A Computational Neuroscience Account of Visual Neglect

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Abstract

On the computational basis of a neurodynamical cortical model, we investigate a specific top-down visual cognitive impairment in brain-damaged patients known as visual spatial neglect. The computational cortical model accounts the neurodynamics underlying selective visual attention, is based on the “biased competition hypothesis" and structured in several network modules which can be related with the different areas of the dorsal and ventral path of the visual cortex. Spatial and object attention are accomplished by a multiplicative gain control that emerges dynamically through intercortical mutual biased coupling. By damaging the model in different ways, a variety of dysfunctions associated with visual neglect can be simulated and explained as disruption of specific subsystems. Essentially, the damage destabilizes the underlying intra- and intermodular mutually biased neurodynamical competition that macroscopically yields the functional deficits observed in visual neglect patients. In particular, we are able to explain the asymmetrical effect of spatial cueing on neglect, and the phenomenon of extinction in the framework of visual search. Even more, we predict that patients with neglect can generate different saccade patterns depending on whether the object under observation is known or not. This finding explicitly shows one of the main characteristics of “active” vision, namely the influence of top-down processing, and its neuropsychological relevance.

1. Introduction

In neurological patients, attention deficiencies can result from damage to different brain regions. Particularly, unilateral parietal damage, could lead to symptoms of the *neglect syndrome*, wherein patients fail to notice the existence of objects or events in the hemispace opposite their lesion site (e.g.: Humphreys and Riddoch, 1994; Vallar and Perani, 1986; Samuelsson et al., 1997). A significant characteristic of the neglect syndrome is *extinction*, the failure to perceive a stimulus contralateral to the lesion (*contralesional*) when simultaneously presented with a stimulus ipsilateral to the lesion (*ipsilesional*). In fact, extinction shows that neglect is not caused by a visual sensory defect but rather by a disorder in visually attending. Visual neglect can be diagnosed by neuropsychological tests. The most popular and simple is the so-called *line cancellation test*. Subjects are given a sheet of paper containing many horizontal lines and asked under free-viewing conditions to bisect the lines precisely in the middle by placing a vertical line. Patients with visual neglect, for example with lesions of the right hemisphere tend to bisect the lines to the right of the midline. They may also miss lines altogether in the direction opposite to the impaired hemisphere.

Unilateral visual neglect is far from a unitary phenomenon. Humphreys and Heinke (1998) analysed different forms of unilateral visual neglect experimentally and theoretically (see also Heinke and Humphreys, 1999). Some patients have deficits in reacting to stimuli presented on one side of space relative to their body (Halligan and Marshall, 1994). Let us call this kind of syndrome space-based neglect. Others show a form of neglect which appears to be independent of the spatial location of the object in the visual field relative to the viewer’s body (Tipper and Behrmann, 1996; Behrmann and Moscovitch, 1994; Arguin and Bub, 1993; Driver and Halligan, 1991; Young et al., 1991). For instance, several studies have now found that

(ORAL PRESENTATION PREFERRED)

patients may neglect the left side of an object (or perceptual group) whether or not it appears in the left or right hemifield with respect to the patient's body (e.g., Humphreys et al., 1996; Walker, 1995; Halligan & Marshall, 1994; Arguin and Bub, 1993; Driver et al., 1992). This kind of visual disorder is called object-based neglect because only the left part of the object is neglected even when the object is rotated so that its left part fell in the right visual hemifield, or the object is translated to the right visual hemifield. In other words, in object-based neglect parts (left or right) relative to the object seem to be ignored.
Despite the ardent interest in the neglect syndrome, the underlying causes leading to this disorder are still obscure and an issue of controversies (Halligan and Marshall, 1994; Rafal and Robertson, 1997). A systemic explanation of the neuropsychological findings on visual neglect would help us to better understand the mechanisms underlying the representation of visual space and the control of visual attention in space, and thus, the cognitive functioning of the brain (Kosslyn, 1996). In this paper, we are interested in an attentional account of the neglect syndrome. We analyse the relation between visual neglect and the underlying neural basis of visual attention in the framework of a computational cortical model. We formulate a neurodynamical model for selective visual attention based on the "biased competition hypothesis" and structured in several network modules which can be related with the different areas of the dorsal and ventral path of the visual cortex. Spatial and object attention are accomplished by a multiplicative gain control that emerges dynamically through intercortical mutual biased coupling. We try to understand which kind of neuropsychological dysfunctions are produced after damage to specific subsystems of this cortical model.

2. Computational Model

We now briefly describe the cortical model of visual attention for object recognition and visual search based on the biased competitive hypothesis (Reynolds et al., 1999) and the corresponding neurodynamical mechanisms. The system is absolutely autonomous and each of its functionalities is explicitly described in a complete mathematical framework. The overall systemic representation of the model is shown in the Figure 1.

