\tau_{ex} = \tau_{in} = 5$ ms, $K^{ex} = K^{in} = 0.02$, $p_{ex} = 0.2$, $p_{in} = 0.7$, $I_0 = 1.001$.

We studied the behaviour of the network with different sets of parameters and found the most important parameters for determining the dynamics to be the relative time-scales of excitation and inhibition. When inhibition was faster than excitation, the variations in the firing rates of the neurons were less pronounced, although the firing remained highly variable. If, however, inhibition was slower than excitation, the network collapsed into sharp synchronized bursts of activity.

Finally, we varied the strength of the input current I_0 from just below threshold to just above threshold. When the strength of the input current crossed the threshold value, the activity in the network jumped from zero to a nonzero rate. This indicates that when the input is close to a threshold, which we suggest is typical during both spontaneous and driven activity, the network is close to a bifurcation point between quiescent and active states, and as a consequence is highly sensitive to the fluctuations of the input. We will return to this point in the context of columnar organization.

3.2. Scaling of the network

Real networks in the cortex contain many more neurons than the simulation of the 400 neuron network. As we increase the size of the model network, how should the parameters be changed to allow comparisons among networks and how should these parameters be

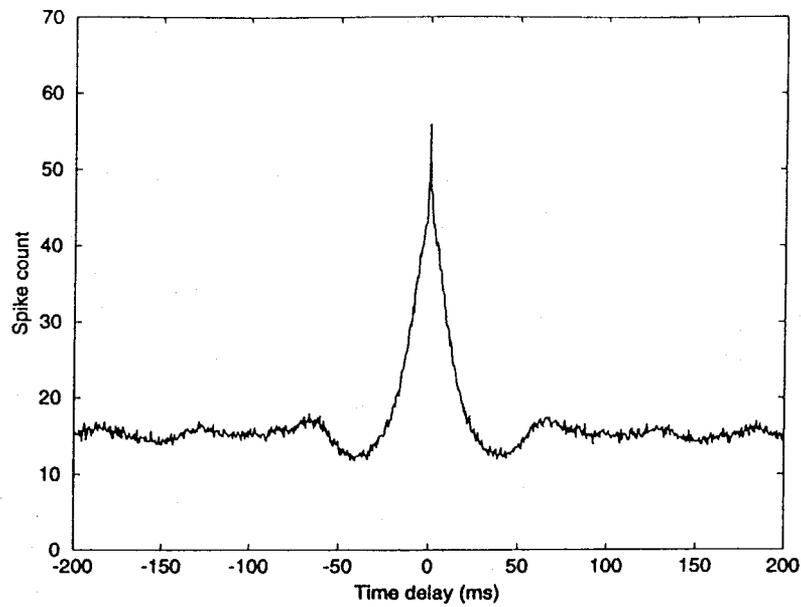


Figure 2. Average cross-correlogram for 100 pairs of excitatory neurons from those shown in the figure 1.

