Interdisciplinary Symposium on Signals & Systems for Medical Applications

Program and abstracts of ISSSMA workshop on Feedback and synchronization in neuroscience

Session 1.1: From neurons to neuronal populations dynamics
June 3rd, 11:00-12:30, amphi Hermite. Chair: W. Pasillas-Lépine

Boris Gutkin - *ENS Paris (France)*

Role of structured neural activity in working memory tasks: differential functional roles for gamma, theta and alpha oscillations.

Working memory (WM) corresponds to the cognitive function of maintaining and manipulating short-term memory rapidly. Electrophysiological recordings have shown that selective persistent activity underlies short-term memory retention. In this framework, WM operations correspond to controlled transitions between persistent and resting states.
Here we propose that modulation of spike-time structure in neural activity provide a mechanism to carry out required WM operations. First we show that spatial correlations in background activity determine the stability of the persistent state in such a network and the ability of a stimulus to activate the persistent state. Second we show that similar effects are seen with synchronous oscillations, and furthermore that the different frequencies have differential effect, notably enabling the network to differentially gate stimuli into the persistent state. We exhibit the mechanisms for these effects. Functionally we show how flexibly shifting oscillatory frequency can appropriately define the gating modes of the working memory networks so as to fully implement all aspects of tasks based on such memory.

Alex Roxin - *CRM (Spain)*

Connectivity motifs and dynamics in cortical network models

Over the past decade there has been increasing evidence that cortical microcircuits exhibit certain connectivity patterns, or motifs, beyond what would be expected from trivial random network models. Such motifs include pairwise reciprocal connections, but also motifs involving three or more neurons. Indeed, these motifs have been cited as evidence for the presence of clusters in cortical networks.
I will discuss how such motifs can be reproduced in network models and to what extent they are dependent on clustering. I will argue that additional network structure, unrelated to clustering and due to ongoing synaptic plasticity, may contribute to these motifs. Finally, I will derive an analytical meanfield model which captures the effect of these motifs on network dynamics.

Guillaume Drion - *Liège Univ. (Belgium)*

Endogenous and exogenous neuronal rhythmicity

The talk will present a simple neuronal model that can switch from an exogenous rhythm to an endogenous rhythm through the regulation of a single parameter. The model is inspired from a detailed conductance-based model of dopaminergic neurons but the mechanism is shared by many other neurons. We will discuss the robustness of this mechanism and its implications for neuronal rhythmicity at the population level.

Session 1.2: Healthy and pathological oscillations in basal ganglia
June 3rd, 16:30-18:00, amphi Hermite. Chair: A. Chaillet

Thomas Boraud - *CNRS, Univ. of Bordeaux (France)*

Beta oscillation and Parkinsonian Motor symptoms: causality or correlation?

Parkinson's disease is caused by a progressive loss of dopaminergic neurons resulting in the manifestation of motor impairment namely akinesia, rigidity and tremor. It has been proposed that beta band (15-30 Hz) oscillatory activity in the basal ganglia, a hallmark of the disease, had a direct causal relationship with the manifestation of PD motor symptoms. However, this hypothesis has not yet been clearly proven. In this talk, we present experimental data from different animal models (rodent and primates of dopamine depletion) that invalidate this hypothesis and propose a systemic approach to conciliate apparently conflicting evidences.

Rafal Bogacz - *Univ. of Bristol (UK)*

Mathematical modelling of abnormal beta oscillations in Parkinson's disease

In Parkinson's disease, increased power of oscillations in firing rate has been observed throughout the cortico-basal-ganglia circuit. In particular, the excessive oscillations in the beta range (13-30Hz) have been shown to be associated with difficulty of movement initiation. However, on the basis of experimental data alone it is difficult to determine where these oscillations are generated, due to complex and recurrent structure of the cortico-basal-ganglia-thalamic circuit. This talk will describe a computational model of a subset of basal-ganglia that is able to reproduce experimentally observed patterns of activity. The analysis of the model suggests where and under which conditions the beta oscillations are produced.

William Pasillas-Lépine - *CNRS, L2S (France)*

Electrical stimulation, firing-rates, and basal ganglia models: are they compatible?

Several models have been proposed recently in order to explain the mechanisms behind basal ganglia oscillations. In the first part of the talk, we will show how a classical result of control theory (the generalized Nyquist criterion) can be applied in order to analyze the stability of such models, in the particular case for which the gain of the feedback loop is strictly decreasing. In the second part of the talk, the impact of electrical stimulation on basal ganglia models will be considered. We will show how several phenomena (the refractory period of neurons, the correlation between inputs, and the transfer functions associated to different neurotransmitters) are difficult to reproduce within the scope of the available models. We will propose an alternative firing-rate model that takes these questions into account. The output of our model will be confronted to recent experimental data available in the literature.

Session 1.3: Synchronization in neuroscience
June 4th, 10:30-12:00, amphi Hermite. Chair: E. Panteley

Constance Hammond - *INSERM, INMED (France)*

Abnormal early synchronization of Pink1-/- M1 cortical neurons is decreased by high frequency stimulation of the subthalamic area in the mouse cortico-subthalamic slice

The activity of brain structures responsible for the automatic execution of learned movements is pathologically synchronized in patients suffering from Parkinson's disease. In a mouse model of a familial form of Parkinson's disease we saw that such synchronization is already present 16 months before the first motor signs (a mouse lives around 24 months). High frequency stimulation of the subthalamic nucleus reduces the synchronization by generating action potentials traveling backward to the motor cortex. This work was performed in brain slices that keep intact some connections between motor cortex and subthalamic nucleus, with electrophysiological and calcium imaging techniques coupled to a mathematical analysis of the recorded signals. These results suggest that frequent episodes of correlated activity is a presymptomatic signature in the motor cortex of the motor disease to come and that high frequency stimulation of the subthalamic nucleus decorrelates motor cortex activity.