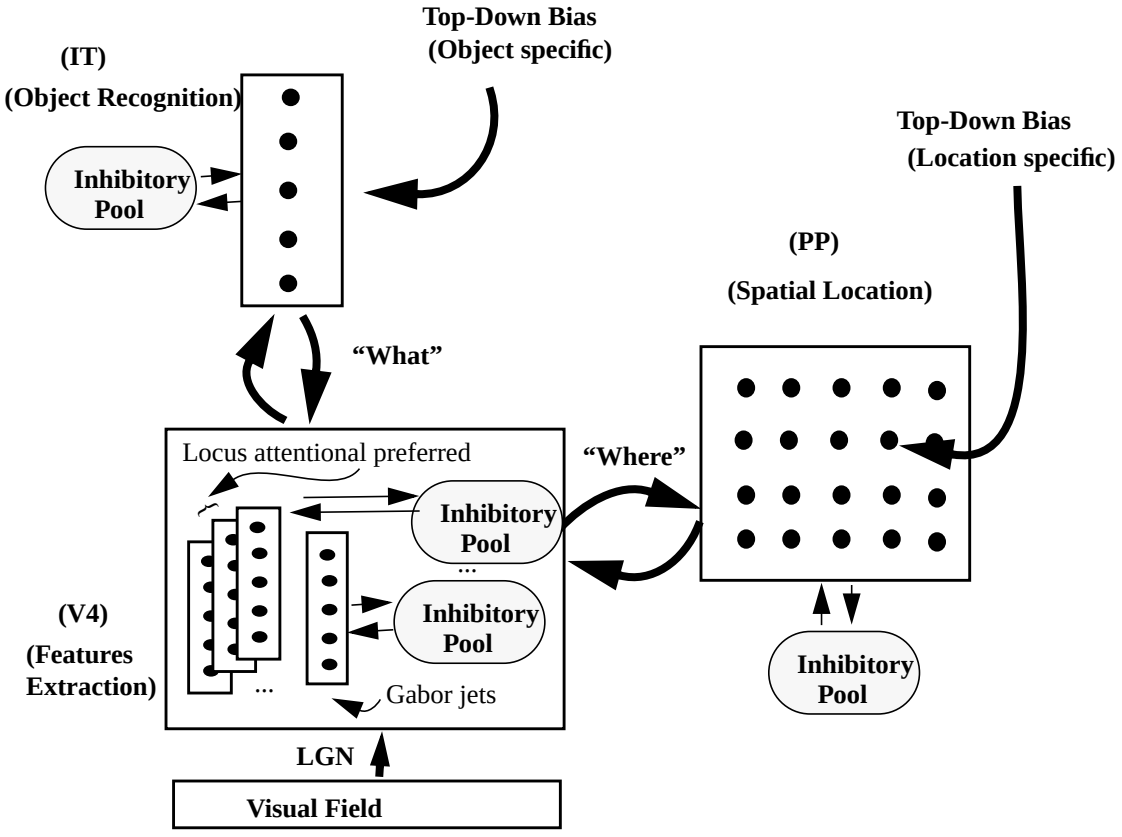

Figure 1. A cortical architecture for visual attention.

The system is essentially composed of three modules structured such that they resemble the two known main visual path of the mammalian visual cortex. Information from the retino-geniculo-striate pathway enters the visual cortex through area V1

in the occipital lobe and proceeds into two processing streams. The occipital-temporal stream (the so-called “what” pathway) leads ventrally through V2, V4 and IT (inferotemporal cortex) and is mainly concerned with object recognition, independently of position and scaling. The occipito-parietal stream (the so-called “where” pathway) leads dorsally into PP (posterior parietal complex) and is concerned with the location and spatial relationships between objects. The first module (V4) of our system is engaged with the extraction of features and consists of pools of neurons with Gabor receptive fields tuned to different positions in the visual field, orientations and spatial frequency resolutions. The “where” pathway is given through the mutual connection with the second (PP) module that consists of pools codifying the position of the stimuli. The connections with the first module origin a top-down biasing attentional modulation associated with the location of the stimuli. At last, the third module (IT) of our system is engaged with the recognition of objects and consists of pools of neurons which are sensitive to the presence of a specific object in the visual field. The pools in (IT) are synaptically connected with translationally invariant receptive fields with pools of the first module (V4) such that based on the Gabor features specific objects are invariantly recognized. The mutual connections between IT and V4 modules represent a top-down biasing attentional modulation associated with specific objects.

The system operates in two different modes: the learning mode and the recognition mode. During the learning mode the synaptic connection between V4 and IT are trained by means of Hebbian learning during several presentations of specific objects at changing random position in the visual field. During the recognition mode there two possibilities of running the system. First, an object can be localized in a scene (visual search) by biasing the system with an external top-down component at the IT module which drives the competition in favour of the pool associated with the specific object to be searched such that the intermodular attentional modulation V4-IT will enhance the activity of the pools in V4 associated with the features of the specific object to be searched and the intermodular attentional modulation V4-PP will drives the competition in favour of the pool localizing the specific object. Second, an object can be identified (object recognition) at a specific spatial location by biasing the system with an external top-down component at the PP module which drives the competition in favour of the pool associated with the specific location such that the intermodular attentional modulation V4-PP will favour the pools in V4 associated with the features of the object at that location and intermodular attentional modulation V4-IT will favour the pool that recognized the object at that location. Both external top-down bias are assumed to come from frontal areas of the cortex that are not explicitly modelled.

Let us now discuss the mathematical formulation of the system. We consider a pixelized grey-scaled image given by a $N \times N$ matrix Γ_{ij}^{orig}. The subindices ij denote the spatial position of the pixel. Each pixel value is given a grey value coded in a scale between 0 (black) and 255 (white). The first step in the preprocessing consists in removing the DC component of the image (i.e. the mean value of the grey-scaled intensity of the pixels) which is probably done in the lateral geniculate nucleus (LGN) of the thalamus. The visual representation in LGN is essentially a contrast invariant pixel representation of the image, i.e. each neuron encodes the relative brightness value at one location in visual space referred to the mean value of the image brightness. Feedforward connections to a layer of V1 neurons perform the extraction of simple features. Theoretical investigations have suggested that simple cells in the primary visual cortex can be modelled by 2D-Gabor functions. The 2D-Gabor functions are local spatial bandpass filters that achieve the theoretical limit for conjoint resolution of spatial and frequency information, i.e. in the 2D-spatial and 2D-Fourier domains. The Gabor receptive fields have five degrees of freedom given essentially by the product of an elliptical Gaussian and a complex plane wave. The first two degrees of freedom are the 2D-location of the receptive field’s centre, the third is the size of the receptive field, the fourth is the orientation of the boundaries separating excitatory and inhibitory regions, and the fifth is the symmetry. This fifth degree of freedom is given in the standard Gabor transformation by the real and imaginary part, i.e by the phase of the complex function representing it, whereas biologically this can be done by combining pairs of neurons with even or odd receptive fields. Let us consider the experimental neurophysiological constraints. There are three constraints fixing the relation between width, height, orientation and spatial frequency. The first constraint postulates that the aspect ratio of the elliptical Gaussian envelope is 2:1. The second constraint postulates that the orientation is aligned with the long axis of the elliptical Gaussian. The third constraint assumes that the half-amplitude bandwidth of the frequency response is about 1 to 1.5 octaves along the optimal orientation. Further, we assume that the mean is zero in order to have an admissible wavelet basis. A family of discretized 2D-Gabor wavelets that satisfy the wavelet theory and the neurophysiological constraints for simple cells as given by Lee (1996)

