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Remarks on Neurodynamics: Spikes, Spike Synchronization, and Spike-Timing-Dependent Plasticity

March 25, 2009

Slides for this talk:

`http://www.cs.brandeis.edu/~bukatin/neuro-talk-mar-25-2009.pdf`

This is a review talk.

Part I. Selected recent results.

Part II. Transition from models of biological neural systems based on firing rates to models based on exact timing of spikes, spike synchronization and neural oscillations, synchronicity detection, and spike-timing-dependent plasticity.

Part III. Can we use this transition to improve artificial neuromorphic systems? Can we use this transition to improve our understanding of higher cognitive functions?

Part I. Selected recent results.

Our understanding of the field changes quite rapidly.

Interpreting fMRI data:

Yevgeniy B. Sirotin, Aniruddha Das (2009). *Anticipatory haemodynamic signals in sensory cortex not predicted by local neuronal activity.* Nature, **457**, 475-479.

(issue: January 22, 2009)

Aniruddha Das, Center for Neurobiology and Behavior Columbia University, gave a talk at MIT on 18 April 2008:

Entraining to Task Expectation in Primary Visual Cortex.

”In functional brain imaging the underlying hemodynamic signals are assumed to reflect cortical metabolic demand driven by local neuronal responses. To test this assumption we developed a dual-wavelength optical imaging technique that simultaneously measures cerebral blood volume and blood oxygenation, continuously, in alert behaving monkeys. Using this technique, we have discovered a novel cortical blood flow signal that appears to be driven by task timing, independent of local neuronal responses. We see this signal in primary visual cortex (V1) of monkeys engaged in periodic fixation tasks, even in total darkness, with little or no measurable underlying neuronal activity. The entrainment to trial period suggests anticipatory timing in the brain.

Given a predicted event, fresh arterial blood appears to be pumped in, in anticipation of the upcoming event, before any spiking driven by the event. This signal could form part of a preparatory mechanism in the brain that is engaged by any periodic or predictable task. It could be governed by a central timing mechanism and mediated through neuromodulatory control of cortical blood flow, independent of local neuronal spiking and related metabolic demands."

At 9th International Conference on Cognitive and Neural Systems (2005), Boston University, Mark Bear from MIT gave a talk "How monocular deprivation shifts ocular dominance in visual cortex". Among other things they demonstrated in mice, that if the retina is chemically turned off (instead of just stiching the eyelid together), the depression of neuronal response in the cortex **does not occur**. So the depression in this case **is not caused by "the lack of use"**, as it is customary to believe, but by **the retinal noise from the eye which is shut.**

The retina of the closed eye still works, and the noise coming from it convinces the downstream cells, that the input is incompetent and the corresponding synapses should be depressed. The lack of input does not cause depression.

Frenkel, M.Y. and Bear, M.F. (2004). *How monocular deprivation shifts ocular dominance in visual cortex of young mice.* Neuron **44**, 917-923.

At the same conference people discussed that superficially similar phenomena might have very different mechanism **and** function. Mark Bear remarked that there are multiple mechanisms of LTD (long-term synaptic depression). During the first day of that conference Nancy Kopell explained that the brain waves of the same frequency might have half a dozen of different generating mechanisms and, correspondingly, different cognitive functions.

For example, a few different gamma rhythms are described here: Christoph Börgers and Steven Epstein and Nancy J. Kopell, "Background gamma rhythmicity and attention in cortical local circuits: A computational study", PNAS 102(19):7002-7007 (2005), available from

`http://cbd.bu.edu/members/nkopell.html`

People started to talk about the tendency to focus the experimental measurements on high-frequency neurons and ignore the majority of neurons.

Bruno Olshausen, David Field.

How close are we to understanding V1?
Neural Computation, 2005;17:1665-1699

`http://redwood.psych.cornell.edu/papers/V1-article.pdf`

The earlier title of this paper was:

What is the other 85% of V1 doing?

