A first step towards in silico neuronal implementation of early-vision map

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I. INTRODUCTION

a) Position of the problem: High-level specification of how the brain represents and categorizes the causes of its sensory input [13], [9], [6] is a link between "what is to be done" (perceptual task) with "how to do it" (neural network calculation). More precisely [20], [18], a general class of cortical map computations can be specified representing what is to be done as optimization problems. One interest of optimization problems is the derivation of a wide range of applications such as image segmentation, optical flow estimation and winner take all mechanisms [1]. It has recently been shown how optimization problems with regularization mechanisms can be related to neural network dynamics [5], even in a rather general case (vector-valued cortical maps, non-linear processing, interaction between several maps, preliminary implementation on spiking networks) [12].

b) Biological plausibility of the framework: This framework has however never been experimented on a detailed biological plausible neuronal network à la Hodgkin-Huxley (HH), for two technical reasons. (i) The required computational resources is really heavy. (ii) At the present stage of the theory, the local Hebbian like adaptation rules do not precisely correspond to usual standard STDP mechanism in this context and several alternatives must be experimented to push the state of the art at this level [10].

Thus a very powerful analog simulator of HH neurons but also very versatile in terms of synaptic weights and axonal delay calculations is required. Such a platform is available thanks to the mixed analog/digital set-up developed by the IMS group. In this set-up, conductance-based and adaptive neural networks are computed in-silico on customized analog and digital integrated circuits [21]. The present preliminary study explores the feasibility of using this unique experimental tool to help understand the biological plausibility of the formal approach, with the hope of proving powerful compilation tools of early-vision functions on such emerging hardware.
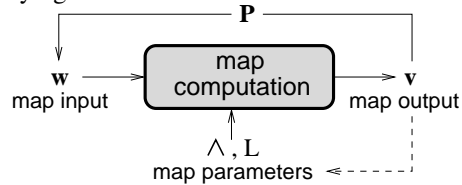
c) What is the contribution about: In the next section we briefly review the variational framework specifications, then present the in-silico simulation platform. We then describe the proposed experiments and conclude about what are the expected results.

II. SPECIFICATION OF A CORTICAL MAP COMPUTATION

Variational formulation

Given an input map $\mathbf{w}$, one look for an output map $\bar{\mathbf{v}}$ verifying

$$\bar{\mathbf{v}} = \underset{\mathbf{v} \in H/\mathbf{c}(\mathbf{v})=0}{argmin} \ \mathcal{L}(\mathbf{v}), \qquad (1)$$

with

$$\mathcal{L}(\mathbf{v}) = \int |\hat{\mathbf{w}} - \mathbf{w}|^2_{\mathbf{\Lambda}} + \int \phi(|\nabla \mathbf{v}|_{\mathbf{L}}) + \int \psi(\mathbf{v}), \quad (2)$$
$$\text{and } \hat{\mathbf{w}} = \mathbf{P}\,\mathbf{v}, \qquad (3)$$

where ∇ stands for the gradient operator, $\phi(\cdot)$, $\psi(\cdot)$, $\mathbf{P}$, $\mathbf{c}(\cdot)$, $\mathbf{\Lambda}$ and $\mathbf{L}$ are commented hereafter. The norms defined in (2) are weighted norms defined by $|\mathbf{u}|_{\mathbf{M}} = \mathbf{u}^T \mathbf{M} \mathbf{u}$, where $\mathbf{M}$ is a given symmetric positive matrix.

The first term in (2) is a fidelity attached term specifying how the output is related to the input, the second term is a smoothing term which defines the regularity of the output and the third term allows to constrain the form of the solution. The equation (3) defines the relation between the input estimation an the output. So the formulation (1)–(3) specifies the cortical map computation in the sense that it explains the "goal", what is to be done, but without any reference to how it is done.

The functions $\mathbf{\Lambda}$ define a so-called *measurement information metric* which represents the *precision of the input* (the higher this precision in a given direction, the higher the value of $\mathbf{\Lambda}$ in this direction) and allows to take into account (in a statistical framework, $\mathbf{\Lambda}$ corresponds to the inverse of a covariance matrix) *partial observations and missing data* (i.e., null precision)

The functions **L** define a *diffusion tensor* modulated by a function ϕ which controls the amount of smoothness required. Low variations, assumed to be "noise", are smoothed (using e.g., quadratic and isotropic smoothing for additive white noise), while high contrasts, assumed to be the "signal", are preserved (e.g. with diffusion only in the direction tangential to the edges). Furthermore, when a problem is ill-posed, adding some a priori on the smoothness of the solution regularizes the problem (e.g. with diffusion from well-defined values to undefined or ill-defined values).