$$G_{kpql}(x,y) = a^{-k}\psi_{\theta_l}(a^{-k}(x-2p)-a^{-k}(y-2q)),\ \psi_{\theta_l} = \psi(x\cos(l\theta_0)+y\sin(l\theta_0), -x\sin(l\theta_0)+y\cos(l\theta_0)) \qquad (1)$$

and the mother wavelet is given by

$$\psi(x, y) = \frac{1}{\sqrt{2\pi}} e^{-\frac{1}{8}(4x^2 + y^2)} \cdot \left[e^{i\kappa x} - e^{-\frac{\kappa^2}{2}} \right]. \tag{2}$$

In the above equations θ_0 denotes the step size of each angular rotation, l the index of rotation corresponding to the preferred orientation θ_l, k denotes the octave, and pq the position of the receptive field centre. In this form, the receptive fields at all levels cover the spatial domain in the same way, i.e. by overlapping always the receptive field in the same fashion. In this work we choose $\kappa = \pi$ that correspond to a spatial frequency bandwidth of one octave. The neurons in the pools in V1 have receptive fields performing a Gabor wavelet transform. Let us denote by I^{V1}_{kpql} the sensorial input activity to a pool in V1 which is sensitive to a determined spatial frequency given at octave k, to a preferred orientation defined by the rotation index l and to stimuli at the centre location specified by the indices pq. The sensorial input activity to a pool in V1 is therefore defined by the module of the convolution between the corresponding receptive fields and the image. Since in our numerical simulations the system needs only to learn a small number of objects (usually 2-4), in our current implementation, for simplicity, we temporarily eliminate the V4 module and fully connect V1 and IT cell assemblies directly together. We implement translation invariance by the attentional intermodular biasing interaction between pools in the modules V1 and PP. For each V1 neuron, the gain modulation observed decreases as the actual point where attention is being focused moves away from the centre of the receptive field in a Gaussian-like form. Consequently, the connections with the pools in the PP module are specified such that the modulation is Gaussian. Let us define in the PP module a pool A^{PP}_{ij} for each location ij in the visual field. The mutual connection (i.e. bilateral) between a pool A^{V1}_{kpql} in V1 (or V4) and a pool A^{PP}_{ij} in PP are therefore defined by

$$W_{pqij} = Ae^{-\frac{(i-p)^2 + (j-q)^2}{2S^2}} - B \tag{3}$$

Let us now define the neurodynamical equations that regulates the evolution of the whole system. The activity level of the input current in the V1 module is given by

$$\begin{aligned} \tau \frac{\partial}{\partial t} A^{V1}_{kpql}(t) = & -A^{V1}_{kpql}(t) + aF(A^{V1}_{kpql}(t)) - bF(A^{I,V1}_{k}(t)) + I^{V1}_{kpql}(t) \\ & + I^{V1-PP}_{pq}(t) + I^{V1-IT}_{kpql}(t) + I_0 + \nu \end{aligned} \tag{4}$$

where the attentional biasing due to the intermodular “where” connections with the pools in the parietal module PP I^{V1-PP}_{ab} is given by

$$I^{V1-PP}_{pq}(t) = \sum_{i,j} W_{pqij} F(A^{PP}_{ij}(t)) \tag{5}$$

and the attentional biasing due to the intermodular “what” connections with the pools in the temporal module IT I^{V1-IT}_{kpql} is defined by

$$I^{V1-IT}_{kpql}(t) = \sum_{c=1} w_{ckpql} F(A^{IT}_{c}(t)) \tag{6}$$

where w_{ckpql} is the connection strength between the V1 pool A^{V1}_{kpql} and the IT pool A^{IT}_{c} corresponding to the coding of an specific object category c. We assume that the IT module has C pools corresponding to different object categories. We implement the resolution hypothesis by assuming different latencies τ in eq. (4) for pools corresponding to different octaves. Higher resolution octaves have a smaller τ than the ones corresponding lower resolutions. For each spatial frequency level, a common inhibitory pool is defined. The current activity of the inhibitory pools obey the following equations:

$$\tau_P \frac{\partial}{\partial t} A^{I,V1}_{k}(t) = -A^{I,V1}_{k}(t) + c \sum_{p,q,l} F(A^{V1}_{kpql}(t)) - dF(A^{I,V1}_{k}(t)) \tag{7}$$

The current activity of the excitatory pools in the posterior parietal module PP are given by

$$\tau\frac{\partial}{\partial t}A_{ij}^{PP}(t) = -A_{ij}^{PP}(t) + aF(A_{ij}^{PP}(t)) - bF(A^{I,PP}(t)) + I_{ij}^{PP-V1}(t) + I_{ij}^{PP,A} + I_0 + \nu \quad (8)$$

where $I_{ij}^{PP,A}$ denotes an external attentional object specific top-down bias and the intermodular attentional biasing I_{ij}^{PP-V1} through the connections with the pools in the module V4 is

$$I_{ij}^{PP-V1}(t) = \sum_{k,p,q,l} W_{pqij}F(A_{kpql}^{V1}(t)) \quad (9)$$

and the activity current of the common PP inhibitory pool evolves according to

$$\tau_P\frac{\partial}{\partial t}A^{I,PP}(t) = -A^{I,PP}(t) + c\sum_{i,j}F(A_{ij}^{PP}(t)) - dF(A^{I,PP}(t)) \quad (10)$$

