http://2009.neurocomp.fr

NEUROCOMP

Computational Neuroscience: From Multiple levels to Multi-level

16-18 Sept. 2009

BORDEAUX

Network/cell dynamics: *Coding and processing*
Neuro-economy: *Value-based decision making*
Implementation: *Hardware and software simulations*
Analysis: *Methods and tools*

Design and Photograph by Olivia Malot

cnrs

UNIVERSITÉ BORDEAUX 1 Sciences Technologies

INRIA

Université Victor Segalen Bordeaux 2

REGION AQUITAINE

Neurocomp'09

Computational Neuroscience:
From Multiple levels to Multi-level

September 16-18, 2009
Bordeaux

Conference program
&
Proceedings

Sponsored by :

Foreword

La communauté bordelaise des neurosciences computationnelles est très heureuse de vous accueillir pour ces 4èmes journées de NeuroComp, **Neurocomp09**, du 16 au 18 septembre 2009. Ce cycle de colloques a été créé pour réunir régulièrement les principaux acteurs français du domaine qu'ils soient francophones ou non. A l'instar des éditions précédentes, ce colloque offre un lieu d'échanges dont le but est de favoriser des collaborations interdisciplinaires entre des équipes relevant des neurosciences, des sciences de l'information, de la physique statistique et des mathématiques.

Le format retenu cette année est celui d'une conférence école avec la présence d'une douzaine de conférenciers internationaux invités, la présentation par les jeunes chercheurs de leurs travaux lors de sessions poster, ainsi que des démonstrations techniques.

La conférence traitera prioritairement de 4 thèmes, représentatifs des activités de la communauté bordelaise et très présentes dans l'actualité internationale des Neurosciences Computationelles, à savoir :

- La dynamique cellulaire et dynamique de réseau : codage et traitement
- La neuro-économie : Prise de décision fondée sur l'estimation de valeurs
- L'implémentation : supports hardware et software de simulation
- L'analyse : Méthodes et outils

Chaque journée du colloque traitera l'ensemble de ces thèmes selon différents aspects qui sont respectivement pour chaque jour « Cognition », « Réseaux » et « Cellulaire ».

Neurocomp'09 a été organisé par un ensemble de chercheurs et enseignants-chercheurs des sites universitaires bordelais, dans le contexte de mise en place du *Neurocampus de Bordeaux*, ensemble immobilier et scientifique dédié aux neurosciences. Les membres du Comité Local d'Organisation sont issus de laboratoire dont les activités sont liées peu ou prou aux Neurosciences Computationnelles.

Nous remercions la société Bio-Logic Science Instruments et les Editions Cambridge University Press pour leur soutien financier. Nous remercions aussi les institutions qui nous ont soutenus, comme à leur habitude la Région Aquitaine, Communauté Urbaine de Bordeaux, Pôle de Recherche et Enseignement Supérieur de Bordeaux, Université Bordeaux 1, Université Bordeaux 2, CNRS, INRIA, ENSEIRB-Matméca, Laboratoire IMS, Laboratoire CNIC.
Nous tenons à remercier plus particulièrement l'ensemble des participants à l'organisation, qu'ils soient chercheurs, personnels administratifs et techniques ou doctorants, qui ont donné de leur temps et sans qui ce projet n'aurait pas pu aboutir.

En vous souhaitant un agréable et fructueux séjour à Bordeaux pour Neurocomp'09,

The third French computational neuroscience conference, **Neurocomp09**, welcomes you in Bordeaux. This conference is organized with the Neurocomp initiative with the aim of bringing together the French research community in this field.

As in the previous editions, this conference is a place to strengthen scientific exchange and interdisciplinary collaborations between neuroscience, computer science, mathematics and engineering.
Neurocomp'2009 is organized with a school format. We are happy to host 11 international tutorials by experts in state-of-the–art topics. In parallel, young researchers will present their latest work during poster sessions, and practical workshops. We thank all the conference speakers and attendees for their participation in Neurocomp'09.

The conference will mainly address 4 topics:

- Network/cell dynamics: Coding and processing
- Neuro-economy: Value-based decision making
- Implementation: Hardware and software simulations
- Analysis: Methods and tools

For each day of the conference, these topics will be addressed through the following aspects: Cognitive, Network, Cellular.

Neurocomp'09 is organized by researchers from different laboratories on the Bordeaux campus. Bordeaux is already recognized as one of the leaders in the field of neurosciences with particular emphasis on cerebral research. The Aquitaine Regional Council has therefore embarked on an ambitious Neuroscience research development project, which will be sited at Bordeaux University and dubbed 'the Neurocampus'. All the members of the conference local organization committee participate in this action through their research activities, directly or indirectly related to computational neuroscience.

We thank Bio-Logic Science Instruments and the Cambridge University Press editions for their financial support. We are also thankful to the following institutions, which supported the conference : Région Aquitaine, Communauté Urbaine de Bordeaux, Pôle de Recherche et Enseignement Supérieur de Bordeaux, University Bordeaux 1, University Bordeaux 2, CNRS, INRIA, ENSEIRB-Matméca, IMS laboratory, CNIC laboratory.
Finally, Neurocomp'09 would not exist without the organization participants' support: members of the scientific committee, local researchers, administrative or technical staff, and students. Many thanks for spending their precious time on the conference scientific or logistic issues.

We wish you a pleasant and fruitful stay in Bordeaux !

Sylvie Renaud
Sylvain Saïghi
IMS Laboratory, Bordeaux

Organizing committee:

- Anne Beuter, IMS (CNRS), Bordeaux
- Thomas Boraud, MAC (CNRS), Bordeaux
- Yannick Bornat, IMS (CNRS), Bordeaux
- Laure Buhry, IMS (CNRS), Bordeaux
- André Garenne, MAC (CNRS), Bordeaux
- Jacques Henry, INRIA, Bordeaux
- Olivia Malot, IMS (CNRS), Bordeaux
- Alain Marchand, CNIC (CNRS), Bordeaux
- Gilles N'Kaoua, IMS (CNRS), Bordeaux
- Sylvie Renaud, IMS (CNRS), Bordeaux
- Sylvain Saïghi, IMS (CNRS), Bordeaux
- Jean-Marc Salotti, IdC, Bordeaux
- Jean Tomas, IMS (CNRS), Bordeaux
- Blaise Yvert, CNIC (CNRS), Bordeaux

Conference program

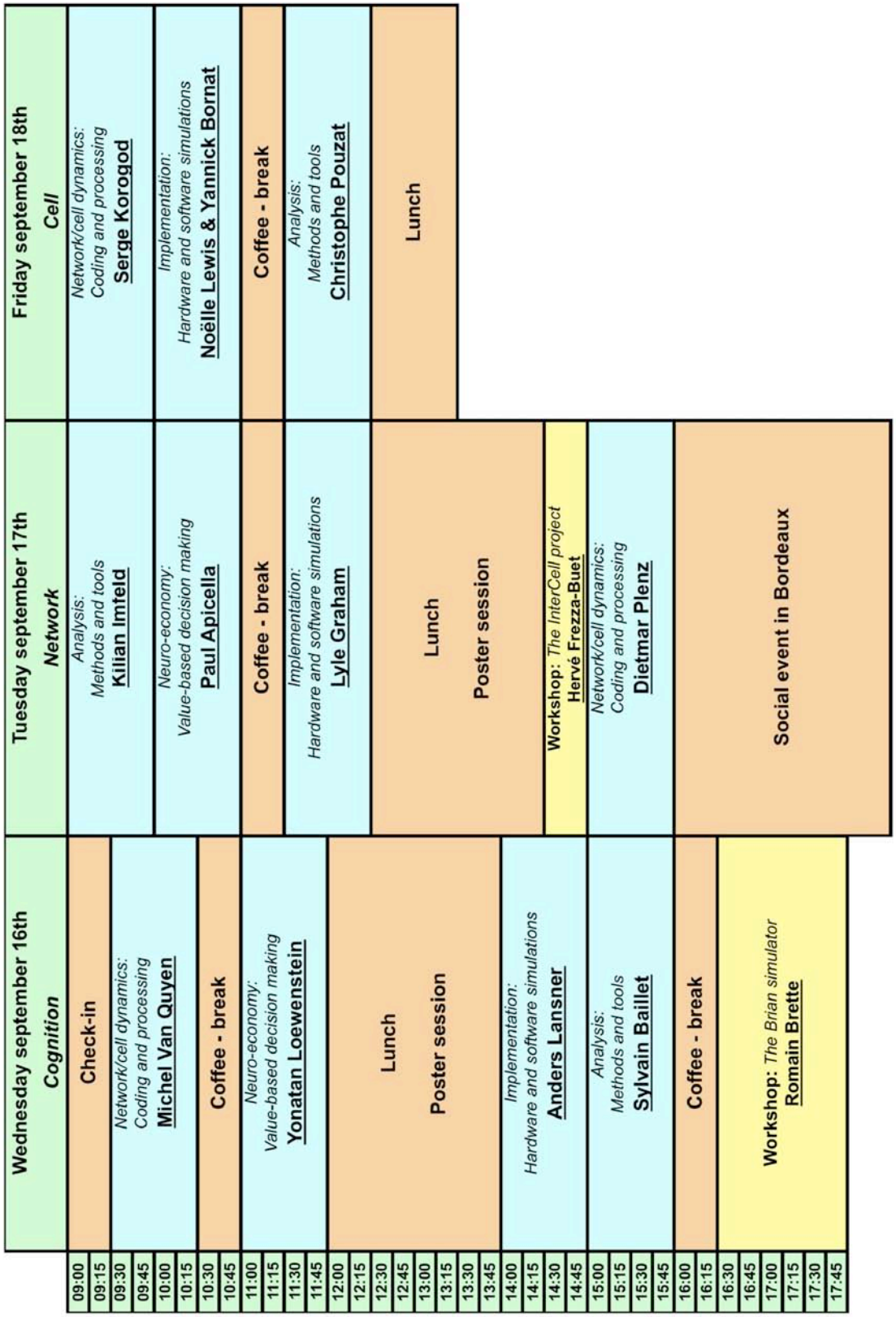


Table of Contents

LECTURES

WORKSHOPS

POSTERS

LECTURES

Large-scale microelectrode recordings of gamma oscillations in human neocortex during sleep

Michel Le Van Quyen[1], Richard Staba[2], Anatol Bragin[2], Jerome Engel Jr.[2]

1- Centre de Recherche de l'ICM, INSERM UMRS 975 - CNRS UMR 7225
Hôpital de la Pitié-Salpêtrière, Paris, France.

[2]Neurology Department, David Geffen School of Medicine at UCLA, Los Angeles, USA

Gamma oscillations (40-120 Hz), usually associated with waking functions can be recorded in the deepest stages of sleep in anesthetized and naturally sleeping animals. The full details of their large-scale coordinations across multiple cortical networks are still unknown. For this purpose, we examined the existence of gamma patterns during polysomnographically defined sleep-wake states using large-scale microelectrode recordings, collected in parallel with macroscopic scalp EEG, in 9 epilepsy patients undergoing presurgical clinical setting. We report that gamma oscillations recurrently emerged over time windows of several hundreds of msec in many cortical sites during slow-wave sleep (SWS) and they are also present, but generally less pronounced, during waking and REM sleep. During SWS, gamma oscillations were correlated with positive peaks of EEG slow oscillations showing that gamma patterns were associated with cortical cells' simultaneous switch to the UP state. Several discrete spectral peaks were systematically identified in the low gamma range, around 50 Hz and 70 Hz, and also, occasionally, in the high gamma range around 90 Hz and 110 Hz, suggesting that multiple gamma generators are present within the human neocortex. Gamma oscillations often involved a single local cortical area but occasionally involved multiple different cortical areas, forming large-scale spatial patterns. Nevertheless, the spatial coherence between cortical foci exhibiting gamma activities was local and fell off quickly with distance when computed between different neocortical sites. After single unit isolation, consistent with a switch to the UP state, we observed that the majority of cells (144 / 206) exhibited a strong increase in spike firing during gamma patterns and that many of these cells (52%) were significantly phase-locked to the oscillations, preferentially around the troughs of the gamma cycle. Furthermore, coincident firings with millisecond precision were specifically enhanced during gamma oscillations between cells over the same cortical area, suggesting that fast oscillations facilitate local neuronal synchronization. Our findings confirm and extend earlier animal studies reporting that gamma oscillations, generally regarded as characterizing states of brain arousal, are transiently expressed during SWS. In particular, we report, for the first time during sleep, that gamma oscillations play a key role in promoting millisecond precise synchrony of unit activities within cortical areas, providing short windows of opportunity for interactions of coactivated groups of cortical neurons. We speculate that these gamma patterns briefly restore “micro-wake” activity patterns and are important for consolidation of memory traces acquired during previous awake periods.

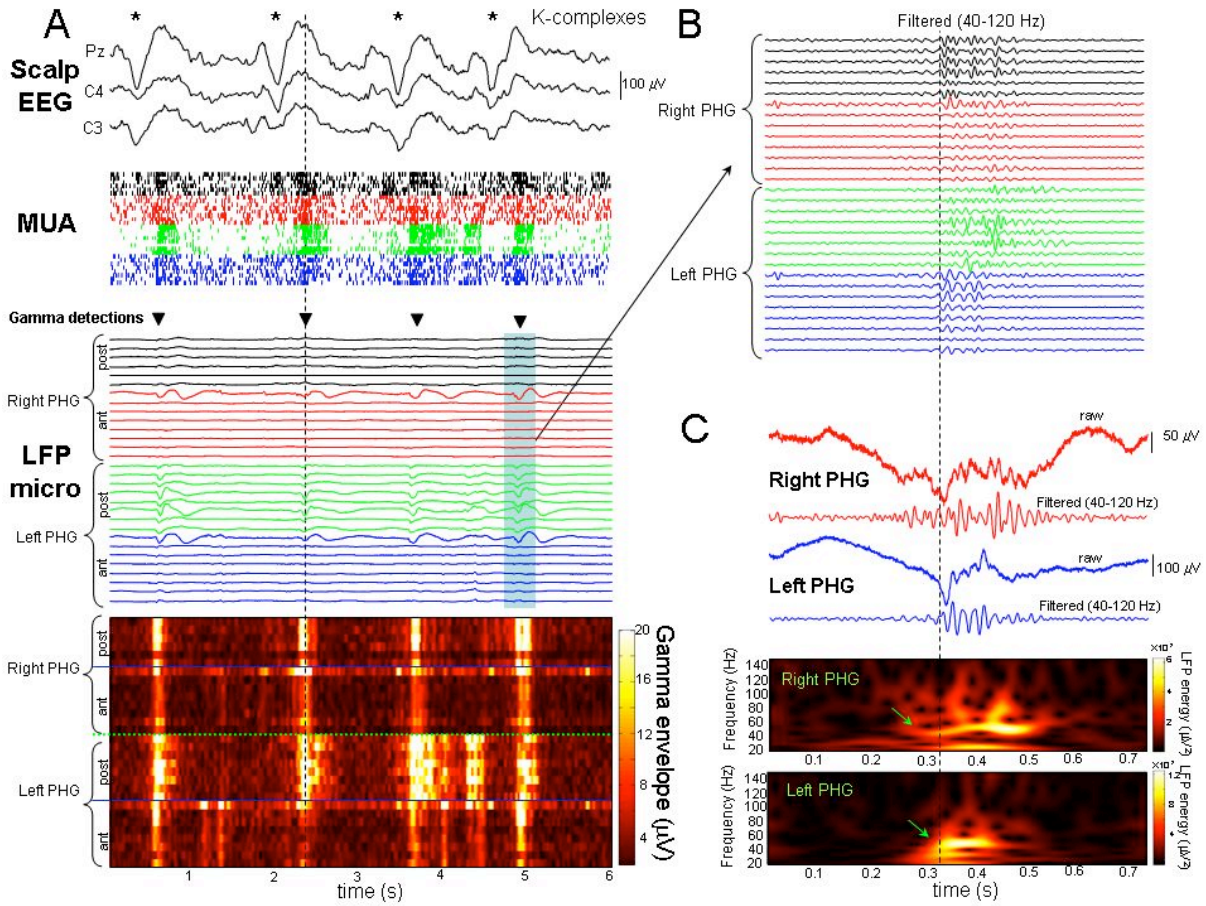


Figure: Gamma patterns recorded during sleep stage 2

A. *Middle*: Examples of detected gamma patterns (black triangles) recorded over 30 microelectrodes in the field potentials (LFP) of the right and left posterior parahippocampal gyri (PHG, *ant*: anterior part and *post*: posterior part) *Top*: Simultaneous recordings of scalp EEG indicate that gamma oscillations were temporally correlated with positive peaks of EEG K-complexes. Strong multi-unit activities (MUA) were observed during these patterns. *Bottom*: Envelope amplitudes of the filtered signals in the gamma range. Note that gamma activities formed large spatial patterns occurring almost simultaneously between all recorded cortical sites.

B. *Top*: Display of one detected gamma pattern in the 40-120 Hz frequency range.

C. Display of two homotopic channels with their corresponding wavelet transforms. Note, in the time-frequency maps, strong gamma oscillations with distinct narrow band frequencies around 50 Hz (*green arrows*).

Utility maximization by reactive policies a game-theoretic approach

Yonatan Loewenstein[1]

Depts. of Neurobiology and Cognitive Sciences and the Interdisciplinary Center for Neural Computation, Hebrew University, Jerusalem, 91904, Israel

According to rational choice theory, subjects make choices as to maximize their well-being or utility. In its standard framework, rational choice theory posits that subjects have complete information about the state of the world and choose their actions as to maximize the expectation value of the sum of present and exponentially discounted future rewards. In this case, the optimal policy is deterministic and can be attained by several simple reinforcement learning algorithms. Whether the brain actually implements these algorithms and what is their neural basis are subjects of intense neuroscience research.

However, in many real-life situations, the state of the world is only partially known. Technically, these situations are referred to as POMDPs. Furthermore, the temporal discounting of future rewards, as measured using intertemporal choice experiments in both human and animal subjects, is typically non-exponential. It is not clear how to define an optimal policy, let alone find it, if the full observability or the exponential discounting assumptions are relaxed. The reason is that in general, the optimal plan at one point in time is not optimal at a later time and therefore will not be followed, leading to a paradoxical intertemproal conflict.

Here we present a novel theory of choice that is based on learned reactions. We consider a subject with an arbitrary discounting function that operates in a POMDP in which an observation at each decision point provides partial information about the state of the world. We consider the set of stochastic reactive policies in which the subject responds to each observation by choosing an action from an observation-dependent probability distribution over all possible actions. To address the intertemporal conflict, we define an intertemporal game in which choices made by a subject over time are viewed as if they are made by different 'selves', each making a single choice at a different point in time. We define optimal policy to be a time-invariant Nash equilibrium of the game between the multiple selves.

How can this Nash equilibrium be attained? We show that the fixed point of a simple on-line reinforcement learning algorithm, previously used to generate a biased estimate of the gradient of the average reward, is the time-invariant Nash equilibrium of the intertemporal game. We show preliminary results that show how such algorithm can be implemented in the brain by a reward-modulated synaptic plasticity rule.

Associative memory properties of a large-scale biophysically detailed neuronal network model of layers 2/3 of mammalian neocortex

Anders Lansner[1]

Dept of Computational Biology
Stockholm University and KTH
Stockholm, Sweden

One of the most prominent theories of cortical memory function is the theory of cell assemblies put forward in 1949 by Donald Hebb. Critical components of his theory are the Hebbian synapse displaying associative plasticity and the cell assembly formed in the recurrent cortical “attractor” neuronal networks by means of such plasticity. The theory addresses the perceptual and associative memory functions of the cortex, but its relevance for the real neuronal networks of the mammalian cortex has been unclear. I will describe simulations of a large-scale biophysically detailed model of layers 2/3 of mammalian neocortex aimed at investigating this and other related issues. The simulated network is composed of multi-compartmental Hodgkin-Huxley type pyramidal cells and two types of inhibitory interneurons connected via glutamatergic and GABA-ergic synapses. This model has been scaled up on a parallel cluster computer to a size of 11 million neurons and 22 billion synapses, corresponding to a cortical patch of about 22x22 mm.

Memories are stored in this cortical network by means of a Hebbian learning rule. The trained network displays appropriate associative memory functionality, like e.g. memory recall as well as perceptual completion and rivalry, on time scales compatible with those measured experimentally. It reproduces several dynamic phenomena seen at the cellular, microcircuit and global network levels, and provides possible explanations for cognitive phenomena like e.g. attentional blink.

In the context of this large-scale cortical network model I will also illustrate and discuss some methodological aspects of performing multi-scale neuronal simulations in general as well as ongoing work within the FACETS project on hardware implementation of spiking large-scale neural networks and cortical computation.

Electromagnetic functional imaging at the speed of brain

Sylvain Baillet[1,2]

1- Department of Neurology, Medical College of Wisconsin, Milwaukee, USA
2- COGIMAGE Brain Imaging Group, Centre de Recherche de l'Institut du Cerveau et de la Moelle, CNRS/INSERM/UPMC, Paris

.

Functional imaging techniques have certainly changed the way neuroscientists and clinicians are looking at the brain. In that respect, positon emission tomography (PET) and functional magnetic resonance imaging (fMRI) have largely contributed to mapping the brain in action.

With increasing numbers of sensors integrated in whole-head arrays, electro and magnetoencephalography (EEG, MEG) have now matured as true functional imaging modalities. With a temporal resolution in the millisecond range, their contribution to the exploration of brain functions has unprecedented potentials. Imaging the neural sources of EEG and MEG scalp signals is essentially a modelling problem. We will discuss how the integration of structural information from MRI contributes to leverage the basic indeterminacy in the modelling of MEG/EEG neural generators.

The fine time resolution of MEG/EEG imaging can take many faces while studying brain functions. We will therefore illustrate how basic evoked brain responses can be complemented by the identification and localization of neural oscillatory components and interactions in specific frequency domains.

From a more technical standpoint, this talk will introduce the basic signal and image processing apparatus being used for MEG/EEG source imaging and also discuss more recent developments dedicated to the identification of propagating patterns of cortical currents using optical flow techniques. A variety of experimental data examples will be provided for illustration.

Representative publications:

Lefèvre, J. & Baillet, S. (2008) *Optical Flow and Advection on 2-Riemannian Manifolds: a Common Framework,* **IEEE Trans. on Pattern Analysis & Machine Intelligence**, June 30(6): 1081-92

Jerbi, K.; Lachaux, J.; N'Diaye, K.; Pantazis, D.; Leahy, R.; Garnero, L., Baillet, S. (2007), *Coherent Neural Representation of Hand Speed in Humans Revealed by MEG Imaging*, **Proceedings of the National Academy of Sciences of the USA**, 104(18):7676-81

C. Sergent, S. Baillet & S. Dehaene (2005), Timing of the Brain Events Underlying Access to Consciousness during the Attentional Blink, **Nature Neuroscience**, Oct; 8(10): 1391_1400.

In vitro Large-Scale Neuronal Networks – Signal Acquisition and Analysis

Kilian Imfeld[1], Luca Berdondini[2], Alessandro Maccione[2], Peter Seitz[1]

1-Centre Suisse d'Electronique et Microtechnique (CSEM), Landquart, Switzerland
2-Italian Institute of Technology (IIT), Genova, Italy

This talk highlights technological aspects in the field of large-scale cultured neuronal networks and outlines a few analysis methods that are used to extract relevant information from the underlying processes. Conventional electrophysiological instrumentation, such as patch-clamp techniques, allows accessing and monitoring a few individual neurons. Functional methodologies, such as implanted electrodes, functional-MRI and electroencenphalography reveal relationships between various brain areas.

However, learning and plasticity processes in a network consisting of thousands of cells are difficult to track and analyze with those methods. In this respect, primary neuronal cell cultures as well as organotypic brain slice cultures represent valuable models of reduced complexity for the study of such neuronal processes and for the screening of potential drugs. Microelectrode arrays (MEA) can be used to monitor these large assemblies of neurons.

