Attractor dynamics in a modular network model of neocortex

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Abstract

Starting from the hypothesis that the mammalian neocortex to a first approximation functions as an associative memory of the attractor network type, we formulate a quantitative computational model of neocortical layers 2/3. The model employs biophysically detailed multi-compartmental model neurons and conductance based synapses and includes pyramidal cells and two types of inhibitory interneurons, i.e., regular spiking non-pyramidal cells and basket cells. The simulated network has a minicolumnar as well as a hypercolumnar modular structure and we propose that minicolumns rather than single cells are the basic computational units in neocortex. The minicolumns are represented in full scale and synaptic input to the different types of model neurons is carefully matched to reproduce experimentally measured values and to allow a quantitative reproduction of dynamic single cell behavior. Several key phenomena seen experimentally \textit{in vitro} and \textit{in vivo} appear as emergent features of this model. It exhibits a robust and fast attractor dynamics with pattern completion and pattern rivalry and it suggests an explanation for the so-called attentional blink phenomenon. During assembly dynamics, the model faithfully reproduces several features of local UP states, as they have been experimentally observed \textit{in vitro}, as well as oscillatory behavior similar to that observed in the neocortex.

Keywords:

Introduction

Many different kinds of computational models of the cerebral cortex have been developed and investigated, ranging from bottom-up ones incorporating extensive biophysical detail, to top-down connectionist type models that mostly address issues at the level of gross cortical plasticity and dynamics. Unfortunately, a gap exists between the two classes of models. Attempts to unify computational paradigms, methodology and results obtained with models of different types for the same system, e.g., the primary visual cortex, are still scarce. At the same time, it is quite clear that in order to enable an understanding of high-level cognitive phenomena in terms of processes at the molecular and cellular level such an approach is necessary.
In the last decades, we have seen a dramatic growth in quantitative experimental data imposing increasingly strict constraints on models. This has triggered a significant convergence of computational theories of cortical function. However, each one of these models is still only capable of addressing part of the complex functionality of the brain, focusing on, e.g., self-organization of neuronal response properties, learning and associative memory dynamics, distributed population coding or oscillatory phenomena related to perceptual processing and binding.

We will here apply a top-down view of the neocortex as an associative attractor memory network, considering the underlying neuronal architecture together with the dynamics of processing and recall in such an associative memory. This view has its origin more than fifty years back in Hebb’s theories of cell assemblies (see, e.g., Fuster 1995 for a review). It has been mathematically instantiated in the form of the Willshaw–Palm (Willshaw & Longuet-Higgins 1969; Palm 1982) and Little-Hopfield models (Hopfield 1982) and has subsequently been elaborated and analyzed in great detail (Amit 1989; Hertz et al. 1991). The olfactory (piriform) cortex (Haberly & Bower 1989) as well as the hippocampus CA3 field (Treves & Rolls 1994) have previously been perceived and modeled as prototypical neuronal auto-associative attractor memory networks. More recently, persistent activity in an attractor memory of a similar kind has been suggested to underlie prefrontal working memory, although in that case the attractor itself and not the connectivity matrix is assumed to hold the memory (Compte et al. 2000).

The attractor memory paradigm of cortex has recently gained further experimental support from in vitro experiments on brain slices (Cossart et al. 2003; Shu et al. 2003) where so-called UP states were observed. In a local UP state, which about 0.5% of the recorded pyramidal cells simultaneously enters, the cells depolarize some 10 mV and increase their spiking frequency to some 20 Hz. Such an UP state builds up in about 60 ms and lasts for several hundreds of milliseconds. It has been suggested that attractor dynamics may explain these local UP states (Cossart et al. 2003). Cossart et al. (2003) also pointed out the similarities between the local UP states and the persistent activity states seen in working memory of behaving monkeys.

Abstract attractor memory models of cortex have typically focused on fix-point dynamics in a network with a structured, learned connectivity matrix. In neural terms this corresponds to a dynamics on a timescale of some hundred milliseconds. In contrast, biophysically detailed models are typically implemented with an unstructured, random or center-surround (“bump-shaped”) connectivity and the dynamics at several different time scales—from milliseconds to seconds—are studied. The focus is often on the dynamics per se like oscillations and spike timing without a strong connection to associative memory retrieval. Only by bridging the gap between such different types of models will we be able to organize and interpret the overwhelming amounts of experimental data at different levels of granularity concerning cortical structure and function. As an example, starting at the network level from a theory of fix-point attractor dynamics, by introducing cellular level mechanisms like accumulated afterhyperpolarization and synaptic depression, attractors may become quasi-stable and one obtains continuously evolving dynamics (Sandberg et al. 2003).

In this spirit, in a previous series of papers we have demonstrated how it is possible to connect a top-down view of auto-associative attractor memory with the details of layer 2/3 cortical memory network architecture and dynamics (Lansner & Fransen 1992; Fransen & Lansner 1995; Fransen & Lansner 1998). We found that such a model could indeed perform critical cell assembly operations, i.e., pattern retrieval, completion and rivalry. In this paper, we continue the same line of reasoning and construct and examine a similar network model that is more faithful to neocortical neurobiology.
On the network level, we have added a hypercolumnar structure on top of the previous minicolumnar one. The hypercolumns act as soft winner-take-all modules typically selecting one of its minicolumns to be active at a time via lateral inhibition. Further, the minicolumns themselves have been scaled up to full scale (relative to their layer 2/3 portions), while the minicolumns in each hypercolumn are subsampled. Due to the full scale minicolumns, we could better reproduce the quantitative behavior and connectivity of its subparts, and each single neuron sees approximately the same synaptic input as it would see in vivo. Further, we have included an additional type of inhibitory neuron, of the vertically projecting regular spiking type, so that both vertically and horizontally projecting, as well as fast and regular, spiking types of interneurons are now present in the model. Finally, we have added fast pyramidal–pyramidal synaptic depression and suggest a role for its presence.

### Model neurons and synapses

The cell models used in the simulations described here are conductance-based and multi-compartmental of intermediate complexity. They are to a large extent similar to those in the previous network model (Fransén & Lansner 1995; Fransén & Lansner 1998). Tables of neuron and synapse parameters can be found in the Appendix.