The dynamics of the inferotemporal module IT is given by

$$\tau\frac{\partial}{\partial t}A_c^{IT}(t) = -A_c^{IT}(t) + aF(A_c^{IT}(t)) - bF(A^{I,IT}(t)) + I_c^{IT-V1}(t) + I_c^{IT,A} + I_0 + \nu \quad (11)$$

where $I_c^{IT,A}$ denotes an external attentional spatial specific top-down bias and the intermodular attentional biasing I_c^{IT-V4} between IT and V1 pools is

$$I_c^{IT-V1}(t) = \sum_{k,p,q,l} w_{ckpql}F(A_{kpql}^{V1}(t)) \quad (12)$$

and the activity current of the common PP inhibitory pool evolves according to

$$\tau_P\frac{\partial}{\partial t}A^{I,IT}(t) = -A^{I,IT}(t) + c\sum_{c}F(A_c^{IT}(t)) - dF(A^{I,IT}(t)) \quad (13)$$

During a learning phase each object is learned. The external attentional location specific bias in PP $I_{ij}^{PP,A}$ is set so that only the pool ij corresponding to the spatial location where the object to be learned is, receives a positive bias. In this way the spatial attention defines the localization of the object to be learnt. The external attentional object specific bias in IT $I_c^{IT,A}$ is similarly set so that only the pool c that will identify the object receives a positive bias. Therefore, we define in a supervised way the identity of the object. After presentation of a given stimulus, i.e. an specific object at an specific location, and the corresponding external bias, the system evolves until convergence. After convergence, the V1-IT connections w_{ckpql} are trained through the following Hebbian rule

$$w_{ckpql} = w_{ckpql} + \eta F(A_c^{IT}(t))F(A_{kpql}^{V1}(t)) \quad (14)$$

being t large enough (i.e. after convergence) and η the learning coefficient. This procedure is repeated for all objects at all possible locations until the weights converge. During the recognition phase there are two possibilities, namely to search for an specific object (visual search) or to identify an object at a given spatial location (object recognition). In the case of visual search the stimuli are presented and the external attentional object specific bias in IT $I_c^{IT,A}$ is set so that only the pool c corresponding to the category of the object to be searched receives a positive bias while the external attentional location specific bias in PP $I_{ij}^{PP,A}$ is set zero everywhere. The external attentional bias $I_c^{IT,A}$ drives the competition in the IT module so that the pool corresponding to the searched object wins. The intermodular attentional modulation between IT and V1 bias the competition in V1 so that all features detected from the retinal inputs at different positions that are compatible with the specific object to be searched will now win. At last, the intermodular attentional bias between V1 and PP drives the competition in V1 and in PP so that only the spatial location in PP and the associated V1 pools compatible with the presented stimulus and with the top-down specific category of the object to be searched will remain active after convergence, i.e. reading the final state in PP the object is found. In this form, the final activation state is neurodynamically driven by stimulus, external top-down bias and intermodular bias, in a fully parallel way. The attention is not a mechanism involved in the competition but just an emergent effect that support the dynamical evolution to a state where all constraints are satisfied. In the case of object recognition, the external attentional bias in PP $I_{ij}^{PP,A}$ is set so that only the pool associated with the spatial location where the object to be identified is, receives a positive bias, i.e. an spatial region will be “illuminated”. The other external bias $I_c^{IT,A}$ is zero everywhere. In this case, the dynamic evolve such that in PP only the pool associated with the top-down biased spatial location will win, and this drives the competition in V1 such that only the pools corresponding to features of the stimulus at

that location will win biasing the dynamics in IT such that only the pool identifying the class of the features at that position will remain active indicating the category of the object at that predefined spatial location.

3. A Model of Visual Spatial Neglect

Theoretical models of visual neglect can be usually divided in approaches based on a representational or an attentional account of the syndrome (Pouget and Driver, 1999; Humphreys and Heinke, 1998; Bisiach, 1996; Driver et al., 1994). A representational account interprets neglect as the result of impairment of one side of a particular spatial representation, whereas an attentional account considers neglect as a deficit in orienting visual attention to the affected hemispace. Nevertheless, the attentional account is strongly favoured by two experimental evidences, namely: 1) the asymmetrical effect of spatial cueing on neglect (Posner et al., 1984; Rafal and Robertson, 1997), and 2) the phenomenon of extinction in the framework of visual search (Eglin et al., 1989). In both cases, the degree of impairment increases when the stimulus at the affected side has to compete with a second stimulus at the unaffected side, referred to the condition when only one stimulus alone is at the affected side. This kind of asymmetry is consistent with the idea that the stimulus on the neglected field attracts attention only in a weak way. In Sections 4 and 5, we explain both experimental results in the theoretical framework of our cortical model based on the biased competition hypothesis. Our account of visual neglect is based on different kinds of damage of the posterior parietal module, which is the module representing and controlling the spatial location of visual attention.

Before modelling visual neglect, let us briefly mention the syndrome of *hemianopia*. Hemianopic patients do not perceive information coming from one hemifield. This kind of disease can be accounted by damage of the early visual sensory system, that is at the level of the LGN cells in the thalamus or the cortical cells in the primary visual cortex. In our model this can be simulated easily by cutting the input coming from the impaired hemifield or by silencing in the primary module V1 the output of the neuronal pools whose receptive fields cover locations in the impaired hemifield. This type of disruption causes the system to behave as a patient with hemianopia, i.e. the subject will not perceive information coming from one hemifield. Objects or parts of objects falling in the impaired hemifield will be ignored as if they would not exist. This kind of visual disorder is not an authentic kind of neglect because only the low-level system is disrupted. In fact, patients with damage to the visual system at this level do not show visual neglect but impaired visually guided oculomotor scanning (Zihl, 1995).