Three kinds of constraints are introduced: *structural constraints* (via $c()$) , to define a nonlinear solution, i.e. to force the solution to belong to a manifold defined by implicit equations; *optimization constraints* (via $\psi()$) to control the form of the solution; *measurement constraints* between both the input and the quantity to estimate (via **P**) to obtain an unbiased estimation [19] in this non-linear case.

Such formalism is in direct link with generative models of the cortical areas processing, for instance in the Bayesian framework, the *a priori* information corresponding to the regularization and choice of constraints and the fidelity term being related to the conditional probability with respect to the input.

Partial differential implementation

The local solution of the previous criterion can be implemented -in the general case- using a network dynamics of the form of Cohen-Grossberg analog network, using linearized integral approximation of a diffusion operator introduced by Cottet, Degond and Mas-Gallic [7], [5], [8], [17]. More precisely, for a neuron of index i, we have

$$\frac{\partial v_i}{\partial t} = -\epsilon_i(v_i)\,v_i + \sum_j \sigma_{i,j}(v_i)\,v_j + \kappa_i\,w_i \quad (4)$$

with:

The term κ_i is a simple gain, while the corrective term $\epsilon_i(v_i)$ is a straight-forward but rather complex non-linear function of the criterion parameters. The term $\sigma_{ij}(v_i)$ corresponds to the synaptic weights, and a linear family of solutions is derived for a given diffusion tensor **L**.

Among those solutions, an optimal one (here the closest discrete approximation with respect to the continuous one, in the least-square sense, given a well-formed distance) is chosen. The obtained derivation turns out to be a short-term adaptive rule and the synaptic weights are related to the related diffusion operator using a Hebbian learning scheme [20]. This describes a biologically plausible mechanism of short-term adaptation [18] (see [20], [18] for details).

Here we are going to map (4) onto a HH spiking neuron state. We consider that v_i and w_i correspond to the last neuron spike with respect to a fixed clock at regular time T. This reference is either generated by an auxiliary neuron (as proposed in e.g. [14]) or related to competitive temporal coding as discussed in e.g. [16]. Here ϵ_i corresponds to the HH dynamics and is thus not derived from the specification.

III. MAPPING ON A HODGKIN-HUXLEY NEURAL NETWORK

Very briefly, the state of a HH neuron (e.g. [10]) is defined by a dynamics of the form:

$$\begin{cases} C\,\dot{V}(t) &= \sum_k \bar{g}_k\,\phi_k(t)^{\alpha_k}\,\psi_k(t)^{\beta_k}\,(V(t) - E_k) \\ &+\; I(t) \\ \tau_{k,\phi}\,\dot{\phi}_k(t) &= \phi_{k,\infty} - \phi_k(t) \\ \tau_{k,\psi}\,\dot{\psi}_k(t) &= \psi_{k,\infty} - \psi_k(t) \end{cases}$$

for the neuronal membrane potential V, input current I, activation ϕ and inactivation ψ fractions of either:
- the maximum conductance $\bar{g}_k$ available at any given time for voltage voltage-gated ion channels or
- the synaptic conductance $\bar{g}_k = w_{ij}\,f(V'(t), t)$ driven by post-synaptic potential induced by another neuron of membrane potential $V'(t)$.
The membrane leak is included in this equation for $\bar{g}_0 = w_{ii}, k = 0, \phi_0 = \psi_0 = 1$. The Nernst potential E_k is the ionic specie reversal potential, $\tau_{k,\phi}$ and $\tau_{k,\psi}$ time-constants and $\phi_{k,\infty}$ and $\psi_{k,\infty}$ steady state values. We write $\dot{V}(t) = F(V(t), t, \cdots)$ this dynamics in the sequel.

Here, we assume that each neuron is *in a periodic regime*, now viewed as interacting oscillator[1], as discussed in e.g. [3]. At the experimental level, the regime is going to be established for each neuron with weak synaptic couplings, considering in this context *regular spiking neurons*, and measuring the current input bounds which guaranty this regime to be preserved (it is known from the theory that this condition is stable). It is thus assumed that this regime is maintained at any time.

