

On the Neurohydrodynamics of Neural Networks

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Abstract

Inspired by De Broglie's pilot wave interpretation of Quantum Mechanics and the subsequent development of Bohm's Quantum Hydrodynamics, we propose a model for the dynamics of a neural network with reaction-diffusion processes described by a modified set of Cohen-Grossberg equations, which we call Neurohydrodynamics. In this approach, a pilot wave interpretation deterministically guides the dynamics of a neural network through the neuropotential that arises biologically from reaction-diffusion processes at the synapses of real neurons. We demonstrate that the neuropotential provides a new type of reinforcement learning useful for characterizing short-term memory and pattern formation in neural networks, and we compare our results to more traditional reinforcement learning methods through an example. Finally, we discuss extending our approach to include learning, memory, cognition and decision-making processes of the mammalian brain that are often modeled by neural networks.

Keywords: Reaction-diffusion, Quantum Hydrodynamics, Cohen-Grossberg equations and neural networks.

1. Introduction

In the famous Solvay Congress of 1927, De Broglie proposed a pilot wave interpretation of Quantum Mechanics but quickly abandoned his approach under severe criticism. About 50 years later, Bohm proposed Quantum Hydrodynamics ([3], [4]), the first complete theory of quantum mechanics, that exploited De Broglie's ideas. Quantum Hydrodynamics ([19]) and the neurodynamics of real neurons in the mammalian brain both involve reaction-diffusion

processes, and should share similar philosophical consequences for self-guidance. More generally, Turing proposed the reaction-diffusion theory of morphogenesis ([20]) that was able to account for pattern formation in chemical and biological processes. It should come as no surprise that the reaction-diffusion processes of real neurons have been shown to aid in pattern formation while exhibiting self-organization, and play a key role in controlling the neurodynamics of the mammalian brain important to perception, attention, memory, learning, and cognition ([1], [2], [7], [9], [12], [14], [15], [16], [18] [23]). Even Artificial Intelligence uses the notion of an "oracle" to instantaneously specify which choices will lead to a solution of a particular problem in a reasonable amount of time, and thereby solving the problem of computational complexity ([11]).

With our motivation established, we propose a mathematical theory of Neurohydrodynamics, and we provide a proof-of-principle example using Hopfield's two neuron model ([10]) with one dimensional diffusion. Finally, we discuss the implications of Neurohydrodynamics to the cognitive processes of memory, learning and decision-making in the mammalian brain.

2. Neurohydrodynamics

In order to develop the foundations of Neurohydrodynamics, we assume the dynamical system perspective given by the Cohen-Grossberg equations ([6]) with the addition of the natural reaction-diffusion processes occurring at the synapse of real neurons. It is well known that the reaction term captures the competition among the stored representation resulting in the selection of only one representation ([23]). Whereas the diffusion term evaluates the similarity of each stored representation to an input with the compari-

son executed locally on each representation. Thus, the Neurohydrodynamics have a wholly deterministic nature that depends stochastically on the local dynamics of individual neurons resulting in some very interesting consequences for the neurodynamics of a neural network.

Let $u_j(\mathbf{x}, t)$ denotes a continuous activation wave over space and time for the j^{th} neuron. Then the equations of motion for a population of n neurons with diffusion can be written as

$$\frac{\partial u_j}{\partial t} = a_j(u_j) \left[b_j(u_j) + \sum_{k=1}^n c_{jk}(\mathbf{x}, t) d_k(u_k) \right] + \mu_j \nabla \cdot [e_j(\mathbf{x}, t) \nabla u_j(\mathbf{x}, t)],$$

where μ_j is the diffusion coefficient of the j^{th} neuron and ∇ is the gradient of a scalar function $u_j(\mathbf{x}, t)$. Suppose that the solution is written in polar form as $u_j(\mathbf{x}, t) = R_j(\mathbf{x}, t) e^{iS_j(\mathbf{x}, t)}$ where $R_j(\mathbf{x}, t)$ and $S_j(\mathbf{x}, t)$ are real functions, $i = \sqrt{-1}$ is the imaginary unit and $\mathbf{x} = \langle x, y, z \rangle$ is a position vector. Upon substitution with $e_j(\mathbf{x}, t) = 1$, we obtain the following equations of motion for the Neurohydrodynamics of a neural network.

