Neuronal Dynamical Systems

Tuesday, Sept. 1: 11:00 – 13:00

Session 1: Audi Max

Contents

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Triggering bursts in all-to-all coupled neurons with global inhibition

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Slow-wave sleep in mammals is characterized by a change of large-scale cortical activity currently paraphrased as cortical up/down states. Recently Shu et al. demonstrated experimentally a bistable collective behavior in ferret brain slices, with the remarkable property that up states can be switched on and off with pulses, or excitations, of same polarity; whereby the effect of the second pulse significantly depends on the time interval between the pulses. We present a time discrete model of a neural network that reproduces this type of stimulation response and quantitatively the time-dependence found in the experiments.

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2

Sequential Desynchronization of Clusters in Spiking Neural Networks

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The brain processes information in networks of neurons that interact by sending and receiving electrical pulses called spikes. The response of a neuron to incoming signals strongly depends on whether or not it has just sent a spike itself. After the initiation of a spike the membrane potential at the cell body (soma) is reset towards some potential and the response to further synaptic input is reduced due to the refractoriness of the neuron. The dendritic part of the neuron where incoming signals are integrated is affected only indirectly by this reset due to intra-neuronal interactions.

Several multi-compartment models have been proposed, in which different parts of a single neuron interact to characterize this effect. For instance, in a two-compartment model [1] of coupled dendrite and soma, the membrane potential at the soma is reset after spike emission while the dendritic dynamics is affected only by the resistive coupling from the soma to the dendrite. This accounts for the fact that in several kinds of neurons residual charge remains

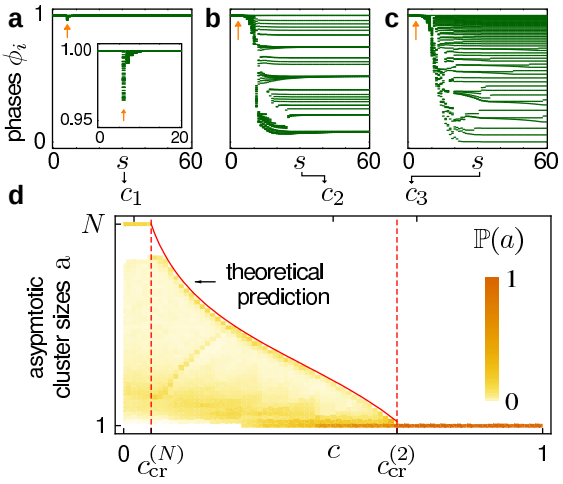

Fig. 2.1: Sequential desynchronization transition in a network of $N = 50$ oscillatory neurons. The phases ϕ_i of all neurons are plotted against the s-th spike of a reference neuron. **(a)** For weak partial reset strength c the synchronous state is stable and coexist with **(b)** cluster states. Increasing c sequentially destabilizes the clusters until only **(c)** the asynchronous state is observed. **(d)** Probability $\mathbb{P}(a)$ of observed cluster sizes a in the asymptotic dynamics starting from random phases uniformly distributed in $[0, 1)$. red line: exact theoretical prediction above which clusters are unstable.

on the dendrite (following the somatic reset), that is then transferred to the soma [2]. Thus the dynamics of the individual neurons is modified which severely affects the collective capabilities of networks of such neurons.

Here we propose a neuron model that after spike emission exhibits a partial response to residual input charges and study its collective network dynamics analytically [3–5]. We uncover a desynchronization mechanism that causes a sequential desynchronization transition [3, 5] (cf. Fig. 2): In globally coupled neurons an increase in the strength of the partial response induces a sequence of bifurcations from coexistent states with large clusters of synchronously firing neurons, through states with smaller clusters to completely asynchronous spiking. We study the consequences of this mechanism in more general networks by connecting our simple model to more realistic biophysical ones using spike time response curves.

This novel mechanism for neural desynchronization differs strongly from known mechanisms that are based, e.g., on heterogeneity, noise, or delayed feedback [6, 7]. Possibly, the mechanism presented here may also be used in modified form to prevent synchronization in neural systems like in Parkinson tremor or in epileptic seizures [7].