Let us now concentrate on visual neglect. We consider in this section two kinds of neglect: 1) space-based, and 2) object-based. Space-based visual neglect can be explained by unilateral damage of the PP module of our cortical architecture. Let us divide the neuronal pools in the PP module in two left and right hemispheres (Figure 2). The left (right) hemisphere is associated with the right (left) visual field. A group of neuronal pools in a certain module can be impaired in an intrinsic way (Humphreys and Heinke, 1998) by damaging only intrinsic inputs within the module. In mathematical terms, the intrinsic lesioning of a neuronal pool in the PP module is described by extending equation (8) to:

$$\tau\frac{\partial}{\partial t}A_{ij}^{PP}(t) = -A_{ij}^{PP}(t) + L_{ij}\{aF(A_{ij}^{PP}(t)) - bF(A^{I,PP}(t)) + I_0\} + I_{ij}^{PP-V1}(t) + I_{ij}^{PP,A} + \nu \tag{15}$$

where L_{ij} is a lesioning factor. Values of L_{ij} equal to one leave the corresponding neuronal pools unaffected, whereas values of L_{ij} smaller than one damage the corresponding neuronal pools by reducing the influence of the intrinsic inputs $A_{ij}^{PP}, A^{I,PP}, I_0$.

Although visual neglect can be present in the left or right hemispace, we refer in the following to the left-sided visual neglect, i.e. the left-visual neglect, because this type is more frequently observed (Vallar and Perani, 1986). However, the model can also be applied to right-sided visual neglect. We consider two different types of lesioning patterns.

The first one is associated with a pure unilateral damage of the PP in the right hemisphere. In this case, the damage factor is given by (Figure 2.b)

$$\begin{cases} L_{ij} = 0.6 & if\,(i < N/2) \\ L_{ij} = 1.0 & if\,(i \geq N/2) \end{cases} \tag{16}$$

The second kind of lesioning pattern considers a gradient impairment of the PP module. The corresponding damage factor is given by (Figure 2.c)

$$L_{ij} = 0.2 + 0.8 \cdot i/N \tag{17}$$

This lesioning pattern refers to the gradient of impairment in neglect following right-parietal injury (Pouget and Sejnowski, 1997; Anderson, 1996; Driver et al., 1994; Mozer and Behrmann, 1990; Kinsbourne, 1987).