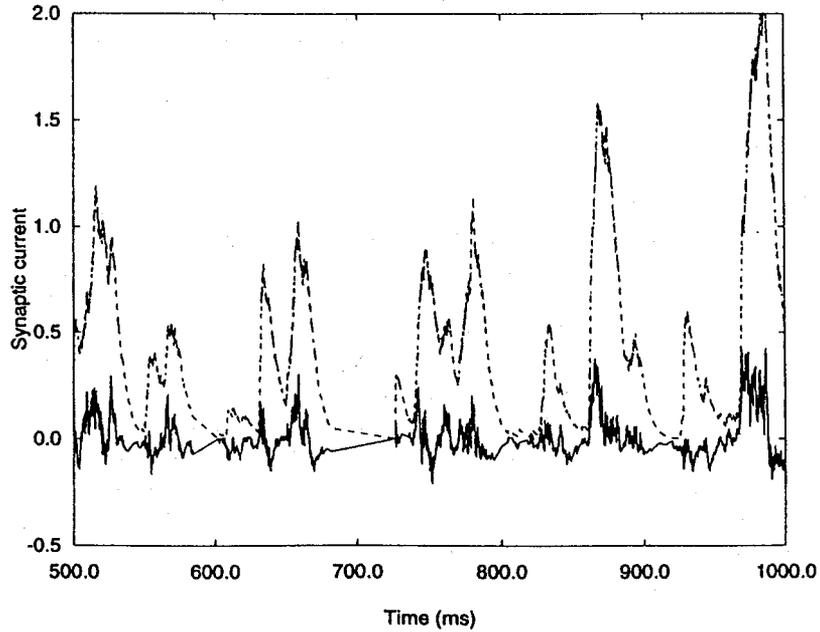


Figure 3. Demonstration of the balance between synaptic currents in a single model neuron from a network of interacting integrate-and-fire neurons. The dashed line is the summed excitatory synaptic currents and the solid line is a plot of the total synaptic current including both inhibitory and excitatory inputs.

compared with experimental measurements? The answer to this question depends on the dynamical state of the cortical network. The usual approach is to decrease the strengths of all synapses with the size of the network to conserve the average synaptic currents to a neuron, assuming that the presynaptic firing rates remain fixed. However, this form of scaling does not maintain the fluctuations of the current, at least under conditions when there is no synchrony. Given the variability observed in cortical neurons, this type of scaling may not be appropriate for modelling a visual column responding to an effective stimulus.

Another possibility would be to scale the reliability of synapses rather than their strength. In this approach, as the size of the network increases, each neuron experiences the same number of synaptic events of the fixed strength, and fluctuations will not change, at least when neurons in the model are not synchronized. In reality the situation is more complicated due to the change of the dynamical state with the growth of the network.

We illustrate the differences between these two ways of scaling by simulating networks with between 350 and 5600 neurons. The parameters in these networks were similar to those in figure 1, and 20% of the neurons in each network were inhibitory. The network with 5600 neurons scaled by synaptic strengths had more regular activity than the network with 350 neurons: the average coefficient of variation of interspike intervals (CV) fell from over 1.0 to about 0.15. In contrast, the network with 5600 neurons scaled by the synaptic reliabilities remained highly irregular with a CV of 0.8. Spike trains from these large networks and the dependence of CV on the size of the network are shown in figure 4.

3.3. Columnar organization

The cortex is topographically organized with a higher degree of connectivity within a vertical column than between columns. Neurons in an orientation column in primary visual cortex are arranged so that the preferred orientation changes gradually across columns except at occasional discontinuities [16]. These architectural features were incorporated into a model with 800 excitatory and 200 inhibitory neurons arranged in one-dimensional array. Each neuron was labelled with a preferred orientation from -90° to 90° , including both excitatory and inhibitory populations, and the neurons at the two boundaries were identified to give the network a circular topology to minimize boundary effects. In contrast to the previous network, synaptic connections were only allowed between neurons with preferred orientations differing by less than 30° for excitatory neurons, and 36° for inhibitory neurons. This included recurrent excitatory connections among the excitatory neurons, recurrent inhibitory connections among the inhibitory neurons, and reciprocal connections between the excitatory and inhibitory neurons. All the allowed connections had same strength, $K = 0.02$.

The input to the network was a broadly-tuned excitatory current close to the firing threshold, so that only a few neurons with preferred orientation near 0° received a superthreshold input, as shown in figure 5(a) (right panel). In the simulation shown in figure 5, the activity was restricted to neurons within 30° of the central peak of the input distribution. However, the firing patterns of the neurons within the column were highly irregular, similar to that observed in the homogeneous network. The average firing rates are plotted as a function of orientation in figure 5(b), which can also be considered the orientation tuning curve for a single neuron as a function of the orientation of the stimulus (position of the peak input). Note that during the continuous 1 s response in figure 5(a), the median of the distribution of spike firing shifted over time as the network fluctuated on a 50 ms time-scale.