$$\frac{1}{R_j(\mathbf{x}, t)} \frac{\partial R_j(\mathbf{x}, t)}{\partial t} = \frac{Re\{f_j(u_j, \mathbf{x}, t)\}}{R_j(\mathbf{x}, t)} + \mu \frac{\nabla^2 R_j}{R_j(\mathbf{x}, t)} - \mu \left[\left(\frac{\partial S_j}{\partial x} \right)^2 + \left(\frac{\partial S_j}{\partial y} \right)^2 + \left(\frac{\partial S_j}{\partial z} \right)^2 \right]$$

$$R_j(\mathbf{x}, t) \frac{\partial S_j(\mathbf{x}, t)}{\partial t} = Im\{f_j(u_j, \mathbf{x}, t)\} + \mu R_j(\mathbf{x}, t) \nabla^2 S_j + 2\mu \left(\frac{\partial R_j}{\partial x} \frac{\partial S_j}{\partial x} + \frac{\partial R_j}{\partial y} \frac{\partial S_j}{\partial y} + \frac{\partial R_j}{\partial z} \frac{\partial S_j}{\partial z} \right)$$

where

$$f_j(u_j, \mathbf{x}, t) = a_j(u_j) e^{-iS_j(\mathbf{x}, t)} \cdot \left[b_j(u_j) + \sum_{k=1}^n c_{jk}(\mathbf{x}, t) d_k(u_k) \right],$$

and $Re\{f_j(u_j, \mathbf{x}, t)\}$ and $Im\{f_j(u_j, \mathbf{x}, t)\}$ are the real and imaginary parts of $f_j(u_j, \mathbf{x}, t)$, respectively. Physically, the real functions $R_j(\mathbf{x}, t)$ and $S_j(\mathbf{x}, t)$ contain information about the magnitude and phase of the activation wave of the neuron distributed over time and space, respectively. Analogous to the quantum potential in Quantum Hydrodynamics, we define the neuropotential as

$$N_j(\mathbf{x}, t) = \mu_j \frac{\nabla^2 R_j}{R_j(\mathbf{x}, t)},$$

which provides guidance of the reaction-diffusion process during the evolution for the neurodynamics of the neural network. The neuropotential provides an organization of spatial and temporal neuronal patterns in the evolution of the neural network. It is important to remark that the activation waves interact producing a spatial and temporal pattern determined by the neuropotential, and not by the interference of the activation waves ([19], [22]). Finally, we incorporate reinforcement learning into Neurohydrodynamics with the following equation of motion for the weights.

$$\frac{dc_{jk}}{dt} = -\eta_{jk} \frac{dN_k}{dt} \int_{t_i}^{t_f} \nabla_{c_{jk}} N_k(c_{jk}, t) e^{\lambda \tau} d\tau,$$

where λ is the learning rate, η_{jk} is a constant and $\nabla_{\mathbf{x}} N_k(c_{jk}, \tau)$ denotes the spatial gradient of the weights that is related to $N_k(\mathbf{x}, t)$ by the chain rule. The neuropotential can be viewed as a measure of the "internal stress" of the activation wave of a neuron. Under a scale change of the amplitude, the neuropotential is independent of the activation wave's height, and thus only depends on the shape of the activation wave. Even though the neuropotential is time dependent, contextuality is introduced into the neurodynamics since the neuropotential remembers its initial conditions. If other potentials were present, such as a thermal potential, these would be counterbalanced by the neuropotential as the system reached equilibrium. The most significant feature of Neurohydrodynamics is that every neuron depends on other neurons with all of them subject to organization by the whole.

3. Numerical Simulations

In this section, we provide a simple example of a one dimensional model for two Hopfield neurons ([10]) with diffusion, which demonstrates some of the key features of the neurohydrodynamics. Once we discretize these equations, one can implement Euler's scheme in a simulation ([22]) to examine the consequences of the neurohydrodynamics for Hopfield's two neuron model with diffusion.

Hopfield's two neuron model with $j, k = 1, 2$ such that $j \neq k$ is described by the following set of Neurohydrodynamic equations.

$$\frac{\partial R_j(x, t)}{\partial t} = Re\{f_j(u_j, x, t)\} + \mu \nabla^2 R_j(x, t) - \mu R_j(x, t) \left(\frac{\partial S_j}{\partial x} \right)^2$$

4. Psychological implications of neurohydrodynamics

$$\frac{\partial S_j(x, t)}{\partial t} = \frac{Im\{f_j(u_j, x, t)\}}{R_j(x, t)} + \mu \nabla^2 S_j + \frac{2\mu}{R_j(x, t)} \left(\frac{\partial R_j}{\partial x} \frac{\partial S_j}{\partial x} \right)$$

$$f_j(u_j, x, t) = 2\alpha c_{jk} e^{-iS_j} \tan^{-1} (\pi\gamma R_{k,t} e^{iS_k} / 2) - \beta R_j$$

$$\frac{dc_{jk}}{dt} = -\eta_{jk} \frac{dN_k}{dt} \int_{t_i}^{t_f} \nabla_{c_{jk}} N_k(c_{jk}, t) e^{\lambda\tau} d\tau$$