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3

Phase Transitions Towards Criticality in a Neural System with Adaptive Interactions

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The concept of self-organized criticality (SOC) [4] describes a variety of phenomena ranging from plate tectonics, the dynamics of granular media, and stick-slip motion to neural avalanches [5]. These examples have in common that a marginally stable dynamics is maintained by self-tuning of parameters towards critical values and that the event sizes obey a characteristic power-law distribution. In neuronal systems the existence of critical avalanches was predicted in a paper of one of the present authors [5] and observed experimentally by Beggs and Plenz [3].

In analogy to the physical systems mentioned above, the strength of the interaction among the neural units by synaptic connections has been identified as a critical parameter [5]. In real neural systems the connection strengths are not static but depend on the relative timing of the neural activity pulses [6]. While the system with static coupling has a classical critical point [5], we show analytically that the adaptive model attains criticality in an extended region of the parameter space that is bounded by phase transitions. It was previously shown that an extended critical interval can be obtained in a neural network by incorporation of depressive synapses [1]. In the present study we scrutinize a more realistic dynamics for the synaptic interactions that can be considered as the state-of-the-art in computational modeling of synaptic interaction. Interestingly, the more complex model does not exclude an analytical treatment and it shows a type of stationary state consisting of a self-organized critical phase and a subcritical phase that has not been described earlier [2]. The critical region of the connectivity parameter is sandwiched between a sub- and a supercritical regime which also can be reached experimentally by a manipulation of the synaptic strengths, c.f. Fig. 3.1 (left). The behavior of the system can be well described by the mean-field approach, c.f. Fig. 3.1 (right). The system exhibits a rich dynamical behavior including a hysteresis between critical and non-critical dynamics, switching of the dynamics in dependence of external inputs, and first- and second-order phase transitions that form a cusp bifurcation. Although presented in the specific context of a neuronal model, this dynamical structure is of more general interest as the first observation of a complex classical bifurcation scenario combined with a SOC phase.

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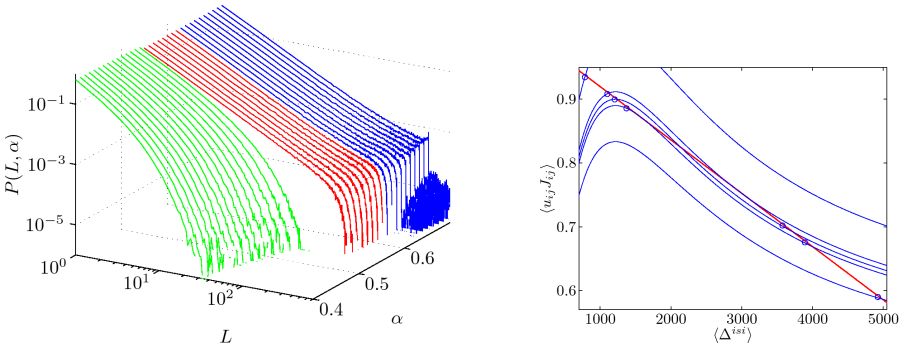

Fig. 3.1: (left) Distributions of avalanche sizes in dependence on the interaction parameter α for a network of size $N = 300$. At $\alpha < \alpha_c \approx 0.55$ the distributions are subcritical (green), while between α_c and $\alpha^c \approx 0.62$ also a critical phase exists (red), which survives beyond α^c, a supercritical distributions are observed for $\alpha > 0.62$ (blue). (right) Average synaptic strength $\langle u_{ij}J_{ij}\rangle$ (virtually straight line) and inter-spike interval $\langle\Delta^{\rm isi}\rangle$(curved lines, from bottom to top). Dependencies are found from the self-consistency equation for $\alpha = 0.5$, $\alpha = \alpha_c$, $\alpha = 0.54$, $\alpha = \alpha^c$, and $\alpha = 0.6$. Intersections of the lines provide solutions of self-consistency equation. Circles represented numerical results for a network with matching α and parameters $N = 300$, $\nu = 10$, $u_0 = 0.1$, and $I_0 = 7.5$.