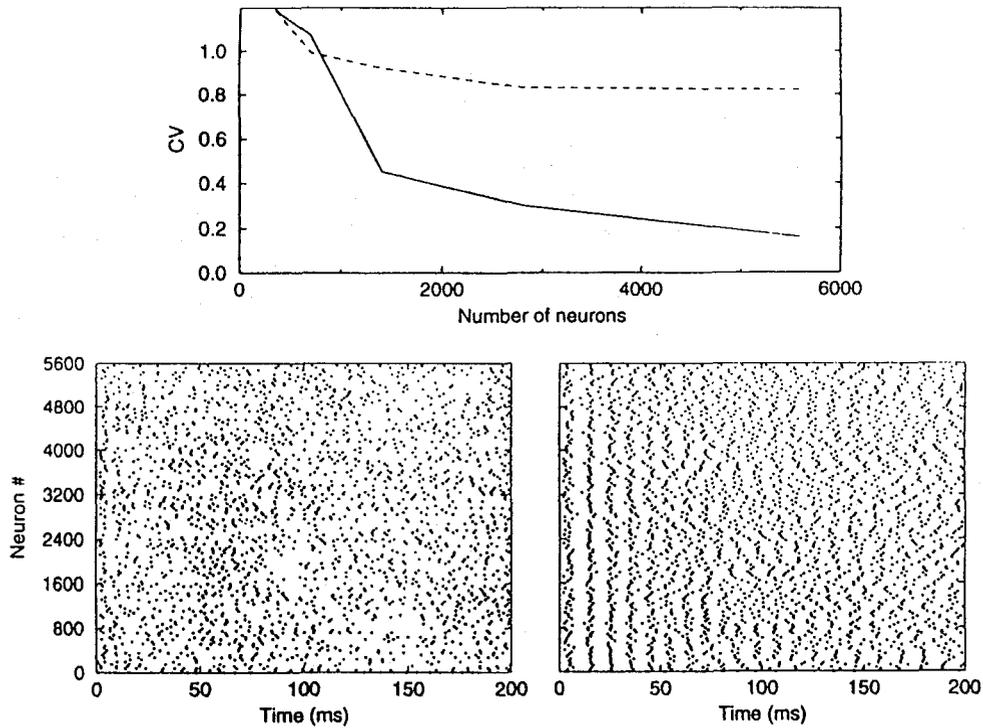


Figure 4. Comparison between two ways of scaling the synaptic parameters with the size of the network. The starting point was a network of 350 neurons (not shown), with parameters as in figure 1, except for $\tau_{ex} = 6$ ms, $K^{ex} = 0.02$, $K^{in} = 0.025$, $p_{in} = 1.0$ and $p_{ex} = 0.285$. Simulations of the networks with 4480 excitatory and 1120 inhibitory neurons are shown on the bottom. On the left the synaptic reliabilities were scaled to the values $p_{ex} = 0.0178$, $p_{in} = 0.0625$, with synaptic strengths left unchanged. On the right, the synaptic strengths were scaled to the values $K^{ex} = 0.0012$, $K^{in} = 0.0016$, without changing the reliabilities. At the top is the average coefficient of variation of interspike intervals (CV) as a function of the size of the network for scaling by synaptic strengths (full) and synaptic reliabilities (broken).

3.4. Dependence on the stimulus amplitude

We investigated the influence of the stimulus amplitude on the orientational selectivity of the network. The input shown in figure 5(a) was modified to keep the baseline constant while the peak excitation was varied from just above the threshold to 10% above threshold to mimic a change in the contrast of the stimulus. The family of orientation tuning curves is plotted in figure 6. Three features should be noted. First, as the input amplitude increased, the amplitude of the tuning curves grew significantly, without a significant increase of the tuning width. The invariance of the tuning width to contrast has been observed experimentally [25]. This effect is discussed in the framework of mean-field model in [26]. Second, as was already noted, when the input first crossed threshold, the activity of the network started with the finite response of about 20 spikes/s, indicating that the network was close to a bifurcation. Finally, as the input amplitude came closer to threshold, the network was activated with increasing delay, which also fluctuated strongly from trial to trial. For the lower curve in figure 6 the delay averaged 50 ms. In our simulations at low input, it took longer for neurons reach their thresholds and to bifurcate to an active state (see discussion).