An important role of the model is to serve as a tool for exploring different possible experimental manipulations. In this work, we use conductance based biophysically detailed model neurons and synapses in which the different ion channels and currents are explicitly represented. This makes it easier to relate to experimental data and to perform informative in silico experiments, like, e.g., blocking or knocking-out of a particular type of ion channel. This is not equally straightforward when using simpler neuron models (Gerstner 2002; Izhikevich 2004), since in this case there is no direct mapping between ion channel properties and parameters.

The cells included are layer 2/3 pyramidal cells and two different types of inhibitory interneurons, assumed to correspond to horizontally projecting basket cells and vertically projecting double bouquet and/or bipolar cells (Kawaguchi & Kubota 1993; Douglas & Martin 2004; Markram et al. 2004). Following Kawaguchi (1995), we will here refer to the latter type as regular spiking non-pyramidal or RSNP cells.

The network connectivity is set up to store a number of memory patterns (attractor states) such as would have resulted from long-term plasticity using a Hebbian learning rule (Sandberg et al. 2002). The synaptic plasticity actually included in the model is restricted to fast synaptic depression of pyramidal to pyramidal synapses, on the same time scale as the network dynamics studied.

### Model neurons

The pyramidal cell is modeled with six compartments (soma, initial segment, basal dendrite compartment and three consecutive apical dendrite compartments). The dendritic tree has one apical and one basal branch separating synaptic input of different origin. The soma diameter is sampled from a distribution within $21 \pm 2 \mu m$ in order to represent biological variation of cell sizes within this population. This distribution in size, together with stochastic noise and random local connections also prevents, e.g., synchronization artifacts due to identical cell excitabilities. The modeled pyramidal cell is of a regularly spiking type. A short supra-threshold stimulation gives a relatively wide spike followed by an early and a late afterhyperpolarization (AHP) separated by a small depolarizing afterpotential (Figure 1). At longer stimulations, the cell has a pronounced adaptation caused by accumulation of
Figure 1. Synaptic input to a postsynaptic model pyramidal cell. A: Spiking of a model pyramidal cell resulting from a current injection of 0.10 nA for 200 ms. B: Spiking of an RSNP cell resulting from a current injection of 0.010 nA for 100 ms. C: Spiking of a basket cell resulting from injection of the same amount of current as in B. Note the lack of adaptation. D: EPSP:s and IPSP:s in a pyramidal cell receiving a depressing glutamatergic synapse from the pyramidal cell in A and inhibitory GABA-ergic synapses from the RSNP and basket cells in B and C, respectively. The resting potential of the postsynaptic cell was −63 mV.

calcium entering via voltage gated Ca-channels and activation of $K_{Ca}$ channels. The decay time constant of the associated $Ca_V$-pool process is 159 ms.

The basket cell is modeled as non-adapting, relatively fast-spiking with three compartments (soma, initial segment and dendritic). The soma diameter is $7 \pm 1 \, \mu m$. The I-f curve is rather linear up to some 200 Hz. The action potential repolarization of the modeled cell is fast followed by a large fast AHP with a depth of about 12 mV but there is virtually no late AHP. Figure 1C shows a synaptically activated basket cell.

The RSNP cell has the same size and passive properties as the basket cell, but a different I-f curve. We have matched the adaptation of the modeled RSNP cells to the RSNP-SS cell in Cauli et al. (2000). The modeled cell reproduces the early adaptation, late adaptation, fast AHP and minimal frequency given there (Figure 1B). The RSNP cell is slightly less adapting than the pyramidal cell. A constant depolarizing current of 0.015 nA will make the RSNP cell fire at 100 Hz initially and at about 40 Hz after 800 ms.

Model synapses

There are both glutamatergic and GABA-ergic synapses in our network model. Glutamatergic pyramidal–pyramidal and pyramidal-RSNP transmission is provided by a mix of kainate/AMPA and voltage dependent excitatory NMDA receptor gated channels in the proportion 80:20. The glutamatergic synapses from pyramidal to basket cells are purely
Figure 2. Connectivity densities and PSP amplitudes of the subsampled network model. Hypercolumns are show with light grey background, minicolumns with dark grey. The percentage is given as the chance of one cell of the pre-population being coupled to one cell of the post-population. Note that global connectivity is exaggerated since both the number of hypercolumns and minicolumns within each hypercolumn is down-scaled. Each cell sees about the same number of active synapses as it would in vivo assuming 1% activity. Holding potential of postsynaptic cells were $-54 \text{ mV}$ for inhibitory cells and $-63 \text{ mV}$ for pyramidal cells.

1Connectivity of pyramid-RSNP cells given the two minicolumns are in different patterns, otherwise 0%.

2Global connectivity of pyramid-pyramid given the two minicolumns are in the same pattern, otherwise 0%.

kainate/AMPA. The inhibitory GABA-ergic transmission is solely exerted by $\text{GABA}_A$. All synapses are saturating such that the postsynaptic receptor pool is fully saturated by one presynaptic spike (Fransén & Lansner 1995). In general, the synaptic conductances are different from those in the earlier model, see connectivity diagram in Figure 2.

A local synapse between pyramidal cells gives an initial EPSP of about 2.4 mV for a postsynaptic membrane potential of $-63 \text{ mV}$, while global pyramid to pyramid synapses give an initial EPSP of 0.3 mV at the same membrane potential. Thomson measures an EPSP of $1.7 \pm 1.3$ at $-68$ to $-80 \text{ mV}$ membrane potential between local layer 2/3 pyramidal cells (Thomson et al. 2002) and long-range connections are estimated to be one order of magnitude weaker (Gilbert et al. 1990). We incorporate $\text{Ca}^{2+}$ permeability in our NMDA synapse; $\text{Ca}^{2+}$ enters through the NMDA-channels and builds up in the $\text{Ca}_{\text{NMDA}}$ pool postsynaptic cell. This will activate the calcium dependent potassium channels, which will hyperpolarize the cell (Shah & Haylett 2002). The pyramidal to pyramidal synapses are further depressing according to the model of Tsodyks and Markram (Reyes et al. 1998; Tsodyks et al. 2000) with $U = 0.25$ and $\tau_{rec} = 575 \text{ ms}$.