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4

Correlations, synchrony and entropy in a pair of neurons

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In order to understand the origin, temporal properties and strength of interneuronal spike correlations it is essential to analyze how neurons subject to correlated synaptic inputs coordinate output spiking activity. We used a simple statistical framework for the analysis of spike correlations and entropy content in pairs of neurons driven by correlated inputs [1]. We examined the synchronization acuity of a pair of neurons subject to a variable percentage of common fluctuating input of different correlation times. First, we calculated the auto conditional firing rate of an individual neuron and analyzed its short and long time asymptotics. For large time lags, we find a substantial influence of the second derivative of the voltage correlation function. In the limit of short times, we find an algebraic rise out of a period of intrinsic silence after each spike which mimics a refractory period. Additionally, we computed the entropy difference between two uncorrelated and two correlated neurons and found that the entropy difference is firing rate dependent and is sensitive to the sign of the correlation strength. Furthermore, we studied the cross conditional firing rate of a pair of neurons for 1) low and 2) high common input fraction and 3) with firing rate heterogeneity. In the low correlation regime, we identified a rate dependence of the rate of synchronous firing corroborating previous observations [2] and predict that spike correlations in this regime reflect detailed properties of input correlations. In the high correlation regime, however, the synchronous rate ceases to depend on the stationary firing rate of individual neurons and the structure of spike correlations is governed by the input correlation time and the coupling strength but is insensitive to firing rate and the detailed form of input correlations. For all strengths of correlations the model predicts the appearance of a systematic delay of firing of the lower rate neuron relative to the higher rate neuron. This effect can significantly decrease spike count correlation coefficient for large time bins. We tested the theoretical predictions of our framework in in vitro experiments in slices of rat visual cortex and injected in pyramidal neurons fluctuating currents with a varying degree of common input. Cross and autoconditional firing rates computed from these recordings, confirmed all basic theoretical predictions 1)-3) of our formalism.

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5

Dynamics of wave segments on curved cortex

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Waves of cortical spreading depression (SD) have been suggested to cause visual field defects in migraine [1]. Despite a precise match in the speed of both phenomena, the spatio-temporal wave patterns of SD observed in flat animal cortex and visual field defects mapped to the curved human cortical surface (Fig. 5.1) ordinarily differ in aspects of size and shape [2].

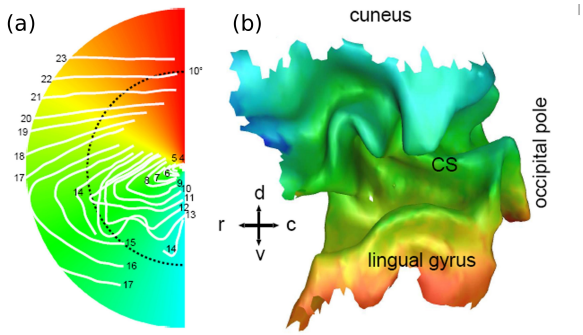

Fig. 5.1: Visual field defects and 3D form of primary visual cortex. (a) Left visual hemifield: the position of the propagating visual field defect, observed during a migraine attack, is indicated by white lines, with numbers denoting the time in minutes after onset. (b) Cortical surface with the representation of the azimuthal coordinate of the left visual hemifield measured by functional magnetic resonance imaging (fMRI) [3]. Color code: hue, value, saturation color model as in (a).

We show that this mismatch in the patterns is reconciled by utilizing that both types of patterns bifurcate from an instability point of a generic reaction-diffusion model. To incorporate features of *in vivo* human cortical physiology not present in animal *in vitro* models, we augment the extended Hodgkin-Grafstein reaction-diffusion model of SD (equivalent to FitzHugh-Nagumo equations, see [4]) with a global time-delayed feedback representing the neurovascular coupling. We model the pattern formation on a curved surface:

$$\partial_t u = u - u^3/3 - v + \Delta_{LB} u \tag{5.1}$$

$$\varepsilon^{-1} \partial_t v = u + \beta - \gamma v + K \int \Theta(u(t-\tau) - u_{th}) \tag{5.2}$$

with the Laplace-Beltrami diffusion operator on a curved surface

$$\Delta_{LB} = \sum_{i,j=1}^{2} g^{-\frac{1}{2}} \frac{\partial}{\partial \alpha^i} \left(g^{\frac{1}{2}} g^{ij} \frac{\partial}{\partial \alpha^j} \right), \tag{5.3}$$

g^{ij} are the components of the inverse of the metric tensor and g denotes the determinant of the metric tensor, furthermore, Θ is the Heaviside function, τ a time delay, and u_{th} a threshold.