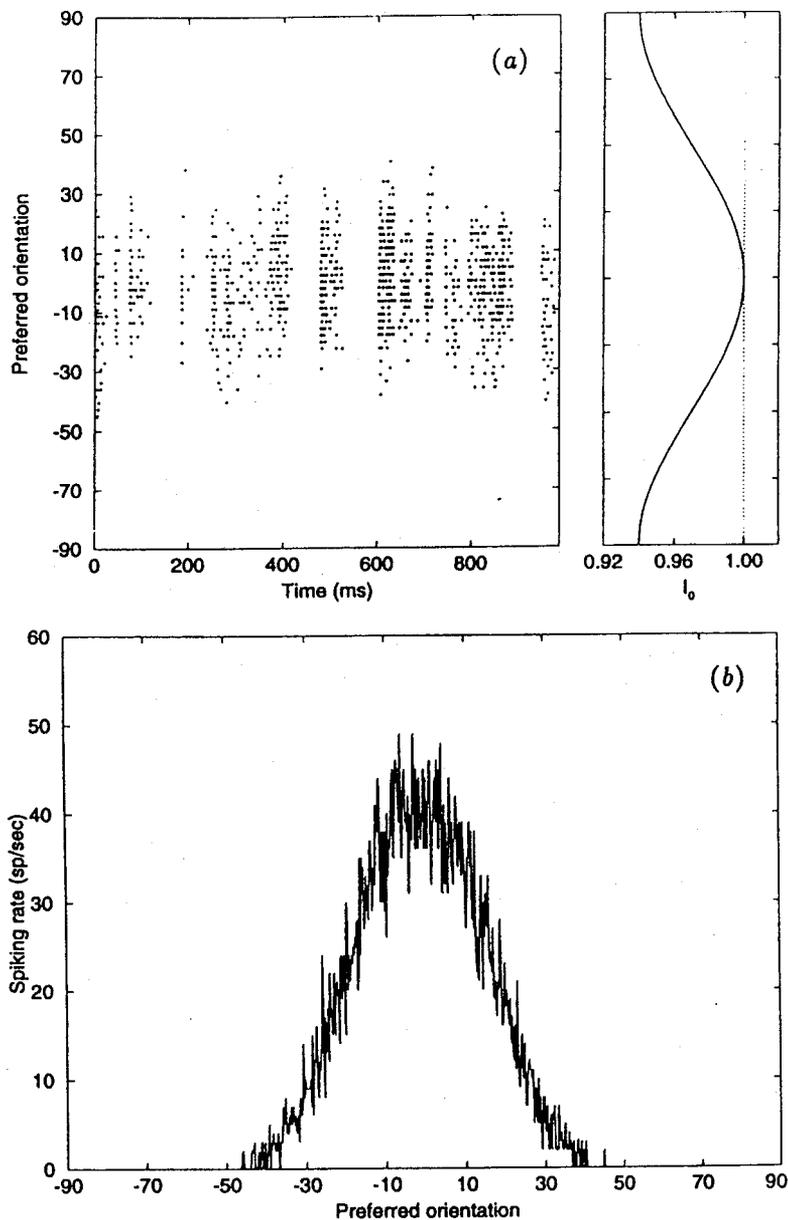


Figure 5. Model of the dynamics of an orientation column in the visual cortex. (a) Activity of a network of 800 excitatory and 200 inhibitory neurons with local connectivity (left panel). Neurons are labelled by their preferred orientation. Only the spikes of the excitatory neurons are shown. The full curve at the right is the external input, broadly tuned around 0° . Parameters are the same as in figure 1 except for $p_{ex} = 0.22$. (b) Average firing rates of the excitatory neurons as a function of their preferred orientation for the input shown in (a).

3.5. Rapid switching

When visual input to the cortex is rapidly switched following a saccade, it is important that the neurons in the primary visual cortex be able to shift rapidly from one configuration to

