ÉCOLE POLYTECHNIQUE FÉDÉRALE DE LAUSANNE

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A Neurocomputational Model of Impaired Imitation

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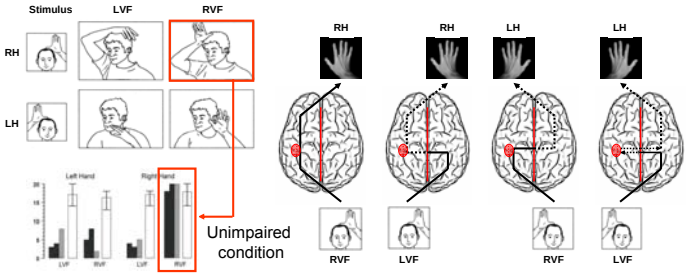
Motivations and Approach

- To understand the basic cognitive and neural mechanisms underlying visuomotor imitation and learning through observation.
- To construct a neurocomputational model of imitation inspired from brain lesion, imaging and neurophysiological data.

A Seminal Study of Imitation and Brain Activation

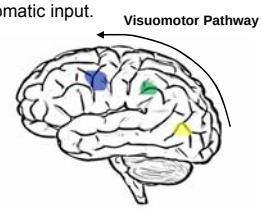
Goldenberg's study of imitation of meaningless gestures with a patient with callosal lesion shows that the pattern of error varies with the hand used (right RH / left LH) and with the side of presentation of the visual stimulus to imitate (RVF/LVF) [1].

We present a very simple neuroanatomical model that accounts for the non uniform flow of information across the two hemispheres. The model suggests a **left lateralization** of the computing process and predicts amelioration in the LVF-LH and deterioration in the RVF-RH condition, which could be verified in [2].



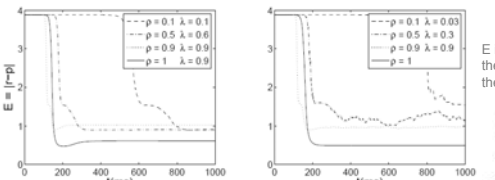
Numerous brain imaging (fMRI,PET) and lesion studies suggest the *left intraparietal sulcus* (parietal cortex, in green) as a neuroanatomical correlate for the left lateralized process, an area that processes mainly somatic input.

The other two consistently activated brain areas in imitation of meaningless gestures are [3]:
- *Extrastriate Body Area* (temporo-occipital cortex, in yellow), specialized in the visual analysis of body postures and movements.
- *Dorsal Premotor Area* (frontal cortex, in blue) responsible for motor preparation.

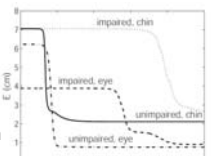


Properties and Predictions of the Model

Even with high impairment rates (90%) the network converges to a good solution:



E is the error between the simulated position *r* and the desired position *p* of the hand



As the face representation is non-uniform, some face parts are more represented (e.g. mouth) as compared to others (e.g. chin) and we observe inhomogeneities in the precision and processing times dependent on the final position.

Focal lesions could lead to impairment of imitation confined to some parts of the face only or to spatial errors shifted along one of the coordinate axes.
Severe lesions correlate with longer processing times. The time needed to do a correct imitation could be used as a measure of the severity of the lesion.

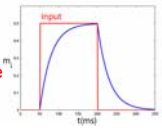
We conduct kinematic and behavioral studies with apraxic patients to provide with data for validation of the model. The learning properties of our model can account for some of the brain reorganization following brain lesion.

This work is done in collaboration with the Laboratory of Cognitive Neuroscience (LNCO) and Geneva University Hospital (HUG).

A Neurocomputational Model of Visomotor Imitation

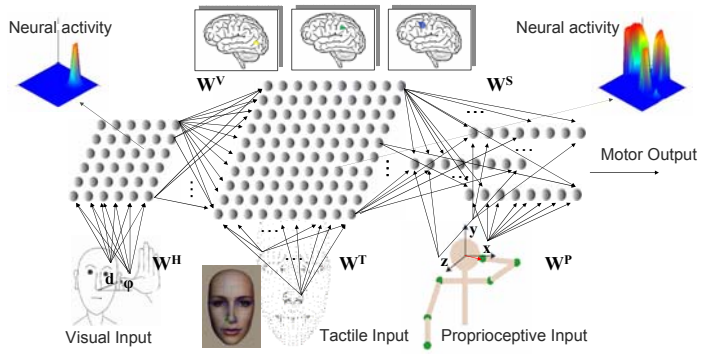
We use **Leaky Integrator Neurons** to account for time. The membrane potential m_i of a neuron with index i is thus governed by the first-order differential equation:

$$\tau \frac{d}{dt} m_i = -m_i + I$$



where τ is a time constant and I is the neuron input. The firing rate of a neuron is a sigmoid function of the membrane potential.

The sensory input involved in imitation of meaningless gestures (i.e. visual, tactile and proprioceptive) is fully connected to the processing networks.



We train the sensory weights $\mathbf{W^H}$, $\mathbf{W^T}$ and $\mathbf{W^P}$ with **Kohonen**'s algorithm to preserve the somatotopic organization [4]. Accordingly, we have defined the input to neuron i as a gaussian function of the distance between sensory input x and the connecting weights w_i:

$$\tau \frac{d}{dt} m_i = -m_i + e^{(-\frac{|x-w_i|^2}{2\sigma^2})}$$

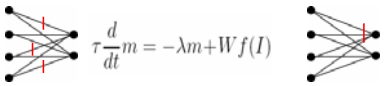
We train the weights $\mathbf{W^V}$ and $\mathbf{W^S}$ to learn the transformations between the sensory inputs. We use a presynaptic-gated **antihebbian learning** rule to associate correlated neural activities:

$$\Delta w_{i,j} = \eta \cdot x_j [y_i - \sum w_{i,k} x_k]$$

where $w_{i,j}$ is the synaptic weight between a presynaptic neuron x_j and a postsynaptic neuron y_i, and η is the learning rate. The neurons develop multimodal properties.

Simulation of the Lesion and Results

We simulate the lesion probabilistically: a function f assigns a probability of impairment ρ to the connection (model 1) or to the neuron input (model 2).
The patient imitates well given unlimited time, so we hypothesize a decay factor $\lambda < 1$.

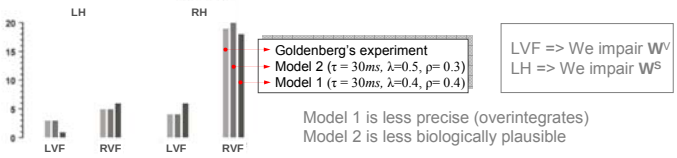


$$\tau \frac{d}{dt} m = -\lambda m + W f(I)$$

$$\tau \frac{d}{dt} m = -\lambda m + f(WI)$$

$$\lambda = 0 \iff m < f(WI)$$

We reproduced the same experimental conditions as in Goldenberg's experiment and found highly plausible parameters that account for Goldenberg's scores.



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References:

[1] Goldenberg G. et al. **Imitation of gestures by disconnected hemispheres.** *Neuropsychologia* (2001) 39:1432-1443
[2] Lausberg H. et al. **Hemispheric specialisation for imitation of hand–head positions and finger configurations.** *Neuropsychologia* (2004) 42(3):320-334
[3] Mühlau M. et al. **Left inferior parietal dominance in gesture imitation: an fMRI study**. *Neuropsychologia* (2005) 43:1086-1098
[4] Buccino G. et al. **Action observation activates premotor and parietal areas in a somatotopic manner: an fMRI study.** *European Journal of Neuroscience* (2001) 5:400-404