Terrence Sejnowski, **Google Brain** talk at MIT, November 7, 2008.

He was also talking about research bias in favor of high-frequency neurons (5-40Hz and more).

He gave an estimate of 1Hz as the average neuron frequency in humans (3-4 Hz in rats).

It is usually more difficult to reproduce experimental results in neurobiology than in the other areas of biophysics and biochemistry.

Great variety of neuron and synapse types, anatomical and physiological context, people always want to extrapolate findings.

So the development we see is very interesting, but we also have to keep in mind that a large part of our theories in this field is quite unreliable at this point.

Part II

Models of biological neural networks

Firing rates vs. exact spike timing

Spike synchronization effects and brain waves

Spike-timing-dependent synaptic plasticity

Old paradigm – the output of a neuron is its **firing rate** (frequency of spikes).

This was the dominant paradigm in neuroscience until late 1990-s or early 2000-s, and this is still the dominant paradigm in the artificial neural networks.

Awful **stability-plasticity** properties (try to teach such a network how to perform a new task, and it will forget the task it knew how to perform before).

One exception: Steve Grossberg and his **Adaptive resonance theory** (classifiers with reasonable stability-plasticity properties based on neural nets):

`http://en.wikipedia.org/wiki/Adaptive_resonance_theory`

Memory and skills in the brain: almost no interference between different memories or different skills (good stability-plasticity). Learning new things is often almost instant (one short exposure).

There were dissidents (the most known of them was probably Karl Pribram), who were saying that there are plenty of wave-like processes in the brain, that it looks like we see Fourier transformers here and there in the brain.

They were also saying that we know a physical architecture which can provide the desired memory properties (robustness against damage **together with** non-interference), and this architecture is storing information in a hologram – **associative holographic memory**.

The new paradigm came during the 1990s from a somewhat different angle: people stressing the importance of **exact spike timing** and of **spike synchronization**.

Natural connection between spike synchronization effects and brain waves.

Conjecture that spike synchronization is the key neurological correlate of consciousness:

Francis Crick and Christof Koch, *Towards a Neurobiological Theory of Consciousness*, Seminars in the Neurosciences (1990): Volume 2, 263-275.

`http://profiles.nlm.nih.gov/SC/B/C/F/D/_/scbcfd.pdf` neurons as leaky integrators and synchronicity detectors

`http://en.wikipedia.org/wiki/`

`Biological_neuron_model#Leaky_integrate-and-fire`

how fast is the relaxation?

Song, S., Miller, K.D. and Abbott, L.F. (2000) Competitive Hebbian Learning Through Spike-Timing Dependent Synaptic Plasticity. Nature Neurosci. 3:919-926

`http://www.neurotheory.columbia.edu/~larry/`

$$dV/dt = (V_{rest} - V)/20ms$$

exact spike timing matters

Spikes: Exploring the Neural Code, by F.Rieke et al, Computational Neurosciences series, MIT Press, 1997

review by Cosma Shalizi:

`http://www.cscs.umich.edu/~crshalizi/reviews/spikes/`

'They close with a "homage to the single spike," as a trustworthy and reliable carrier of a substantial amount of information, sometimes even responsible by itself for conscious sensations. "The individual spike, so often averaged in with its neighbors, deserves more respect."'

Spike-timing-dependent synaptic plasticity

Bi G.Q., Poo M.M. Synaptic modifications in cultured hippocampal neurons: dependence on spike timing, synaptic strength, and postsynaptic cell type. Journal of Neuroscience 18, 10464-72 (1998)