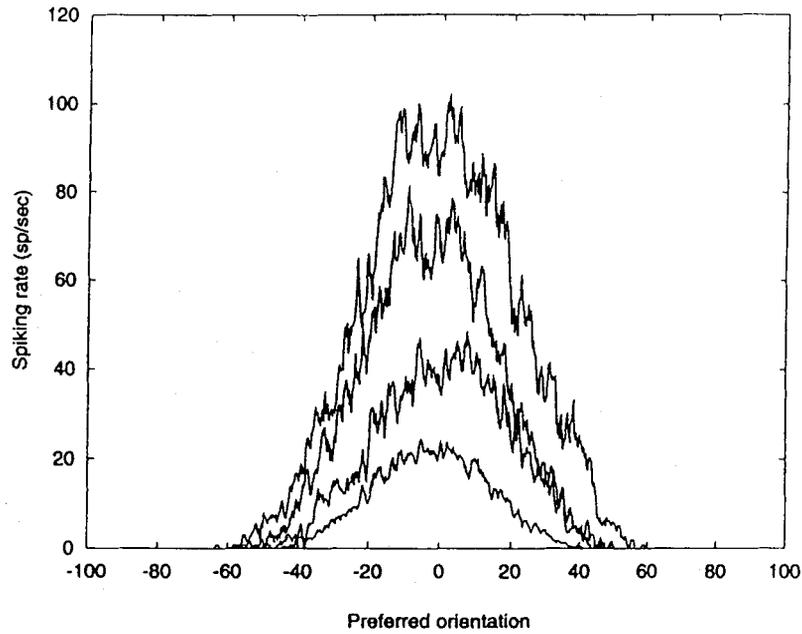


Figure 6. Tuning curves for neurons in the orientation column model. The same plot as in figure 5(b) with four different peak amplitudes of external input: 0.1%, 1%, 5% and 10% from bottom to top, respectively.

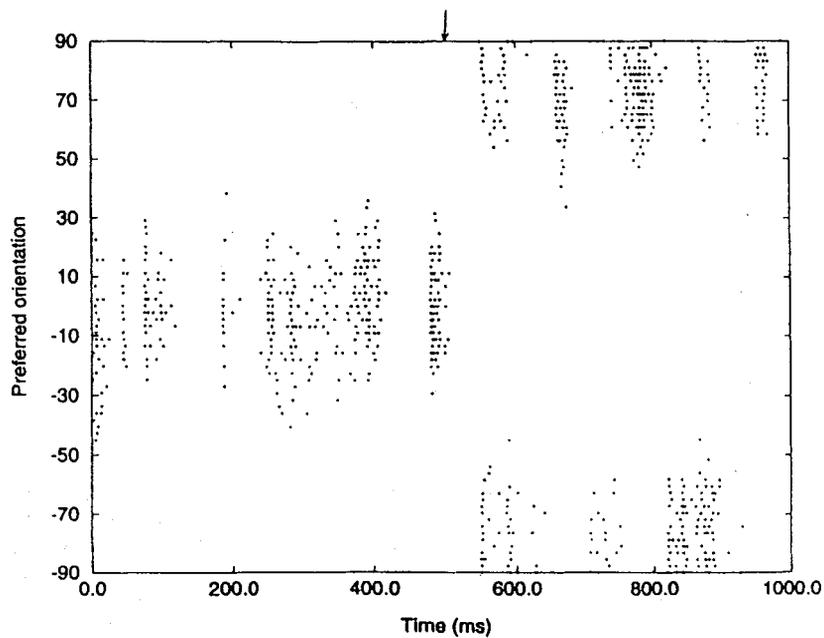


Figure 7. Rapid switching in a locally connected network, as in figure 5(a). The tuning curve of the external input instantaneously shifted from 0° to 90° at $t = 500$ ms. The arrow at the top indicates the time when the shift occurred.