We can define an *instantaneous phase* v for each neuron, the phase reduction being formally defined by the partial differential equation:

$$\frac{\partial v(V(t))}{\partial t} = \frac{\partial v(V(t))}{\partial V}\,F(V(t), t, \cdots) \quad (5)$$

(see e.g. [3] for a development). The periodic current input $I(t)$ is similarly related to its instantaneous phase w.

In practice, between the occurrence of two consecutive spikes, after the spike of index n,

$$\dot{v}(t) \simeq \frac{2\Pi}{t_n - t_{n-1}}, t_n \leq t < t_{n+1}$$

is going to be measured as the inverse of the instantaneous period. Please note that we do not use the instantaneous firing rate as coding variable but the instantaneous phase, the former being the temporal derivative of the latter.

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[1]This view is not to be mixed with other mean-field coupled oscillator models, e.g. [15]

With this setting, equation (4) simply represents the *1st order expansion of the instantaneous phase equation*, with the following correspondence from the previous setting:

$$
\begin{aligned}
\kappa_i &= \nabla v/C \, \frac{\partial I(w(t))}{\partial w} \\
\sigma_{i,j} &= w_{i,j} \nabla v/C \, \phi_k^{\alpha_k} \, \psi_k^{\beta_k} \, \frac{\partial f(V_j(v_j))}{\partial v_j} \\
&= w_{i,j} \, \lambda_{ij} \\
\epsilon_i &= -\nabla v/C \left[w_{i,i} + \sum_{k>0} \bar{g}_k \, \phi_k^{\alpha_k} \, \psi_k^{\beta_k} \right] \\
&= -[w_{i,i} + \mu_i] \, \lambda_{ii}
\end{aligned}
\tag{6}
$$

the time dependency being dropped. Here the neuronal leak is used to control the corrective term; it is trick in this preliminary set-up, whereas in truth more plausible mechanisms (e.g. the global conductance state) are likely in action, but with an equivalent effect in this case.

At the experimental level, the quantities κ_i, λ_{ij}, μ_i and λ_{ii} are estimated in a given context of periodic activity. So that it is then possible to adjust the synaptic weight to realize the given function in this regime.

IV. PRESENTING THE HARDWARE SIMULATION PLATFORM

The set-up we designed is dedicated to the temporal simulation of spiking neural networks. It is architectured to process in biological real-time adaptive neural networks of conductance-based models of neurons. Computation is distributed on analog hardware (custom-made integrated circuits), digital hardware (configurable logic devices) and software. Therefore, the simulation platform is organized in 3 layers, as shown in figure 1.
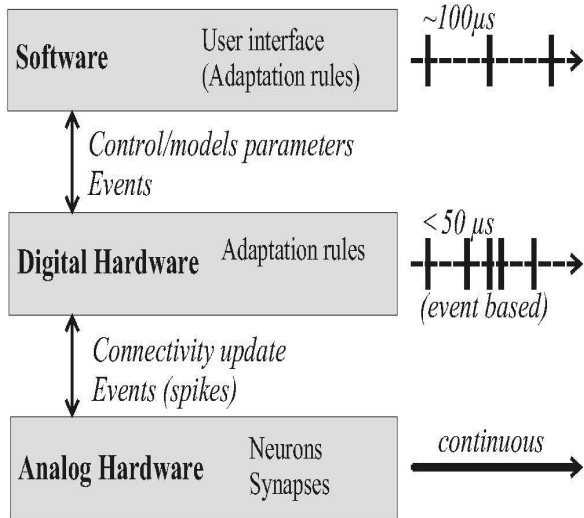

Fig. 1.   Architecture of the simulation platform.

The analog hardware layer comprises full custom specific integrated circuits (ASICs), that were designed using a proprietary library of electronic modules [2]. The ASICs run in continuous and real-time the computation of the neurons and synapses ionic currents. Neurons can be modeled by up to 5 ionic conductances, following the HH formalism. Each mathematical function appearing in the neuron model corresponds to an analog module in the library, with inputs for tunable parameters, stored on-chip in analog memory cells. The ASICs output the neural spikes as time-stamped events. They receive pre-synaptic events as binary pulses, which length encodes the synaptic strength. The digital hardware layer is in charge of the neural network connectivity: it processes spike events from the analog neurons, and computes the synaptic weights, applied back on the ASICs. The adaptation rules of the network, that dynamically influence these weights, are computed by the FPGAs of the digital layer, provided that the computation respect the real-time constraints: in the current configuration of the platform, each synaptic weight has to be refreshed every 50 μs. Predefined stimulation patterns can also be applied to individual neurons; such patterns are useful to emulate background cortical activity. The third layer includes the software driver and interface, in charge of controlling the data bi-directional transfer to the software via a PCI bus. The personal computer runs a real-time operating system. It is available to host software functions that compute adaptive connectivity functions that would not be supported by the FPGAs. The software also includes user interface functions to control (off-line and on-line) the simulation parameters and results.