Our pyramidal to inhibitory cell synapses are neither depressing nor facilitating. The conductance of the pyramidal to basket and RSNP cell synapses gives an EPSP of 2.5 mV at a membrane potential of $-54 \text{ mV}$, to be compared to experimental data for layer 2/3 pyramidal to layer 2/3 inhibitory cells which gives $1.9 \pm 1.6 \text{ mV}$ at $-60$ to $-80 \text{ mV}$ (Thomson et al. 2002).

The inhibitory connections in the model are local within the mini- or hypercolumn and act only on pyramidal cells (Figure 2), following the experimental observation that synapses between inhibitory interneurons are known to be quite rare in cortex (Markram et al. 2004). Thomson et al. (2002) describe interneuron to pyramidal IPSP:s in layer 2/3 to have an amplitude of $0.65 \pm 0.44 \text{ mV}$ at $-55$ to $-65 \text{ mV}$ in rat neocortex. In our model, the IPSP:s
from the RSNP cells are slightly stronger, $-1.5 \text{ mV}$ and $-1.2 \text{ mV}$ for the basket cells, both measured in a pyramidal cell at a membrane potential of $-63 \text{ mV}$. An important difference between the two types is that basket cells target the soma of the pyramidal cells, while the RSNP cells target their second apical dendritic compartment.

**Architecture of the network model**

Our abstract view of cortical associative memory has been expressed in the form of a connectionist type of attractor memory network (Sandberg et al. 2002; Lansner et al. 2003; Sandberg et al. 2003; Djurfeldt et al. 2006). The computational units of this network have been mapped in the present study not to individual neurons but to groups of neurons. The groups correspond to minicolumns, such as the orientation minicolumn in the primary visual cortex. This abstract framework also suggests modularity in terms of a hypercolumnar organization of a similar kind to that described by Hubel & Wiesel (1977) for the primary visual cortex, i.e., bundles of about 100–200 minicolumns forming a hypercolumn.

Each hypercolumn operates like a soft winner-take-all module in which activity is normalized among the minicolumns, such that their summed activity is kept approximately constant. We propose that the lateral inhibition mediated by basket cells may achieve this normalization in cortex. Normalization models of a similar kind have recently been proposed for the primary visual cortex (Blakeslee & McCourt 2004).

**The full-scale conceptual model**

Our minicolumnar network model is restricted to the layer 2/3 portion of neocortex. Each minicolumn is assumed to have a diameter of about 30 $\mu \text{m}$ and to be comprised of 30 pyramidal and two RSNP cells (Peters & Sethares 1997). The RSNP cells are innervated from outside the local hypercolumn and provide inhibition to the pyramidal cells in their minicolumn. Since they inhibit locally, they can act as inverters, changing the sign of incoming long-range connections. We propose that this is how the equivalent of the negative components in an abstract connection matrix is set up in cortex. The long-range excitatory connections, from a pyramidal cell in one minicolumn to a pyramidal cell in a minicolumn of a different hypercolumn implement the positive weights between units.

Our full-scale conceptual network model has about one hundred such minicolumns bundled into a hypercolumn with a diameter of 500 $\mu \text{m}$ (Figure 3) laid out irregularly on a 2D surface patch. The number of minicolumns per hypercolumn is motivated both by geometrical constraints and by the activity density of 0.5–1 % often given for cortex (Cossart et al. 2003; Lennie 2003). Since the hypercolumn normalizes activity to correspond to one fully active minicolumn in a hypercolumn this gives such a number.

According to Markram et al. (2004) there is about 150 large basket cells within the layer 2/3 portion of a hypercolumn. According to Binzegger et al. (2004), between 37% and 54% of inhibitory cells in layer 2/3 of cat V1 are RSNP cells, 3.4% are Chandelier cells (not included in the model) and the rest (42–60%) are mainly basket cells, where not all are included in the model. Our model thus recreates this composition well since the full hypercolumn model has 3000 pyramidal cells, 200 RSNP cells and 100 basket cells. The ratio of excitatory to inhibitory neurons in the hypercolumn and the entire network model is 90:10. This number of inhibitory neurons is slightly on the lower side compared to what is known from the data. This is because not all types of inhibitory cells have been included in the model.

A network corresponding to a cortical area could be regarded as a mosaic of such hypercolumns. For instance, a network with the size of cat striate cortex would be comprised
of some 320000 minicolumns organized in around three thousand hypercolumns (Peters & Yilmaz 1993). It would consist of some 20 million neurons and hundred billion synaptic connections. Such a network would have about a thousand active minicolumns in each pattern. The density of the long-range associative connectivity would be about 0.02% (Johansson 2004; Johansson & Lansner 2004).
The subsampled simulation model

The full-scale albeit simplified model of the layer 2/3 network in an entire cortical area is beyond what could be simulated on today’s readily available computers. We have therefore developed a subsampled network model from the conceptual full-scale model described above. This has been done while carefully conserving as much as possible the summed synaptic conductances of active synapses onto postsynaptic cells of different types. Since the number of incoming connections tends to shrink as the network size is decreased some compensation is needed. One solution is to increase synaptic conductances, but that tends to distort the dynamics of the network since the input looks like coming from (artificially) synchronized presynaptic neurons. An alternative way is to increase the connection density between cells which counteracts such a possible artifact. The latter is the path we have chosen.

Our model is subsampled compared to a full cortical area in terms of the total number of hypercolumns and in terms of the number of minicolumns in each hypercolumn. The subsampled network we use comprises nine adjacent hypercolumns in each of which eight minicolumns and eight basket cells are represented (Figure 3). This makes a total of 2160 pyramidal cells, 144 RSNP cells, and 72 basket cells connected by 257000 synapses of which 94% are excitatory. The high percentage of excitatory synapses is a result of the subsampling of the number of hypercolumns, which has lead to an exaggeration of the density of long-range connections, which are all excitatory. The basket cells included comprise the local population that is realistically within reach of the eight minicolumns in each hypercolumn (Holmgren et al. 2003). Eight orthogonal patterns were stored in the network, implying that activity in an attractor state engages one minicolumn in each hypercolumn. Though this is certainly an artificial configuration it is not qualitatively unlike the situation in a large sparse distributed attractor memory storing randomly generated patterns. The underlying assumption is that the preprocessing stages in thalamus and cortical layer 4 have decorrelated the input patterns to such an extent that they have a statistics similar to that of random patterns.