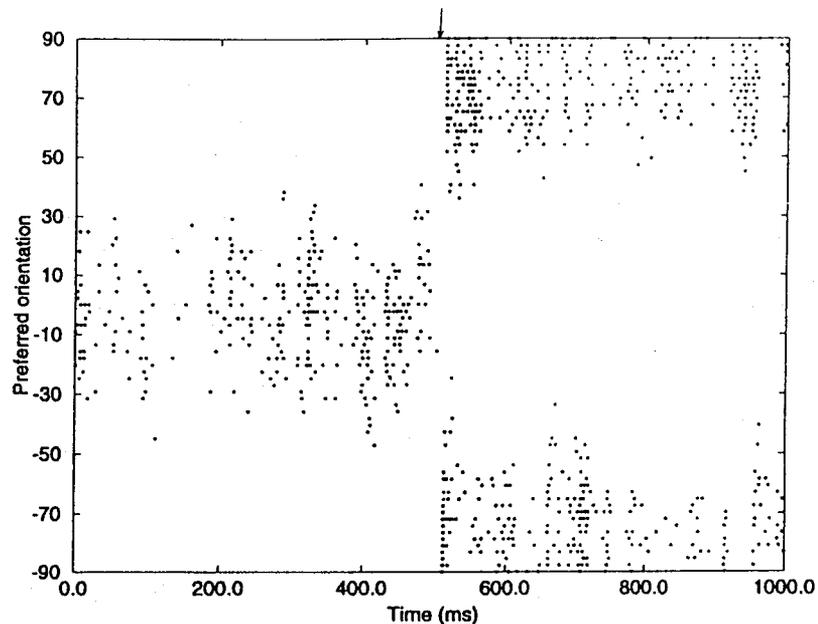


Figure 8. Rapid switching in a locally connected network as in figure 7, but with inhibition 20% faster than excitation. Parameters were the same as in figure 5 except for $\tau_{ex} = 6$ ms, $K^{in} = 0.025$ and $I_0 = 1.01$.

another. This was simulated by instantaneously shifting the peak of the input curve shown in figure 5(a) from 0° to 90° , as shown in figure 7. The network reorganized in less than 50 ms to a new firing pattern in response to the new input without activity moving through neurons with intermediate orientation preferences. The shift shown in figure 5 occurred during a transient burst of activity in the population around 0° , which was abruptly terminated. As in the homogeneous case, the speed with which the network shifted from one configuration to another was sensitive to the relative time constants of excitation and inhibition. The transitions were faster when inhibition was faster than excitation. In the simulations shown in figure 8, the transition time was about 10 ms when the time constant of excitation was 20% slower than that of inhibition.

4. Discussion

The simulations presented here demonstrate a new regime for neural network dynamics that occurs under conditions that match *in vivo* recordings from cortical neurons. This includes quantitative measures of the irregular firing patterns observed in cortical neurons and the cross-correlations between them. In agreement with experimental results of [17, 18], the orientation tuning of the excitatory and inhibitory input currents to a neuron had the same orientation tuning. Although external input currents were weak, the currents generated through feedback connections within the network were substantial. In the balanced regime, fluctuations of the synaptic input to a given neuron were large even if there was no correlation of the presynaptic activity.

4.1. Rapid state switching

The cerebral cortex receives a rapidly shifting report from the external environment and must extract fleeting information from this input stream. The balanced network can rapidly switch to a new state within a few tens of ms following a sudden shift in the input. This property of the network allows feedback-mediated cortical dynamics to contribute to the information processing on a short time-scale (see also [13]). This is compatible with experiments showing that the response properties of neurons in visual cortex were already specific within the first few tens of ms after the response onset [19, 20].

The balance condition, which was essential for permitting rapid state switching, depended on several features of the network architecture. Many conditions were tested, but the most robust results were obtained when the range of excitatory and inhibitory interactions were similar. In addition, it was important that the external inputs were only strong enough to bring the neurons to threshold; this insured that even those neurons not in an active state were fluctuating near threshold. Thus, only small shifts in the input were needed to produce a large change in the activity. These conditions illustrate the importance of network parameters, as well as internal properties such as a small reset, for achieving the balanced state of the neurons in the cortical network model.

A network with a similar architecture but with long-range inhibition, and operating in a mean-field regime was analyzed in [21, 26]. The mean-field model produced a distribution of *average* firing rates similar to that in the present model but without the large fluctuations observed in our model. A more dramatic difference between the two models emerged when the input was suddenly shifted. As shown in [26], the activity continuously 'moved' through neurons with intermediate preferred orientations until it matched the peak of the new input distribution (this was called 'mental rotation' in [22]). This effect was also demonstrated in a more realistic model of spiking neurons [23], where the cause of rotation was the active modulation of synapses rather than the shift of the input.