On any spatial interval, all amplitudes vanish except for the two neurons located at their respective positions, which have a prescribed amplitude. All phases are random numbers from the interval $[-\pi, \pi]$. Let $\delta = \mu\Delta t / \Delta x^2$. Upon application of Euler's scheme over some prescribed time step, we obtain the following set of discretized equations for Hopfield's two neuron model.

$$\begin{aligned} R_{j,t+1} &= R_{j,t} - \beta R_{j,t} \Delta t \\ &+ 2\alpha c_{jk} Re \left\{ e^{-iS_{j,t}} \tan^{-1} (\pi\gamma R_{k,t} e^{iS_{k,t}} / 2) \right\} \Delta t \\ &+ \delta (R_{j,x+1} - 2R_{j,x} + R_{j,x-1}) \\ &- \delta R_j (S_{j,x+1} - S_{j,x})^2 \end{aligned}$$

$$\begin{aligned} S_{j,t+1} &= S_{j,t} + \delta (S_{j,x+1} - 2S_{j,x} + S_{j,x-1}) \\ &+ \frac{2\delta}{R_j} [(R_{j,x+1} - R_{j,x})(S_{j,x+1} - S_{j,x})] \\ &+ \frac{2\alpha c_{jk}}{R_{j,t}} Im \left\{ e^{-iS_{j,t}} \tan^{-1} (\pi\gamma R_{k,t} e^{iS_{k,t}} / 2) \right\} \Delta t \end{aligned}$$

$$N_k = \frac{\mu_k}{R_k(\mathbf{x}, t)} \left(\frac{R_{k,x+1} - 2R_{k,x} + R_{k,x-1}}{\Delta x^2} \right)$$

$$\begin{aligned} c_{jk,t+1} &= c_{jk,t} - \eta_{jk} (N_{k,t+1} - N_{k,t}) \\ &\cdot \sum_{k=1}^t e^{\lambda k \Delta t} \left(\frac{N_{k,x+1} - N_{k,x}}{c_{jk,x+1} - c_{jk,x}} \right) \Delta t \end{aligned}$$

In [24], this system can achieve stability with both neuron's activation wave decaying exponentially in time while spatially broadening. Hopfield's two neuron model is well known to decay exponentially with time. It is the neuropotential that guides the whole neural network along with neuron interdependence. To accomplish guidance, the weights undergo reinforcement learning using gradient descent along maximum changes of the neuropotential. It is important to realize that the resulting pattern of activation is formed by the neuropotential, and not by the interference of activation waves.

In Neurohydrodynamics, competition between sensory representations is described by the reaction terms with the similarity between a sensory representation and a representation in memory described by the diffusion term. The "pilot wave" guides the dynamics of a neural network through the neuropotential that arises biologically from the reaction-diffusion processes at the synapses of real neurons. For example, the striatum is well known to have extensive arborization of dendrites and axons creating a network of distance dependent laterally inhibited neurons ([21]). Thalamic disinhibition of sensory information occurs when striatal neurons inhibit the GPi permitting the cortex to engage in an action selected by the striatal neurons. Hence, the cortical-striatal loop through the thalamus clearly plays a role in the motor-sensory decision-making processes utilizing Reinforcement Learning ([5], [21]). In the acquisition of a motor sequence ([8]), simultaneous activity is observed in the cortico-striatal and cortico-cerebellar systems. Both are active during the early stages of learning but a decrease in the activity of the cortico-cerebellar system occurs with practice. Then when performance is achieved the cortico-striatal system remains active suggesting that this region is critical for long-term retention of practiced movements.

Another cortico-strio-pallidal systems involves the association cortex, the nucleus accumbens, amygdala which are responsible for the higher-order decision-making processes of the mammalian brain. Here the regulation of neuromodulators, such as dopamine or serotonin, present at the synapse of neurons, are among the most active in those brain structures responsible for fear and anxiety ([13]). Dopaminergic transmission in the nucleus accumbens septi have been shown to be important for maintaining responses in conditions of intermittent reward. Even the basolateral amygdala seems to have the ability to pair stimuli with reward when the primary reward has occurred in the remote past ([17]). The association cortices are implicated in executive control and is reciprocally connected to the hippocampus, the site of working or short-term memory. The hippocampus is known to be strongly engaged in early training but it has little effect in the latter stages of learning. On the other hand, the medial prefrontal cortex (mPFC) has been shown to have a profound effect on the latter stages of learning but little effect on the earlier stages of learning. Hence, these structures are complementary in function by virtue of their intervention in the learning process albeit at different times. We conjecture that the slow reaction-diffusion processes of the mPFC may explain its executive function and involvement in the latter stages of learning.

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