Our results suggest that SD in humans is much closer to a bifurcating instability point of pattern formation than in nonprimate mammals. From a synergetics point of view, the brain is in general viewed as a self-organizing pattern forming system that operates close to instability points [5]. We conclude that in the case of migraine SD waves, the important point in state space is a 2D critical solution of the reaction-diffusion system in the shape of a wave segment with exactly one negative eigenvalue whose center-stable manifold of co-dimension 1 defines the threshold surface in state space separating initial conditions belonging to the stable homogeneous state (healthy cortical state) from those leading to the initiation of SD (migraine attack). Being close to this instability point in the presence of augmented transmission capabilities dramatically changes the dynamical repertoire of pattern formation in the cortex. This factor, as will be discussed, could have important implications for the design of biomedically engineered devices that intelligently target the occurrence of SD waves based on methods adapted from chaos control [6].

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6

Controlling effective connectivity in networks of coupled cortical areas

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The anatomy of long-range synaptic connections between local cortical circuits or distinct cortical areas constrains at a large extent the spatio-temporal complexity of neural responses and, more specifically, of brain rhythmic activity [1, 2]. However such *structural* connectivity does not necessarily coincide with *effective* connectivity, related to the more elusive question "Which areas drive the activity of which others?" [3]. Effective connectivity is directed and is often task-dependent, evolving even across different stages of a single task [4, 5]. These fast changes are incompatible with the slow variation of anatomical connections in a mature brain and might be explained as dynamical transitions in the collective organization of neural activity.

We consider here small fully-connected networks of interacting cortical areas ($N = 2 \div 4$), modeled both as mean-field rate units and as large populations of spiking neurons. Intra-areal local couplings can be inhibitory or excitatory while inter-areal longer-range couplings are purely excitatory. All these interactions are delayed. Sufficiently strong local delayed inhibition induces synchronous fast oscillations and phase-locked multi-areal polyrhythms are obtained for weak long-range excitation [6, 7].

Even if these small structural networks are fully symmetric, varying the strength of local inhibition and the delays of local and long-range interactions generates dynamical states which spontaneously break the symmetry under permutation of the areas. The simplest example is provided by the $N = 2$ network in which transitions from in-phase or anti-phase to out-of-phase lockings with intermediate equilibrium phase-shifts are identified [7]. Areas *leading* in phase over *laggard* areas can therefore be unambiguously pinpointed, introducing naturally a directionality in inter-areal communication.

Remarkably, asymmetries in phase-locked polyrhythms are amplified in the associated chaotic states seen for strengthened inter-areal couplings. In these cases indeed, the firing rate oscillations of laggard areas undergoes significantly stronger amplitude fluctuations than leading areas. Asymmetric chaotic states can be described as conditions of *effective entrainment* in which laggard areas are driven into chaos by the more periodic firing of leader areas (see Fig.6.1).

Fully symmetric structural networks can thus give thus rise to multiple alternative effective networks with reduced symmetry. Transitions between different effective connectivities are achieved via transient perturbations of the dynamics without need for costly rearrangements of the structural connections.

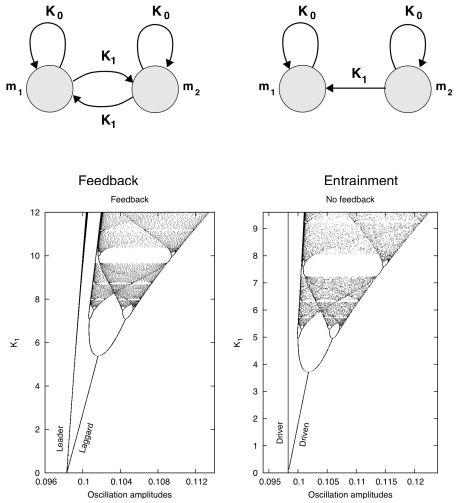

Fig. 6.1: An example of effective entrainment in a symmetric network of $N = 2$ coupled areas, compared with direct entrainment in an asymmetric network.

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7

A mathematical model of homeostatic regulation of sleep-wake cycles by hypocretin/orexin

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A generally accepted concept of sleep regulation postulates that sleep-wake transitions result from the interaction between the circadian and homeostatic processes [1]. The circadian process is ascribed to the activity of the suprachiasmatic nucleus of the hypothalamus, while the mechanisms of the homeostatic process are still unclear.

In this study we present a concept of hypocretin/orexin-based control of sleep homeostasis. Hypocretin/orexin (hcrt/orx) is a neuropeptide which is produced in the lateral hypothalamus and its absence leads to the well-known sleep disorder narcolepsy. We propose that (1) high frequent impulse activity of the hcrt/ox neurons during wakefulness is sustained by reciprocal excitatory connections with other, e.g. local glutamate neurons; (2) the transition to a silent state (sleep) is going along with a weakening of the hcrt/ox synaptic efficacy.