Statistical methods are applied to extract relevant parameters from the bulk of data in order to characterize the network under different biological conditions. Cross-correlation and mutual information between neurons are computed to access the functional dependence of groups of neurons within a network. In the end, this would lead to a functional mapping of the underlying network, however it is computationally very expensive. An alternative method for the characterization of a neuronal culture is the use of some global parameters that describe the overall state of a network (e.g. center of activity trajectory). Such parameters are of particular interest for high-throughput pharmaceutical screenings.

An important shortcoming of the current MEA technology is its limited spatial resolution. Only a few tens to a few hundreds of neurons can be monitored with current implementations and therefore a large spatial undersampling of the activity is expected. Recently, CMOS-based MEAs were introduced and they enable the integration of thousands of electrodes (figure 1). The spatial resolution is increased down to a single cell and at the same time a large network consisting of many thousands of neurons can still be monitored. In analogy to conventional imaging techniques, these new MEA devices can thus provide and visualize data as a sequence of images (figure 1). Hence, analysis concepts inspired from the imaging field are introduced. As an illustrative example, the center of gravity of activity can efficiently describe the different burst behaviors of an *in vitro* neuronal network, however without trading off for temporal resolution (figure 2).

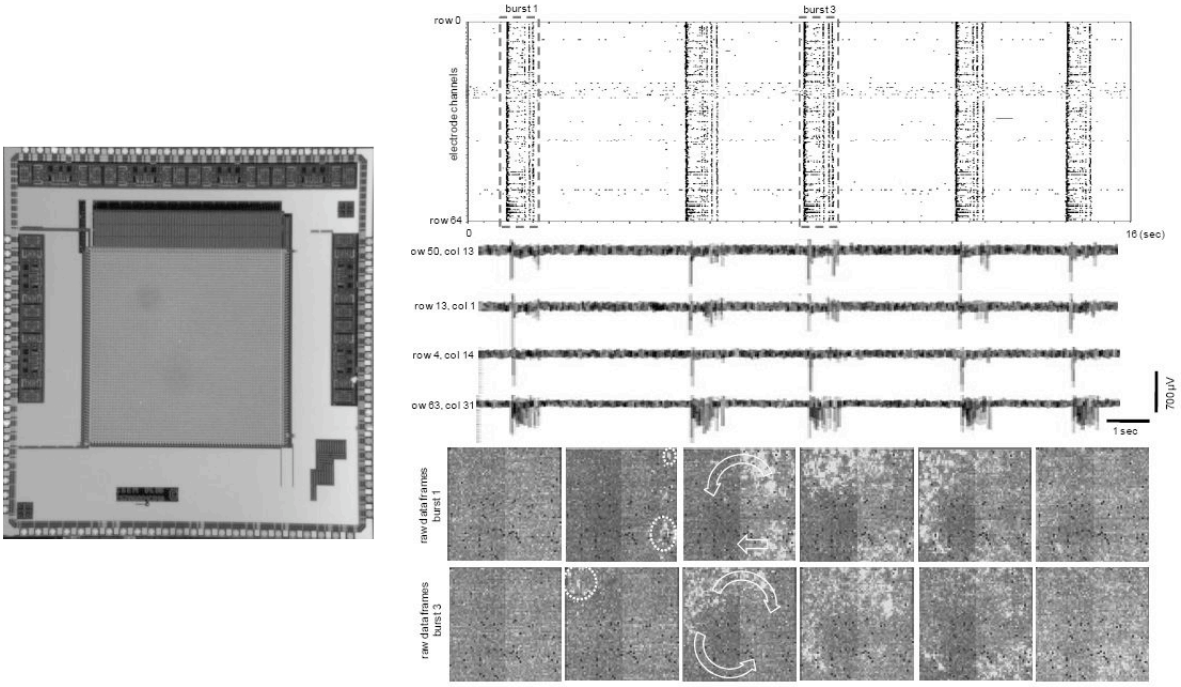


Figure 1: High-resolution large-scale MEA
The sensor integrates 4096 electrodes on an area of 7 mm^2 (left). Measured burst activity of a dissociated hippocampal culture (rat, DIV 27) is depicted. Two specific bursts are shown as a sequence of images (right).

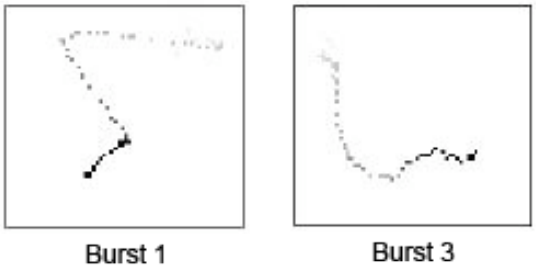


Figure 2: Center of Activity
Trajectory of the Center of Gravity of Activity of burst 1 and 3 from figure 1 (temporal evolution from white to black).

Economic views of reward influences at the neuronal level

Paul Apicella

Laboratoire de Neurobiologie de la Cognition UMR6155, Université de Provence-CNRS
3 place Victor Hugo, 13331 Marseille Cedex 3, France

How primates make decisions or choices in various contexts is currently the subject of intense investigations in neurosciences. Many of these decisions are important to flexibly adapt behavior based on the expected outcome of the choice. One topic of interest in this field is the role of rewards on decision making. Animal research and human brain imaging studies suggest that a number of interacting brain regions contribute to reward-based decision making. Also, psychiatric disorders are associated with disturbances in decision processes as a consequence of impaired evaluation of future rewards as well as uncontrollable translation into unwanted or inappropriate actions (compulsions). The complementary strengths of neurophysiological approaches, in humans (fMRI) and animals (neuronal recordings), with economic approaches that take into account the probability of reward, the value of reward, and the cost of the work to obtain reward, provide a framework for understanding the computations in the brain during decision making. I will briefly review the neuronal evidence for the role of specific brain regions, particularly orbitofrontal cortex, striatum and amygdala, during reward-based decision making. Computational models have yielded insights about the contribution of each of these key structures to evaluation and action selection processes and how their interactions may allow the brain to choose among several possible options. In particular, the role of the striatum and midbrain dopamine neurons in reward processing is well established. It has been shown that striatal neurons encode the reward value of stimuli and/or actions. In our recent work, we found that a population of striatal neurons, thought to be interneurons, are sensitive to changes in the probability of a rewarding outcome, with a subset of them being candidates for encoding a reward prediction error (Figure 1). Because an important aspect of decision making concerns the ability to detect prediction errors and adjust behavior accordingly, these findings suggest that a local circuit system in the striatum has a specialized role in combining prediction error signal input with motor, cognitive, and motivational signals carried by striatal output neurons during decision making. Establishing the direction and nature of interactions between cortical and subcortical areas involved in reward processing and action selection remains an important goal for understanding how neuronal circuits implement decisions about reward. In this regard, the combination of experimental findings and theories of economic rationality has proven extremely valuable in guiding investigations into our ability to process various alternatives and make a "good" decision.

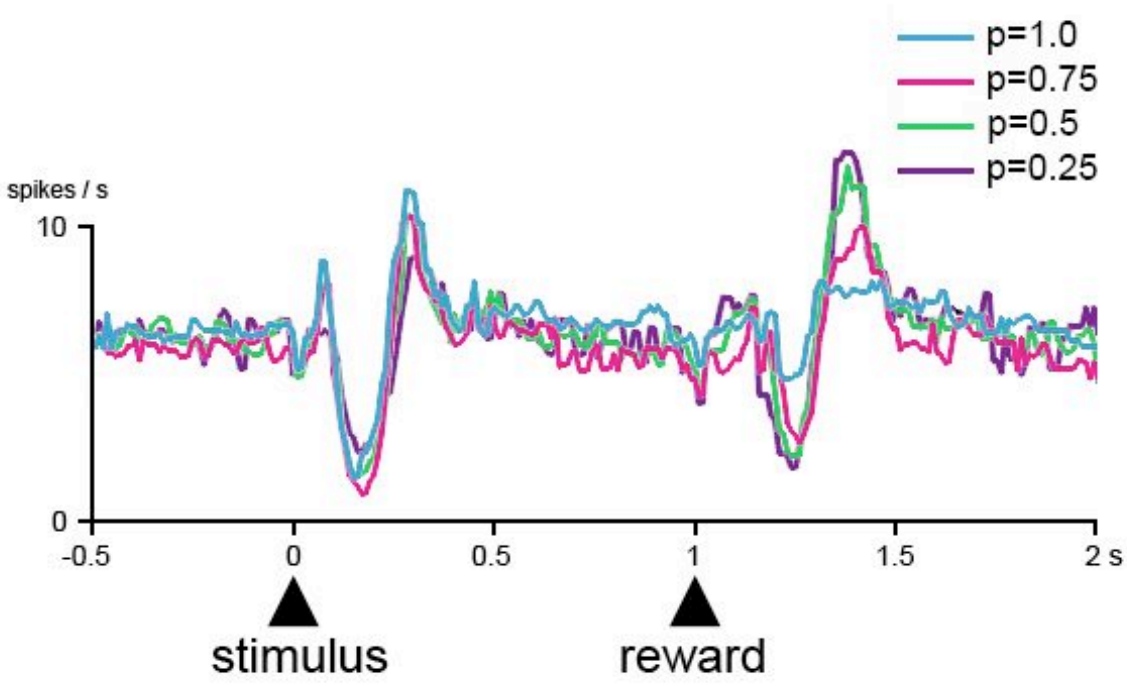


Figure1. The averaged activity of presumed striatal interneurons was modulated by the probability of reward in a classical conditioning task

The study was conducted on macaque monkeys subjected to a Pavlovian procedure in which reward delivery (a drop of fruit juice) was predicted by a visual stimulus. In different trial blocks, rewards occurred at four different probabilities. Population response histograms with reward probabilities ranging from p = 1.0 to p = 0.25 are shown from top to bottom and included only rewarded trials. These data were obtained from samples of 56-87 putative interneurons recorded in the striatum. Arrows indicate the onset of the conditioned stimulus and the delivery of reward 1 s later. The biphasic neuronal response (pause-rebound) to reward is maximal at p = 0.25 and p = 0.5 and less prominent at higher probabilities.With increasing probability of reward, the neurons also respond somewhat stronger to the conditioned stimulus. Such a response profile across reward probabilities may reflect a positive prediction error.

Exploring the Functional Roles of Membrane Conductances with Experiments and Models – In Vivo Dynamic Clamp Study of Shunting Inhibition and the BK Current in Visual Cortex

Lyle J. Graham [1]

1-Neurophysiology of Visual Computation Group, Laboratory of Neurophysics and Physiology, CNRS UMR8119, Université Paris Descartes, Paris, France

The essential dynamics of neurons underlying synaptic integration and spike generation are understood to define these cells' unique computational role within the neural net. Yet, in the context of the intact system and physiological function the quantitative and qualitative identification of specific biophysical mechanisms is still at an early stage. In this talk I will review our recent work in addressing these questions using in vivo dynamic-clamp recordings that are strongly tied to cellular and membrane models. In particular, we are characterizing the influence of shunting synaptic inhibition and the BK potassium current on the neuron's transfer function, probed with both artificial and functional, visual stimuli. In comparison to previous experimental and theoretical studies, we find that realistic shunting inhibition has a significant divisive effect on firing gain, as well as important effects on threshold and saturation. Shunting inhibition also has a non-linear effect on visual responses, reducing response amplitude but as well tightening response timing. We confirm predictions that the BK current facilitates spike firing, despite being a hyperpolarizing current. This effect is demonstrated by an increase in the gain of the f/I curve and of visual responses.

Neuronal Avalanches in the Cortex: A Case for Criticality

Dietmar Plenz[1]

1-NIMH, Sect Critical Brain Dynamics, BETHESDA, MD, USA

Complex systems, when poised near a critical point of a phase transition between order and disorder, exhibit scale-free, power law dynamics. Critical systems are highly adaptive and flexibly process and store information, which prompted the conjecture that the cortex might operate at criticality. This view is supported by the recent discovery of neuronal avalanches in superficial layers of cortex. The spatiotemporal, synchronized activity patterns of avalanches form a scale-free organization that spontaneously emerges in vitro as well as in vivo in the anesthetized rat and awake monkeys. Avalanches are established at the time of superficial layer differentiation, require balanced fast excitation and inhibition, and are regulated via an inverted-U profile of NMDA/dopamine-D1 interaction. Neuronal synchronization in the form of avalanches naturally incorporates driven conditions such as found in nested theta/gamma-oscillations. Neuronal avalanches also pose a general stochastic framework that for sequential activations similar to synfire chains. Importantly, a single avalanche is not an isolated network event, but rather its specific occurrence in time, its spatial spread, and overall size is part of an elementary organization of the dynamics that is described by three fundamental power laws. Overall, these results suggest that neuronal avalanches indicate a critical network dynamics at which the cortex gains universal properties found at criticality. These properties constitute a novel framework that allow for a precise quantification of cortex function such as the absolute discrimination of pathological from non-pathological synchronization, and the identification of maximal dynamic range for input-output processing.

Spatial aspects of neuronal coding and processing at cellular level

Sergiy Korogod[1]

1-Dnipropetrovsk National University and Dnipropetrovsk Division of International Center for Molecular Physiology, National Academy of Sciences of the Ukraine

Spatial aspect of the cell dynamics refers to snapshots of electrical states mapped on structural elements of a neuron displaying how these elements act together to produce characteristic patterns of action potentials at the cell output, the neuronal codes. Complexity and diversity of cell shapes defined mainly by the dendrites are generally thought of as structural prerequisites for complex and diverse functions of neurons and their networks in the brain. Complex structure and nonlinear membrane properties of neurons impede relating dynamics of individual cells and their network. A usual compromise is reduction of the cell structure that affects both the spatial and temporal aspects of the cellular dynamics. Well-known examples are the "point" neuron reduced to a single-compartment, the soma and the "ball-and-stick" neuron composed of the soma with attached non-branching dendrite. In the first case the spatial aspect is absent and the output codes result from temporal processing of synaptic inputs targeted at the soma. In the second case the output depends on temporal and spatial processing of inputs situated at various path distances from the soma. Here the spatial aspect includes the "distal-to-proximal" relations between sites communicating by lateral currents transferred in somatopetal (forward) and somatofugal (backward) directions. In cells with branching dendrites the spatial relations are significantly enriched by the "path-to-path" relations between sites situated at the same path distances from the soma but on different dendritic paths. These relations crucially depend on such structural feature of the dendrites as metrical asymmetry due to difference in lengths and/or diameters between the dendritic branches and paths emerging from the common origin. Size and complexity (lengths and number of branches) also do matter. Demonstrative examples are neocortical pyramidal and cerebellar Purkinje neurons obviously different in both geometry of the dendritic arborizations and population of ion channels. The structure-defined features of cellular dynamics were highlighted in the model neurons exposed to synaptic input of the same type: tonic excitation homogeneously distributed over the dendrites. Spatial electrical maps were explored for different discharge patterns, i.e. sequences of elementary events such as single spikes or bursts (spike doublets, triplets, quadruplets, etc.) generated in various combinations at different intensities of synaptic activation. In the both models, the patterns were regular with different number of elementary events in the periodically repeating sequence or irregular, stochastic. Each pattern had its distinct spatial signature: a characteristic combination of states of high (upstate) or low (downstate) depolarization of metrically asymmetrical dendritic domains. Depending on synaptic intensity, the asymmetrical dendritic sub-trees united or disunited to form larger or smaller cooperatively behaving spatial domains. Pyramidal and Purkinje cells showed the same transformations of patterns in different ranges of synaptic intensities indicating size-dependence of the cellular dynamics. The disclosed spatial heterogeneity of electrical states during generation of different patters is important for the network dynamics: for signal processing it does matter not only "when in time", but also "where in space" of the dendritic arborisation the cell receives phasic synaptic inputs.

Spiking Neural Network Hardware implementation: from single neurons to networks

N. Lewis, Y. Bornat, S. Renaud

University of Bordeaux, IMS Laboratory (CNRS/ENSEIRB), Talence, France

Spiking Neural Networks (SNN) are used in computational neurosciences to simulate information processing in the brain. SNN implement biologically plausible models of neural networks, from the detailed neuron physiology to network adaptation and plasticity rules. Various implementation solutions exist for SNN, including software, hardware or mixed systems. In this presentation, we will focus on hardware-based SNN and propose a review of the most recently developed platforms as well as pioneer platforms. These hardware-based platforms often use analog cores to emulate the neuron-level behavior while other computation or configuration tasks are distributed on digital hardware or software units.

First we will introduce the principle of hardware implementation, from the primitive computational elements (transistors, Boolean operators) to the description of ASIC (Application Specific Integrated Circuit) design flow. Typical performance criteria will be pointed out to compare analog/digital hardware designs, supported by a detailed example of a real analog solver (naturally continuous- and real-time).

Before reviewing the hardware SNN solutions, we will present an overview of standard computational models, from the cellular or sub-cellular level, to the network level. At the neuron-level, models can be classified in two main categories: conductance-based or threshold-type, with several levels of abstraction in each category. At the network level, connectivity between neurons can be managed in different ways and plasticity rules can be also implemented with more or less details and parameters. This brief overview will highlight the trade-off between biological plausibility and computational cost. It finally shows how the models choice is closely related to the SNN application field.

The reviewed hardware-based SNN will be compared in terms of: network size, neuron model accuracy, ability to real-time operation, multi-scale configurability and ability to hybrid living-artificial connection. A few systems will be described in a more detailed fashion, within their specific application context.

Finally, some projects which are currently in progress using hardware SNN will be exposed. Their goal is to explore novel paradigms for information processing and overcome the related technological limitations.

Spike Train Analysis

Christophe Pouzat[1]

1- Laboratoire de Physiologie Cérébrale, CNRS UMR 8118 , Université Paris-Descartes, Paris, France

Since the seminal work of Adrian and Zotterman (1926, J Physiol, 61: 465-483), a central working hypothesis of systems neuroscience has been that action potential or spike occurrence times, as opposed to spike waveforms, are the sole information carrier between brain regions. This hypothesis legitimates and leads to the study of spike trains per se. It also encourages the development of models whose goal is to predict the probability of occurrence of a spike at a given time, without necessarily considering the biophysical spike generation mechanisms. In short it leads to the application of the point process / counting process formalism to neuronal spike trains introduced by Perkel, Gerstein and Moore (1967, Biophys J, 7: 391-418).

We will start by reviewing classical and well know spike train analysis methods like the inter-spike interval histogram, the auto- and cross-correlation functions (Perkel et al, 1967) as well as less well know ones (Fitzugh, 1958, J Gen Physiol 41: 675-692). We will then introduce and discuss methods aiming at the estimation of the conditional intensity of spike trains, the key quantity involved in the point process / counting process formalism. These methods were first introduce by Brillinger (1988, Biol Cybern, 59: 189-200) and have generated a lot of activity during the last decade.

Emphasis will be but on methods implementations in software and on available goodness of fit tests.

WORKSHOPS

The Brian simulator

Romain Brette (1,2) and Dan Goodman (1,2)
1. Laboratoire Psychologie de la Perception, CNRS and Université Paris Descartes, Paris, France
2. Département d'Etudes Cognitives, Ecole Normale Supérieure, Paris, France
Email: romain.brette@ens.fr

Brian is a simulator for spiking neural networks written in Python (http://www.briansimulator.org). The focus is on making the writing of simulation code as quick and easy as possible, and on flexibility: new and non-standard models are no more difficult to define than standard ones. This allows scientists to spend more time on the details of their models, and less on their implementation. Neuron models are defined by writing differential equations in standard mathematical notation, facilitating scientific communication.
In this tutorial, we will introduce the Brian simulator with concrete examples. Anyone interested should bring a laptop, if possible with the software installed as explained on the web site: http://www.briansimulator.org/download/ (otherwise we will help you with the installation). No previous knowledge of Python is required.

The InterCell Project

Hervé Frezza-Buet
IMS, Supélec, Metz, France

The InterCell project aims at offering a cluster of 256 PCs to scientists who need to experiment large scale fine grain computing. It implements on such a cluster a generic neural paradigm: computational units linked by an arbitrary connectivity, updated in parallel. One of the targeted applications is the use of such a paradigm in computational neuroscience, for modelling biologically inspired robot controllers. Such a goal implies that the execution of the neural network on the cluster can actually be coupled, through the robotic device, to some real environment. Allowing situated computation on a cluster is an originality of the project, since the middleware offers client-server facilities to interact on-line with the running simulation.

The project is supported by INRIA and the Region Lorraine (CPER MISN/MIS 2007), and managed at the Metz campus of Supélec.

Links:
http://intercell.metz.supelec.fr
http://www.loria.fr/~falex/index.php/Adm/CperMis

POSTERS

Plasticity Computation in a Multiboard System Simulator Dedicated to SNN

B. Belhadj, J. Tomas, O. Malot, Y. Bornat, S. Saïghi and S. Renaud
IMS Labs, University of Bordeaux, France
bilel.belhadj@ims-bordeaux.fr

***Keywords*—Hardware spiking neural network simulator, Mutltiboard system, Real-time distributed STDP computation, FPGA processing, Analog Hodgkin-Huxley neurons.**

I. Introduction

This work falls into the perspective of developing hardware systems to simulate adaptive variability in biologically plausible neural networks. A realistic STDP model is digitally implemented onto commercial FPGAs while Hodgkin-Huxley neuron model is mapped onto analog VLSI circuit. The system operates in biological time scale and releases accurate simulation results compared with floating-point software computation.

II. System Overview

The global system consists of a system rack connecting a series of similar electronic boards. The current version of the system can host up to 140 neurons spread across 7 boards. Each board is a six layers full-custom board which hosts 4 analog ASICs and one *Xilinx Spartan3*™ FPGA. Each ASIC incorporates 5 neurons which compute in analog mode conductance-based models following the Hodgkin-Huxley formalism [1]. Individual neurons produce in continuous time action potentials that express their intrinsic dynamic properties. Ionic channel properties are fully reconfigurable [2]. When the neuron output comparator detects an action potential, a digital 1-bit event is transmitted to the FPGA. In turn, the FPGA computes the plasticity as it relates to the incoming events and generates a digital pulse whose width encodes the synaptic weight. This pulse triggers the transition to the opening state of the synaptic channels in each postsynaptic neuron.

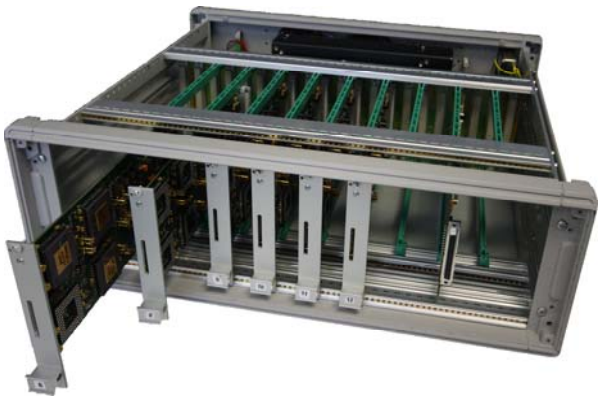


Fig. 1. The system rack is composed by a series of electronic boards connected via a common 64-bit wide bus. A token-ring based communication protocol assures information exchange between boards.

III. STDP computation

A. STDP model

The STDP model encodes the timing and amplitude of the mutual influence between pre- and postsynaptic spikes. Furthermore, it modulates the synaptic strength according to the previous history of spike times (spike efficacy). In order to avoid infinite evolution of the synaptic strength, saturating factors attenuate synaptic weights as the higher or lower boundary approaches. Synaptic weights are then updated according to the mathematical equation of the STDP model.

B. Distributed STDP computation

The system distributes plasticity computation over several FPGAs located on different boards. In addition to local plasticity processing, each FPGA computes plastic changes of afferent events released by external presynaptic neurons. Exchanged events are multiplexed in time before their transmission through communication bus. The communication protocol guarantees the integrity and the required arrival time of travelling events.

C. Simulation results

The system provides accurate simulation results compared with software floating-point computation implemented on a PC using C language. The system operates in real-time mode and the execution time is similar to the biological counterpart. To assess the functional robustness of the system, we provoke the worst case situation that can happen in SNNs (simultaneous activity in all-to-all connected network). The system is then able to simulate STDP related applications for any network configuration.