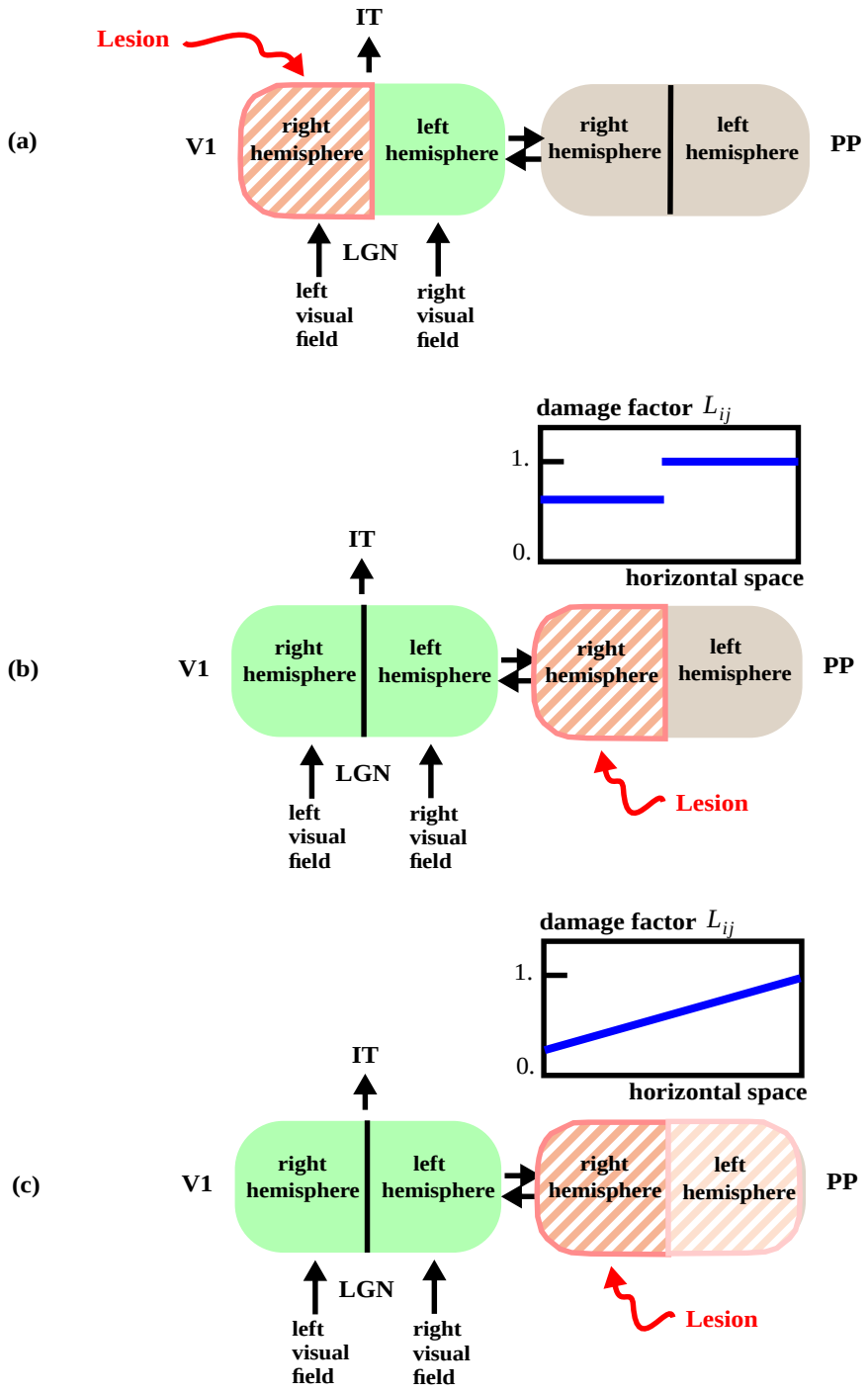

Figure 2. Lesioning patterns. (a) Hemianopia resulting from damage of the V1 module (primary visual cortex); (b) Left-sided space-based visual neglect resulting from a pure unilateral damage of the right hemisphere of the PP module (posterior parietal cortex); (c) Left-sided object-based visual neglect resulting from a gradient impairment damage of the right hemisphere of the PP module.

The damage factor reflects the decreasing number of parietal cells in the lesioned system for positions toward the retinal left, in accordance with neurophysiological data on the distribution of receptive fields between the hemispheres (see Pouget and Sejnowski, 1997). The neural response to stimuli is increasingly impaired toward the patient's egocentric left.

We will show now that the syndrome of local-based visual neglect can be explained by pure unilateral damage of the posterior parietal network of the cortical architecture. Likewise, object-based visual neglect can be explained by a gradient impairment of the posterior parietal network. We damage the PP module of our model according to equations (15) and (16) for simulating local-based neglect, and to equations (15) and (17) for simulating object-based neglect. In order to visualize the results of the different types of damage, we plot the final state of the PP module after dynamical convergence, which shows a spatial map of locations where attention has been deployed. In addition, we plot the topographically ordered activation final state of the PP module overlapped with the input image, so that the attentional spatial map can be easily interpreted. We symbolize with a red point the pixels at locations that correspond to a PP neuronal pool that have an activity larger than a certain threshold $\vartheta = 0.08$. Hence, we can relate the attentional spatial map with the scan path of the fixation locations during the perception of an object. High activity spatial locations in the PP module correspond to spatial attended regions that attract a potential fixation.

We use an input display given by a pixelized 66×66 image ($N = 66$) showing an object ("Paolina" of the Carnegie Mellon Database) at different translated locations. The V1 hypercolumns cover the entire image uniformly. They are distributed in 33×33 locations ($P = 33$) and each hypercolumn is sensitive to three spatial frequencies and to eight different orientations (i.e., $K = 3$ and $L = 8$). The PP module contains 4356 pools corresponding to each possible spatial location, i.e. to each of the $66x66$ pixels, and a common inhibitory pool. We first simulate the case of unilateral local-based neglect by disrupting the PP module with a pure unilateral damage of the right hemisphere. Figure 3 shows the neglect of the entire left part of the visual field and therefore the disrupted processing of objects (Figures 3.a) or parts of an object (Figure 3.b) in the impaired left hemifield. If the object is completely in the intact right hemifield, the scan path is absolutely normal (Figure 3.c) and consequently spatial attention is deployed on both sides of the object. The neuronal pools corresponding to the right hemisphere are not able at all of achieving high activity due to the unilateral pure disruption affecting them. This occurs in spite of the eventually positive bias coming from pools in the right hemisphere of V1 that excite the respective pools in the right hemisphere of PP. Hence, their low activation can be interpreted as neglect for all sensory informations coming from the left visual hemifield.

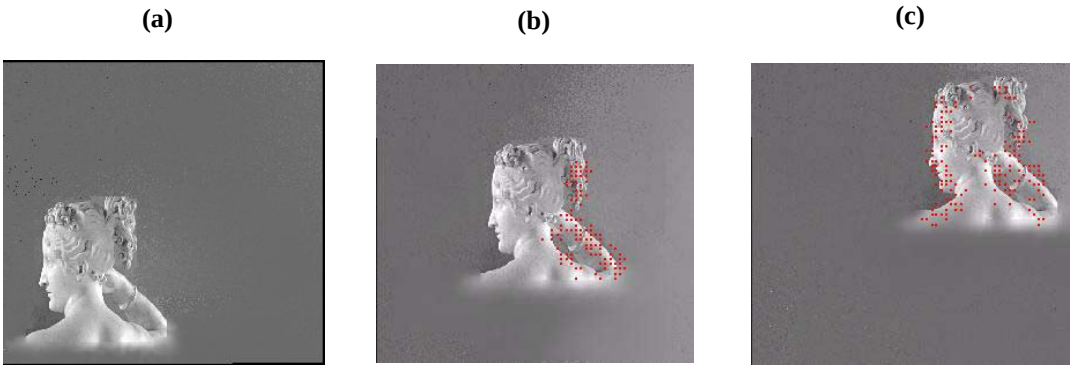

Figure 3. Left-sided local-based visual hemi-neglect for an object at different translated positions. See text for details.

Figure 4 shows the attentional spatial map obtained after a gradient impairment of the PP module. In this case a pure object-based neglect is obtained. Translation of the object has no effect: the location of the neuronal pools in module PP with high activation is always restricted to the right part of the object with respect to the object frame. Although the absolute level of activity is lower when the object is in the left versus right visual field, in both cases the left side of the object receives a weaker response than its right side, due to the horizontal gradient across the PP module. The intramodular inhibition between neuronal pools in PP drives the dynamical competition always in favour of the pools corresponding to the right side of the object, suppressing at the same time the activity of the pools associated with the left side of the object. Hence, in this case the underlying biased competition causes an attentional spatial map that explains the scan path of the fixation locations observed by object-based visual neglect.