Another interesting dynamical aspect of the network is a bifurcation between the quiescent and active states. We found that as the external input amplitude crossed the threshold value, the network activity started with finite firing rates, but at threshold there was a longer delay that had variable duration. A dependence of the delay on the contrast of the stimulus has been observed in recording from the cortex [24]. In our simulations, the fluctuating input from thalamus and remote cortical areas was modelled as a constant input current. In the real situation, it may be that as the contrast is reduced to just noticeable values, the network experiences randomly positioned bifurcations to the active state with finite firing rates. The biological implications of this observation deserve further study with experimental and modelling techniques.

4.2. Network scaling

One way to characterize the collective properties of a network is to increase its size and observe its behaviour in the limit as it becomes infinite. Increasing the size of the present network with fixed values of synaptic strengths leads to increased variability. If the strengths of the synapses are normalized with the size of the network, the mean-field approximation becomes exact in this limit and the fluctuations are reduced to zero (called the thermodynamic limit for physical systems). This normalization can be justified only if fluctuations are small ($k \ll 1$, see (4)). We propose another way of scaling up the network by normalizing the synaptic reliability. Our simulations demonstrate that these two ways of scaling may lead to very different behaviours as the network size grows. The probabilities

for excitatory neurotransmitter release in our model ranged from $p_{ex} = 0.2$ to 0.01 (see figure 4). These small values are representative for the majority of excitatory synapses in the hippocampus [27, 28]. Our model demonstrates how reliable active states can emerge from highly unreliable synapses.

The network was stable over a wide range of parameters in our simulations. This was quite surprising since positive feedback from recurrent collateral between excitatory neurons tends to produce an explosion of activity. The key to self-stabilizing the network was fast feedback inhibition that balanced the excitation. The high gain of the circuit can amplify weak input signals without sacrificing steady-state stability. This feature of the network connectivity is similar to that of the canonical cortical microcircuit introduced by Douglas and Martin [18].

The synaptic strengths in the model were fixed, but in biological systems, the synaptic strengths may be modulated on fast and slow time-scales, depending on the firing rates of the neurons and other conditions [29]. The reliability of synapses may also be regulated. It would be interesting to investigate the behaviour of the model network with dynamically changing parameters. By changing some parameters, such as the reset amplitude, the network could switch between the mean-field regime and the strongly fluctuating one described in this paper and adjust its sensitivity to information on different time-scales. One of the most important properties of the input is its rate of change and we have already shown how the network with one set of parameters reacts to an instantaneous change. It will be of interest to explore other sets of parameters and other types of time-varying inputs.

The focus of this paper has been on a greatly simplified version of cortical architecture. The goal was to study the stability and switching behaviour in a new dynamical regime where fluctuations are strong. There are many extensions of the model that would make it more biologically realistic, including two-dimensional topography and additional populations of neurons representing cortical layers. In the cortex there is a variety of neuronal types that differ in their intrinsic properties as well as their connectivity and these need to be included to more accurately account for processing in visual cortex. These extensions are under investigation.

Acknowledgments

We greatly benefited from discussions with T Bell, C Koch, Z Mainen, M Shadlen and H Sompolinsky. We are also grateful to L Abbott, D Amit, H Sompolinsky and Z Mainen for critical reading of the manuscript.

Note added in proof. A model of orientation selectivity in cat visual cortex [30], based on recurrent excitation and similar in spirit to the more simplified model studied here, reached similar conclusions regarding cortical dynamics.

References

- [1] Smith D R and Smith G K 1965 A statistical analysis of the continual activity of single cortical neurons in the cat unanesthetized forebrain *Biophys. J.* **5** 47–74
- [2] Shadlen M and Newsome W 1994 Noise, neural codes and cortical organization *Current Biology* **4** 569–79
- [3] Softky W R and Koch C 1993 The highly irregular firing of cortical cells is inconsistent with temporal integration of random EPSPs *J. Neurosci.* **13** 334–450.
- [4] Abeles M 1991 *Corticonics* (New York: Cambridge University Press)
- [5] Douglas R J, Martin K A C and Whitteridge D 1991 An intracellular analysis of the visual responses of neurons in cat visual cortex *J. Physiol.* **440** 659–96
- [6] Sejnowski T J 1976 On the stochastic dynamics of neuronal interaction *Biol. Cybern.* **22** 203–11.