Local and global connectivity

As can be seen in Figure 2, the connection density between pyramidal cells in the same minicolumn is set to 25% (Thomson et al. 2002). The two RSNP-cells in a minicolumn project vertically to 70% of the pyramidal cells. The basket cells receive input from 70% of the pyramidal cells in the hypercolumn and project back horizontally to 70% of them.

Pyramidal cells that share pattern have a long-range connectivity of 30%. Since there are no connections between pyramidal cells not sharing pattern, the average pyramidal–pyramidal long-range connectivity is 3.75%. As the number of the minicolumns and hypercolumns is scaled up, this connectivity will decrease further, while it will increase somewhat if non-orthogonal patterns are introduced. RSNP cells also receive long-range connections from pyramidal cells.

A pyramidal cell in an active pattern receives excitatory input from on average about a hundred pyramidal cells, 9 local and 90 long-range. The RSNP-cells are in turn excited by on average 42 pyramidal cells given its minicolumn is not part of the active pattern. Since we found no data on the estimated number of synapses onto RSNP-cells \textit{in vivo}, we have assumed that the number of pyramidal cells in one minicolumn contacting another minicolumn is constant whether they contact pyramidal or RSNP-cells.
Noise and input from layer 4

In addition to the synapses originating from other cells in the model, the pyramidal cells also receive synapses carrying spikes from Poisson processes. These synapses simulate unspecific input from various other areas and structures. The noise level was set to create a background firing rate of the modeled pyramidal cells. The level of background activity is 2.1 Hz with all other excitation blocked and 3.5 Hz with the inhibitory synaptic input blocked as well.

To simulate input from layer 4, we have five pyramidal cells in each minicolumn projecting onto the layer 2/3 pyramidal cells. These layer 4 pyramidal cells can be selectively activated by a stimulating current giving them a specific firing frequency over some period of time. Their spiking frequency was calculated from the number of cells in layer 4 likely to provide input to a layer 2/3 pyramidal, estimated to \( \sim 30 \) with a spiking frequency of \( \sim 10 \) Hz, and a connectivity density of 25% (Thomson et al. 2002). There are no recurrent connections back from layer 2/3 or between layer 4 pyramidal cells. The synapses of layer 4 to layer 2/3 connections are of a mixed NMDA and kainite/AMPA type and have the same strengths as the local layer 2/3 excitatory connections.

Simulation software and execution time

Our network model is implemented using SPLIT, a MPI based simulation library in C++ developed by our group to allow for large scale parallel simulations of conductance based neurons (Hammarlund & Ekeberg 1998). We ran the model both on single PCs and on small clusters of computers. Simulating 1s of the subsampled model (2460 neurons and 257000 synapses) on a PC with a 2.4 GHz Pentium IV processor and 1 GB RAM took about 10 minutes. SPLIT allows for simulation of networks with millions of neurons and billions of synapses on high-end cluster computers (Djurfeldt et al. 2006).

Results and model analysis

We have applied several methods to analyze and characterize the activity in the model network. Our ambition has been to apply standard methods, in order that our results should be comparable to existing analyses of other simulation and experimental data. We also study the network’s ability to perform basic attractor network operations like pattern completion and pattern rivalry. In particular the following aspects of network behavior have been studied:

1. Attractor state properties and UP states.
2. Pattern completion and rivalry.
3. Attentional blink.
4. Temporal fine structure.

In addition, the model’s sensitivity to variation of some critical parameter values was evaluated. Next, we describe briefly methods for each of these and the results from the analysis of simulation output from the model.

Attractor state properties and UP states

We investigated the properties of our network model that allow it to function as an attractor memory. For instance, how do cellular and network parameters affect attractor function and its properties, such as the settling time and duration (dwell time) of an attractor state.
To that end, we aggregated and low pass filtered the spike output of the pyramidal cells in a minicolumn and used this measure to determine whether the network was in an UP or DOWN state. We then characterized the states based on spike rate, membrane potential and the nature of state transitions, and compare to experimental data.

One of our main hypotheses is that UP and DOWN states are expressions of the underlying attractor dynamics: when an attractor is active its cells are in an UP state, when the attractor is not active its cells are in a DOWN state. To test this hypothesis, we studied the quantitative behavior of the pyramidal cells during pattern retrieval in order to compare it to the UP and DOWN state behaviors seen in vitro.

When subjected to a low level of random input noise, the network model exhibits spontaneous activity, alternating between the stored patterns. The network visits one to two states per second (Figure 4A). For individual neurons, the global alternation between patterns means that their synaptic inputs vary accordingly. When a pattern of which a given neuron takes part is active, the number of depolarizing synaptic events is high and we find a raised membrane potential as well as a significant increase in spike rate.

Figure 4B–F shows typical firing patterns of the different cell types in the model network during such activity. As can be seen, the pyramidal cells fire irregularly and with a low average spike rate, whereas both types of inhibitory interneurons fire more continuously and with a higher rate. The main difference between the basket and RSNP cells is the tendency of the latter to make occasional pauses of some hundred milliseconds duration. This happens when the minicolumn in which the cell is located participates in an attractor state.

Looking closer at the pyramidal cells, we find that they in fact display most of the prominent characteristics of UP and DOWN states as observed experimentally (Steriade et al. 1996; Cossart et al. 2003). In a DOWN state, the average soma potential is $-64.6 \pm 0.2$ mV and the spike frequency is 0.06 Hz; in an UP state these values are $-57.8 \pm 0.1$ mV and 5.6 Hz. The UP states lasts for some 500–1000 ms and the rise of soma potential at their onset happens during about 50 ms. We did not see an exponential decay after termination of an UP state; this is overshadowed by inputs from the next active pattern. We did, however, see more activity in the beginning of UP states compared to the middle and the end, as described by Cossart et al. (2003), and the peak frequency was at about 14 Hz (see Figure 4G).

**Pattern completion and rivalry**

Pattern completion is a basic property of associative memory. In our case, when some of the minicolumns in a pattern are stimulated or activated, it is desired that this activity spread to activate the inactive minicolumns in the pattern. We test this by stimulating just a subset of the minicolumns participating in a pattern by simulated layer 4 input. Equally important is pattern rivalry; competition between patterns. If two or more patterns are simultaneously stimulated, one would expect the pattern that receives the strongest stimulation to be activated. We study this by injecting ambiguous layer 4 input, stimulating more than one pattern.