IV. Bibliography

[1] T. Lévi, N. Lewis, S. Saïghi, J. Tomas, Y. Bornat, and S. Renaud, "Neuromimetic Integrated Circuits," in VLSI Circuits for Biomedical Applications, K. Iniewski, Ed. Boston: Artech House, 241-264 (2008).

[2] L. Buhry et al. , "Parameter estimation of the Hodgkin-Huxley model using metaheuristics: application to neuromimetic analog integrated circuits," IEEE Biomedical circuits and systems, 173-176 (2008).

[3] A. Destexhe and Z. F. Mainen, “Plasticity in Single Neuron and Circuit Computation,” Nature, 789–795 (2004).

Modeling Classical and Instrumental Conditioning in Computational Neuroscience

Pierre Berthet and Jean-Marc Salotti
Institut de Cognitique, EA-487 Laboratory
berthetp@gmail.com salotti@idc-bordeaux.fr

***Keywords*—Conditioning, developmental robotics, learning, neural network.**

I.Introduction

What is the contribution of conditioning in the general learning process? This type of learning is important as it could be a basic element used in more complex learning mechanisms of animals or robots. This work is therefore related to the growing field of developmental robotics (Lungarella et al., 2004).

We already had a conditioning model for classical and operant conditioning but the aim of this work was to adapt it within the computational neuroscience framework (Salotti & Lepretre, 2008). The idea is to change our view and to improve the model thanks to the synergy of different disciplines.

II.Development

A.Starting model

Our model is based on the learning of a Bayesian network. It is a predictive model in the sense that it does not focus on the action selection mechanism but mainly on the probability that reinforcement will follow a given event. An important assumption of the model is that conditioning is strictly event based: It does not matter if a stimulus is present or not (i.e. On or Off). What matters is the existence of variations in the representation of the environment (stimulus On/Off or action event).

B.Proposed modeling

It is similar to the critic part of the Actor-critic approach (Barto, 1995) in that a prediction is proposed first and updates of synaptic weights are made depending on the outcome. The main originality of our model is that there are two variable synaptic weights per stimulus, the first between layer 1 and layer 2 and the second between layer 2 and the outcome (see figure 1). The propagation is strictly feed-forward. Updates rules are based on Hebb's rule and an adapted version of the Rescorla-Wagner function. Intra-layer 2 connections are both excitatory and inhibitory with constant weights. However, the activity of layer-2 neurons strongly depends on the timing of its neighbors (which neuron is activated first). Layer-2 connections enable blocking and secondary conditioning.

III.Results

We managed to fully reproduce active and passive extinction, blocking (strict), latent inhibition, secondary and even n-ary conditioning and reacquisition. Furthermore, we also added a forgotten rule, which slowly decreases or increases synaptic weights towards their original value. Such rules make it possible to reproduce spontaneous recovery, which is a well known complex conditioning feature.

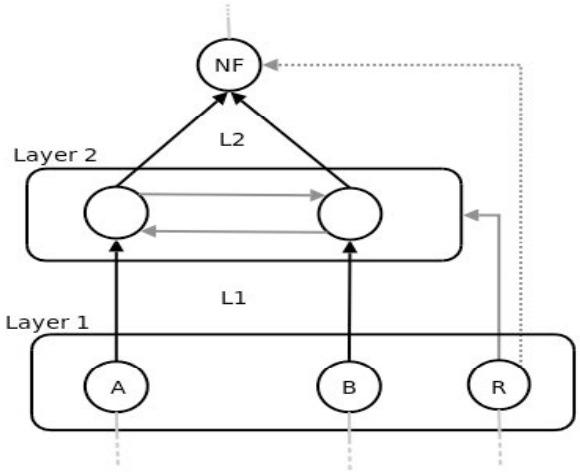


Fig. 1. Example of neural network with only 2 units per layer. Neurons in layer 1 are activated when events corresponding to onsets of stimuli or triggered actions are detected. R is the reward unit (dopamine like). NF is a predictor of the reward.

IV.Conclusion

The synergy is successful. Complex conditioning phenomena, such as spontaneous recovery, can be handled by our model with two adaptive weights per stimulus.

V.Bibliography

Barto, A. G. (1995). Adaptive Critics and the Basal Ganglia. In: Houk, J. C., Davis, J. and Beiser, D., editor, *Models of Information Processing in the Basal Ganglia*, Cambridge, MA: MIT Press : 215-232.

Joel, D., Niv, Y. and Ruppin, E. (2002). Actor-Critic Models of the Basal Ganglia: New Anatomical and Computational Perspectives. *Neural Networks*, 15 : 532-547.

Lungarella, M., Metta, G., Pfeifer, R. and Sandini, G. (2004). Developmental Robotics: a Survey. *Connection Science*, 0 : 1-40.

Rescorla, R. A. and Wagner, A. R. (1972). A Theory of Pavlovian Conditioning: Variations in the Effectiveness of Reinforcement and Nonreinforcement. In Black, A. H. & Prokasy, W. F. (eds.), *Classical Conditioning II*, New York Appleton-Century-Crofts : 64-99.

Salotti, J.M. and Lepretre, F. (2008). A Bayesian Approach for Classical and Operant Conditioning. Actes du Workshop « From Motor to Interaction Learning in Robots », IEEE/RSJ International Conference on Intelligent Robots and Systems, 22 – 26 Septembre 2008.

A context-specific independence model of multisensory perception

Patricia Besson[1], Jonas Richiardi[2]
[1] ISM, UMR-CNRS 6233, France, [2] PatternLab SARL, Switzerland
Patricia.Besson@univmed.fr

***Keywords*— audiovisual perception, mutual information, Bayesian network, cross-modal, segregation**

I. Introduction

Human beings process the information coming from their different senses to finely grasp their environment. The perception of temporally co-occurring multisensory (MS) stimuli may lead to either cross-modal effects (the percept depends on the two stimuli) or segregation effects (one of the stimuli is detected independently of the other).

This work aims at showing how an accurate MS perception model, including both of these effects, can be built using the notion of context-specific independence (CSI).

II. Experimental protocol and results

Subjects were sat in complete darkness and had to localize a primary stimulus which might come with a temporally coincident but possibly spatially discrepant secondary stimulus. In the acoustic perception task (APT), the primary and secondary stimuli were auditory and visual signals respectively (beeping buzzer and flashing diode) and vice versa in the visual perception task (VPT).

Subjects did experience both cross-modal and segregation effects. Indeed, their judgements about primary stimulus locations were strongly impacted by the secondary stimulus in the APT whereas this secondary stimulus effect is nearly absent in the VPT.

III. Bayesian network model

A. Finding the model's structure

To build a Bayesian network (BN) model of MS perception, the observable events are modelled by random variables (rv's). These are S_1, S_2, and S_1*, denoting respectively the primary and secondary stimuli, and the subjects' judgment. Another rv N should be introduced to model the subject's perceptual mode (induced by the acoustic or visual nature of the perception task). Mutual information (MI) and conditional MI [MacKay, 2003] give means for analyzing the dependence and independence between these rv's [Besson et al., 2009]. This set of statistical conditional independence statements can be expressed as a graph (BN).

As expected, subjects' judgements exhibit a stronger dependence with primary than with secondary stimuli. The model thus catches the cross-modal effect. However the segregation effect observed in the VPT does not explicitly appear since no conditional independence between S_2 and S_1* can be stated.

B. Context-specific independence for a finer model

For the model to account for the segregation effect, we can use CSI [Boutilier et al., 1996]. CSI represents conditional independences that hold only for specific values of an rv in the conditioning set (a "context"). The context N=1, denoting the VPT, leads to a conditional independence between S_2 and S_1*. As a result, both the integration and the segregation effects are captured by the model (Fig. 1). The latter explicitly handles the perceptual mode subjects are using, thus fits better the physical process. Eventually, the conditional probability distributions for S_1* can be written $P(S_1^*|S_1,S_2,N=0)$ and $P(S_1^*|S_1,N=1)$ for the APT and VPT respectively.

IV. Conclusion

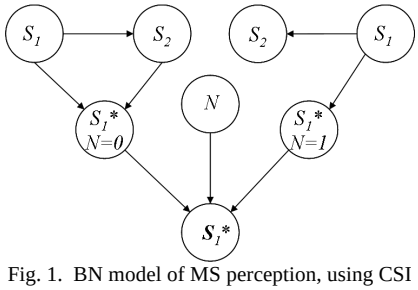


Fig. 1. BN model of MS perception, using CSI

A CSI analysis added to traditional MI approaches for determining a BN model's structure provides means for a complete and efficient representation of MS perception. The resulting model affords a better interpretation of the involved physical process since the perceptual mode used by the subject is explicitly captured by the model. Furthermore, S_1* prediction residuals are improved over a fully-connected model.

V. Bibliography

MacKay, D. J., "Information Theory, Inference, and Learning Algorithms", *Cambridge University Press*, 2003.

Besson P. et al., "A Bayesian network model of integration and segregation e□ects in multisensory perception". *Neural Computation*, 2008 (submitted).

Boutilier C. et al., "Context-specific independence in Bayesian networks", In *Proc. UAI*, 1996.

A mixed excitatory/inhibitory model of neural growth reproduces cortical cultures' activity during development

Bologna Luca Leonardo[1,*], Nieus Thierry[1], Martinoia Sergio[1,2]

[1] Italian Institute of Technology (IIT), NBT Department, Genoa, Italy;
[2] University of Genoa, NBT Lab, Department of Biophysical and Electronic Engineering, Genoa, Italy;
* *Current address*: UPMC, ANC Group, Laboratory of Neurobiology of Adaptive Processes, Paris, France;
luca.bologna@upmc.fr

***Keywords* — bursting activity, neuronal growth, overshoot**

I. Introduction

Dissociated cortical neurons grow their processes after plating and form a newly structured network since the first *days in vitro*. Over weeks, cultures undergo several developmental stages showing strong changes in activity. The dynamics stabilizes after a maturation period during which an activity overshoot in the network firing is often observed [1]. Here we present a model of neuronal growth reproducing both the bursting dynamics and the overshoot found in the experimental recordings, thanks to an *ad hoc* single cell intrinsic firing rate and the introduction of inhibitory cells.

II. Material and Methods

A. Cultures of dissociated neurons

Primary neuronal cultures were obtained from cerebral cortices of Sprague-Dawley rats at day 18 of gestation. Embryonic cortices were dissociated and suspended in culture medium at the concentration of 1500−2000 cells/µl. Cells were plated upon a 60-channel Micro-Electrode Array (MCS, Reutlingen) at a final density of 1200-2000 cells/mm^2.

B. Model of neural growth

A model of neuronal growth [2] was considered for studying firing dynamics at the network level. Briefly, each neuron is represented by a growing circle which shrinks (stretches) if its internal Calcium concentration, intrinsically decreasing to zero, is higher (lower) than a reference value [3]. Single cell firing rate relaxes to an intrinsic value *f0*. Each spike triggers an influx of Calcium. The area of overlap gives connection strength between neurons. With respect to the purely excitatory model proposed in [2], we decreased the value of *f0* and introduced uniformly disposed inhibitory cells.

III. Results

A lower *f0* value drove the dynamics of the model to a bursting behaviour highly resembling the one observed in experiments (~ 10 bursts / min). The introduction of inhibitory cells led the model to undergo the same firing overshoot observed *in vitro* (see Fig. 1).

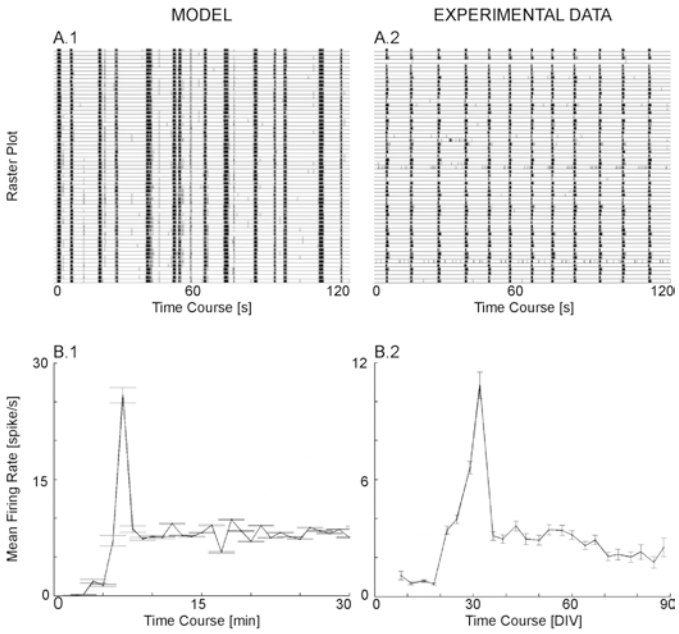


Fig. 1. Raster plot of modeled data (A.1) compared with experimental ones (A.2) (different electrodes/neurons are reported in different rows: thick lines are bursts, thin lines are spikes). Overshoot phenomenon is present in both the activity of the model (B.1) and the cultures (B.2).

IV. Conclusions

A model of neuronal growth is presented. Simulations showed that an appropriate value of the individual intrinsic firing rate is needed to reproduce a plausible bursting behavior. Furthermore, inhibitory dynamics introduced the *in vitro* firing overshoot, unobserved in a purely excitatory network.

V. Bibliography

1. Chiappalone, M., Bove, M., Vato, A., Tedesco, M., and Martinoia, S. (2006). Dissociated cortical networks show spontaneously correlated activity patterns during in vitro development. Brain Res, 1093(1).
2. Abbott, L. F. and Rohrkemper, R. (2007). A simple growth model constructs critical avalanche networks. Prog Brain Res, 165.
3. Kater, S. and Mills, L. (1991). Regulation of growth cone behavior by calcium. J. Neurosci., 11(4).

Fast encoding/decoding of haptic microneurography data based on first spike latencies

Romain Brasselet [1], Roland S. Johansson [2], Angelo Arleo [1]

1 CNRS - UPMC Univ Paris 6, UMR 7102, F-75005, Paris, France
2 UMEA Univ, Dept Integr Medical Biology, SE-901 87 Umeå, Sweden
romain.brasselet@upmc.fr

***Mots-clefs / Keywords*— Cuneate nucleus, Information theory, Mechanoreceptors, Spike train metrics**

I. Introduction

During haptic exploration tasks, forces are applied to the fingertips, which constitute the most sensitive parts of the hand and are prominently involved in object manipulation/recognition tasks. The epidermis is innervated with thousands of sensory cells, called mechanoreceptors, which encode the mechanical indentations and deformations of the skin. These cells project directly to a dorsal column nucleus called the cuneate nucleus (CN), which constitutes the first synaptic relay to the central nervous system. Recent microneurography studies in humans [1] suggest that the relative timing of impulses from ensembles of mechanoreceptor afferents can convey information about important contact parameters faster than the fastest possible rate code, and fast enough to account for the use of tactile signals in natural manipulation.

II. Working Hypothesis

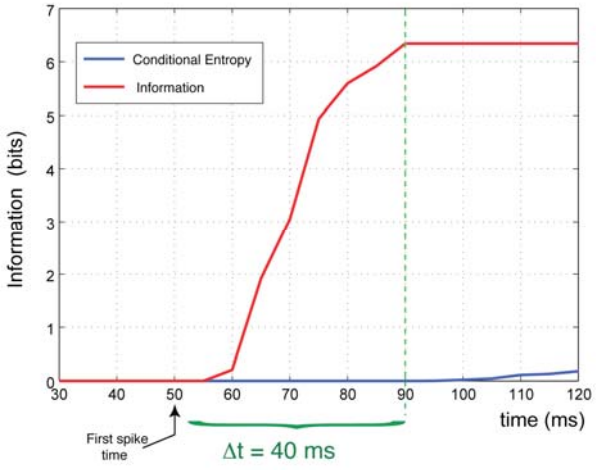
The rationale behind this study is to corroborate our working hypothesis that the CN does not constitute a mere synaptic relay, but it rather conveys an optimal contextual account (in terms of both fast and reliable information transfer) of peripheral tactile inputs to downstream structures (in particular to the thalamus and the cerebellum). Therefore, the CN may play a relevant role in the early processing of haptic information and it would constitute an important component of the haptic classification process (e.g., by facilitating fast discrimination of haptic contexts, minimising destructive interference over lifelong learning, and maximising memory capacity).

III. Methods

Here, we study a biologically-plausible encoding/decoding process accounting for the relative spike timing of the signals propagating from peripheral nerve fibres onto second-order CN neurons. The CN is modelled as a population of 450 spiking neurons receiving as inputs the spatiotemporal responses of real mechanoreceptors obtained via microneurography recordings in humans. An information-theoretic approach is used to quantify the efficiency of the haptic discrimination process. To this extent, a novel entropy definition has been derived analytically. This measure constitutes a promising decoding scheme to generalize the classical Shannon's entropy for spiking neural codes, and it allows us to compute mutual information (MI) in the presence of a large output space (i.e., 450 CN spike train responses) with a 1-ms temporal precision. We also use a plasticity rule designed to maximise information transfer explicitly [2].

IV. Results

The discrimination capacity of the model CN layer has been assessed when considering only one spike per mechanoreceptor afferent. Population coding permit a complete discrimination of 81 tactile stimuli already within 40 ms after the first afferent spike. Partial discrimination (80% of the maximum MI) is possible as rapidly as 20 ms.



V. Bibliography

[1] Johansson, R. and Birznieks, I. (2004) First spikes in ensembles of human tactile afferents code complex spatial fingertip events. *Nat Neurosci*, 7:170–177.

[2] Toyoizumi, T., Pfister, J.-P., Aihara, K. and Gerstner, W. (2005) Generalized Bienenstock Cooper Munro rule for spiking neurons that maximizes information transmission. *Proc Natl Acad Sci USA*, 102(14):5239-5244.

Granted by the EC Project SENSOPAC (SENSOrimotor structuring of Perception and Action for emergent Cognition), IST-027819-IP.

The neural computations of arbitrary visuomotor learning

Andrea Brovelli & Driss Boussaoud
Institut de Neurosciences Cognitives de la Méditerranée (INCM), UMR 6193 CNRS-Université de la Méditerrané, Marseille
andrea.brovelli@incm.cnrs-mrs.fr

***Mots-clefs / Keywords*— Associative learning, goal-direct system, habitual system, model-based fMRI, reinforcement learning, reward, decision-making.**

I. Introduction

Learning the consequences of our actions in their context is a fundamental cognitive ability, because it allows us and other animals to anticipate relevant events and to adapt to varying environments. If the relation between the visual stimulus, or context, the action, and its outcome is arbitrary and causal, we refer to as arbitrary visuomotor learning (Wise and Murray 2000). Associative theory (Dickinson 1980) postulates that learning the consequences of our actions is represented as stimulus-response-outcome associations that evolve according to prediction error signals (the discrepancy between the observed and predicted outcome). With further training, goal-directed behaviors are transformed into habitual responses that gradually become elicited by the antecedent stimuli (Yin and Knowlton, 2006). The aim of our work is to test these theories on functional magnetic resonance imaging (fMRI) data acquired from human participants and to provide quantitative descriptions of the neural computation mediating the acquisition of instrumental behaviors.

II. Methods

A. Task Design and fMRI analysis

Our implementation of arbitrary visuomotor learning required subjects to find by trial-and-error the correct associations between 3 colored circles and 5 finger movements. We developed a new task that systematically manipulates learning and induces highly reproducible performances.

We performed two type of analyses: the first looked for the neural computations related to the processing of outcomes during learning (Brovelli et al., 2008); the second looked for the brain correlates of the decision-making processes that are deploited during the different phases of learning.

III. Results

Outcome-related brain responses

Consistent with the Rescorla-Wagner model, prediction-error signals are computed in the human brain and selectively engage the ventral striatum. In addition, we found evidence of computations not formally predicted by the Rescorla-Wagner model. The dorsal fronto-parietal network, the dorsal striatum, and the ventrolateral prefrontal cortex are activated both on the incorrect and first correct trials and may reflect the processing of relevant visuomotor mappings during the early phases of learning. The left dorsolateral prefrontal cortex is selectively activated on the first correct outcome (Brovelli et al., 2008).

Decision-related brain responses

We tested the hypothesis that the selection of action during the different phases of instrumental leaning is mediated by complementary fronto-striatal loops (the associative and sensorimotor newtorks) transforming goal-directed actions into stimulus-driven habits (Yin and Knowlton, 2006). Preliminary results showed that the fMRI activity in the dorsal caudate nucleus increases during the exploratory phase and it correlates with the learning curve. In parallel, the activaty in the putamen increases slower during learning and it correlates with the probability that the stimulus is a good predictor of the correct action.

IV. Conclusion

The results provide quantitative evidence of the neural computations mediating arbitrary visuomotor learning and suggest new directions for future computational models.

V. References

Brovelli A, Laksiri N, Nazarian B, Meunier M, Boussaoud D 2008. Understanding the neural computations of arbitrary visuomotor learning through fMRI and associative learning theory , Cereb Cortex, 18(7):1485-95

Dickinson A. 1980. Contemporary animal learning theory. UK: Cambridge University Press.

Wise SP, Murray EA. 2000. Arbitrary associations between antecedents and actions. Trends Neurosci. 23:271-276.

Yin HH, Knowlton BJ. 2006. The role of the basal ganglia in habit formation. Nat Rev Neurosci. 7:464-476.

French Ministère de la Recherche (ACI Neurosciences intégratives et computationnelles); and 2-year post doc fellowship awarded by the Fondation pour la Recherche Médicale (Paris, France) to A.B.

Real time control of a CPG-based model of the human trunk in different walking conditions

Jean-Charles Ceccato, Christine Azevedo-Coste, Jean-René Cazalets

***Keyword*—Neural network, control**

I. INTRODUCTION

Central pattern generators (CPGs) are dedicated to neuronal networks that generate rhythmic motor behavior such as locomotion [1]. There are evidences that such neural circuits also exist in human [2]. In a previous study, we demonstrated the existence of a metachronal (segment by segment) descending wave of trunk muscles activity during human walking [3]. This pattern of propagation is similar to the one observed in other vertebrates like rats [4], lamprey [5] and salamander [6] and it has been suggested that it relies on the existence of a CPG dedicated to trunk control [4]. It may be therefore interesting to model a human CPG in order to reproduce the trunk metachronal descending wave.

II. METHOD

A. Data acquisition of trunk activity

We recorded 10 young subjects (27±6 years), they wore shorts and sandals and were asked to perform walking and gait initiation trials. Bilateral EMG and kinematics of the trunk were recorded as well as resultant forces under feet in the laboratory movement analysis platform.

B. Adaptation of the salamander model

In order to modelize its supposed activity, we adapted an oscillator network developed by Ijspeert [2] for salamander. The command of the network was then developed on two levels: voluntary command (walking, running ± speed) and adaptative command (inclined pathway, stairs).

III. RESULTS

A. Trunk activity during walking and gait initiation

With the recorded data we were able to extend some feature about trunk activity during walking:

- The presence of a metachronal (segment by segments) descendant wave in ES from C7 to L3 during both walking and gait initiation.
- A concordance between muscular activity and trunk flexion/rotation during walking, whose anticipatory aspect let think that it could help rising the pelvis and the leg.

B. Adaptive command

As shown in Ijspeert [7], voluntary command allows easily speed and locomotor mode variation then it appears interesting to adapt CPG activity with external conditions. We used vertical acceleration collected from an accelerometer to synchronize he phase of the oscillator network with an adapted controller. Data were recorded during flat floor walking and up/downward stairs walking. Adapting the principle of PLL (Phase locked loop), we synchronized CPG model and chosen acceleration signal.

IV. CONCLUSION AND FUTURE WORK

We developed a model of trunk CPGs that is able to express different type of synchronization under different voluntary and external solicitation. Then in further study we developed a protocol aiming to explore pattern change in muscular and kinematics activity of the trunk during running, cycling and hopping. Each of those activities is rhythmic but present wide differences. New patterns could then improve our model.

The model control could be improved on the adaptative part; in fact the used network controller deduced the phase in a discrete way that induces a lag in external change adaptation (gait initiation, slope, stairs). To improve this, we propose to detect gait initiation and to estimate continuously the phase of walking cycle with sensors placed on the trunk.