Henk Nijmeijer - *Tec. Univ. Eindhoven (The Netherlands)*

An experimental neuronal network: does it (partially) synchronize?

We discuss synchronization in networks of Hindmarsh-Rose neurons that interact via gap junctions, also known as electrical synapses. We provide a theoretical framework for analysing synchronization in such diffusively coupled networks where we consider the case where either no time-delay is in the coupling between neuronal cells, or when also coupling is allowed. The results show that, depending on the particular network, a definite route towards synchronization can be found, with as typical intermediate stages different types of partial synchronization. The latter may have interesting connections in neuronal behavior as, for instance, epilepsy is typically found to be linked with a similar phenomenon. Experiments on an electronic brain network illustrate some of the theoretical findings.

Michael Rosenblum - *Potsdam Univ. (Germany)*

Collective dynamics in large neuronal ensembles: synchronization and control

We discuss dynamics of large ensembles of oscillatory units, e.g. of spiking or bursting neurons. The well-known effect here is synchronization and appearance of a collective mode with increase of coupling; however, this scenario is not general. We concentrate on two problems. First, we demonstrate that increase of coupling between elements of the network does not always facilitates synchrony, but can result in its breakup and emergence of complex, e.g. quasiperiodic states, where the mean field frequency is larger than frequencies of all oscillators. We discuss two different scenaria of desynchronization transitions and analyze effects of common periodic forcing of such ensembles. The theory and numerical simulations are illustrated by the results of physical experiment. Second, we discuss control of collective dynamics; this problem is relevant for developing efficient techniques for suppression of pathological rhythms, e.g. of parkinsonian activity by means of deep brain stimulation. We discuss algorithms of global feedback control which provide efficient suppression via desynchronization of the network. Important feature of the technique is that the feedback signal vanishes as soon as the collective dynamics is suppressed, and, thus, the intervention into the system is minimized.

Session 1.4: Identification and observer design in neuroscience
June 4th, 15:30-17:00, amphi Hermite. Chair: W. Pasillas-Lépine

Sridevi V. Sarma - *John Hopkins Univ. (USA)*

On the Therapeutic Mechanisms of Deep Brain Stimulation for Parkinson's Disease: Annihilation or Restoration of Basal Ganglia Feedback?

High-frequency deep brain stimulation (DBS) (>100 Hz) of the Basal Ganglia (BG) is a clinically recognized treatment for movement disorders in Parkinson's disease (PD), but requires extensive tuning of the DBS signal, high power, and several surgical battery replacements. Improvements to DBS therapy are hindered because its therapeutic mechanisms are still not understood. Many people have observed abnormally prevalent 10-30 Hz oscillations in the BG in PD patients when no DBS is applied, which causes motor symptoms such as tremor and rigidity. One hypothesis is that high frequency DBS may be "jamming" this pathological activity, thus annihilating the faulty BG output (mimicking a lesion in the area), to ultimately restore movements.
In this talk, we challenge the annihilation hypothesis by studying the dynamics in neurophysiological signals using unique primate data and novel computational models. First, we show that: (i) in health, the output of the BG exhibits 10-30 Hz oscillations (slow) when the subject does not move, and (ii) then switches to exhibit 30-70 Hz oscillations (fast) when the subject plans movement. This "crossover effect" (slow oscillations decrease while fast oscillations increase) has been reported in cortical areas before but not in deep structures such as the BG, and is a critical form of neural communication to generate healthy movements.
It has been well documented that in PD, the BG exhibits excessive 10-30Hz oscillations. Therefore, it may be harder to suppress these slow rhythms during movement planning, thereby compromising the crossover effect and movements in patients. In the second part of the talk, we show that high frequency DBS (i) reinforces activity in the BG input structure (via backwards and forwards propagation through the BG circuit), which then ii) increases the propensity of the BG output to generate fast oscillations. This suggests that HF DBS may restore the crossover effect seen in healthy, as opposed to "jamming" the BG output.
Finally, we explain why the crossover is needed to generate healthy movements using a computational model of motor thalamic neurons. Specifically, we show that the BG output (which projects to thalamus) must generate fast rhythms when information from cortical areas should be relayed by the thalamus to motor cortex for proper execution of movements. This study is the first to precisely quantify thalamic relay and explains in more details BG output in health, in PD with and without DBS.

Romain Postoyan - CNRS, CRAN (France)

Parameter and state estimation for a class of neural mass models

While various procedures are available to measure brain activity such as electroencephalography (EEG), magnetic resonance imaging (MRI), magnetoencephalography (MEG), the underlying (neo) cortical mechanisms remain largely unknown in particular for pathologies like epilepsy. Recent advances in computational modeling might cast new lights on our understanding of these internal mechanisms. In particular, some neural mass models are able to reproduce signals which mimic EEG recordings. They have been developed so far to investigate physiological assumptions, but they might be used for `predictive' purpose. Indeed, if we could estimate on-line their states and parameters, we would have access to neurophysiological data which is currently not available and which may thus bring new insight into the pathology dynamics. In addition, they could help for the development of feedback controllers. However, the nonlinear nature of these models makes the estimation problem challenging. In this talk, we will briefly review the considered class of neural mass models. Afterwards, we will present the estimation schemes we have recently synthesized in collaboration with researchers from the University of Melbourne (Australia). We will notably show their ability to estimate the states and the parameters of a single cortical column model in simulations.

Elena Panteley - *CNRS, L2S (France)*

Diffusively coupled neural networks: estimation of emergent behavior.