To test pattern completion, we stimulate 4 out of 9 minicolumns in a pattern with simulated layer 4 input for 120 ms. Figure 5 shows a spike raster plot of the pyramidal cells participating in the stimulated assembly and also the intracellular potential of three of the cells, two directly stimulated and one indirectly activated through pattern completion. It turns out that completion is very robust and fast; after just one gamma burst from the four stimulated minicolumns, the others are activated. Table 1 shows that the latency from stimulus onset to activation for stimulated and non-stimulated cells was around 15 ms and 45 ms, respectively.
Figure 4. Spontaneous network activity. A: Raster plot of spike activity in the entire network; the topmost, rapidly firing cells are the RSNP cells, then follows the pyramidal cells (sorted by hypercolumn and minicolumn) and finally the basket cells. Simulated time is 5 seconds and different patterns take turns being active, with short transitional periods in between. B, C: Soma potential of two pyramidal cells in pattern #1. Membrane potential and spike rate are clearly elevated in the UP state. D: Soma potential of a pyramidal cell active in a different pattern. E: Soma potential of an RSNP cell from the same minicolumn as the pyramidal cell shown in B. F: Soma potential of a basket cell from the same hypercolumns as the pyramidal cells in B and C. G: Mean spike frequency of the pyramidal cells in pattern #1, showing UP and DOWN states, the spike frequency being highest at the beginning of UP states.
Figure 5. Pattern completion when only part of a pattern is directly stimulated. Upper panel: Raster plot of the pyramidal cells from two different assemblies; one that is initially active (crosses) and one that is activated by an external stimulus (dots). Lower panel: Soma potentials for three pyramidal cells in the stimulated pattern. The upper two cells are from minicolumns that were directly stimulated; the third cell is taken from a recruited minicolumn. Stimulation is marked grey.

Similarly, we test for pattern rivalry by stimulating different numbers of minicolumns from two different assemblies. We find that the assembly with more stimulation becomes active; the others activity is terminated. Figure 6 shows a case where an assembly that receives stimulation to four minicolumns wins over one receiving input to three, which is always the outcome of this case. Table 1 shows that the latency from stimulus onset to activation of the winning assembly for stimulated and non-stimulated cells was around 40 ms and 60 ms, respectively. The suppressed assembly gets active only when the winning one has terminated its activity.

<table>
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<th>Condition</th>
<th>Minicolumns</th>
<th>From start, mean</th>
<th>From start, stdev</th>
<th>From rise, mean</th>
<th>From rise, stdev</th>
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<tr>
<td>Compl., strong</td>
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<td>3</td>
<td>13</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Stim</td>
<td>14</td>
<td>3</td>
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<tr>
<td></td>
<td>Stimul</td>
<td>24</td>
<td>11</td>
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We also tested some more intricate cases. What happens for example when two assemblies receive exactly the same stimulation, but in one it is spread out over more minicolumns, i.e., one of the inputs is more redundant than the other? We stimulated a total of 81 cells in each of two different assemblies. Which of the two would win was now dependent on the distribution of the stimulation. For weaker stimulation, the assembly which had a more concentrated input defeated the other one. If the strength of the input was increased, however, the one with more spread-out input was the one that survived. This relation depended on the local connectivity. If that was decreased from our standard 25% connectivity between pyramidal cells in a minicolumn to 10%, the more concentrated input gained in effectiveness relative to the more spread-out one. We can explain these results in the following way: When input was weak, input concentrated to a group of cells with high connectivity helped the minicolumn to become active, and the activity spread from there once established locally. But if the strength and intensity of the input was increased, the local connectivity and interaction became less important. The stimulated minicolumn would then always be activated, but once that was the case, the further increase in activity level would not increase linearly to the increase in input. This is because the stimulated pyramidal cells will drive the basket cells reciprocally connected to them. The stable activity level of a minicolumn is more or less fixed. Instead, the spread-out activity would in this case activate a larger number of minicolumns and the
corresponding assembly would win. To complicate this static picture, there were also dynamic effects such that an assembly that was recently active would be less easy to reactivate.

**Input sensitivity and attentional blink**

“Attentional blink” is a phenomenon studied by experimental psychologists (Shapiro et al. 1994; Marois & Ivanoff 2005). Subjects are, for instance, told to look for two different target letters (T1, T2) in a flow of letters blinking on a screen at a steady pace. If this pace is quick enough and if T2 follows shortly after T1 (with a short stimulus onset asynchrony, SOA), T2 is not perceived. Similar phenomena can also be elicited in the auditory modality and it thus appears to be a general perceptual bottleneck. The neural basis of the attentional blink effect remains unclear, but it is correlated with the P300 peak in ERP, a neuro-physiological marker of working memory updating.

We suggest here that the attentional blink phenomenon is due to the subject’s working memory entering an attractor state in response to T1. The afferent activity triggered by the closely following letter T2 is not able to terminate the newly entered attractor state. In the model, this translates to layer 4 input being unable to terminate an active attractor state if it follows shortly after such a state was activated.

We test the sensitivity to new input by injecting two bursts of layer 4 spikes, checking for the shortest time interval where both bursts lead to activation of the respective patterns (Figure 7). The strength of the stimulus on pattern 1 was held fixed, five out of nine minicolumns stimulated, so that the network always entered attractor 1. The stimulus strength of the second pattern was varied. For delays of 450 ms or greater, stimulation of one single

![Figure 7. Attentional blink.](image)

Figure 7. Attentional blink. In the period of time closely after activation of one stored pattern in the network, triggering another pattern requires more stimulation than otherwise. Here, one pattern was first activated at \( t = 0 \) by stimulating the layer four cells in five out of the nine hypercolumns. After some delay, a second stimulus was applied to the layer four units of a different pattern, attempting to trigger its activation. Two data series (rings and crosses) are shown, corresponding to separate experiments. Each data point shows the minimum number of minicolumns that have to be stimulated in order to activate the second pattern after a given delay. Third degree polynomials have been fitted to the data points.
Dynamics in a cortical model

minicolumn sufficed to switch the network state to the second pattern. On the other hand, for delays greater than 20 ms but smaller than 100 ms, the stimulus failed to trigger activation of the second pattern, even when all hypercolumns were stimulated. For delays less than 20 ms, the second stimulus was sometimes activated, thus entirely suppressing the first stimulus.