REFERENCES

[1]S. Grillner and P. Wallen, "Central pattern generators for locomotion, with special reference to vertebrates," *Annu Rev Neurosci,* vol. 8, pp. 233-61, 1985.

[2]J. Duysens and H. W. Van de Crommert, "Neural control of locomotion; The central pattern generator from cats to humans," *Gait Posture,* vol. 7, pp. 131-141, Mar 1 1998.

[3]M. de Seze, M. Falgairolle, S. Viel, C. Assaiante, and J. R. Cazalets, "Sequential activation of axial muscles during different forms of rhythmic behavior in man," *Exp Brain Res,* vol. 185, pp. 237-47, Feb 2008.

[4]M. Falgairolle and J. R. Cazalets, "Metachronal coupling between spinal neuronal networks during locomotor activity in newborn rat," *J Physiol,* vol. 580, pp. 87-102, Apr 1 2007.

[5]S. S. Islam, P. V. Zelenin, G. N. Orlovsky, S. Grillner, and T. G. Deliagina, "Pattern of motor coordination underlying backward swimming in the lamprey," *J Neurophysiol,* vol. 96, pp. 451-60, Jul 2006.

[6]T. Bem, J. M. Cabelguen, O. Ekeberg, and S. Grillner, "From swimming to walking: a single basic network for two different behaviors," *Biol Cybern,* vol. 88, pp. 79-90, Feb 2003.

[7]A. J. Ijspeert, A. Crespi, and J. M. Cabelguen, "Simulation and robotics studies of salamander locomotion: applying neurobiological principles to the control of locomotion in robots," *Neuroinformatics,* vol. 3, pp. 171-95, 2005.

Visual pattern classification by neural fields

Mauricio Cerda and Bernard Girau
LORIA - INRIA Nancy Grand Est
cerdavim@loria.fr

***Keywords*— biological motion, pattern recognition, neural fields, STS, EBA.**

I. Introduction

The recognition of visual motion patterns such as walking, fighting and face gestures among others, is remarkably efficient in humans and many other species. Experiments have already given some clues about the nature of the internal mechanisms of recognition. These experiments are based on point-light stimuli in psychophysics [1], electro-physiological data and functional imaging [3] techniques. In this work, we study some of the identified properties and we propose to model them by means of asymmetric neural fields.

II. Biological data

A. Properties and coding

Visual motion pattern recognition in the human brain seems to be extremely sensitive to temporal correlations [2]. On the contrary, it appears spatially robust: even though the visual input can be severely diminished, stimuli as simple as point-lights [1], where only joints (or random points) are enlighten, can be recognized. Similarly, the observer angle may be perturbed by up to 20 degrees [2].

Experiments [3] indicate that a 2D representation is sufficient to explain brain coding schemes for 3D body actions, indicating a possible template based coding.

B. Biological arguments

The existence of template units in motion information has not yet been proved. Nevertheless, there is a direct analogy with "snapshot" units found in the ventral pathway. Moreover, these templates could be the input for decision units that may be similar to some single neurons observed in areas EBA and OFA that are sensitive to human actions such as walking [2].

III. Modeling

Taking into account the tolerance to diminished stimuli, we consider motion information that could be available from areas V1/MT as relevant to model. To extract *discriminative* information, we build local flow pattern detectors as observed in area STS (motion as: spirals, expansions, translations, etc.). These two operations approximate a "joints-like" detector.

From the *discriminative* points' information $B(\vec{x},t)$ (see Fig. 1) we generate a population of units to simultaneously track the different trajectories. We use one of these populations $m(\vec{x},t)$ for each motion pattern. To achieve temporal selectivity with high sensitivity, we use 2D asymmetric neural fields (extending the 1D model of [4]):

$$\tau \frac{\partial m(\vec{x},t)}{\partial t} + m(\vec{x},t) = \left[\int_0^{x_f} w(\vec{x},\vec{x}')m(\vec{x},t)d\vec{x}' + B(\vec{x},t) \right]^+$$

where the asymmetric kernel $w(\vec{x},\vec{x}')$ gives the direction selectivity in time. Our model implicitly has all "snapshots" in the same population, eliminating global comparators as in [2].

IV. Results & Conclusion

Simulations show that our model is able to classify synthetic patterns and we are currently working with real videos, from noisy environments to test our model further.

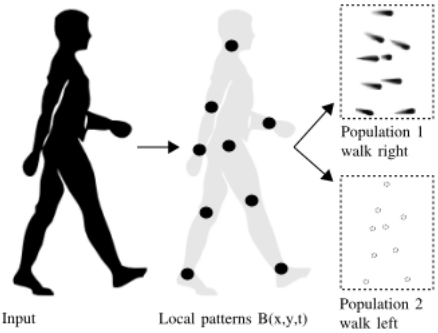


Fig. 1. Schematic view of our model for the left/right discrimination task.

In this work we show that several key features of the human recognition of visual motion patterns may be modeled using 2D asymmetric neural fields. Additionally, we conclude that the key evidence to support template-based recognition from the dorsal pathway is the existence of units or populations acting as snapshots.

V. Bibliography

[1] Johansson, G., "Visual perception of biological motion and model for its analysis". Percept. Psycho. 14 195—204, 1973.

[2] Giese, M. and Poggio, T., "Neural mechanisms for the recognition of biological movements". Nat. Rev. Neurosci. 4 179—192, 2003.

[3] Bulthoff, I., Bulthoff, H. and Sinha, P., "Top-down influences on stereoscopic depth-perception". Nat. Neurosci. 3 254—257, July 1998.

[4] Xie, X. and Giese, M.., "Nonlinear dynamics of direction-selective recurrent neural media". Phys Rev E 65, 2002.

BioMEA: A Modular 256-channel Micro Electrode Array System for Stimulation and Acquisition

G. Charvet[1]; S. Gharbi[1]; R. Guillemaud[1]; B. Yvert[2] ;
T. Kauffman[3]; C. Georges[3]; A. Pellissier[3]; M. Heuschkel[4]; S. Makohliso[4]
1. CEA/LETI/MINATEC, Grenoble, France
2. Université de Bordeaux and CNRS, Bordeaux, France
3. Bio-Logic SAS, Claix, France
4. Ayanda Biosystems SA, Lausanne, Switzerland
cedric.georges@bio-logic.info

***Keywords*— Micro Electrode Arrays, Neural Network, Electrophysiology, Electrical Stimulation.**

I. Introduction

In order to understand the dynamics of large neural networks, where information is widely distributed over thousands of cells, one of today's challenges is to successfully monitor the simultaneous activity of as many neurons as possible. This is made possible by using the Micro-Electrode Array (MEA) technology allowing neural cell culture and/or tissue slice experimentation in vitro. Thanks to development of microelectronics' technologies, a novel data acquisition system based on MEA technology has been developed, the BioMEA. It combines the most advanced MEA biochips with integrated electronics, and a novel user-friendly software interface.

II. Development

BioMEA is the result of a research project (NEUROCOM) where the first prototype was specified and tested by CEA/LETI/MINATEC and CNRS Bordeaux, France. BioMEA is both a stimulus generator, and a high sensitivity data acquisition system, which permits 256 electrodes to be stimulated and monitored simultaneously. To move from prototype to manufactured product, a number of changes have been made: BioMEA offers a novel user-friendly interface allowing rapid and easy setting of stimulation and acquisition parameters; a spike detection algorithm for identification of active electrodes; an adjustable gain of each electrode depending on electrical cell activity measured; and all 256 electrodes can be selected simultaneously for recording and stimulation. Furthermore, all data from the 256 channels can be saved and reloaded with BioMEA software or further analyzed using Spike2 software (Cambridge Electronic Design Limited, UK).

BioMEA has been designed modularly, allowing the use of MEA biochips including 64, 128 or 256 recording/stimulation electrodes on the same system. A wide choice of MEA biochips adapted to BioMEA including various electrode geometries (planar and 3D tip-shaped electrodes for an optimized tissue penetration) and single or multi-well format (up to 9 wells) configurations are currently available.

III. BioMEA Application

BioMEA offers unprecedented capabilities to address applications such as evaluation of neural plasticity, functional screening and toxicology/safety pharmacology.

Fig. 1. BioMEA instrument.

Mature alternating motor output may be generated using immature chloride mediated inhibition.

Alain Delpy*, Daniel Cattaert*, Pascal Branchereau, Pierre Meyrand
UMR 5228 CNIC, CNRS- Univ. Bordeaux 1, Talence, France
(Equal contribution)*
d.cattaert@cnic.u-bordeaux1.fr

***Mots-clefs / Keywords*— Development; spinal cord; shunting inhibition; locomotor pattern; GABA/glycine**

I. INTRODUCTION

In mature spinal network, alternation between ipsilateral and contralateral motor networks is supported by reciprocal inhibition mediated by GABA/glycine. As soon as embryo day 17.5 (E17.5), alternation is expressed when mouse spinal networks are activated by exogenous application of 5HT/NMDA (Branchereau et al. 2002). However, at this stage, even though the equilibrium potential for chloride ions (ECl) has dropped dramatically, GABA and glycine still evoke depolarizing responses in motoneurons (MNs) via the activation of their ionotropic receptors (Delpy et al. 2008). In order to understand this paradox (how can inhibition be mediated via depolarizing events, which are generally excitatory?), we performed both physiological experiments and modeling.

II. GABA SYNAPSES: FROM EXCITATION TO INHIBITION DURING EMBRYO DEVELOPMENT

A. Single synaptic responses

At the MN level, we analyzed the characteristics of responses evoked by $GABA_AR$ activation after isoguvacine pressure application. These responses include two components: an inhibition (shunt) and an excitation (depolarization). Inhibition is present during raising phase of the GABA response, whereas excitation is present during the repolarizing phase of the GABA response. We found that, whereas at E13.5 GABA response is mainly excitatory and spike inducing, at E17.5 the response remained depolarizing but prevented spiking. Between these two stages, three main changes were observed: MN size (capacitance) increased, input resistance decreased and ECl dropped. However, to assess the role played by each of these parameters in the excitatory/inhibitory effect of the GABA response, it is necessary to study them independently. This is not possible using the only physiological approach. This is why we used a combination of physiological analysis and simulations.

Références éventuelles sur les financements des travaux / Eventual references about grants

In order to test the respective roles of these three parameters in the ontogenetic maturation of inhibition, we developed realistic models of E13.5 and E16.5-E17.5 MNs in the NEURON environment (Hines and Carnevale, 1997, 2000). These simulations showed that: 1) the ontogenetic capacitance increase favored the excitation; 2) the input resistance decrease favored shunting inhibition; 3) the ECl drop played the major role in the occurrence of shunting inhibition.

Using experimental approach, in which E16.5-E17.5 MNs were recorded, the change of ECl from physiological value to E13.5 value reversed the $GABA_AR$ induced shunting inhibition to excitation, highlighting the preponderant role of ECl in maturation of inhibition.

B. Trains of synaptic events

IPSPs generally occur in burst. Therefore, the two components (inhibition and excitation) will interact during bursts of IPSPs. In order to understand how MNs integrated bursts of chloride-mediated post-synaptic responses, we analyzed the effect of trains of GAB_AR depolarizing responses, using simulation. This simulation showed that, for each frequency (between 5 to 100 Hz), there was a critical ECl value for which trains of GABA events switched from excitatory to inhibitory. Interestingly, these critical ECl values were in a restricted domain (-45 to –50 mV) that is crossed during the ontogenetic ECl drop from E13.5 to E17.5

III. CONCLUSION

The increase in frequency discharge could therefore be crucial to elicit clear-cut alternating rhythmic activities observed at E17.5 but not at E16.5.

IV. BIBLIOGRAPHY

Branchereau P, Chapron J, Meyrand P (2002) Descending 5-hydroxytryptamine raphe inputs repress the expression of serotonergic neurons and slow the maturation of inhibitory systems in mouse embryonic spinal cord. J Neurosci 22: 2598-2606

Delpy A, Allain AE, Meyrand P, Branchereau P (2008) NKCC1 cotransporter inactivation underlies embryonic development of chloride-mediated inhibition in mouse spinal motoneuron. J Physiol 586: 1059-1075

Hines ML, Carnevale NT (1997) The NEURON simulation environment. Neural Comput 9:1179 -1209.

Hines ML, Carnevale NT (2000) Expanding NEURON's repertoire of mechanisms with NMODL. Neural Comput12 : 995-1007.

Analysis of behaviour of a neural population

Gregory Dumont, Jacques Henry, and Carmen Oana Tarniceriu
INRIA
Oana.Tarniceriu@inria,fr

Keywords: **Izhikevich model, Population density approach, Synchronization.**

I. INTRODUCTION

Understanding the behaviour of a neural network in response to various stimulations is nowadays of great interest. We propose here a mathematical model describing the dynamics of a population of neurons; the evolution of the state of a single neuron in the population is given by the Izhikevich model [1].

II. THE MODEL

The model we use is the following:

$$\frac{\partial}{\partial t}p(t,v,u) = -\mathrm{div}\left[F(v,u)p(t,v,u) + \sigma(t)\bar{e}_v \int_{v-\epsilon}^{v} p(t,\dot{v},u)\,\mathrm{d}\dot{v}\right]$$

Where p(t,v,u) is the population density at time t in the state (v,u); F(v,u)p(t,v,u) is the neural flux flowing trough the state (v,u) at time t, and σ(t) is the average reception rate for a single neuron. The expression of F is given by the Izhikevich model:

$$F = (F^v, F^u)$$

where

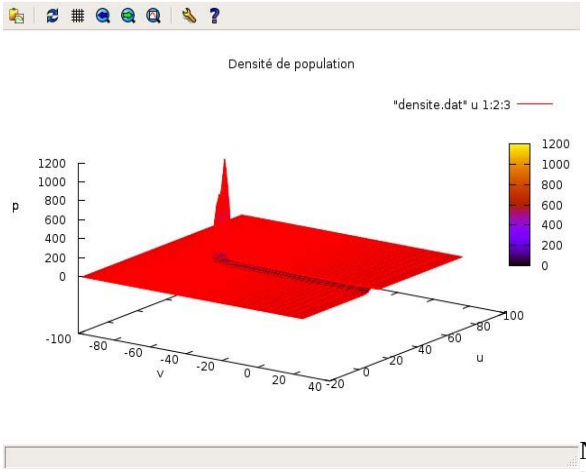
$$F^v = 0.04v^2 + 5v + 140 - u + I(t)$$
$$F^u = a(bv - u)$$

In the expression above, I represents the injected current.

The reset mechanism from the Izhikevich model is naturally translated in our case to a periodic boundary condition for the neural flux.

The model we presented has been introduced by J. Modolo [2] in his Ph.D. thesis. The main inconvenient lays in the supposition that the neurons in the population are identical, therefore all of them have the same behaviour. Nevertheless, it is important to stress that this approach is totally independent of the number of neurons of the population, therefore, the size of the population does not influences the computation time in a simulation.

We are interested first in the study and numerical simulation of the model using conservative schemes. The second objective we have is to describe the phenomenon of synchronization; this is done in the case when a weakly connected neurons network is considered. To this end, we have used the analytical tools of the weakly connected systems theory [3].



Nu

Nu

merical simulation of the mathematical model via a upwind conservative scheme

III. CONCLUSIONS

This method, although based on strongly simplifying assumptions, has the advantage of using a very concise model for the mathematical analysis and numerical simulations, and computation times totally independent of the number of neurons of the population.

IV. REFERENCES

[1] E. Izhikevich : Dynamical Systems in Neuroscience, The MIT Press, 2007
[2] J. Modolo : Modélisation et analyse mathématique des effets de la stimulation cérébrale profonde dans la maladie de Parkinson, thčse 2008
[3] E. Izhikevich, F. C. Hoppensteadt : Weakly Connected Neural Network, Springer 1997
[4] J. Modolo, A. Garenne, J. Henry, A. Beuter: Development and validation of a neural population model based on the dynamics of discontinuous membrane potential neuron model, J. Integr. Neurosci., vol , no. (2007), 625655
[5] J. Modolo, E. Mosekilde, A. Beuter: New insights offered by a computational model of deep brain stimulation, J Physiol Paris 101 (2007) 5663

This project has been financed by the neuroinformatic program of CNRS

Computational model of nicotine addiction: basic approaches

[1]Saïd ECH CHADI, [2]Ali QUYOU, [2]Mohamed Khaled CHOULLI
[1] ENSA-Safi (Ecole Nationale des Sciences Appliquées de Safi) Université CADI AYYAD BP : 63 Route de Sidi Bouzid Safi –
[2] UFR des Sciences Biologiques et Pharmaceutiques - Faculté des Sciences Université IBN TOFAIL BP : 133 Kénitra –
lab_fsk@yahoo.fr, said.echchadi@ucam.ac.ma

***Mots-clefs / Keywords*— mathematical modelling, behavioural sensitization, nicotine dependance.**

I. Introduction

Changes in the external environment, occurring repeatedly, can cause neuro-adaptations and changes to long-term in the functioning of the nervous system. Behavioral sensitization induced by nicotine is a good example of these phenomena desadaptation. A dozen injections of nicotine are sufficient to change long-term behavior of rats. This behavioral sensitization depends on the activation of nicotinic receptors $\alpha_2\beta_4$ increasing psychostimulants and disinhibitors (anxiolytic) effects of nicotine [1] [2]. It is associated with morphological changes [3] and the long-term alteration of dopaminergic, glutamatergic and serotonin activity [4] [5] [6]. The classical approach is to compare locomotor activity elicited by nicotine and saline in both sexes [7].

II. Mathematical Model

The analytical model describes the kinetic profile of the evolution of the locomotor activity, with two parameters:

- So expresses the initial activity, and hence a high state of awareness caused by the first contact with the environment, and exploration.
- λ which expresses the speed with which the locomotor activity decreases, i.e. the loss of motivation to explore and the decrease in the level of arousal.

The long-term model expresses the changing parameter λ from the first model assessed during daily sessions over a long period. The theory of neuroadaptation shows the brain's response to chronic treatment, characterized by direct influences on the way locomotor activity declines. By iteration, we calculate two indices λ_{max} and L (see Fig. 1).

III. Results

With this new approach, significant differences were observed. Indeed λ_{max} which expresses the maximum speed of decrease in locomotor activity differed significantly between the four groups with a small gap between saline / nicotine male groups and a an important gap saline / nicotined female groups. The second parameter L, which expresses the habituation, is identical in males' saline and nicotine, whereas there is a difference in value between nicotined and saline females. The first correlations of this model (Spearman and Kendall coefficients), give good results in four cases: nicotined females, saline females, nicotined males and saline males, which gives great validity to our model.

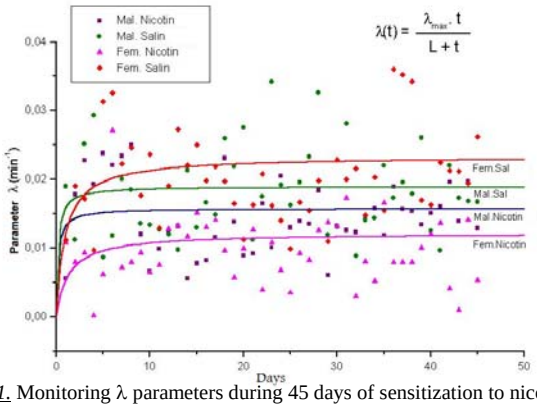


Fig. 1. Monitoring λ parameters during 45 days of sensitization to nicotine.

Indeed, the gender differences are clearly significant, the maximum speeds differ among the four groups, this index correlates with basic biological differences between male and female. On the other hand, the index of habituation is very different between sex, as it can detect the effect of nicotine awareness among females, but not in males.

IV. Conclusion

With the approach of analysis of the locomotor activity, these results were confirmed by experiments involving the intravenous administration of nicotine, but could not be in the presence of a subcutaneous administration.

V. References

[1] M ERICSON, P OLAUSSON, JA ENGEL, SODERPALM B. Nicotine induces disinhibitory behavior in the rat after subchronic peripheral nicotinic acetylcholine receptor blockade. Eur J Pharmacol 2000b, 397: 103-111

[2] FE KEMPSILL, JA PRATT. Mecamylamine but not the alpha7 receptor antagonist alphabungarotoxin blocks sensitization to the locomotor stimulant effects of nicotine. Br J Pharmacol 2000, 131 : 997-1003

[3] RW BROWN, B KOLB. Nicotine sensitization increases dendritic length and spine density in the nucleus accumbens and cingulate cortex. Brain Res 2001, 899 : 94-100

[4] C CADONI, G DI CHIARA. Differential changes in accumbens shell and core dopamine in behavioral sensitization to nicotine. Eur J Pharmacol 2000, 387 : R23-25

[5] KA TRUJILLO, H AKIL. Excitatory amino acids and drugs of abuse : a role for N-methyl-D-aspartate receptors in drug tolerance, sensitization and physical dependence. Drug Alcohol Depend 1995, 38 : 139-154

[6] JE KELSEY, T BEER, E LEE, A WAGNER. Low doses of dizocilpine block the development and subsequent expression of locomotor sensitization to nicotine in rats. Psychopharmacol (Berl) 2002, 161 : 370-378

[7] L KANYT., I.P STOLERMAN., C.J CHANDLER., T SAIGUSA., S POGUN. Influence of sex and female hormones on nicotine-induced changes in locomotor activity in rats. Pharmacol Biochem Behav. 62(1999): 179-187.

Learning to perceive image contours in the context of Neural Fields

Mathieu GALTIER, and Olivier FAUGERAS
NeuroMathComp Team, INRIA Sophia / ENS Paris
mathieu.galtier@sophia.inria.fr

***Keywords* — Neural fields, correlation based learning, contour detection, gestalt connectivity.**

I. Introduction

One approach to neuroscience is to detail very accurately each mechanism observed at the microscopic level hoping that this might help understanding the emergence of macroscopic behaviors. Without denying the importance of such an approach, we do think that developing large scale models of idealized neurons is potentially very fruitful and has not been explored enough. We use a combination of mathematics and computer simulations, well-grounded in biology, to drive our research toward a macroscopic understanding of the brain.

II. Neural Fields Formalism

Neural fields are an interesting option for modeling macroscopic parts of the cortex. Indeed, by considering neural masses (i.e. populations of similar neurons) as building blocks, the neural fields formalism makes it possible to handle large pieces of cortical layers and study their dynamics. They provide a mathematically elegant, well posed and powerful framework for studying generic neural mass networks: a single continuous functional nonlinear integro-differential equation governs the neural field evolution (for a given input). This formalism provides a versatile method to model efficiently any biological neural network.

III. Learning in large scale neural fields

We think that correlation based learning is the organizing principle of sensory areas. To build the connectivity within a neural field (exposed to a certain input statistics), one should therefore couple dynamical learning and the neural field evolution. This results in a set of coupled functional nonlinear integro-differential equation. A rigorous mathematical analysis of large scale learning implies studying the well posedness of such a system. We have systematically analysed the global existence and uniqueness of the solution for several learning rules (e.g. Hebb with different normalizations, BCM), using functional analysis tools. Other general properties such as the boundedness of the solution and its stability are also obtained. Moreover, by identifying the problem as a slow-fast dynamical system, we mathematically justify a number of computational algorithms that are commonly used to perform learning, proving that the approximation that they imply can be bounded.

IV. Application to visual perception

To illustrate the way this large scale learning process may shape neural fields, we model the visual contour detection task performed in the visual cortex. We consider 2 cortical layers with more than 100,000 neural masses that interact in a recurrent fashion. Neurons of the first layer are tuned to orientation and scale at some particular places of the visual field. Lateral connectivities in the 2 layers implement the classical mexican hat pattern. The connections between the 2 layers are assumed to be symmetric and evolve according to a correlation based learning rule (e.g. BCM). This leads to an unsupervised learning of the connectivity between the 2 layers that eventually defines the receptive field of the neural masses in the 2nd layer. The system is also constrained with isotropy and homogeneity arguments and the final receptive fields correspond to regularly distributed and oriented smooth pieces of curves. The corresponding neurons are complex cells. More precisely, the connectivity between the 2 layers strongly links co-circular neural masses of the 1st layer (see figure below). It implies that co-circular edges are particulary well detected by this neural field, clearly implementing the Gestalt good continuation principle.