(a) **(b)** **(c)**

Figure 4. Left-sided object-based visual hemi-neglect for an object at different translated positions. See text for details.

4. Spatial Cueing Effect on Neglect

A biased competition account of the underlying disturbances of attentional mechanisms that induce visual neglect is decisively supported by the asymmetrical repercussion of spatial cueing on neglect patients. In a typical spatial attention experiment using the precueing method, the subject is asked to respond by pressing a key, to the appearance of a target at a peripheral location (Posner et al., 1984). The target is preceded by a precue that indicates to which visual hemifield the subject is to covertly attend. The cue is then followed by a target in either the correctly (valid cue) or the incorrectly (invalid cue) cued attention.

This kind of experiments for measuring cover shifts of attention have been applied using simple reaction time paradigms in patients with unilateral lesions of the parietal cortex. Albeit under normal conditions these patients may neglect contralesional stimuli, they can often be commanded to attend to the neglected field. In fact, in the precueing paradigm, reaction times to valid cued targets in the hemifield contralateral to the lesion were almost normal in the sense that they were not much slower than those for targets that occurred in the ipsilesional hemifield when that field was precued. On the contrary, if the ipsilesional field was precued, they were unusually slow to respond to the target when it unexpectedly appeared in the contralesional hemifield. In other words, patients are able to orient contralesionally to benefit from a valid cue, but they are impaired when attention is summoned by an invalid cue in the ipsilesional field and the target occurs in the contralesional hemifield. Figure 5.a shows an example of this measured experimental asymmetry by plotting the detection time difference between the contralesional and ipsilesional fields for the two cueing conditions (Rafal and Robertson, 1997).

These asymmetric reaction time pattern have been described as an *extinction-like* reaction time pattern to indicate its similarity to the clinical finding of extinction in these patients. The extinction-like asymmetric reaction times pattern has now been found in several experimental studies (Posner et al., 1984, 1987; Baynes et al. 1986; Rafal and Robertson, 1987). These studies demonstrate that the degree of asymmetry correlates with the severity of the clinical neglect (Morrow and Ratcliff, 1988).

In order to understand the underlying attentional dynamical mechanisms involved in this extinction-like phenomena, we simulate the precueing experiments with the cortical architecture introduced in this paper. We perform the numerical experiments by using a pure unilateral damage of the right hemisphere of the PP module (equations (15) and (16)) in order to simulate the behaviour of left neglect patients. The input display is given by a pixelized 66×66 image ($N = 66$). The target is a letter "E" of 6×9 pixels that could appear centred in the left or right visual hemifield. The V1 hypercolumns cover the entire image uniformly. They are distributed in 33×33 locations ($P = 33$) and each hypercolumn is sensitive to three spatial frequencies and to eight different orientations (i.e., $K = 3$ and $L = 4$). The PP module contains $66x66$ pools, and a common inhibitory pool. During the first precueing period of 250 ms no stimulus is presented in the visual field and the left or right hemifields are cued by applying an external attentional location specific bias $I_{ij}^{PP,A} = 0.07$ to the neuronal pools in PP corresponding to a 9×9 square centred in the right or left hemisphere, respectively. After 250 ms, the object to be recognized (letter "E") is presented as stimulus in the left or right visual hemifield. Two pools in the IT module were previously trained to learn the identity of the two stimuli "E" and "X", respectively. Four conditions are of interest: contralesional presentation of the stimulus by valid cue (top-down bias at the right PP hemisphere) or invalid cue (top-down bias at the left PP hemisphere),

and ipsilesional presentation of the stimulus by valid cue (top-down bias at the left PP hemisphere) or invalid cue (top-down bias at the right PP hemisphere). The reaction times were calculated by detecting the time at which the IT pools are sufficiently polarized in favour of the target, i.e. when the difference between the maximal activity of the IT pools corresponding to the target and non-targets $A_T^{IT} - A_D^{IT}$ is larger than a certain threshold ς (in our case, we chose $\varsigma = 0.05$). Figure 5.b shows the corresponding numerical results. It is clear from the figure, that the computational model reproduces perfectly the extinction-like asymmetric precueing effect.

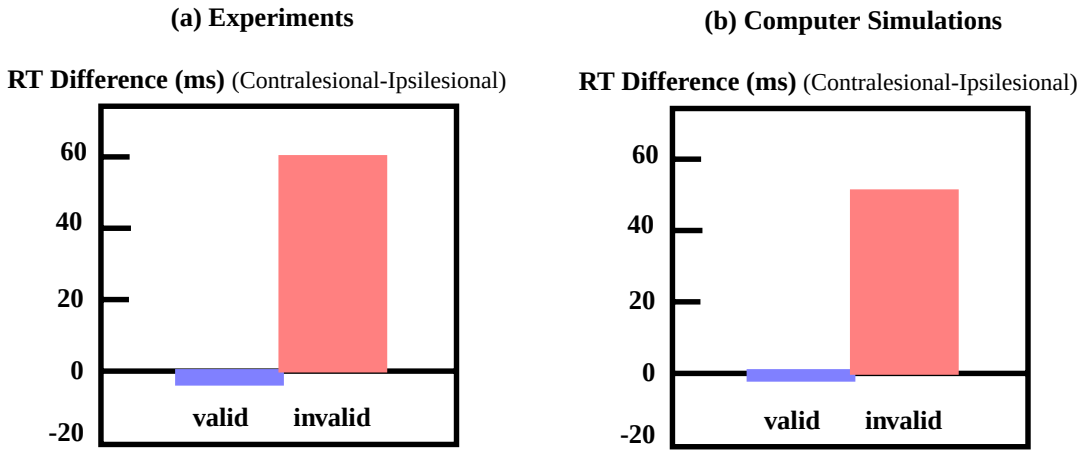

Figure 5. Extinction-like reaction time patterns obtained in spatial precueing experiments: (a) in patients with unilateral lesions of the parietal cortex (adapted from Rafal and Robertson, 1997); (b) computational simulation results. Plotted is the detection time difference between the contralesional and ipsilesional fields for the two cueing conditions.