The relation between stimulus strength and SOA observed in our simulation replicates semi-quantitatively the corresponding experimental findings (Marois & Ivanoff 2005). Note that the attractor memory model explains why only searched for letters mask each other; only when an attractor state has actually been entered is the network insensitive to input; previous stimulation that did not lead to pattern activation—working memory updating—has no such effect. Our simulation explains the phenomenon as arising from the activation of an attractor representing the first stimulus and the fact that the UP state activity is higher in the beginning of the attractor retrieval than towards the end.

Parameter sensitivity of attractor state properties

The characteristics of the attractor or UP-state, such as dwell time, rise time and firing frequency may vary depending on the setting of some key parameters in the model. In Figure 8, we show the quantitative effects on these properties when a number of parameters are varied, one at a time. We find that the network dynamics express qualitatively the same behavior for a wide range of parameter values even though the quantitative measures vary significantly. This shows that the same basic network properties can easily be modulated to satisfy the needs of a specialized cortical area or a given mode of operation. Regarding dwell time, there are three candidate mechanisms in the model that might terminate an active pattern; adaptation of pyramidal and RSNP cells respectively, and synaptic depression between pyramidal cells. Our simulations show that the calcium dynamics, mainly the Ca\textsubscript{NMDA} build-up, of the pyramidal cells is the most important factor determining the attractor dwell time. Since calcium influx is also dependent on firing rates, the ratio between excitation and inhibition matters as well. If excitation is high, the pyramidal cells adapt faster. More pronounced synaptic depression significantly lowers the UP state firing frequency without affecting rise-time. However, if synaptic depression is increased too much, rise time increases and firing frequency becomes so low that the attractor activity becomes unstable.

The average firing frequency in UP states was stable at around 6 Hz for most parameter values tested, and did depend only slightly on calcium dynamics. But it was strongly dependent on excitation. An increase in global pyramidal–pyramidal synaptic conductance by a factor of two led to an almost fourfold increase of the average firing frequency. The 6 Hz average firing frequency most commonly displayed was close to the minimal frequency of the network, the lowest frequency for which stable attractor dynamics could be sustained.

Temporal fine structure

There is a wealth of empirical EEG data, recorded from human subjects under different conditions. Particularly, the gamma rhythm has been shown to correlate with delay activity in a memory task (Tallon-Baudry et al. 1998). Local field potential is the microscopic version of EEG. We create an artificial local field potential trace from the network simulation, intended to be similar to the actual one that would be recorded if the model was embedded in real cortex (Protopapas et al. 1998). The main source of the local field potential and EEG signals is believed to be currents in the apical dendrites of pyramidal cells; the exact mechanisms are likely quite complicated. We use the current entering the soma of the pyramidal cells from
Figure 8. Parameter changes and UP state properties. This figure shows how the network behavior changes when key parameters are varied. The parameters are; the decay time constants for the fast (decay $Ca_{V}$) and the slow (decay $Ca_{NMDA}$) calcium pools; the potassium conductances gated by the fast ($g_K \, Ca_{V}$) and slow ($g_K \, Ca_{NMDA}$) calcium pools; maximum conductances for pyramidal-pyramidal synapses within minicolumns (local pyr–pyr), for basket-pyramidal synapses (local ba–pyr) and for pyramidal-pyramidal synapses in between hypercolumns (global pyr–pyr); the synaptic depression parameter in the synaptic dynamics (syn. depr. “U”). Each parameter is varied by a factor of two, except for those pertaining to slow calcium dynamics, which are varied by a factor of 1.25 (grey shaded). This is because the UP states were found never to terminate when the decay time of the slow calcium pool was increased beyond a factor of about 1.5 or when the conductance gated by that pool was similarly decreased. Otherwise, the qualitative behavior of the system is very robust to parameter changes.

the apical dendrite as the source for local field potentials. This signal, aggregated from all pyramidal neurons in the model, is low-pass filtered to generate the simulated EEG signal. Following practice in the field, the EEG signal is further divided into shorter segments, to each of which a cosine-shaped Hanning filter is applied to regularize the signal; smoothly
bringing it to zero at the beginning and end of the segments (Dressler et al. 2004). For each subpart, the power spectrum is calculated, and these spectra are added together.

Looking at the artificial EEG (or local field potential) produced from spontaneous activity, the most prominent finding is that of a gamma-like oscillation with a frequency around 25–30 Hz (Figure 9B, 9C). This signal is almost exclusively generated by the activity in the UP states, consistent with experimental data, relating gamma patterns in human EEG to memory matches. Similar oscillations have also been found in local field potentials of awake behaving monkeys (Brovelli et al. 2004).

One standard measure to analyze spike recordings from multiple units is to form auto- and crosscorrelograms of the spike trains. We apply this technique to create averaged crosscorrelograms for pairs of neurons belonging to the same minicolumn and for pairs participating in the same pattern, but located in different minicolumns. We apply a correction, similar to the shift predictor, by subtracting from the raw correlations the correlogram for low pass filtered spike trains, removing correlations related to the gross temporal dynamics but preserving temporal fine structure (Gerstein & Perkel 1969).

A very clear synchronization of pyramidal spikes within the minicolumn is evident from the averaged crosscorrelogram between such local neurons. It shows a well defined central peak, with the peaks corresponding to one oscillatory period at about ±40 ms clearly visible and peaks for two periods at ±80 ms being less prominent (Figure 9E). This synchronization is the source of the gamma frequency apparent in the EEG. In Figure 9F, we see that synchronization is much weaker between different minicolumns, even those belonging to the same cell assembly. This is consistent with what has been found experimentally (Steriade et al. 1996). As in the case of the EEG signal, the temporal correlations found originate from activity in the UP state. On a longer time scale, we find a non-zero correlation, appearing after about seven seconds. This relates to a pattern being reactivated after a DOWN period (Figure 9D). With a larger number of attractors in the system, these peaks can be expected to disappear altogether.