V. Conclusion

We have built the basis of a rigorous Mathematical analysis of large scale learning in Neural Fields. Much work remains to be done, for instance to qualify the attractors of such a system or to extend the theory to time-based learning. On the other hand, the implementation of these macroscopic networks already shows significant results since it suggests a mechanism underlying the good continuation principle. The next step is to go higher in the cortical hierarchy to unravel the structure of other receptive fields and identify functional properties of neurons.

VI. Bibliography

[1] O. Faugeras, F. Grimbert, J.-J. Slotine, Absolute Stability and Complete Synchronization in Neural Fields , SIAM Journal of Applied Mathematics, 69: 1, pp. 205-250 (2008)
[2] E. Bienenstock, L. Cooper, P. Munro, Theory for the development of neuron selectivity: orientation specificity and binocular interaction in visual cortex, Journal of Neuroscience, Vol 2, 32-48 (1982)
[3] R. Miikkulainen, J. Bednar, Y. Choe, J. Sirosh , Computational Maps in the Visual Cortex , Springer (2005)

The research leading to these results has received funding from the EC IP project Facets and the PACA region.

Temporal sequence learning and adjustment in cerebellar-like network architectures.

André Garenne[1,2], Thomas Boraud[1,2]
[1] UMR-CNRS 5227, [2] Université de Bordeaux 2
andre.garenne@u-bordeaux2.fr

***Keywords*—large-scale neural network, motor trajectory, temporal learning.**

I. Introduction

The general ability to learn delayed responses and more specifically temporal sequences of signals (spikes or bursts) is associated in part with the properties of cerebellar neural networks. In the last few years, several attempts have been made to clarify the relationship between the neural network architecture and its capacity and to deal with timing sequences using spiking neuron networks models (Suri & Schultz, 1999; Medina et al., 2000; Garenne & Chauvet, 2004; Yamazaki & Tanaka, 2007, Goodman & brette, 2008). Indeed, learning of temporal sequences of signals plays a key role in both cognitive and motor acquisition processes. In the case of complex motor sequence, this model has to take into account one important property which is often put aside: a capacity to adjust the motion speed (and thus the emitted signal sequence speed) to the dynamics of the task to achieve. A closed-loop approach is presented here which involves a cerebellar-like neural network, a robotic arm, a sensory-motor interface linking both and precise timing constraints in a behavioural reaching task.

II. Materials and methods

A. Neural network model

The cerebellar network architecture used here relies on network connectivity data from literature as well as on some synaptic plasticity properties experimentally observed and based on relative presynaptic and postsynaptic event spike timing plasticity. The neuron representations are interconnected Izhikevich models (Izhikevich et al., 2004).

B. Sensory-motor interface

A two degree of freedom (DOF) arm is connected to the neural network output and thus driven by its spiking activity. The main goal of this effector is to show the ability of the whole system to adapt motor trajectory speed to various sensory inputs (in this case, to catch a moving prey by touching it with its extremity). The moving target that has to be caught, always follows the same trajectory but at different speeds. This speed is encoded linearly as an input firing rate to the network, arm configuration is used as a proprioceptive signal and the final distance between target and arm extremity is encoded as an error signal.

III. Results

Temporal sequence learning and "grasp" position reaching

The network learns supervised spike sequences. It shows the ability of cerebellar-like neural networks to make use of the large frequency time domain exhibited by the granule cell activity and of the high degree of convergence between parallel fibres and Purkinje cells. Using the proper error signal, the trajectory learning rapidly converges.

Arm motion speed adaptation

The robotic arm is then trained to adjust its motion speed to the fastest and slowest moving targets and exhibits then a perfect capacity to anticipate its passage past the grasping position.

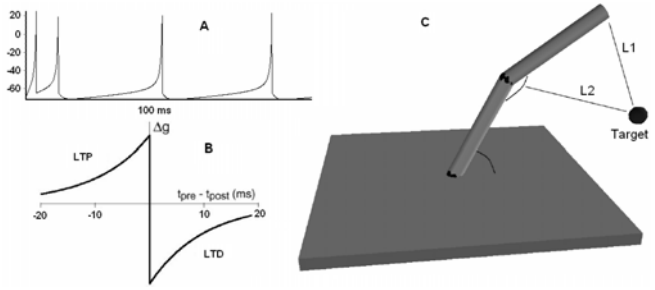


Fig. 1. A: activity sample of an Izhikevich neuron membrane potential model, B: sample of Spike-Timing Dependent Plasticity profile used in the network model, C: Two DOF robotic arm, L1 and L2 are respective joint distances to the target used as an error signal.

IV. Conclusion

We show that a properly designed neural network, taking into account realistic biological features and embedded into a sensori-motor closed-loop, is able to learn temporal sequences of spiking events and to apply them to the control of a robotic arm at the adequate speed in a dynamical reaching task.

V. Bibliography

Garenne A, Chauvet GA (2004) A discrete approach for a model of temporal learning by the cerebellum: in silico classical conditioning of the eyeblink reflex. J Integr Neurosci 3:301-318.

Goodman DF, Brette R (2008), Brian: a simulator for spiking neural networks in python. Front Neuroinformatics. 2008;2:5. Epub 2008 Nov 18.

Izhikevich EM, Gally JA, Edelman GM (2004) Spike-timing dynamics of neuronal groups. Cereb Cortex 14:933-944.

Medina JF, Nores WL, Ohyama T, Mauk MD (2000) Mechanisms of cerebellar learning suggested by eyelid conditioning. Curr Opin Neurobiol 10:717-724.

Suri RE, Schultz W (1999) A neural network model with dopamine-like reinforcement signal that learns a spatial delayed response task. Neuroscience 91:871-890.

Yamazaki T, Tanaka S (2007) A spiking network model for passage-of-time representation in the cerebellum. Eur J Neurosci 26:2279-2292.

Two-Stage Action Selection in a Computational Model of the Basal Ganglia

M. Guthrie, B. Bioulac, C. Gross, T. Boraud
UMR CNRS 5227
martin.guthrie@u-bordeaux2.fr

***Keywords*—Basal ganglia, action selection, segregated loops.**

I. Introduction

There is continued debate about the role of cortex-basal ganglia loops in decision making and action selection processes. In particular, it has been shown that there are several parallel functional loops through the basal ganglia connecting back to distinct areas of cortex. The picture of segregated loops is complicated by studies showing axonal divergence that imply that the loops are not completely segregated. Here we propose a unified computational model of cortical – basal ganglia loops that has two functional loops, one cognitive and the other motor (Figure 1). There are two pathways through each loop, the direct pathway through the striatum to the GPi and the hyperdirect pathway via the STN to the GPi. The separate loops interact only via divergent projections to GPi from striatum and STN.

II. Methods

A. Task modeled

The model is used to simulate a two-choice decision task that has been shown, in monkeys, to have two distinct phases[1]; a first stage in which a target is selected and a second in which a direction of movement is selected. The monkey is shown two targets that have different probabilities of reward and, using a joystick, selects one target.

B. Model architecture

The two loops in the model receive input from different areas of cortex. The first loop in the model is considered to be cognitive, receives cortical input regarding the value of the two targets shown and acts in deciding which of the two targets to select. The second loop is considered to be more motor related and receives cortical input regarding the two possible directions of movement that could be taken to reach a target (although there is no information on the value of the targets directly available to structures in this loop) and acts to choose the direction of movement. The two model loops perform the two stages of the task in parallel, with the decision in the target selection phase feeding forward via the divergent projections to influence the decision in the direction selection phase. Reconvergence from GPi to thalamus integrates the information to perform the action selection.

III. Results

Action selection

In a simulation of 200 runs, 4 (2%) did not complete when the model made no choice. Of the other 196 runs, 100% chose the correct target and 165 (86.2%) chose the correct direction.

Activation levels

Using 2-way ANOVA we tested which units responded to the choice value (Cv), the action value (Av), the action alone (A) or were not responding (NR) in each task phase (Table 1).

Table 1. Proportion of units responding to task parameters

Phase	Cv	Av	A	NR
Target selection	33%			67%
Movement decision	25%	50%	17%	8%

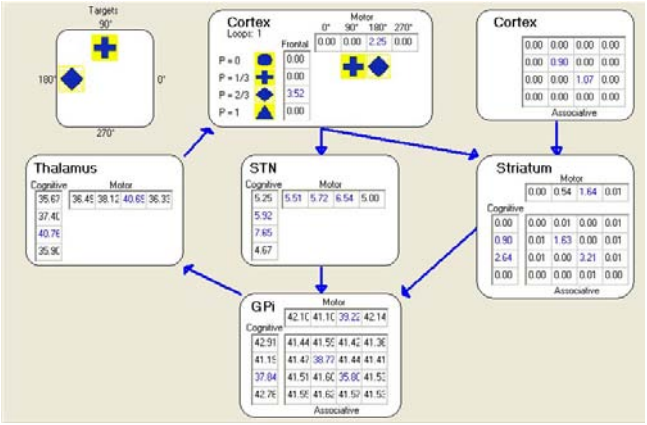


Fig. 1. Typical run of the model. Two targets, a cross and a rhombus are presented at 90° and 180° respectively. The values of these targets are transferred to cognitive cortex at the start of the run. In the first (decision) phase, the target with a higher value (rhombus) is chosen. In the second phase the direction corresponding to that target (180°) is chosen.

IV. Conclusion

The model is able to perform a two-stage action selection task with a high rate of success. The proportions of units that responded to the various parameters of the task in GPi in each phase of the task were in accordance with the proportions found in electrophysiological recordings in monkeys[1].

V. Bibliography

1. Pasquereau, B. et al. (2007). Shaping of Motor Responses by Incentive Values through the Basal Ganglia. *Journal of Neuroscience*, **27**(5), 1176-1183.

Quantitative estimation of calcium dynamics from ratiometric measurements: a direct, non-ratioing, method

Sébastien Joucla, Andreas Pippow, Peter Kloppenburg and Christophe Pouzat
CNRS, Université Paris-Descartes / Laboratoire de Physiologie Cérébrale (UMR 8118)
sebastien.joucla@parisdescartes.fr

***Keywords* — calcium imaging, parametric model, square root transformation, constraints.**

I. Introduction

MEASURING variations of intracellular free calcium concentration ($[Ca^{2+}]$) through the fluorescence changes of a calcium sensitive dye is a ubiquitous technique in neuroscience. Some dyes like Fura-2 present different absorption spectra between their calcium-bound and calcium-free forms. This leads to ratiometric measurements giving an estimate of $[Ca^{2+}]$ with minimal processing [1]. We focus here on the construction of meaningful confidence intervals (CIs) on calcium dynamics parameters obtained from ratiometric measurements.

II. The Classical View: The Ratiometric Transformation

According to the *ratiometric* transformation, $[Ca^{2+}] = K_{eff} \times (R–R_{min}) / (R_{max}–R)$, where $R = (F_{340}–F_{340,bg}) / (F_{380}–F_{380,bg})$. R_{min} and R_{max}, the respective minimum and maximum fluorescence ratios of the dye, and K_{eff}, the effective dissociation constant of the dye in the cell, are obtained from calibration experiments.

Mono- and bi-exponential calcium dynamics models are commonly fitted to the $[Ca^{2+}]$ transient deduced from the *ratiometric* transformation, theoretically giving estimates (and CIs) of the $[Ca^{2+}]$ baseline, influx and time constants.

Using Monte-Carlo simulations, we found that the CIs provided by this *ratiometric* approach were meaningful for the time constants only, and largely underestimated for $[Ca^{2+}]$ baseline and influx. This was mainly due to the fact that the measurement uncertainty of the calibration experiments was not taken into account.

III. A New Approach: The Direct Method

To overcome this limitation, we were led to develop a new "*direct*" method. This method embeds a calcium dynamics model within a full data generation model. The raw fluorescence data read out of the CCD camera at the two wavelengths are predicted simultaneously by the model, without any data ratioing. The use of a probabilistic model of the camera led us to the construction of meaningful CIs on the calcium dynamics parameters. These CIs take into account the finite precision with which the calibrated parameters are known. Moreover, we show how to handle a time-dependent buffer concentration, improving thereby considerably our goodness of fit (Fig. 1).

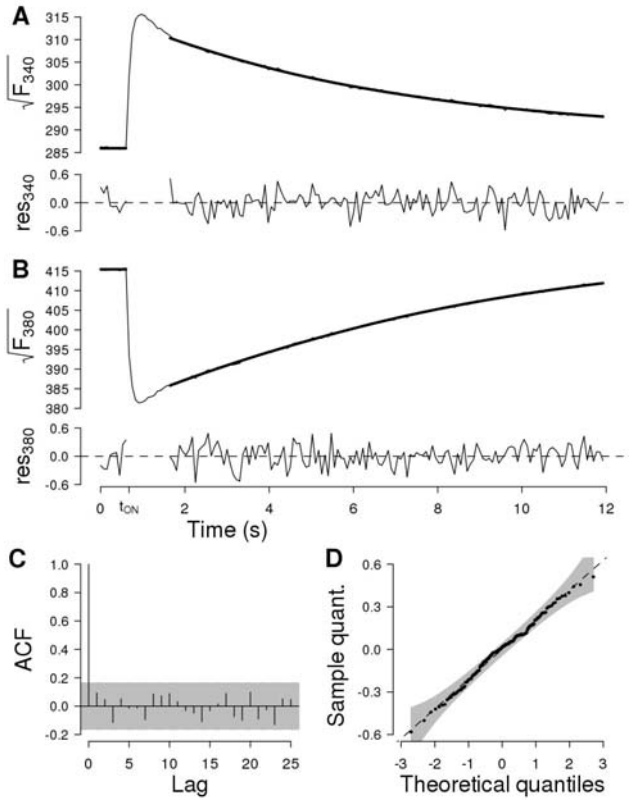


Fig. 1. Calcium transients were evoked in insect olfactory interneurons. The fluorescence transients recorded at 340 and 380 nm were simultaneously fitted using the *direct* method. The goodness of fit was assessed by the autocorrelation of the residuals (**C**) and the quantile-quantile plot (**D**).

IV. Conclusion

The *direct* method has been implemented in the open-source environment `R` and is freely distributed in the `CalciOMatic` package. Hence, we warmly encourage calcium imagers to analyze their data using the *direct* method.

V. Bibliography

[1] Grynkiewicz G, Poenie M, Tsien RY (1985) *A new generation of Ca2+ indicators with greatly improved fluorescence properties*. J Biol Chem, 260(6):3440-3450.

[2] Joucla S, Pippow A, Kloppenburg P, Pouzat C (2009) *Quantitative estimation of calcium dynamics from ratiometric measurements: a direct, non-ratioing, method*. J Neurophysiol, submitted.

The “mirror” estimate: An intuitive predictor of membrane polarization during extracellular stimulation

Sébastien Joucla and Blaise Yvert
CNRS, Université Bordeaux 1 / Centre de Neurosciences Intégratives et Cognitives (UMR 5228)
s.joucla@cnic.u-bordeaux1.fr

***Keywords* — central nervous system, functional electrical microstimulation, prosthesis, implants, activating function.**

I. Introduction

EXTRACELLULAR electrical stimulation of excitable tissues has been used empirically for decades, both with fundamental and clinical goals. New advances in microelectrode arrays allowing interfacing neural networks with hundreds of recording and stimulating sites require finding pertinent paradigms of extracellular microstimulation to modify and even control the dynamics and plasticity of neural networks. To address this question, the first step is to understand the direct effect of an extracellular stimulation on the membrane response of a single cell within the tissue.

II. The Classical View: The Activating Function

The cable equation (CE) formalism was originally proposed by McNeal (1) and extended by Rattay (2). These pioneering works have shown that the spatio-temporal variations of the membrane potential are driven by the following CE:

$$\tau \frac{\partial V_m(s)}{\partial t} + V_m(s) - \lambda^2 \frac{\partial^2 V_m(s)}{\partial s^2} = \lambda^2 \frac{\partial^2 V_{ext}(s)}{\partial s^2}.$$

The source term of this equation, called the activating function ($AF = \lambda^2 * \partial^2 V_{ext}/\partial s^2$), is proportional to the second derivative of the extracellular potential field V_{ext} along the fiber. The *AF* has long been used as an intuitive estimate of the membrane polarization in response to an extracellular stimulation. It was particularly useful to determine excitation and inhibition sites without requiring a full knowledge of biophysical properties of the cell. However, subsequent studies have pointed out several limitations of the *AF*, mainly due to the importance of longitudinal intracellular currents (neglected when considering the *AF* as the solution to the CE) and boundary fields playing an important role at the fiber terminations.

III. A New Approach: The Mirror Estimate

We show here that a very simple analytical steady-state solution to the CE can often be used as an alternative to the *AF*. Figure 1 illustrates the fact that while the *AF* approximates well the steady-state solution to the CE for very small space constants λ, this is no longer the case for high space constants. In the latter case, the membrane polarization is well predicted by a *mirror* estimate: $V_m(s) = -V_{ext}(s) + \langle V_{ext} \rangle$, where $\langle V_{ext} \rangle$ is the spatial mean of V_{ext} along the cell arborization.

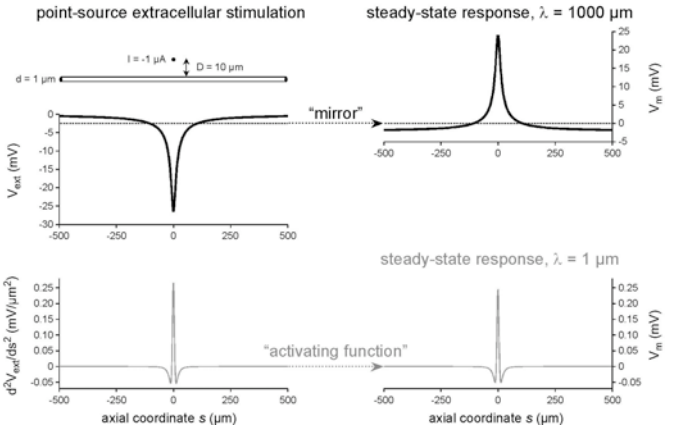


Fig. 1. *The steady-state response of a uniform passive fiber to an extracellular potential field depends on the space constant λ.* For λ = 1 µm, the steady-state membrane potential profile is close -in shape and amplitude- to the *activating function*, while for λ = 1000 µm, the membrane polarization becomes the opposite of the centered extracellular potential (*mirror*).

We numerically examined the domains – in terms of space constants, stimulation durations, fiber lengths, and electrode-fiber distances – where either the *mirror* or the *AF* estimates are most adequate. We found that the *mirror* domain includes a wide range of these parameters encountered in practice, and that this simple analytical estimate can advantageously be used in practice to predict the response of fibers as well as complex neurons to extracellular stimulation.

IV. Conclusion

The *mirror* estimate can often be preferred to the *activating function* to intuitively predict membrane polarization during extracellular stimulation.

V. Bibliography

1. McNeal DR (1976) Analysis of a model for excitation of myelinated nerve. IEEE Trans Biomed Eng 23:329-337.
2. Rattay F (1986) Analysis of models for external stimulation of axons. IEEE Trans Biomed Eng 33:974-977.
3. Joucla S and Yvert B (2009) The “mirror” estimate: An intuitive predictor of membrane polarization during extracellular stimulation. Biophys J, 96(9):3495-3508.

Support : French Ministry for Research and Technology (ACI, RMNT), ANR Blanc & TecSan, Fyssen, FRM, IRME, and Région Aquitaine (France).

A model of integration between reinforcement learning and task monitoring in the anterior cingulate and dorsolateral prefrontal cortices

Mehdi Khamassi[1,2], **René Quilodran**[1,2], **Emmanuel Procyk**[1,2], **Peter F. Dominey**[1,2]
[1]INSERM U846 Stem Cell and Brain Research Institute, Bron France, [2]Université Lyon I, France

I. INTRODUCTION

Growing evidence indicates that the anterior cingulate (ACC) and dorsolateral (DLPFC) prefrontal cortices play complementary roles in performance monitoring and cognitive control. However, the precise mechanisms at stake are not yet known. Our recent electrophysiological data from a primate visuo-motor task alternating exploration and exploitation periods suggest the existence within the ACC of neural activities related to 1) reinforcement learning (RL), 2) feedback categorization (e.g. responses to the first correct trial (COR1), responses to errors (ERR)), 3) task monitoring signals (e.g. cells that distinguish between exploration and exploitation) (Quilodran et al., 2008). Here we propose a new computational model which integrates reinforcement learning and task-monitoring. We use the model to predict monkey behavior and to analyze single-unit data recorded from the DLPFC-ACC network.

II. REINFORCEMENT LEARNING MODEL

We extend an existing RL model called 'Q-learning' to predict monkey behavior in this task (Sutton and Barto, 1998). The model performs quasi-optimally but can predict only 60% of monkeys' specific choices, primarily because a behavioral shift occurs when a new exploration phase starts. By adding a component that enables flexible reset of action values (taking into account individual spatial preferences) when detecting the need to switch to exploration, the model can reproduce 74% of the monkeys' choices.

III. MODEL-BASED ANALYSIS OF BRAIN ACTIVITY

We tested the correlation of some model variables such as Q-values and the reward prediction error (RPE) with single-unit activity recorded in ACC and DLPFC (Quilodran et al., 2008). We analyzed a selected sample of 76 neurons. We found 18 cells (20%) correlated with one of the model's Q-values. These neurons could encode the value of spatial targets and contribute to decision-making. 20 cells (25%) were correlated to the RPE. However, only 2 cells quantitatively encoded RPEs and could be interpreted as participating to RL. The other 18 cells were either neurons that respond to errors, neurons that respond to correct trials, or neurons that respond to the first correct trial of each problem. We interpret the latter neurons as participating to task-monitoring.

IV. NEURAL-NETWORK MODEL

We use these principles to develop a neural network model extending our previous cortico-striatal loop model (Dominey 2005). In our ACC component, prediction error signals are extracted to produce COR1 and ERR signals. The latter are used to update a modulatory variable (MV) which boosts the DLPFC system after errors during exploration, and which attenuates it during exploitation, when the cortico-striatal pathway assures action repetition.

This model performs the task optimally, and enabled us to reinterpret additional ACC single unit observations such as cells that respond both to errors and to cues signalling the beginning of exploration phases (which require an increase of MV), and cells that respond more during exploitation phases (where MV is low). The model also explains several key characteristics of DLPFC including increased spatial selectivity as exploration progresses, and a drop of spatial selectivity during exploitation.

V. CONCLUSION

This work provides a formal link between theoretical (Q value, TD) models, their neural network implementation, and the underlying neurophysiology. Our model proposes a testable mechanism of integration between RL and task-monitoring within the ACC-DLPFC network.

BIBLIOGRAPHY

Dominey, P.F. (2005) From sensorimotor sequence to grammatical construction: Evidence from simulation and neurophysiology. Adaptive Behavior, 13(4):347-62.

Quilodran, R., Rothe, M., and Procyk, E. (2008) Behavioral shifts and action valuation in the anterior cingulate cortex. Neuron, 57, 314–25.

Sutton, R. and Barto, A. (1998) Reinforcement Learning: An Introduction. MIT Press.

Learning behaviour in a two alternative decision task in primate follows a gradient based learning rule

Laquitaine S.1, Loewenstein Y.2,4, Gross C.1, Hansel D.3,4, Boraud T.1,4
(1) Bordeaux 2 CNRS, UMR 5227, Bordeaux, France. (2) Dept. of Neurobiology and Cognitive Sciences, Jerusalem, Israel. (3) Neurophysique et physiologie, Paris Descartes, France. (4) Interdisciplinary Center for Neural Computation, Hebrew University of Jerusalem, Israel Steeve_laquitaine@msn.com.