- [7] Amit D J, Evans M R and Abeles M 1990 Attractor neural networks with biological probe neurons *Network* **1** 381-405
- [8] Amit D J and Tsodyks M V 1992 Effective neurons and attractor neural networks in cortical environment *Network* **3** 121-37
- [9] Tsodyks M, Bell A, Mainen Z F and Sejnowski T J 1994 Why do cortical neurons spike irregularly? *Soc. Neurosci. Abs.* **20** 1527
- [10] Gerstein G and Mandelbrot B 1964 Random walk models for the spike activity of a single neuron *Biophys. J.* **4** 41-68
- [11] Wilson H R and Cowan J D 1974 Excitatory and inhibitory interactions of localized populations of model neurons *Biophys. J.* **12** 1-24
- [12] Amit D J and Tsodyks M V 1991 Quantitative study of attractor neural network retrieving at low spike rates I: Substrate—spikes, rates and neuronal gain *Network* **2** 259-73
- [13] Treves A 1993 Mean-field analysis of neuronal spike dynamics *Network* **4** 259-84
- [14] Stern P, Edwards F A and Sakmann B 1992 Fast and slow components of unitary EPSCs on stellate cells elicited by focal stimulation in slices of rat visual cortex *J. Physiol.* **449** 247-78
- [15] Abbott L F and van Vreeswijk C 1993 Asynchronous states in networks of pulse-coupled oscillators *Phys. Rev. E* **48** 1483-90
- [16] Hubel D H and Wiesel T N 1968 Receptive fields and functional architecture of monkey striate cortex *J. Physiol.* **195** 215-43
- [17] Ferster D 1986 Orientational selectivity of synaptic potentials in neurons of cat primary visual cortex *J. Neurosci.* **6** 1284-301
- [18] Douglas R J and Martin K A C 1991 A functional microcircuit for cat visual cortex *J. Physiol.* **440** 735-69
- [19] Tovee M J, Rolls E T, Treves A and Bellis R P 1993 Information encoding and the responses of single neurons in the primate temporal visual cortex *J. Neurophysiol.* **70** 640-54
- [20] Celebrini S, Thorpe S, Trotter Y and Imbert M 1993 Dynamics of orientation coding in area VI of the awake primate *Visual Neurosci.* **10** 811-25
- [21] Lukashin A V and Georgopoulos A P 1993 A dynamical neural network model for motor cortical activity during movement: population coding of movement trajectories *Biol. Cybern.* **69** 517-24
- [22] Georgopoulos A P, Lurito J T, Petrides M, Schwartz A B and Massey J T 1989 Mental rotation of the neuronal population vector *Science* **260** 47-52
- [23] Lukashin A V and Georgopoulos A P 1994 Directional operations in the motor cortex modelled by a neural network of spiking neurons *Biol. Cybern.* **71** 79-85
- [24] Dean A F and Tolhurst D J 1986 Factors influencing the temporal phase of response to bar and grating stimuli for simple cells in the cat striate cortex *Exp. Brain Res.* **62** 143-51
- [25] Skottun B C, Bradley A, Sclar G, Ohzawa I and Freeman R D 1987 The effect of contrast on visual orientation and spatial frequency *J. Neurophysiol.* **57** 773-86.
- [26] Ben-Yishai R, Lev Bar-Or R and Sompolinsky H 1994 Theory of Orientation Tuning in Visual Cortex *Hebrew University of Jerusalem Preprint*
- [27] Hessler N A, Shirke A M and Malinow R 1993 The probability of transmitter release at a mammalian central synapse *Nature* **366** 569-72
- [28] Rosenmund C, Clements J D and Westbrook G L 1993 Nonuniform probability of glutamate release at a hippocampal synapse *Science* **262** 754-7
- [29] Thompson A M and Deuchars J 1994 Temporal and spatial properties of local circuits in neocortex *Trends in Neuroscience* **17** 119-26
- [30] Somers D C, Nelson S B and Sur M 1995 An emergent model of orientation selectivity in cat visual cortical simple cells *J. Neurosci.* in press