`http://www.jneurosci.org/cgi/reprint/18/24/10464`

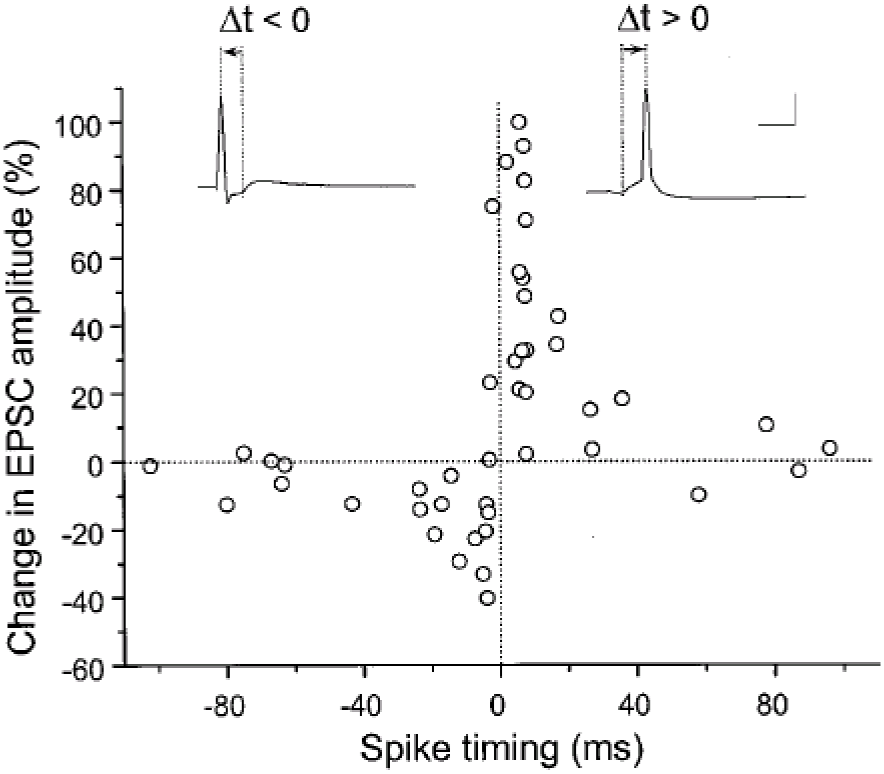

Fig.7 from Bi, Poo (1998)

Change in the Excitatory Post Synaptic Current depending on the difference between post-synaptic and pre-synaptic spike timing.

Wulfram Gerstner and Werner M. Kistler, Spiking Neuron Models: Single Neurons, Populations, Plasticity, Cambridge University Press (August 2002)

`http://lcn.epfl.ch/~gerstner/BUCH.html`

Peter Dayan, L. F. Abbott, Theoretical Neuroscience: Computational and Mathematical Modeling of Neural Systems, MIT Press, 2001.

Part III

a) Spikes and oscillations in artificial neural networks

b) Can spikes and oscillations help us to understand higher cognitive functions?

From "backpropagation" to biorealistic learning?

Spike-timing-dependent synaptic plasticity is Hebbian in spirit.

Can we use the fact that these are waves, and implement something like holographic or patch-holographic memory along these lines?

"Diffraction of neural waves on synapses"? (Where neural waves are presumably based on something like synchronized spikes.)

Related efforts: various biorealistic and not biorealistic developments in spiking neural networks and complex-valued neural networks.

Various oscillatory neural models by Roman Borisyuk:

`http://www.tech.plym.ac.uk/soc/staff/ROMAN/selected_publications.htm`

Various efforts of Eugene Izhikevich, Editor-in-Chief of

`http://www.scholarpedia.org/`

`http://vesicle.nsi.edu/users/izhikevich/publications/index.htm`

S. Grossberg, S., M. Versace, *Spikes, synchrony, and attentive learning by laminar thalamocortical circuits,* Brain Research, **1218**, 278–312 (2008).

`http://www.maxversace.com/papers/BRAIN_RESEARCH_Versace_Grossberg_2008.pdf`

D. Gabor, *Associative Holographic Memories*, IBM Journal of Research and Development, **13**(2), 156-159 (1969).

`http://www.research.ibm.com/journal/rd/132/ibmrd1302C.pdf`

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