Keywords—behavioural model, linear reward inaction algorithm, choice learning task, primates

INTRODUCTION

To describe choice learning behaviour in decision Markov processes, artificial intelligence proposes different approaches in which choice behaviour can be based on dynamic action evaluation. The limit of action value based approaches is the complex computation necessary to integrate the agent initial preference for one of the alternative. A simpler learning rule directly updates choices based on past reward and choices, and authorizes an initial preference for one alternative without extensive computation. This approach consists in finding the optimal policy by policy iteration and is not based on action evaluation. In this work we test if this simple linear mathematical equation based on linear reward inaction properties can partially reproduce experimentally observed behavioural results in a two alternatives decision task in which 2 monkeys had to learn new associations of reward probabilities and targets randomly presented in 4 directions.

I. METHODS

A. *Behavioural experiment:*

2 female monkeys (4 kg) performed a 2 armed bandit free choice learning task

B. *Model:*

$Paj(t+1) = \eta * r(t) * (A(t) - Paj(t))$.

Paj (t+1): probability of choosing alternative Aj on the next trial; Paj (t): probability of choosing Aj in the current trial; η: learning factor or plasticity rate; A (t): current choice; r (t): reinforcement received.

The error rule driving probability of choice is: $\delta Wj(t) = \eta(t) * r(t) * (Ai(t) - Paj(t))$

II. DISCUSSION

We first show that probabilistic conditions modulate performance of the monkey while no directional bias was observed.

Learning was characterized by two phases: a dynamic phase where the preference of the monkey progressively builds up, then a suboptimal stationary phase.

We then used computational methods to extract the simplest mathematical parameters that could support the behavioural results and showed that a simple linear mathematical equation based on reward inaction properties could reproduce the behavioural results with 67-70 % likelihood. Where the model couldn't reproduce a similar level of exploration to the late behaviour, it fitted with its general dynamic.

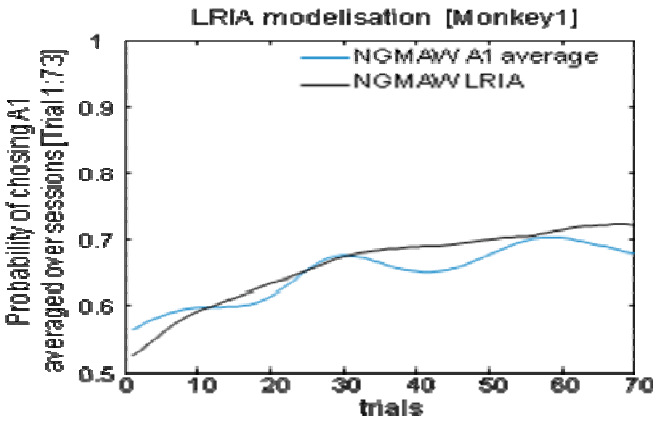


Fig. 1. Comparison between model and monkey learning curve

III. CONCLUSION

The present study shows that a primate complex learning behaviour in a probabilistic decision task, can mechanistically be described by a one free parameter gradient based model.

BIBLIOGRAPHY

K. S. Narendra and M. A. L. Thatachar, "Learning automata-A survey," IEEE. Trans. Syst. Man, Cybern., vol. SMC-4, pp. 323-324, 1974.

R.S.Sutton and A.G. Barto, "Reinforcement learning", MIT press, Cambridge, MA, A Bradford Book, 1998

This work was supported by the Université Victor Segalen , the CNRS and french israeli neurorobotic fund (embassade de France en Israel) and PID neuroinformatique, CNRS and the french-Israel neuroscience lab (CNRS HUJI).

Feedback modulation of BCM's neurons in multi modal environment

Mathieu Lefort, Yann Boniface and Bernard Girau
LORIA
mathieu.lefort@loria.fr

***Keywords*—BCM, feedback modulation, multi modalities, neural network.**

Abstract

In Gibson's theory, an object is defined by its interactions with people, named affordances. Affordances raise from the association of the object's sensory perceptions. Bienenstock Cooper and Munro's (BCM's) cells converge to one of the input patterns with a decentralized and unsupervised learning (refer to [1]). We want to extend this mechanism in order to develop a generic incremental modalities association paradigm. We introduce a feedback modulation of BCM's neurons to obtain a spatial self-organization. This feedback will be the reflect of the multi modal constraints.

I. BCM's neurons

A. Biological facts

Some experiments (see [2]) show that the LTD (Long-Term Depression) / LTP (Long-Term Potentiation) threshold is sliding. This threshold between hebbian and anti hebbian learning depends on the past history of neuron spiking. This mechanism restrains synaptic weights and induces the specialization of the neuron to one input pattern.

B. Mathematical equations

The neuron potential u is equal to the dot product of its synaptic weights and its input x.

The synaptic weights are modified according to the following equation:

$$\Delta w = \eta * x * u * (u - \theta) \quad (1)$$

with η the learning rate and θ the sliding LTD/LTP threshold. θ is equal to the exponential filtering of u^2.

Under conditions for convergence, the neuron output will be $1/p$ for the discriminate input pattern, with p the probability of its apparition, and 0 for the others. These two outputs are the stable states of the Δw equation: $u=0$ and $u=\theta$. See [3] for more informations.

II. Introduction of feedback modulation

A. Motivation

Our work deals with the association of several modalities with a multi levels and multi maps neuronal architecture defined in [4]. Each modal map has to self organize itself considering the multi modal environment. One activity bump per map, coherent with the others, appears in a competitive layer. This bump will be used as feedback modulation to obtain an self organization of the map.

B. Principle

The aim is to encourage the LTP under the bump. To do this, we multiply u by a sigmoidal feedback in order to help the potential u to overcome the θ threshold.

The u equation becomes:

$$u = (w \,.\, x) * \frac{\alpha}{1 + (\alpha - 1) * e^{-\beta * feedback}}$$

III. Results and conclusion

We obtain a spatial self organization of a BCM's neurons isolated map modulated with a bump feedback. This positive and negative bump appears in a cnft competitive layer and influences the spatial convergence of the map. Multi modal constraints will have to influence the position of each modal bump.

IV. Bibliography

[1] Leon N. Cooper, Nathan Intrator, Brian S. Blais, and Harel Z. Shouval. Theory of Cortical Plasticity. World Scientific, Singapore, 2004.

[2] A. Kirkwood, M. G. Rioult, and M. F. Bear. Experience-dependent modification of synaptic plasticity in visual cortex. , 381 :526–528, June 1996.

[3] B.S. Blais. The Role of the Environment in Synaptic Plasticity : Towards an Understanding of Learning and Memory. PhD thesis, Brown University, 1998.

[4] Olivier Ménard and Hervé Frezza-Buet. Model of multimodal cortical processing : coherent learning in self-organizing modules. Neural Netw., 18(5-6) :646–655, 2005.

Characterization of spontaneous activity patterns during development of whole embryonic mouse hindbrain and spinal cord in organotypic cultures

Yanling Li, Oussama Abdoun, Sébastien Joucla, Blaise Yvert
CNRS, Université Bordeaux 1 / Centre de Neurosciences Intégratives et Cognitives (UMR 5228)
y.li@cnic.u-bordeaux1.fr

***Keywords* — Maturation, neural networks, activity-dependent plasticity, microelectrode arrays.**

I. Introduction

SPONTANEOUS activity is a common feature of immature neural networks (1–4) observed in various species and parts of the developing central nervous system (CNS), and known to play a critical role in the maturation of neural systems. However, the spatiotemporal (ST) patterns of spontaneous activity have rarely been described at the level of the whole immature CNS. Moreover, how ST patterns evolve along the maturation of the CNS, and whether stereotyped patterns can be identified as specific landmarks of development stages, remain unknown.

II. Results

Here, we address this question by studying the day-by-day evolution of spontaneous activity in organotypic cultures of whole embryonic mouse hindbrain-spinal cord preparations. Microelectrodes arrays (MEAs) composed of 60 electrodes were used to record spontaneous activity over this whole system. Hindbrain and spinal cord were isolated at embryonic day 12 (E12), and maintained in culture on MEAs for at least seven days. Spontaneous activity was recorded every day for at least one hour. Both local field potentials (LFP) and spiking bursts activity were considered.

We developed specific algorithms inspired from image processing and automatic clustering to detect and classify the different spatiotemporal patterns expressed during episodes of LFPs and/or bursts of spikes. In order to link these patterns with the anatomy, we also designed a cartographic software based on spline interpolation to generate maps of activity. The clustering analysis revealed that the numerous episodes of spontaneous activity occurring in the cultured preparations expressed a limited number of spatiotemporal patterns. Another critical feature of spontaneous activity is its evolution over time, in terms of pattern variability, frequency of occurrence, single/multiple events ratio, as well as amplitude and spatial extent. A thorough analysis of these parameters brought to light a stereotyped scheme of evolution of spontaneous activity across all our preparations. Especially, episodes composed of single LFPs were observed at early stages (from day in culture DIC1), while multiple-LFPs episodes occurred later on (from DIC 3 on). Moreover, thereafter DIC3, a rostro-caudal maturation of activity was observed.

III. Conclusion

Altogether, these results contribute to define a detailed characterization of spontaneous activity during development in culture of entire structures of the CNS.

IV. Bibliography

1. Penn AA, Riquelme PA, Feller MB, Shatz CJ (1998) Competition in retinogeniculate patterning driven by spontaneous activity. Science 279:2108-2112.
2. O'Donovan MJ (1999) The origin of spontaneous activity in developing networks of the vertebrate nervous system. Curr Opin Neurobiol 9:94-104.
3. Ben-Ari Y (2001) Developing networks play a similar melody. Trends Neurosci 24:353-360.
4. Yvert B, Branchereau P, Meyrand P (2004) Multiple Spontaneous Rhythmic Activity Patterns Generated by the Embryonic Mouse Spinal Cord Occur Within a Specific Developmental Time Window. J Neurophysiol 91:2101-2109.

Support : French Ministry for Research and Technology (ACI, RMNT), ANR Blanc & TecSan, Fyssen, FRM, IRME, and Région Aquitaine (France).

Modeling AP-evoked Ca^{2+} transients in cerebellar cortex interneurons

Tiffany Lieury, Sébastien Joucla, Christophe Pouzat and Thibault Collin
Laboratory of cerebral physiology, CNRS, UMR 8118
tiffany.lieury@parisdescartes.fr

***Keywords*— Cerebellar Cortex Interneurons, Calcium Dynamics, Modeling.**

I. Introduction

The kinetics of presynaptic Ca^{2+} signaling play a major role in the control of neurotransmitter release, and thus, in the communication between neurons. Endogenous Ca^{2+} buffers such as parvalbumin (PV), calretinin and calbindin, modify the shape of presynaptic Ca^{2+} transients, and can therefore interfere with synaptic neurotransmitter release. In cerebellar molecular layer interneurons, PV has been shown to be responsible for the biphasic decay of action potential-evoked (AP-evoked) Ca^{2+} signals in single axonal varicosities (see [3] and Fig.1).

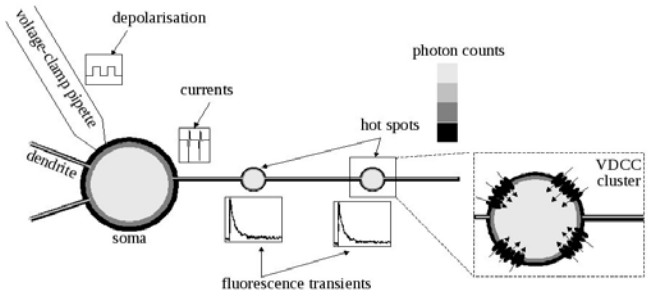


Fig. 1. Depolarization-induced $[Ca^{2+}]_i$ transients in stellate/basket cells have a marked spatial heterogeneity, being much larger in discrete spots of the axon than in dendrites and soma [1,2]. The Ca^{2+} signal presents local maxima at

Unfortunately, some parameters such as the kinetics of the Ca^{2+} extrusion mechanisms, the intracellular concentration of PV and the Ca^{2+} influx associated with each AP are hardly measurable experimentally. Moreover, fluorescence recording techniques require the use of a probe which is also an exogenous buffer that alters presynaptic Ca^{2+} decay kinetics. To overcome experimental limitations, we have developed a mathematical model allowing us to further investigate the influence of PV on AP-evoked Ca^{2+} signals in single axonal varicosities.

II. Calcium Dynamics Model

Our single compartment model describes the kinetic effects of endogenous buffers (ATP, PV, Mg2+) in the presence of an exogenous buffer (fluorescent probe P) using first-order ordinary differential equations. All extrusion systems (Na+-dependent Ca2+ efflux and ATP-fueled Ca2+ pump) have been lumped as a single, voltage-independent system bearing Michaelian kinetics [4]. AP-evoked Ca2+ transients are simulated by an instantaneous Ca2+ rise (Ca2+ pulse).

III. Parameters optimization

Our Ca2+ dynamics model is embedded into a fluorescence model which takes into account the properties of the CDD camera (gain and readout noise). This direct approach allows us to directly compare simulations results with raw fluorescence data instead of using fluorescence ratios (F/F0), as usually done. The parameters of the Ca2+ dynamics model were optimized so as to minimize the weighted least squares error between experimental and simulated fluorescence transients. We focused on the optimization of three unknown parameters i.e: the total concentration of PV, the maximal Ca2+ efflux velocity (or extrusion rate) and the Ca2+ influx.

Experiments were performed in cultured cerebellar interneurons. We compared Ca2+ from wild-type and PV(-/-) mice at two stages: postnatal days 10-12 (P10-P12) and P19-P21.

IV. Conclusion

Using this model, we investigate Ca^{2+} dynamics in presynaptic axonal varicosities in the presence of PV. It allows us to quantify Ca^{2+} extrusion kinetics, PV intracellular concentration and AP-induced Ca^{2+} influx.

The direct approach, taking into account a probabilistic model of the acquisition system, will enable us to assess meaningful confidence intervals on our estimated parameters.

Moreover, this optimized model of Ca2+ dynamics will allow us to predict the actual time course of Ca2+ signal in the absence of exogenous Ca2+ probes.

V. Bibliography

[1] Llano I., Tan Y., Caputo C., *Spatial heterogeneity of intracellular Ca2+ signals in axons of basket cells from rat cerebellar slices,* J. Physiol., vol.502, 1997.
[2] Forti L., Pouzat C., Llano I., *The spatial distribution of action potential-evoked Ca2+ signals in axons of developing rat cerebellar interneurons,* J. Physiol., vol.527, 2000.
[3] Collin T., Chat M., Lucas MG., Moreno H., Racay P., Schwaller B., Marty A., Llano I., *Developmental changes in parvalbumin regulate presynaptic Ca2+ signaling,* J. Neurosci., vol.25, 2005.
[4] Sala F., Hernández-Cruz A., *Calcium diffusion modeling in a spherical neuron. Relevance of buffering properties, Biophys. J., vol.*57, 1990.

Denoising Two-Photon Calcium Imaging Data

Wasim Q. Malik[1,2], James Schummers[1,3], Beata Jarosiewicz[1,3], Mriganka Sur[1,3], and Emery N. Brown[1,2]
[1] Department of Brain and Cognitive Sciences, Massachusetts Institute of Technology, Cambridge, MA 02139, USA.
[2] Department of Anesthesia and Critical Care, Massachusetts General Hospital, Harvard Medical School, Boston, MA 02114, USA.
[3] Picower Institute for Learning and Memory, Massachusetts Institute of Technology, Cambridge, MA 02139, USA.
wqm@mit.edu

***Keywords*—Calcium imaging, statistical modeling, two-photon.**

I. Introduction

TWO-photon laser scanning microscopy is a useful tool that facilitates very high-resolution *in vivo* imaging of neuronal populations [1]. We present a novel statistical framework for extracting functional information from two-photon calcium fluorescence images. Our results show that the proposed technique substantially outperforms the existing approaches.

II. Methods

A. Experiment

Neurons in the visual cortex of anesthetized ferrets were bulk-loaded with the fluorescent calcium indicator OGB. *In vivo* imaging was performed using a two-photon microscope. Time-series traces with a 250x250μm field-of-view were obtained at 1 Hz. The visual stimulus consisted of three repetitions of a square-wave grating with 100% contrast rotating by 10˚ in successive data frames. The traces at each pixel in the frame were used in the time-series analysis below.

B. Signal Model

As the stimulus described above is periodic, we can express the neuronal response in terms of its Fourier series expansion. We thus use a multiple harmonic model to estimate the baseline and stimulus-evoked response, with the number of harmonics determined by model order selection criteria. The residual stimulus-free response, which represents various colored noise processes, is modeled with an autoregressive (AR) process of the appropriate order. The stimulus-evoked component is a noise-free estimate of the neuronal response.

C. Parameter Estimation

By separating the signal and noise components, we can recast the estimation problem into two multiple regression problems which can be solved efficiently. We use an ordinary least squares procedure to estimate the harmonic coefficients, and the Burg algorithm to estimate the AR coefficients. A cyclic descent algorithm is developed that iteratively estimates the harmonic and AR coefficients jointly, converging rapidly.

This work was supported by NIH Grants DP1 OD003646 and EY07023.

III. Results

These methods are applied to *in vivo* visual cortex data to study the orientation selectivity of neurons and astrocytes. We find that a sufficiently low-order model, consisting of 4 harmonics and 8 AR terms, is sufficient to represent the data accurately. The cyclic descent converges after about 5 iterations, and is thus computationally efficient. The estimates thus obtained for each pixel of the frame are used to construct denoised neuronal response to the stimulus applied. Fig. 1 shows the response of a single neuron as an example, whose preferred orientation is at about 180˚ (corresponding to the third frame). Our approach achieves remarkable denoising and contrast improvement, making the cell boundary and dendritic structure clearly observable while suppressing the noise at nonpreferred orientations and from the surrounding neuropil.

IV. Conclusion

Our statistical model and algorithms provide a principled and efficient method for obtaining improved neuronal response estimates from high-resolution two-photon images of the brain. The resulting denoised images enable the analysis of inter- and intra-neuronal structures and their functional characteristics with substantially improved clarity and reliability.

V. References

[1] J. Schummers, H. Yu, and M. Sur, "Tuned responses of astrocytes and their influence on hemodynamic signals in the visual cortex," *Science*, vol. 320, no. 5883, Jun. 2008.

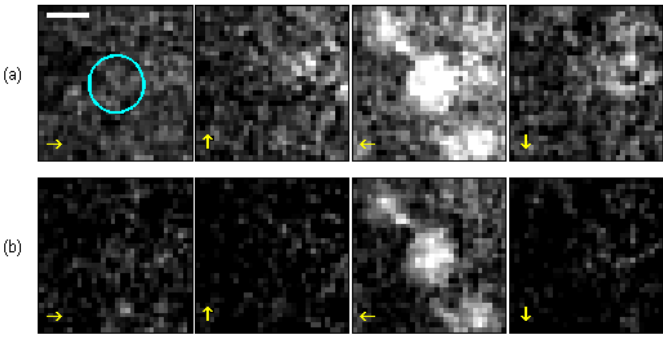


Fig. 1. Two-photon images showing the functional response of a cell (location marked with blue circle) to a visual stimulus with orientation indicated by the arrow in each frame: (a) With conventional processing based on averaging across multiple trials; (b) Estimate from our technique. (Scale bar represents 10 μm; brightness scale represents $\Delta F = [0, F_{max}]$).

A cortical column model for studying spatial navigation planning

Louis-Emmanuel Martinet[1,2], Denis Sheynikhovich[1], Angelo Arleo[1]
[1] CNRS - UPMC Univ Paris 6, UMR 7102, F-75005, Paris, France
[2] CNRS - UPMC Univ Paris 6, UMR 7222, ISIR, F-75005, Paris, France
louis-emmanuel.martinet@upmc.fr

***Keywords*—hippocampus, neuromimetic modelling, prefrontal cortex, spatial navigation planning.**

I. Introduction

According to experimental evidence, spatial navigation planning is likely to rely upon a distributed neural network spanning limbic and cortical brain structures. This network includes (i) the hippocampus, which mediates robust spatial representations, and (ii) neocortical structures, such as the prefrontal cortex, which participate to the elaboration of more abstract contextual descriptions (e.g., accounting for motivation-dependent memories and action cost/risk constraints).

II. Methods

In order to investigate this working hypothesis, we model the interaction between the hippocampus [1] and the prefrontal cortex [2]. We focus on the cortical columnar organisation to study a neuromimetic architecture suitable for spatial navigation planning. We validate the system's learning performance on a classical spatial behavioural task, the Tolman & Honzik's detour protocol [3], which suggests that rodents can plan flexible goal-directed trajectories in the presence of blocked pathways. We also put forth a set of statistical analysis to assess the spatial coding properties of the model hippocampal place and cortical column cells.

Here, we couple our hippocampal place cell [1] and columnar cortical [2] models to provide a better understanding of the dynamics of the action planning neural network. We also improve the biological plausibility of the cortical model [2], by explicitly identifying the subpopulations of neurones that encode different information (e.g., current spatial state, goal-related and prospective memory signals, local actions). The response of each subpopulation being more specific, it makes it possible to perform a series of analyses of multiple neural activity correlates.

III. Results

The spatial planning model reproduces the experimental results by Tolman & Honzik [3]. It also unravels the possible links between the single unit level and the behavioural level relevant to the learning of the task (e.g., to the selection of the shortest path to the reward, and to the prediction of future state sequences). Finally, our neural response analysis suggests how the interplay between the model hippocampus and the prefrontal cortex can yield to the encoding of manifold information pertinent to the spatial planning function (e.g., prospective and distance-to-goal correlates).

IV. Discussion

Extensions of the model are being currently explored. In particular, multidimensional encoding (such as motivational information and mutiscale spatial correlates) is being introduced in the columnar network model. This will increase its representational capacity so that it will be able to mediate more complex decision-taking processes (e.g. cost-benefit analysis, large maze solving). In addition, the model is being validated by comparing the simulated neural responses against those obtained by *in vivo* electrophysiological recordings from the hippocampus and the prefrontal cortex of freely moving rats [4]. This comparative study aims at providing new insights on the interaction between the hippocampus and the prefrontal cortex. It can also lead to testable predictions about the learning processes related to spatial memory, such as declarative memory consolidation occurring during sleep.

V. References

1. Sheynikhovich D, Chavarriaga R, Stroesslin T, Arleo A, Gerstner W (2009) Is there a geometric module for spatial orientation? Insights from a rodent navigation model. *Psychol Rev (in press)*.
2. Martinet LE, Passot JB, Fouque B, Meyer JA, Arleo A (2008) Map-based spatial navigation: A cortical column model for action planning. *LNAI Springer-Verlag*, 5248:39–55.
3. Tolman EC, Honzik CH (1930) "Insight" in rats. *Univ Calif Publ Psychol*, 4(14):215–232.
4. Benchenane K, Peyrache A, Khamassi M, Battaglia FP, Wiener SI (2008) Coherence of theta rhythm between hippocampus and medial prefrontal cortex modulates prefrontal network activity during learning in rats. *Soc Neurosci Abstr*, No. 690.15.

Acknowledgements: Granted by EC Project ICEA (Integrating Cognition, Emotion and Autonomy), IST-027819-IP.

A BMI application to control a robotic digit

[1,3]Ouanezar S., [1,2]Eskiizmirliler S. and [1,2]Maier M.A.
[1]Université Paris Descartes / LNRS - CNRS UMR7060, [2]Université Denis Diderot , UFR Sciences du Vivant
[3]TELECOM ParisTech / Département TSI Signal-Images
ouanezar@tsi.enst.fr

***Keywords*—BMI, neurobotics, motor cortex, EMG, bio-inspired control, computational neuroscience.**

I. Introduction

Various recent brain-machine interfaces (BMI) have emphasized the potential of using cortical signals for the kinematic control of artificial devices such as robotic arms or legs [1]. However, very few studies are available for dynamic control as well as for the control of grasp movements.

In this work our main goal is to first provide a proof-of-concept that a 4 Dof anthropomorphic robot finger can be controlled by intra-cortical signals recorded in monkeys and second to replicate the applied force.

II. Method

The biological signal is composed of the activity of 33 corticomotoneuronal (CM) cells and multiunit EMG signals recorded from up to eight intrinsic hand and forearm muscles as the monkey performed a low force precision grip task as shown in Figure 1 [2].