We applied another standard technique, unitary event analysis, to study the network’s spiking behavior on a very fine time scale. This method calculates a “surprise” value for the precise instantaneous spike patterns (Grün et al. 2002a, 2002b). Because of the large number of neurons in our model, we chose a version of the method based on binning (Grün et al. 1999). Further, we extended the method by calculating a separate confidence interval for the “surprise” measure, at each moment in time; otherwise unitary events would be found exclusively at peak spike frequencies.

With this method, we find few bins containing significant surprise according to our refined measure, provided the spike rate of the network is tracked sufficiently closely when calculating the significance level. This means that on a very fine time scale, on the order of 1–3 ms, the pyramidal cells fire irregularly. The few unitary events found occur in the beginning and towards the end of an attractor state period (not shown). One condition in which unitary events have been found experimentally is towards the end of the delay period in a delayed response task, but it remains to be determined if our results are related to that observation (Riehle et al. 2000).

Discussion

We have developed and investigated a quantitative scale model of neocortical associative layers 2/3, employing multi-compartmental and conductance based model neurons. The network modeled includes layer 2/3 pyramidal cells and two types of inhibitory interneurons,
Figure 9. UP and DOWN states. A: Raster plot of spike activity in the entire network; the topmost, rapidly firing cells are the RSNP cells, then follow the pyramidal cells (sorted by hypercolumn and minicolumn) and finally the basket cells. Simulated time is 4 seconds and different patterns take turns being active, with short transitional periods in between. B: Local field potential; note asynchronous spindles at the UP state onsets. C: Frequency spectrum of artificial EEG, generated by pyramidal cell currents. There is a peak energy around 25–30 Hz. D: Average autocorrelation between spiking activity within a minicolumn. UP state duration is reflected in peak width, pattern recurrence in the side peak spacings. E: Autocorrelation within a minicolumn on a short time scale, corrected for slow dynamics. The synchronization giving rise to the 25–30 Hz oscillation is evident. F: Average crosscorrelation between different minicolumns, belonging to the same pattern. An imprecise synchronization is evident, but with a tendency towards inverting the patterns seen within a minicolumn on the 10 ms time scale.
basket cells and RSNP cells, with distinctly different properties and roles in dynamic network function. Glutamatergic synaptic transmission includes AMPA as well as NMDA receptor gated channels and our pyramidal to pyramidal synapses undergo significant synaptic depression. The network has a distinct modular structure featuring minicolumns and hypercolumns and its functional organization builds on a top-down perspective of cortical attractor memory. However, we expect many results obtained here to hold even for a network with a more diffuse modularization.

The present model represents an extension of a previous one (Fransen & Lansner 1998) which was based on a minicolumnar structure but lacking hypercolumns and basket cell feedback inhibition. The most prominent effect of introducing the hypercolumns was a marked reduction in pyramidal cell firing rates in an attractor state, from about 50–90 Hz down to about 5–20 Hz, together with the emergence of prominent gamma oscillations. Yet, the associative memory and pattern processing capabilities of the network remained qualitatively intact. One of the main aims of the current work was to investigate whether a recurrent network with connectivity, synaptic weights and firing rates seen in vivo could sustain attractor dynamics. This was indeed found to be the case. Minicolumns enter clear on or off states while this switching between states is not always clearly visible on the cellular level. This conforms well to data on low in vivo firing rates (Abeles et al. 1990; Amit & Brunel 1997) and to a view of minicolumns rather than single cells as the units responsible for cortical memory and pattern processing.

We investigated to what degree our model was capable of displaying sustained activity reminiscent of cortical UP states. We found that the recurrent mutual excitation between pyramidal cells in a distributed cell assembly (active attractor state) is sufficiently strong to support such activity, giving a depolarization plateau of about 10 mV. The UP state in the model terminates after a couple of hundred milliseconds, due to the build-up of hyperpolarization via calcium activated potassium channels. An active cell assembly also produces a powerful lateral inhibition that effectively prevents other assemblies to activate. As the active assembly terminates, the others are disinhibited and one of them may get active. In the absence of input the network displays a “free recall mode” in which the network state spontaneously jumps between different attractors with a frequency in the order of a few Hz. This frequency is quite sensitive to several parameters that would most likely be affected by, e.g., neuromodulation. We speculate that this kind of quasi-stable attractor dynamics may be a generator for phenomena seen in EEG recordings like the theta rhythm and cortical microstates (Lehmann et al. 1998).

In the presence of input, the model network performs some important pattern processing operations like pattern completion, noise reduction and pattern rivalry. Despite the low firing rates of individual neurons, these operations occur robustly and on a time scale of some tens of milliseconds, i.e., within the time frames suggested by psychophysical experimental data relating to the corresponding operations (Thorpe et al. 1996). Even though in reality there are several connected cortical areas involved in such visual processing, the process itself would not be strictly serial. In a cascade of serially connected recurrent networks of IF-neurons, Panzeri et al. (2001) showed that recurrent networks contributed to the downstream calculations 15 ms after they received input. We observe here the same short latency per stage given strong and crisp stimulus conditions. For weak or incomplete input or in case of rivalry, the latency increases to some 40–60 ms. Thus, the recurrent connections at each stage do not necessarily make processing slower, but rather allows for a refined processing over time and it also buffers the result of the computation over a period of time as persistent activity.
We further propose and demonstrate that the model suggests a possible connection between cognitive phenomena like the attentional blink and the dynamics of the underlying cortical attractor networks and properties of their constituent neurons and synapses. Obviously, the full attentional blink phenomenon is quite complex and involves the interaction between several cortical areas. Building a more elaborate model comprised of network modules such as the one studied here will be necessary in order to make progress towards a more complete quantitative model of this fascinating phenomenon.

The attractors stored in our subsampled network were simple, consisting of the eight possible orthogonal patterns. Due to the way activity is structured in the network and due to the low overlap that would exist between cell assemblies generated from decorrelated input to layer 2/3 from earlier processing stages, we argue that the dynamics seen here is at least qualitatively representative of that of a full-scale network. To verify this is, however, an important matter for further investigation.