This concept has been realized in a mathematical model with Hodgkin-Huxley-type neurons and physiology-based synapses. The model is capable to simulate the neuronal activity which corresponds to the sleep-wake transitions and the effects of various disturbances. It offers a new approach for further evaluation of the physiological, especially homeostatic mechanisms of sleep-wake cycles on the basis of neuronal activity and synaptic transmission.

The work was supported by the European Union through the Network of Excellence BioSim contract No LSHB-CT-2004-005137.

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8

The dynamics of a model system for invariant object recognition

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An important problem in neuroscience is object recognition invariant to transformations, such as translation, rotation and scale. Many dynamic models have been proposed to perform this task with considerable success [1, 2]. In order to gain insights on the recognition dynamics and the organization of stored objects, we here propose a model system as an abstraction of invariant recognition.

Assume there are q objects $M^k \in R^n$, $k = 1, \cdots, q$, in the gallery, and p possible transformations Γ^k, $k = 1, \cdots, p$. An input image $I \in R^n$ is generated by one of the objects through a transformation. The task is to recover the object and the transformation that generate I. The system variables are $C = (c_1 c_2 \cdots c_p)^T$ for the selection of transformations, and $D = (d_1 d_2 \cdots d_q)^T$ for object selection.

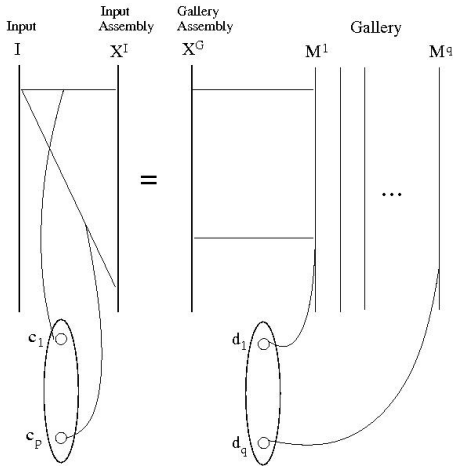

Fig. 8.1: The structure of a model system for invariant recognition.

The structure of our model system is shown in Fig. 8.1. Between the input image and the gallery are two assembly layers. The image assembly layer X^I is a combination of transformed images, while the gallery assembly layer X^G is a combination of models. If both combinations are linear, we have $X^I = (\sum_k c_k \Gamma^k) I = \sum_k c_k P^k$, and $X^G = \sum_k d_k M^k$. The goal is to determine C and D, such that $X^I = X^G$.

When $p + q > n$, the system is underdetermined, having many solutions. Because 0 is always a solution, we impose a constraint on the total activity of

the system, and define the energy function

$$E = \|X^I - X^G\|^2 + \lambda(C^T C - 1)^2 + \lambda(D^T D - 1)^2. \tag{8.1}$$

The dynamics follow the gradient of the energy function. Unfortunately the constraint on total activity is not sufficient to warrant a unique solution. An example solution is shown in Fig. 8.2 for $n = 20 \times 20, p = 18 \times 18 \times 2, q = 8$. The ground truth is $c_1 = 1, d_5 = 1$ and all other variables 0.

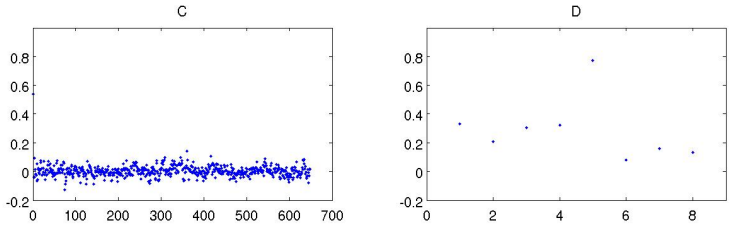

Fig. 8.2: An example solution different from true cause (0 except for $c_1 = 1, d_5 = 1$).

We propose a coarse-to-fine strategy where variables are first grouped (share the same dynamics) such that a unique solution can be obtained. Then variables within the winning groups resume their own dynamics, using the group solution as initial value. We show that this coarse-to-fine dynamics can recover true sparse causes.

Acknowledgement: supported by EU project SECO and the Hertie Foundation.

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