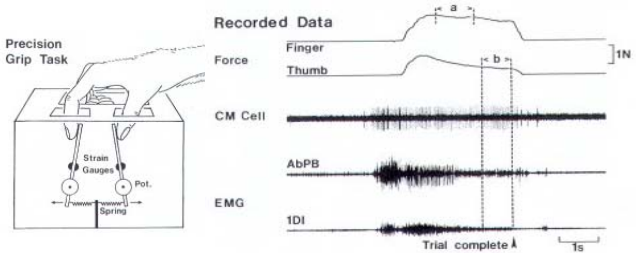


Fig. 1. Schematic diagram of the manipulandum used for the precision grip task and the examples of the recorded signals.

In subsequent work [3] it has been demonstrated that the muscle activity can be predicted by means of artificial neural networks. However, only the correlation between a given muscle activity and one of the corresponding CM cells was investigated. The average estimated performances in predicting EMG signals were 8.9% and 29.37% for TDMLP and TempUnit (two different types of artificial neural networks ANN) respectively. In the current work we first tried to increase the reported performances by using all available CM cell activities as input to the ANNs, i.e. all CM cells that facilitated the same EMG. Figure 2 shows an example of the used experimental data during one trial as well as the real and estimated EMG signals obtained during successive trials. In the training phase we set the number of hidden layer neurons to 25 and used the adaptive learning rate backpropagation algorithm. We showed that the estimation performance can be increased to 32.7% and 48% with the same types of ANNs.

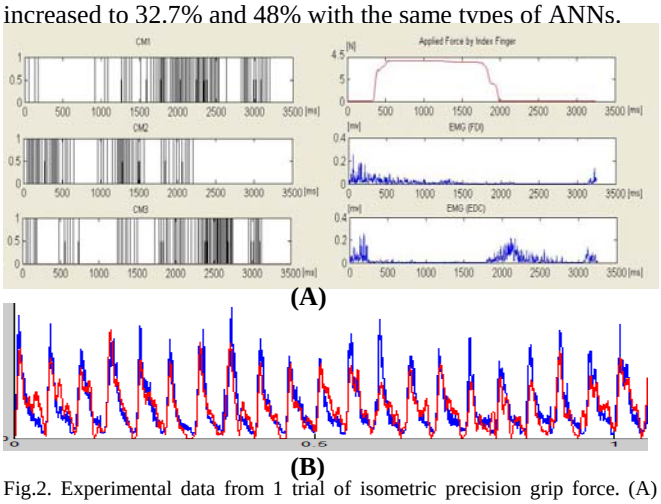


Fig.2. Experimental data from 1 trial of isometric precision grip force. (A) Right, from top to bottom: applied force and the rectified EMG signals of the muscles (FDI and EDC). Left: corresponding CM cell activities which facilitated the FDI as determined by spike-triggered averaging. (B) The real (blue) and estimated (red) EMG signals of several successive trials.

The second step of the work will comprise the application of this EMG estimator to control an anthropomorphic robotic finger actuated by antagonist artificial muscles to replicate the recorded monkey index finger movements as shown in Fig. 3

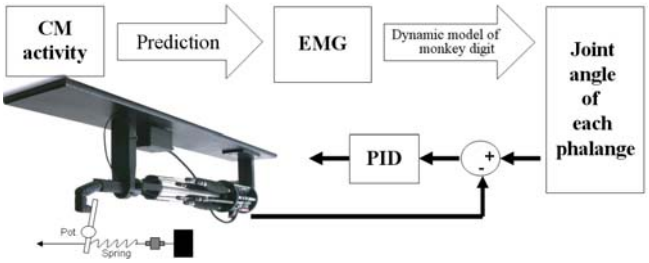


Fig.3. The schematic representation of successive steps for the biomimetic control of an anthropomorphic robotic finger unit (Shadow Robot Co.)

III. Bibliography

[1] Lebedev M.A. and Nicolelis M.A.L. "Brain-machine interfaces: past, present and future.", Trends in Neurosciences 2006; 29(9):536-546.
[2] Maier M A; Bennett K M; Hepp-Reymond M C; Lemon R N "**Contribution of the monkey corticomotoneuronal system to the control of force in precision grip.**" Journal of Neurophysiology 1993;69(3):772-85.
[3] Manette OF, Maier MA."**Temporal processing in primate motor control: relation between cortical and EMG activity**", IEEE Trans Neural Netw. 2004 Sep;15(5):1260-7.

This work is supported by the FRM project DBC20080713368

Studying the role of the cerebellum in spatial cognition through a neurocomputational approach

J-B. Passot, A. Arabo, D. Sheynikhovich, L. Rondi-Reig, A. Arleo

CNRS – UPMC Univ Paris 6, UMR 7102, F-75005 Paris, France
jbpassot@snv.jussieu.fr

***Keywords*—Cerebellar microcomplex, spatial navigation, spiking network model.**

I. Context

Recent experimental findings have begun to unravel the implication of the cerebellum in high-level functions such as spatial cognition [1,2]. We focus on behavioural genetic data showing that L7-PKCI mice (lacking LTD at parallel fibre–Purkinje cell synapses) have a spatial learning impairment in the Morris Watermaze (MWM), whereas they exhibit normal performances in the Starmaze, a paradigm that reduces the procedural demand of the task [3]. These results suggest that cerebellar learning may prominently contribute to the procedural component of spatial learning [3].

II. Methods

We model the main information processing components of the cerebellar microcomplex via a large-scale network of spiking neurons. We test the performances of artificial L7-PKCI mice in simulated MWM and Starmaze environments. Importantly, we isolate the purely procedural component of the learning process by endowing simulated controls and mutants with identical declarative learning capabilities.

III. Results

The model reproduces most of the experimental results on the learning impairments of L7-PKCI mice: in the MWM, the mean escape latency and the mean angular deviation between the optimal direction to the target and the actual motion direction of the animal are both significantly larger compared to controls. These differences are not due to swim capability deficits. Furthermore - consistent with experimental data - simulated mutants and controls exhibit comparable learning capabilities in the Starmaze paradigm.

On the other hand, our simulations cannot reproduce the experimentally observed difference between the goal-searching behaviour of mutants and controls in the MWM [3]. In fact, our results suggest that a purely local impairment of the procedural component cannot explain this latter deficit.

IV. The cerebellum and the exploration - exploitation trade-off

To explain the experimental discrepancy between control and L7-PKCI, we have put forth a hypothesis according to which the mutants' impairment in optimizing their goal-searching behaviour could be due to a deficit in trading-off exploration and exploitation strategies [4]. This hypothesis has been tested by perturbating the ability of simulated mutants to properly balance their exploration-exploitation behaviour when solving the MWM and Starmaze tasks. By simulating this deficit, we could reproduce all the differences observed experimentally between control and mutant performances [3].

V. Cerebellum and spatial learning

The cerebellum plays an important role in integrating proprioceptive information to predict future state variables, such as body orientation and position, given a motor command [5]. A hypothesis being tested with our model is that the ability of L7-PKCI mice to integrate idiothetic (e.g. proprioceptive) signals might be impaired. This would indirectly affect the path integration process [6,7]. Since the latter contributes to the learning of stable spatial representations [6,8], we therefore propose that L7-PKCI mice might have a deficit in acquiring spatial representations that remain stable under different environmental conditions. This hypothesis is being evaluated by coupling our cerebellar model to an existing model of the hippocampal spatial learning function [8,9]. Preliminary results suggest that the cerebellum might be critical to build declarative spatial knowledge when idiothetic inputs are the most reliable source of information (e.g. when navigating in darkness).

VI. References

1. Rondi-Reig L, et al. *Prog Brain Res.* 2005, **148**:199-212
2. Petrosini L, et al. *Prog Neurobiol.* 1998, **56(2)**:191-210
3. Burguière E, et al. *Nat Neurosci.* 2005, **10**:1292-1294.
4. Cohen J, et al., *Philos Trans R Soc Lond B Biol Sci.* 2007, **362(148)**:933-42.
5. Wolpert DM, Kawato M, *Neural Netw* 1998, **11**:1317-29
6. Etienne AS, Jeffrey KJ. *Hippocampus* 2004, **14(2)**:180-92.
7. Leggio MG, et al. *Brain Res.* 1999, **127**:1–11
8. Arleo A, et al. *Biol Cybern.* 2000, **83(3)**:287-99.
9. Sheynikhovich D, et al. *Psychol Rev.* 2009, **in press**

Prediction along a line in a probabilistic model of motion perception

Laurent U. Perrinet and Guillaume S. Masson
Institut de Neurosciences Cognitives de la Méditerranée (INCM)
31, ch. Joseph Aiguier, 13402 Marseille Cedex 20, France
CNRS / Université de la Méditérrannée

Short presentation of a large moving pattern elicits an Ocular Following Response (OFR) that exhibits many of the properties attributed to low-level motion processing such as spatial and temporal integration, contrast gain control and divisive interaction between competing motions. Similar mechanisms have been demonstrated in V1 cortical activity in response to center-surround gratings patterns measured with real-time optical imaging in awake monkeys. More recent experiments of OFR have used disk gratings and bipartite stimuli which are optimized to study the dynamics of center-surround integration. We quantified two main characteristics of the global spatial integration of motion from an intermediate map of possible local translation velocities: (i) a finite optimal stimulus size for driving OFR, surrounded by an antagonistic modulation and (ii) a direction selective suppressive effect of the surround on the contrast gain control of the central stimuli (Barthélemy et al., 2007).

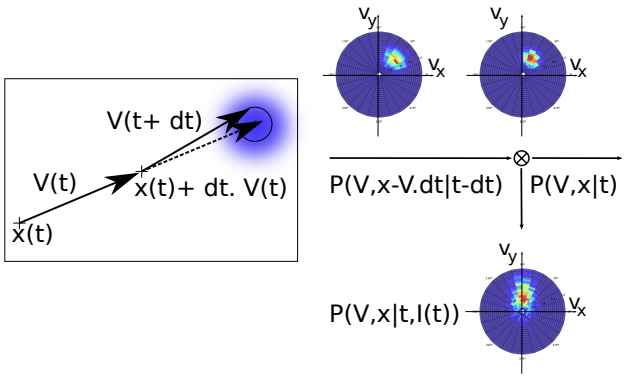


FIGURE: Architecture of the model. The system selects a priori continuous direction according to the advection equation. Implementation as a Markov Chain.

We explore here an hypothesis where center-surround interactions could be understood as the effect of velocity prediction. In fact, the integration step in previous models (see (Perrinet and Masson, 2007)) assumes independence of the local information while natural scenes are very predictable due to the rigidity and inertia of physical objects in visual space. We implement this in a retinotopic velocity map, where predictions are propagated by lateral recurrent interactions (Rao and Ballard, 1999; Bayerl and Neumann, 2004). Similarly to (Cremers, 2007), writing the velocity field as a vector field, velocity is conserved along path-lines:

$$\nabla V.V + \Delta_t V = \mathcal{N}_V$$

This leads to the auto-advection term in the Navier-stokes equations and adds-up to the equation for intensity conservation.

We implement this in a realistic model of a layer representing velocities in a map of cortical columns, where predictions are implemented by lateral interactions within the cortical area. First, raw velocities are estimated locally from images and are propagated to this area in a feed-forward manner. Using this velocity map, we progressively learn the dependance of local velocities in a second layer of the model. Results show that this simple model is sufficient to disambiguate characteristic patterns such as the Barber-Pole illusion. Due to the recursive network which is modulating the input to the velocity map, it also explains that the representation may exhibit some memory, such as when an object suddenly "disappears" or when presenting a dot followed by a superimposed line (line-motion illusion). This model may be applied to algorithms for the efficient representation of video sequences since it gives a Partial Derivate Equation (PDE) for an optimal dynamical evolution of the representation for natural scenes.

References

F. Barthélemy, L. U. Perrinet, E. Castet, and G. S. Masson. Dynamics of distributed 1D and 2D motion representations for short-latency ocular following. *Vision Research*, 48(4):501–22, feb 2007. doi: 10.1016/j.visres.2007.10.020.

P. Bayerl and H. Neumann. Disambiguating Visual Motion Through Contextual Feedback Modulation. *Neural Computation*, 16:2041–66, 2004.

D. Cremers. Bayesian approaches to motion-based image and video segmentation. pages 104–123. 2007. doi: 10.1007/978-3-540-69866-1. URL `http://dx.doi.org/10.1007/978-3-540-69866-1`.

L. U. Perrinet and G. S. Masson. Modeling spatial integration in the ocular following response using a probabilistic framework. *Journal of Physiology (Paris)*, 101(1–3): 46–55, 2007. doi: 10.1016/j.jphysparis.2007.10.011. URL `http://incm.cnrs-mrs.fr/LaurentPerrinet/Publications/Perrinet07neurocomp`.

R. Rao and D. H. Ballard. Predictive Coding in the Visual Cortex: A Functional Interpretation of Some Extra-Classical Receptive-Field Effects. *Nature Neuroscience*, 2(1):79–87, 1999.

This work was supported by EC IP project FP6-015879, "FACETS". Scripts reproducing these results may be obtained at `http://incm.cnrs-mrs.fr/LaurentPerrinet`.

EEG segmentation through time-varying PCA

Maxime Rio, Axel Hutt and Bernard Girau
Loria, Campus Scientifique, BP329
54506 Vandoeuvre-lès-Nancy, France
maxime.rio@loria.fr

***Keywords*— Evoked related potentials, principal components analysis, hidden Markov model, variational inference**

I. Introduction

EEG recordings have been used as a tool to measure the brain activity for a long time. Especially, through the stimulus evoked experimental paradigm, numerous cortical activation patterns have been discovered, as sequences of evoked related potentials (ERP) components.

Our aim is to use tools from machine learning in order to systematically investigate ERP. More precisely, we focus on detecting cognitive components and localize them in time and space. The main issue is to deal with as few trials as possible.

II. Method

A. Current methods

Generally, ERP are assumed to be generated by a set of sources inside the brain. In order to extract these hidden sources, principal components analysis (PCA) and, more recently, independent components analysis (ICA) are two main approaches.

Both techniques project data on a lower dimensional space, representing the signals of hidden sources. Data records are a linear combination of these signals. The weights are given by a matrix which represents the contribution of each hidden source to each recorded channel. It may be considered as a kind of *activity map* w.r.t. the hidden sources.

Usually, these techniques are applied on a truncated dataset, within a specific time-window where ERP components are expected. It brings limitations since the time-window has to be known in advance. In addition, only one activity map and a fixed number of hidden sources are computed for the dataset.

B. Proposed probabilistic model

In order to overcome these limitations, we have developed a new technique relying on probabilistic inference. The goal is to extract the same kind of information as by conventional techniques, within several time windows, that are detected automatically. Thus, the activity map and the sources may change over time.

Our algorithm is based on a continuous hidden Markov model (HMM) while, in contrast to previous studies, here we propose to use probabilistic PCA analyzers instead of Gaussian components. The HMM segments time-series into time-windows. Each time-window is modeled by a probabilistic PCA analyzer.

This model has been set up within a Bayesian framework. Therefore, with appropriate prior probabilities, an automatic relevance determination (ARD) mechanism infers the number of PCA analyzers and, for each analyzer, the number of dimensions (i.e. the number of hidden sources) [1][2]. This approach tends to avoid overfitting. Moreover, The inference of the Bayesian model is conducted using the variational approximation [2].

III. Results

A. Synthetic data

A first experiment has been made on with 10-dimensional synthetic time-series. The model performed very well to isolate the different time-windows, i.e. the different states of the HMM.

B. BCI P300 dataset

The model was also used on a trial average of EEG records representing P300 evoked potentials [3].

As expected, the time-series was split into several parts. Interestingly, these parts were correlated to several known ERP components. Furthermore, the different activity maps extracted by each PCA analyzer correspond to possible activated areas on the scalp for the observed task.

IV. Conclusion

We propose a new probabilistic model combining two known approaches (HMM and PCA) in order to add the time information to the conventional PCA approach.

First results, on synthetic and real datasets, are promising. However, more tests have to be done on real datasets, in order to assess the correlation of inferred transitions and ERP components. Future work is also concentrated on the robustness of the model w.r.t. to the number of trials used.

V. Bibliography

[1] Bishop, C. Variational principal components. In Proceedings Ninth International Conference on Artificial Neural Networks, ICANN'99, Volume 1, pp. 509–514. IEE.

[2] Beal, M.J. Variational Algorithms for Approximate Bayesian Inference. PhD. Thesis, Gatsby Computational Neuroscience Unit, University College London, 2003

[3] Wolpaw, J. R., Schalk, G. and Krusiensk, D. Wadsworth. BCI Competition III, dataset II, “P300 speller paradigm”. 2004

On deterministic reservoir computing: network complexity and algorithm

H. Rostro-Gonzalez[1], B. Cessac[1,2], J.C. Vasquez[1] and T. Vieville[3]
[1]INRIA-Neuromathcomp research team, [2]LJAD-University of Nice-Sophia Antipolis and
[3]INRIA-Cortex research team
hrostro@sophia.inria.fr

Keywords: Reservoir computing, Computing with spikes, Discretized integrate and fire neuron models and Spiking neural networks.

I. Introduction

E consider a deterministic case of reservoir computing W[2] with spiking neurons (e.g. gIF [1]) with connection weights and delays. The purpose is to study a class of algorithmic methods able to calculate the proper parameters (weights and delayed weights) allowing the exact reproduction of a spike train produced by an unknown neural network.

II. Methods

The problem is known as NP-hard when delays are to be calculated. We propose here a reformulation, now expressed as a Linear-Programming (LP) problem, thus allowing to provide an efficient resolution. It is clear that this does not change the maximal complexity of the problem, whereas the practical complexity is now dramatically reduced at the implementation level.
More precisely we make explicit the fact that the back-engineering of a spike train (i.e., finding out a set parameters, given a set of initial conditions), is a Linear (L) problem if the membrane potentials are observed and a LP problem if only spike times are observed, for a gIF model. Numerical robustness is discussed. We also explain how it is the use of a generalized IF neuron model instead of a leaky IF model that allows to derive this algorithm.
Furthermore, we point out how the L or LP adjustment mechanism is distributed and has the same architecture as a "Hebbian" rule. A step further, this paradigm is easily generalizable to the design of input-output spike train transformations.

III. Results

Numerical implementations are proposed in order to verify that is always possible to simulate a expected spike train. The results obtained shows that this is true, expect for singular cases.
In a first experiment, we consider the linear problem and use the singular value decomposition (SVD) mechanism in order to obtain a solution, in the least-square sense, from the observation of spikes and membrane potential, allowing also to better understand the geometric nature of the problem.
When the aim is to find the proper parameters from the observation of spikes only, we consider the related LP problem and the numerical solutions are derived thanks to the well-established improved simplex method as implemented in GLPK library.
Several variants and generalizations are carefully discussed showing the versatility of the method.

IV. Discussion

The neural network model parameters learning is a complex issue. In biological context, this learning mechanisms mainly related to synaptic weights plasticity and as far as spiking neural network are concerned STDP [3].
In the present study, the point of view is quite different since we consider supervised learning, in order to implement the previous capabilities. To which extends we can "back-engineer" the neural network parameters in order to constraint the neural network activity is the key question addressed here.

V. References

[1] B. Cessac and T. Vi'eville. **On dynamics of integrate-and-fire neural networks with adaptive conductances**. Frontiers in neuroscience, 2(2), jul 2008.
[2] B. Schrauwen, D. Verstraeten, J. Van Campenhout, **An overview of reservoir computing: theory, applications and implementations**, in: M. Verleysen (Ed.), ESANN'2007, Advances in Computational Intelligence and Learning, 2007.
[3] S. M. Bohte and M. C. Mozer. **Reducing the variability of neural responses: A computational theory of spike-timing-dependent plasticity.** Neural Computation, 19(2):371–403, 2007.
[4] P. Baudot. **Nature is the code: high temporal precision and low noise in V1**. PhD thesis, 2007.

Partially supported by the CONACYT and the ANR MAPS & the MACCAC ARC projects.

Role of dopamine for long-term plasticity in the rat prefrontal cortex: a computational model.

Denis Sheynikhovich[1], Satoru Otani[2], and Angelo Arleo[1]
[1]CNRS-UPMC Univ Paris 6, UMR7102, F-75005 Paris, France
[2]Laboratory of Psychopatology of Central Nervous System Diseases,
INSERM U952 | CNRS-UMR7224, Univ Paris 6, F-75005, Paris, France
denis.sheynikhovich@upmc.fr

***Keywords* — computational model, dopamine, long-term potentiation, prefrontal cortex.**

I. Introduction

The prefrontal cortex (PFC) is thought to mediate executive functions, including strategic organization of behavior [1]. These functions rely on long-term plasticity within the PFC [1,2]. Dopamine (DA) input to the PFC has been shown to modulate neural activity in this area and affect long-term plasticity [3,4].

DA signaling in the PFC occurs in two distinct modes. Transient or *phasic* release of DA is caused by a short-term activation of dopamine cell firing in response to behaviorally relevant stimuli. The phasic DA is important for encoding of reward signals, and long-term potentiation. In contrast, background or *tonic* dopamine is characterized by an ambient concentration of dopamine within the PFC, which is not directly related to momentary sensory stimuli. Tonic DA is thought to exhibit control over higher cognitive functions mediated by the PFC. Several computational studies have addressed the roles of tonic *vs* phasic DA for short-term working memory during delay-period activity [5]. The present theoretical study is focused on the reproduction of *long-term* effects of DA signaling in the PFC, including long-term potentiation and depression (LTP/LTD) [3,4].

II. Computational model of dopamine influence on prefrontal cell activity

Using a detailed computational model of a single pyramidal cell in layer V of rat PFC, we study the neuronal properties that may be responsible for the changes in synaptic efficacy following tetanic stimulation in the presence of DA [4]. Our model is based on the Hodgkin-Huxley formalism for spike generation and includes 6 ionic conductances, excitatory and inhibitory synapses. We fit the properties of the model neuron to the electrophysiological data provided by S. Otani and collaborators and determine the potential changes in the ionic and synaptic conductances and/or internal parameters of the neuron (e.g. Ca^{2+} concentration) that can eventually give rise to the observed changes in the EPSP slope [4]. The underlying hypothesis in this study is that phasic release of endogenous DA is necessary for the induction of long-term changes in synaptic efficacy, while the concentration of tonic DA determines the direction (i.e. LTP or LTD) of these changes (Fig. 1) [4]. The computational approach adopted in our study helps to determine changes in neuronal properties required by this hypothesis as well as predict the impact of this hypothesis on a network level.

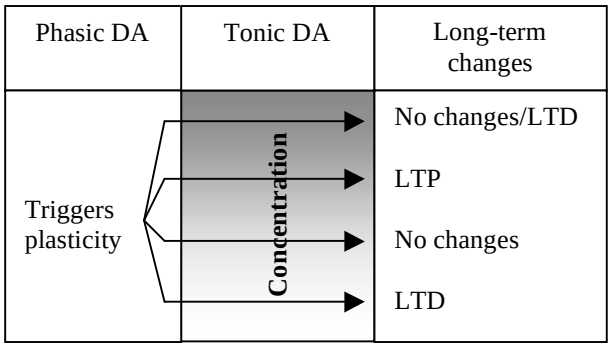


Fig. 1. Phasic *vs* tonic dopamine and synaptic plasticity [4].