We observed a fine-structure in the firing patterns, during an UP state, in the form of a synchronization of firing within a minicolumn. Because of the high level of mutual excitation within a minicolumn the system is easily kicked into co-activity. But since the pyramidal cells in each minicolumn are recurrently connected to a population of inhibitory basket cells, they are synchronized into a bursting behavior on the minicolumnar level. In this way, after-activity in a cell assembly can be maintained for several hundred milliseconds with an overlay of spike synchrony between local cells at a frequency around 20–30 Hz. We found evidence for weaker synchronization between distant minicolumns also showing up in the synthetic EEG as a collective high frequency oscillation of the entire active assembly. The synchronization observed was, however, transient and not necessary for the pattern processing operations studied. Yet, more subtle effects on network processing is likely to occur but were not further investigated here.

Our studies of the parameter sensitivity of the model demonstrated that its qualitative dynamic behavior was quite robust, though quantitative changes, especially in UP-state dwell time and firing frequency could be significantly modulated. This part of our study can be interpreted as a set of model predictions relating to how these features of cortical neurodynamics should depend on experimental pharmacological manipulation of the corresponding cellular and synaptic properties.

Our model is undoubtedly still incomplete and approximate in many respects. We regard it as scaffolding for building more extensive models of neocortex in which the other cortical layers and additional types of interneurons are explicitly represented. Peters & Yilmaz (1993) suggested that the cells in layer 2/3 together with the large pyramidal cells in layer 5 form a basic module of the cat striate cortex. One obvious way to extend our model is thus to add layer 5 pyramidal cells to the minicolumns. Together with the intermediate range intracortical horizontal connections in layer 2/3, running up to some 3 mm horizontally (Stettler et al. 2002), they form the long-range connectivity and the “excitatory core” of an active attractor state. Another important addition would be a layer 4 model (Tao et al. 2004).

The size of the model is currently being scaled up significantly by the use of a parallel simulator, running on a cluster computer (Hammarlund & Ekeberg 1998). This model comprises a large array of full-scale hypercolumns and will allow us to better represent the geometric extent and configuration of the cortical networks and to study in detail the influence on the dynamics of the spatial extent of a cell assembly over cortical areas. The first simulations of a network of the same type as described here but with eight million neurons and four billion synapses has already been performed successfully (Djurfeldt et al. 2006).

Using such simulations as tools it will be possible, e.g., to build a full-scale virtual cortical slice and to simulate various pharmacological manipulation of the slice, e.g., raised
extracellular potassium and block of GABA\textsubscript{A} and compare results with those experimentally obtained. Further, by including in a large-scale geometrically accurate model of neocortex a measure of local field potential, energy consumption, etc. due to neuronal and synaptic activation, it would be possible to synthesize along with EEG also MEG and fMRI signals. Such synthetic measures could then be related to the corresponding experimental data and via the model be connected with cellular and synaptic physiology. Such tools would significantly enhance our abilities to better understand the very intricate and powerful operation of the human neocortex.

**Conclusions**

The primary aim of this study was to investigate the dynamics of a biophysical model of a piece of neocortex with a long-range connectivity set up according to the principles suggested by abstract attractor memory models. We found that such a model indeed performs as an attractor memory, exhibiting pattern completion and rivalry. It displayed spontaneous activity reminiscent of UP and DOWN states as well as a temporal fine structure of activity similar to phenomena experimentally observed. Future work includes adding further cortical layers, scaling up the network model by means of parallel simulation and synthesizing further experimental signals like MEG and fMRI from the model.

**Acknowledgements**

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**References**


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Appendix

Cell and synapse parameters

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<th>Parameter</th>
<th>Pyramid</th>
<th>RSNP</th>
<th>Basket</th>
<th>Unit</th>
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<td>7 ± 0.7</td>
<td>7 ± 0.7</td>
<td>(\mu m)</td>
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Table A2. Table of synapse parameters. Synapse model equations as in Fransén & Lansner (1998). Parameters describing the synaptic depression are $U$ and $\tau_{rec}$. $U$ is the fractional decrease of the maximum conductance after a spike is received, and $\tau_{rec}$ is the characteristic time constant for recovery of the conductance.

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Queries

Q1. Au: Pls. provide article dates (received; revised; accepted).
Q2. Au: Pls. provide 3–5 article keywords.
Proof corrections

1. Row 5 on front page: “Department of Numerical and Computer Science (NADA)” should be changed to “School of Computer Science and Communication (CSC)”.
2. Row 12 on front page: “and conductance” should be changed to “with conductance”.
3. On row 17 on front page “dynamic single cell behavior” should be changed to ”single cell recordings”.
4. Fig 2 text: Add after second sentence: “Pyramidal cells are shown as triangles, basket cells as circles and RSNP cells as rhombs.”
5. Row 154: after “pool”, add “in the”.
6. Row 158: space between “τ_{rec}” and “=” is too wide.
7. Row 168 add “-“ before 0.65.
9. Row 189: too large spacing between “µ” and “m”.
10. Row 198: too large spacing between “µ” and “m”.
11. Row 203: change “is” to “are”
12. Row 210: change “lower” to “low”
13. Fig 3 text: in row 3 “eight blue” should be changed to “eight dark”.
14. Fig 3: on left hand side it says: “4C/Art” for some reason – please remove.
16. Row 330: change “lasts” to “last”.
17. Fig 4 text: row 6: “cell shown in B” should be changed to “cells shown in B and C”.
18. Row 352: change “others” to “others’”
19. Row 372: change “increase linearly to” to “scale linearly with”.
20. Fig 8: a (very small) part of fig 8 is cut off (lowest part).
21. Row 477: add “expected” between “the” and “”surprise””.
22. Fig 9 text: row 5: change “D: Average autocorrelation between spiking activity within a minicolumn” to “D: Average crosscorrelation between pyramidal cells in the same minicolumn”
23. Fig 9 text: row 7: after “peak spacings.” add “The Δt = 0 bin has been removed.”
24. Fig 9 text: row 7: change sentence starting “E: Autocorrelation within … “ to “E: Average crosscorrelation between pyramidal cells in the same minicolumn on a short timescale, corrected for slow dynamics”
25. Row 500: change “introducing the hypercolumns” to “these changes”.
26. Row 585: change “has” to “have”.
28. Row 590: change “a measure of local field potential, energy consumption, etc” to “in addition to the local field potential, also energy consumption and other measurements”.

Query replies

Q1 Date received: Jun 19 2005   Date revised: Feb 24 2006   Date accepted: April 26 2006
Q2 Keywords: Cortex, UP state, attentional blink, attractor dynamics, synchronization.