V. EXPERIMENTING 1D NON-LINEAR DIFFUSION

In order to show the interest of the previous platform, let us revisit an edge-preserving smoothing approach proposed by Cottet and Ayyadi [5] which corresponds to the framework presented in this paper, here in 1D. Here, (2) corresponds to a 1D non-linear diffusion, depending on the norm of the gradient of the intensity. The smoothing term infers two kinds of behaviors:

- For low contrasts, the smoothing term is quadratic which corresponds to an isotropic smoothing.
- For high contrasts, the smoothing term vanishes in order to preserve edges.

and considering a simple nearest-neighbor connectivity as illustrated in Fig. 2 we can directly write:

$$
\begin{aligned}
w_{i,i} &= \gamma[w_{i,i-1} + w_{i,i+1}] - \mu_i \\
w_{i,i-1} = w_{i,i+1} &+= \psi(v_{i+1} - v_{i-1})
\end{aligned}
\tag{7}
$$

with $\psi(t) = \alpha \, \mathrm{H}(2 - |t|) \, (1 + |t|/\beta) \, \frac{(2 - |t|/\beta - 2)^2}{4}$ where κ (the input gain), α, β and γ are tunable parameters. Here the $\psi(t)$ profile is not critical, providing it is

a sigmoid like profile[2] (we write H(t) the Heaviside function).

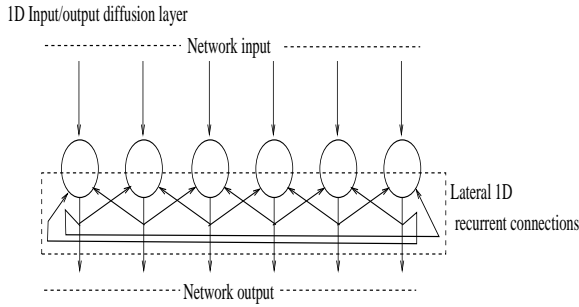1D Input/output diffusion layer

Fig. 2. Schematic representation of the experimental network. Each input is combined with local nearest-neighbor left-right diffusion, to drive the network output.

In other words, this mechanism is designed to weakly poly-synchronize the neurons as observed in simulated or biological neural networks [11], [4]. s

Such a mechanism has already been experimented as reported in [12] (see Fig. 3) considering a software simulation of a restrained spiking neuronal network, while in the present study we plan to experiment it on biological plausible HH neurons, where the membrane potential dynamics is explicitly considered.
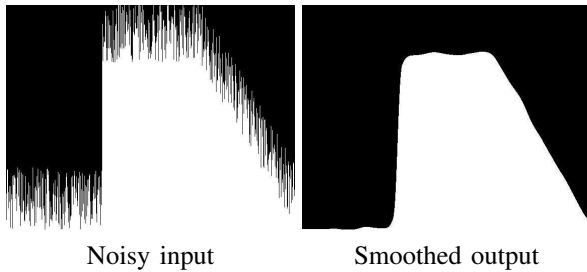

Noisy input   Smoothed output

Fig. 3. Example of 1D implementation of anisotropic edge-preserving smoothing using a spiking network, from [12].

The proposal is to implement mechanisms for different families of parameter and eventually different weight adjustment profiles.

VI. CONCLUSION

We propose to experiment how non-linear diffusion processes which allow to implement rather sophisticated visual functions as derived from a variational approach can be mapped onto a biologically plausible HH neuronal network. To attain this goal the in-silico implementation of such a network is the optimal tool to experiment many variant of such mechanisms and to understand how the non-linear diffusion parameters map onto the neuronal dynamics.

It must be clear that this preliminary proposal of such implementation is to be tested, adapted and

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[2]typically with:
$\psi(0) = \alpha, \psi'(0) = 0, \psi(+\infty) = 0, \psi(\beta) = \alpha/2, \psi''(\beta) = 0$.

validated on the network. Here we are in a situation where the neuronal dynamics is complicated enough for analytical methods to be virtually usable. Therefore, the experimentation is mandatory, as proposed here.

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