III. References

1. Fuster JM (1995) Memory in the cerebral cortex. Boston: A Bradford Book, The MIT Press.
2. Touzani K, Puthanveettil SV, Kandel ER (2007) Consolidation of learning strategies during spatial working memory task requires protein synthesis in the prefrontal cortex. *Proc Natl Acad Sci USA.* 104:5632-5637
3. Matsuda Y, Marzo A, Otani S (2006) The presence of background dopamine signal converts long-term synaptic depression to potentiation in rat prefrontal cortex. *J Neurosci* 26:4803-4810
4. Kolomiets B, Marzo A, Caboche J, Vanhoutte P, Otani S (2009) Background dopamine concentration dependently facilitates long-term potentiation in rat prefrontal cortex through postsynaptic activation of extracellular signal-regulated kinases. *Cereb Cortex (E-pub ahead of print)*
5. Cohen JD, Braver TS, Brown JW (2002) Computational perspectives on dopamine function in prefrontal cortex. *Curr Opin Neurobiol.* 12:223-229

Head compensation reflexes in flying insects

Charles Thurat, Stéphane Viollet and Nicolas Franceschini

Equipe Biorobotique, Institut des Sciences du Mouvement,
UMR 6233, CP 938, 163 av de Luminy, 13288 Marseille
stephane.viollet@univmed.fr

Keywords: **gaze stabilization, oculomotor reflexes, flying insects**

I. IN-FLIGHT GAZE STABILISATION

DURING their locomotion, animals move their eyes and head for various reasons. Flies rotate both their head and body around the three axes: yaw, pitch and roll (fig.1). Several studies have shown that a freely flying fly exhibits pure translational phases lasting between 100 and 200ms, which are interspersed with sudden changes in trajectory (yaw saccades) (Collett & Land, 1975; Wagner, 1986; Schilstra & van Hateren, 1998). Such saccadic behaviour minimizes the time during which rotational optical flow (OF) occurs, at the benefit of the translational OF phases. A pure *translational* OF is a reliable sensory cue for insects to avoid obstacles (e.g., Wagner, 1986; Franceschini & al., 2007).

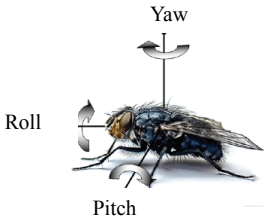


Fig. 1: Schematics of the three axes of rotation of the blowfly's thorax; the head can move independently about 3 rotational degrees of freedom as well. The arrows give the direction of positive angles used for the experiments.

II. HEAD STABILIZATION IN FLIES

The control of the blowfly's head orientation relies on no less than 23 pairs of muscles, and on several proprioceptive (visual, inertial and hair) sensors. There exists a typical head roll compensatory reflex in flies, which results from the fusion of visual and inertial signals (Hengstenberg & al; 1986). The inertial signals are delivered by the halteres, which act as genuine micro-rate gyrometers, able to measure the animal's rotational speed around the three axes (Nallbach, 1993).

III. GOAL OF THE STUDY AND METHOD

Our aim is to characterize and identify the *dynamic* properties of the head compensatory roll reflex in flies.

The set-up consists of a servomotor able to roll the insect accurately and reproducibly. The thorax is glued to a tiny patch of cardboard supported by a micro tweezers movable in X and Y by a tiny micromanipulator. The animal is illuminated frontally by a coaxial ring of infrared LEDs that are flashed in synchrony with the electronic shutter of a high speed camera (up to 500 f/s). The flying insect is then subjected to stepwise or sinusoidal rolls about the thorax axis, while the camera captures the orientation of an infrared reflecting marker (a narrow white line) painted on the insect's front. The whole apparatus is synchronized by an acquisition board controlled by a computer. Using a custom-made image-processing software, the successive frames acquired by the camera allow the angular orientation of the head to be followed and the frequency response of the head compensatory system to be determined.

A

IV. RESULTS

Stepwise rolls of the thorax in darkness were observed to induce conspicuous head rolls in the opposite direction, provided the insect was flying. We assessed the spatial and temporal resolution and the dynamics of this head compensatory reflex in the tethered flying blowfly in total darkness, to exclude any visual cues and to focus on the purely inertial part of the reflex. Fig. 3 shows some preliminary results that indicate that the fly compensate only partially the thorax movements of amplitude larger than 30 degrees, irrespective of their frequencies.

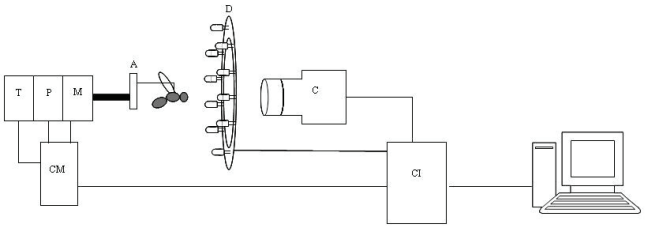


Fig.2: Roll generator inspired by Hengstenberg & Stange (1996). A, rotational axis with micromanipulator adjusted tweezers; C, infrared camera with IR filter; D, ring of IR-emitting LEDs; M, micromotor and reduction gear; P, potentiometer; T, tachometer; CM, motor control board ; CI, control board.

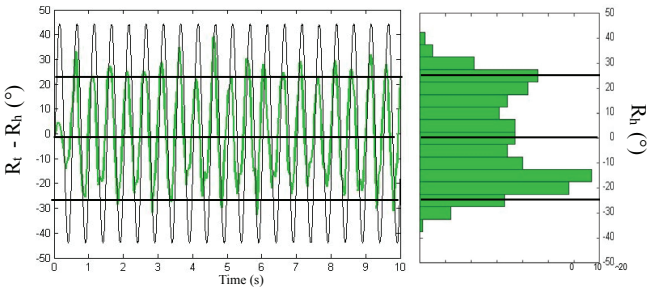


Fig.3: A, typical curve of the fly's response to a sinusoidal roll-stimulus in the dark. (Black curve) Sinusoidal rotational motion imposed on the thorax (R_t); (green curve) Angular position of the fly's head (R_h). B, distribution of head orientation (R_h) during the same experiment: the head stays within the +/-20° range despite the rotational movemement (perturbation) imposed to its body (amplitude peak-to-peak +/-45° and frequency 2Hz).

V. BIBLIOGRAPHY

• Collett T & Land, M., *J. comp. Physiol.* 99: 1-66 (1975)
• Franceschini & al., *Curr. Biol.* 17: 329-335 (2007)
• Hengstenberg R. & al. *Proc.. Roy. Soc. B*,: 455-482 (1986)
• Nallbach G., *J. comp. Physiol.* A 173: 293-300 (1993)
• Schilstra & Van Hateren, *Nature,* 395: 654 (1998)
• Stange, G. & Hengstenberg, R. *J. Neurosci. Meth.* 64: 209-218 (1996)
• Wagner, H., *Phil. Trans. R. Soc. Lond.* B, 312: 553-579 (1986)
• Zeil & al., *Curr Biol* , 18: R320-R323 (2008)

Gibbs Distributions and Spike-Time Dependent Plasticity

Juan C Vasquez[1], Bruno Cessac[1,2], Horacio Rostro-Gonzalez[1], Thierry Vieville[3]

1 NEUROMATHCOMP, INRIA Sophia-Antipolis ;

2 Laboratoire J.A Dieudonné, U. de Nice-Sophia ; 3 CORTEX, INRIA-LORIA

I. INTRODUCTION

Recently we have shown how Gibbs distributions are natural probability measures to describe the statistics of spike trains, given the data of known empirical averages and, how a Gibbs distribution may arise when considering 'slow' synaptic adaptation rules[1]. Here we illustrate numerically these results in the case of a recurrent neural network with discrete time current based dynamics under the action of a STDP rule.

II. THEORY AND METHODS

Neuron/Network Model: We consider a simple implementation of the leaky Integrate and Fire model, where time has been discretized. Call V_i the membrane potential of neuron i. Fix a firing threshold $\theta > 0$. Then dynamics is given by:

(1)
$$V_i(t+1) = \gamma V_i(t)\,(1 - Z[V_i(t)]) + \sum_{j=1}^{N} W_{ij} Z[V_j(t)] + I_i^e$$

$i = 1 \dots N$, where the "leak rate" $\gamma \in [0,1[$ and $Z(x) = \chi(x \geq \theta)$ where χ is the indicatrix function namely, $Z(x) = 1$ whenever $x \geq \theta$ and $Z(x) = 0$ otherwise. W_{ij} models the synaptic weight from j to i. The network is fully connected and weights are Gaussian.

STDP Model: Call $\omega_i(t) \in \{0,1\}$ the activity of neuron i at time t. Then, we consider the plasticity rule:

(2)
$$W'_{ij} = \epsilon \left[r_d W_{ij} + \frac{1}{T} \sum_{t=T_s}^{T+T_s} \omega_j(t) \sum_{u=-T_s}^{T_s} f(u)\omega_i(t+u) \right]$$

where $-1 < r_d < 0$ is a term corresponding to passive LTD, T a large time, corresponding to averaging spike activity for the synaptic weights update, and

(3)
$$f(x) = \begin{cases} A_- e^{\frac{x}{\tau_-}}, & x < 0, \quad A_- < 0; \\ A_+ e^{-\frac{x}{\tau_+}}, & x > 0, \quad A_+ > 0; \\ 0, & x = 0; \end{cases}$$

with $A_- < 0$ and $A_+ > 0$, is the STDP function as derived by Bi and Poo,1998. $T_s \stackrel{\text{def}}{=} 2\max(\tau_+, \tau_-)$ is a characteristic time scale.

Gibbs Distributions: We make the assumption that at each adaptation step, the time-empirical measure for the spike-statistics $\pi^{(T)}_{\tilde{\omega}^{(\tau)}}$ can be approximated by a Gibbs measure $\nu_{\boldsymbol{\psi}^{(\tau)}}$ with potential $\boldsymbol{\psi}^{(\tau)}$ and topological pressure $P\left[\boldsymbol{\psi}^{(\tau)}\right]$. Then, under small variations, this adaptation rule is *gradient system* for

(4)
$$\mathcal{F}^{(\tau)}_{\boldsymbol{\phi}}(\mathcal{W}) = P\left[\boldsymbol{\psi}^{(\tau)} + (\mathcal{W} - \mathcal{W}^{(\tau)}).\boldsymbol{\phi}(\mathcal{W}^{(\tau)})\right] - P\left[\boldsymbol{\psi}^{(\tau)}\right],$$

which asymptotically reachs its minimum corresponding to a *static distribution* for the synaptic weights, on a spike-statistics given by a *Gibbs distribution* with potential: (See references for details)

(5)
$$\Psi^{\text{STDP}} = \sum_{i=1}^{N}\sum_{j=1}^{N} \lambda_{ij} \sum_{t=0}^{n-1} \sum_{u=-T_s}^{T_s} f(u)\omega_j(t)\omega_i(u+t).$$

III. RESULTS

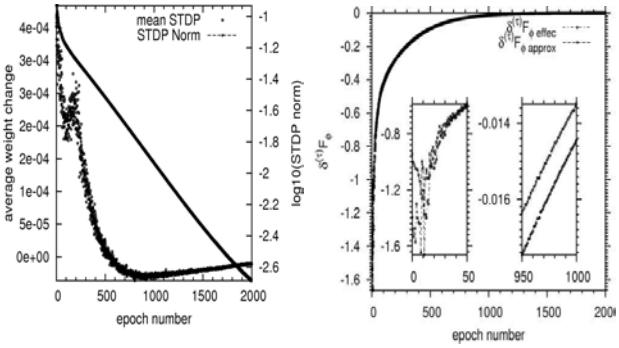


Figure 1: **(Left)** Evolution of the average synaptic modification (mean STDP) and of the Frobenius norm of the total change on the whole synaptic matrix (STDP norm). **(Right)**Evolution of the variation of the topological Pressure calculated by two estimation methods.

IV. CONCLUSIONS

This work shows: *(i)* that certain form of palsticity (STDP) are variational; *(ii)* that Gibbs distributions are natural candidates for modeling spike statistics. Note that the form (5) generalizes the form proposed in [2] under the name ofr Ising distribution.

Acknowledgments : Partially supported by the ANR MAPS and the MACCAC ARC projects.

VI. REFERENCES

1. B. Cessac et al. How Gibbs distributions may naturally arise from synaptic adaptation mechanisms, to appear in J. Stat. Phys. (2009)

2. Schneidman et al. Weak pairwise correlations imply strongly correlated network states in a neural population, Nature Vol. 440,(2006)

3. ENAS library, http://enas.gforge.inria.fr

Computation of neural illusions

Veltz Romain, Faugeras Olivier
NeuroMathComp-CERTIS
romain.veltz@sophia.inria.fr

***Keywords*— neural illusions, neural fields, bifurcations, numerical continuation**

I. Introduction

Everybody has ever seen visual illusions or movement illusions in which multiple interpretations can arise from a single stimulus. [1] is one of the first that gives a mathematical way to compute planforms (which are visual states) using bifurcation theory in very good agreement to what is reported by people experiencing planforms. These planforms result from the dynamics of the cortex when there are few cortical inputs. Here we address the question of the computation of these cortical states when a visual stimulus is applied. We will then interpret these multiple persistent states as neural illusions.

II. Neural field equations (NFE)

A. Introduction

The NFE modelling the evolution of the membrane potentials of the different populations of cortical columns are :

$$\frac{d}{dt}\mathbf{V}(\mathbf{r},t) = -\alpha\mathbf{V}(\mathbf{r},t) + \int_\Omega \mathbf{W}(\mathbf{r},\mathbf{r}')\mathbf{S}(\mathbf{V}(\mathbf{r}',t))\,d\mathbf{r}' + \mathbf{I}^{\text{ext}}(\mathbf{r})$$

where W is the connectivity matrix, S is a non-linear function of sigmoidal shape, I is the external input, the cortex Ω is supposed to be finite. Within this formalism, we can cast the equations of [1].

For any given initial cortical state , there exists a unique evolution V(t) satisfying the previous equations, which can converges to a persistent state.

Persistent states Vf are solutions of the previous equations not depending upon the time, hence satisfying

$$0 = -\mathbf{L}\cdot\mathbf{V}^f + \mathbf{W}\cdot\mathbf{S}(\lambda\mathbf{V}^f) + \mathbf{I}_{\text{ext}}$$

They have been studied in [2].

Neural illusions were analysed in [1] by varying a global weight μ in the description of the connectivity matrix W when I = 0. However we cope with a problem when I≠ 0 because in the formulation of the NFE, the ratio between I and W is not given. We decide to solve this problem by scaling I with ε and computing the persistent states Vf for different couples (μ,ε) using the multi-parameter numerical continuation library TRILINOS [3].

III. Persistent states calculus

We have to solve the persistent state equations numerically, hence we need to discretize the problem. Discretizing the space Ω will generate too many variables which will slow down the numerical continuation.

We choose a particular form of connectivity function whose range is finite dimensional, they are called Pincherle-Goursat kernels :

$$\mathbf{W}(\mathbf{r},\mathbf{r}') = \sum_{k,l=0}^{N-1} a_{kl} X_k(\mathbf{r}) \otimes Y_l(\mathbf{r}')$$

Their advantage is to directly look for persistents states of the form :

$$\sum_k v_k X_k$$

which proves to be useful when N is small. The numerical continuation also provides clues for the linear stability saying which cortical states are plausible.

IV. Conclusion

We believe that such computations could help to validate/invalidate the NFE modelling of cortical processes. The biggest challenge will be to reproduce some simple well-known neural illusions not involving higher area than V1.

V. Bibliography

[1] Bressloff, Cowan, Golubitsky, Geometric visual hallucinations, Euclidian symmetry and the functional architecture of striate cortex, The royal society 2001

[2] Faugeras & al Persistent neural states: stationary localized activity patterns in nonlinear continuous n-population q-dimensional neural networks, Neural

[3] http://trilinos.sandia.gov/

Delays in Neural fields equations

Veltz Romain, Faugeras Olivier
NeuroMathComp-CERTIS
romain.veltz@sophia.inria.fr

***Keywords*— delays, neural fields, stability**

I. Introduction

Delays are an important aspect in the modeling of cognition processes such as vision and motion analysis. Delayed continuum neural networks have been investigated by many authors [1,2], and more particularly the stability of persistent states. But all the given analytical bounds regarding this stability are independent of the delays. Here we present a new analytical bound involving the delays that is very easy to compute. We then compare this bound to the predictions of the linear stability analysis in numerical applications.

II. Delayed neural field equations (DNFE)

A. Introduction

The DNFE modelling the evolution of the membrane potential are :

$$\begin{cases} \dot{\mathbf{V}}(\mathbf{r},t) = \mathbf{L}\mathbf{V}(\mathbf{r},t) + \int_\Omega d\bar{\mathbf{r}}\mathbf{W}(\mathbf{r},\bar{\mathbf{r}})\mathbf{S}[\mathbf{V}\left(\bar{\mathbf{r}},t-\tau(\mathbf{r},\bar{\mathbf{r}})\right)] + \mathbf{I}(\mathbf{r},t), \quad t \geq 0 \\ \mathbf{V}(t) = \phi(t), \quad t \in [-\tau,\ 0] \end{cases}$$

where W is the connectivity matrix, S is a non-linear function of sigmoidal shape, I is the external input, L is a diagonal matrix describing the dynamics of a single cortical column. The delays are given by the function τ. The cortex Ω is supposed to be finite.

For any given initial cortical state , there exists a unique evolution V(t) satisfying the previous equations.

Persistent states Vf are solutions of the previous equations not depending upon the time, hence satisfying

$$0 = -\mathbf{L}\cdot\mathbf{V}^f + \mathbf{W}\cdot\mathbf{S}(\lambda\mathbf{V}^f) + \mathbf{I}_{\text{ext}}$$

They have been studied in [3].

B. Stability

The question addressed concerns the stability of the states Vf when we intoduce delays. If the following conditions (1-2 or 3) are satisfied, then Vf is stable :

1. $\sqrt{\tau}\|\tilde{\mathbf{W}}\tau\|_{\Omega^2} < \frac{1}{2}$
2. $\rho(\mathbf{L}^{-1/2}\tilde{\mathbf{W}}\mathbf{L}^{-1/2}) < 1$

or

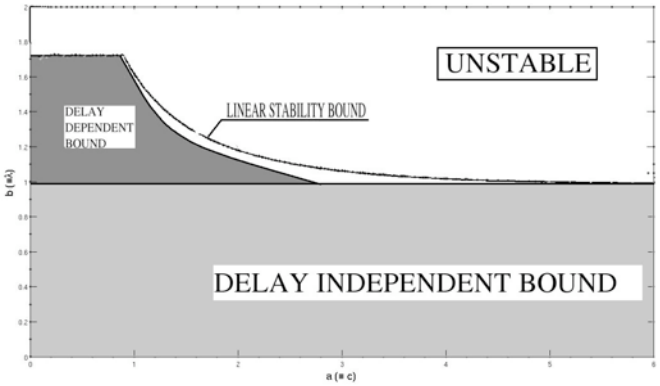
3. $\|\tilde{\mathbf{W}}\|_{\Omega^2} < \min_i \mathbf{L}_{ii}$

where ~W is a modified connectivity. Therefore, there are two integrals to compute. If we put a scaling in the delays, hence considering a delay term like c.τ(r,r'), we find the following power law for the stability :

$$c^{3/2} < Cte$$

III. Numerical application

We look at the following model with a “mexican hat connectivity” where w(x,y)=(1-|x|)exp(-|x|) with Ω=[-1,1] and compare the previous bounds to the (non analytical) results from linear stability.



We see that even if we still underestimate the stability, we have improved the previous results which gave the delay independent bound.

IV. Conclusion

These analytical bounds provide a quick way to analyse the complicated DNFE. When building a non-delayed model, we can easily have clues about the instabilities induced by the introduction of delays. Moreover the power law could be an intesting prediction for biological experiments.

V. Bibliography

[1] Atay & al Stability and bifurcations in neural fields with finite propagation speed and general connectivity, 2005

[2] Bressloff & al Nonlocal Ginzburg-Landau equation for cortical pattern formation, 2008

[3] Faugeras & al Persistent neural states: stationary localized activity patterns in nonlinear continuous n-population q-dimensional neural networks, Neural Computation 2009

Mechanical control of spontaneous activity in developing neural networks

Blaise Yvert, Claire Mazzocco, Adeline Langla, Sébastien Joucla, Oussama Abdoun, Pierre Meyrand
CNRS, Université Bordeaux 1 / Centre de Neurosciences Intégratives et Cognitives (UMR 5228)
b.yvert@cnic.u-bordeaux1.fr

***Keywords* — Maturation, neural networks, hindbrain, cortex, mouse, activity-dependent plasticity, mechanotransduction, microelectrode arrays.**

I. Introduction

SPONTANEOUS activity is a ubiquitous feature of developing neural networks, which has been described in many immature structures of the central nervous system (CNS), and shown to be essential for the maturation of functional connectivity (1–4). However, the origin of spontaneous activity remains largely unknown.

II. Results

Here, we show that a flow of artificial cerebrospinal fluid (aCSF) running over the inner ventricules or around the CNS controls the emergence of rhythmic activity in developing neural structures. This phenomenon was indeed observed in two different regions of the CNS.

First, whole embryonic mouse hindbrain-spinal cord preparations (embryonic stages E12.5-E16.5) were isolated on microelectrode arrays (MEAs). Spontaneous waves of activity originated in the hindbrain and propagated in the spinal cord under aCSF perfusion flow over the inner wall of the fourth ventricle (Figure 1). During development, the frequency of this rhythm increased, while the duration of activity episodes decreased. This rhythmic activity was observed with perfusion rate as low as a few tens of microliters per minute, and the frequency of the rhythm increased with the speed of the aCSF flow. However, at all stages of development considered, hindbrain activity was abolished as the perfusion was stopped.

Spontaneous rhythmic activity was also recorded in a second preparation. Embryonic or newborn (P0-P8) mouse cortices were isolated on MEAs or recorded extracellularly using glass pipettes and spontaneous activity was observed as perfusion flowed either over the inner wall of the lateral ventricle or around the meninges-removed cortex. Again, activity was abolished when the perfusion flow was stopped. This phenomenon, both in the hindbrain and the cortex, could not be attributed to changes in temperature, oxygenation level, pressure, or pH of the aCSF.

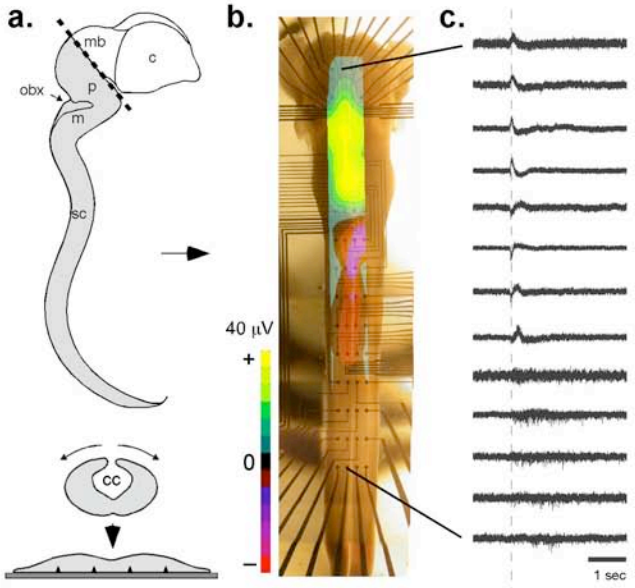


Figure 1. a) Schematics of the embryonic preparation layed on MEAs. b) Color-coded map of the peak of hindbrain activity with respect to the anatomy of a E14.5 preparation. c) Rostro-caudal wave of activity initiated in the hindbrain and propagating down the spinal cord.

III. Conclusion

Altogether, these results suggest that the movement of CSF in the ventricles and around the brain during development may control the genesis of spontaneous rhythmic activity in the developing CNS.

IV. Bibliography

1. Penn AA, Riquelme PA, Feller MB, Shatz CJ (1998) Competition in retinogeniculate patterning driven by spontaneous activity. Science 279:2108-2112.
2. O'Donovan MJ (1999) The origin of spontaneous activity in developing networks of the vertebrate nervous system. Curr Opin Neurobiol 9:94-104.
3. Ben-Ari Y (2001) Developing networks play a similar melody. Trends Neurosci 24:353-360.
4. Yvert B, Branchereau P, Meyrand P (2004) Multiple Spontaneous Rhythmic Activity Patterns Generated by the Embryonic Mouse Spinal Cord Occur Within a Specific Developmental Time Window. J Neurophysiol 91:2101-2109.

Support : French Ministry for Research and Technology (ACI, RMNT), ANR Blanc & TecSan, Fyssen, FRM, IRME, and Région Aquitaine (France).

Author Index

Notes

Notes

Notes