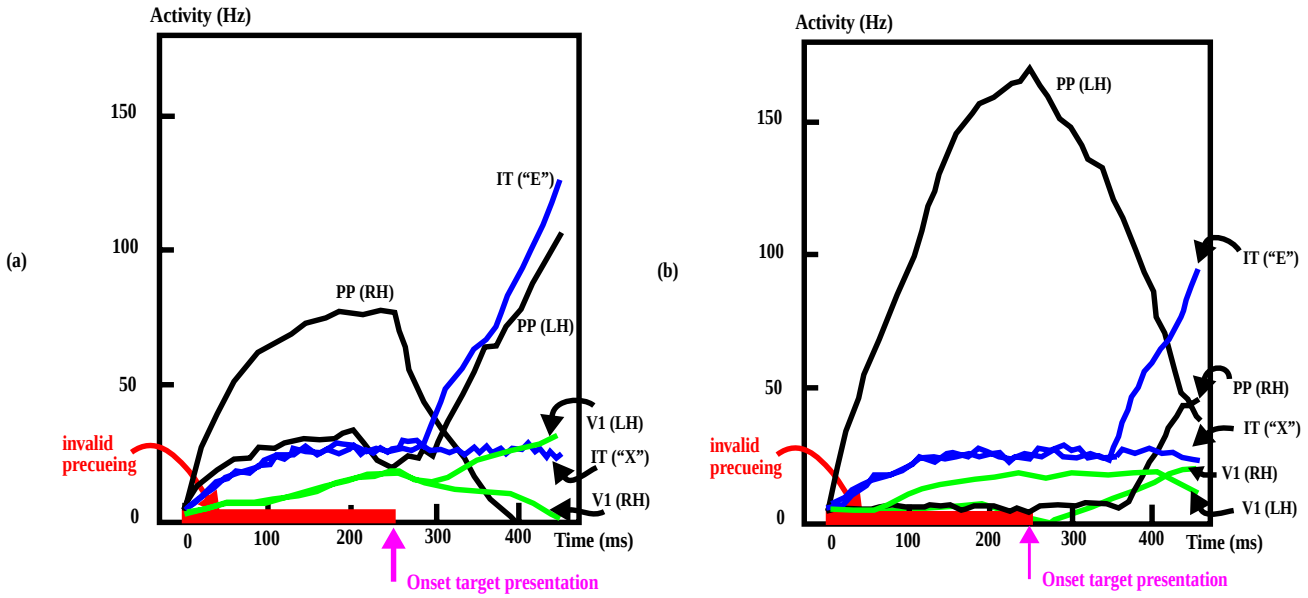

Figure 6. Dynamical evolution of the computational cortical system with pure unilateral damage of the PP module during the spatial precueing experiment. (a) ipsilesional stimulus presentation and invalid precueing; (b) contralesional stimulus presentation and invalid precueing. LH and RH denote left and right hemisphere respectively. The red bars indicate precuing duration.

Figure 6 shows the evolution of the underlying dynamics by plotting the maximum activities of target assemblies and the distractor assemblies in the different modules IT, V1 and PP. The case of invalid precueing for the presentation of a contralesional and ipsilesional stimulus is shown. In the ipsilesional stimulus case, the invalid precueing of the damaged right

PP hemisphere polarizes the PP activity in favour of the right hemisphere but very moderately, so that V1 is not even polarized at all (left versus right hemisphere). This is due to the fact that the pools in the PP right hemisphere do not react to the cue so efficient and fast as usually because of the intrinsic damage. On the other hand, in the contralesional case, the invalid precueing of the left PP hemisphere polarizes the PP activity strongly in favour of the left hemisphere and consequently the activity in V1 is also strongly polarized in favour of the left hemisphere. Hence, after presentation of the stimulus in the contralesional field, the dynamics of the cortical system takes much more time to invert the polarization in favour of the stimulus side in V1 and in the damaged and therefore slow right side of PP. Hence, in the contralesional stimulus case, the underlying biased intra- and intermodular competition in V1 and PP explains the unusual delayed polarization of the IT pools that causes the asymmetric spatial precueing effect shown in Figure 5.

5. Extinction Phenomenon and Visual Search

Another kind of extinction-like effect observed in patients with unilateral neglect is evidenced in the context of visual search experiments. Eglin et al. (1989) studied the behaviour of patients with unilateral parietal lesions during a visual search task. When visual search arrays were presented to only the contralesional or ipsilesional hemifields, the patients did not perform differently in the two hemifields. However, when search arrays were presented to both hemifields simultaneously, the patients' reaction time for target detection differed between the two hemifields. The reaction time for target detection in the intact hemifield was not affected by the presence of distractor stimuli in the contralesional hemifield. On the contrary, the reaction time for target detection in the contralesional hemifield was much slower when distractor stimuli were presented in the intact field. Thus, in spatial cueing and visual search tasks, patients with unilateral lesions of the parietal lobe perform poorly at target detection on the contralesional side, and the performance declines even more when stimuli are presented to the ipsilesional side, or when they are precued to expect a target to be in the ipsilesional hemifield.

The damaged underlying neurodynamical biased competition causes that attention to items in the intact hemifield affect processing in the contralesional hemifield. We simulate a simple version of visual search by using our computational model. Like in the last section, we impair the computational model by posing a pure unilateral damage of the right hemisphere of the PP module (equations (15) and (16)) in order to simulate the behaviour of patients with left-sided neglect. We use the same parameters and dimensions as in the last section. The system functions in an object attention mode. During the visual search task, the stimulus corresponding to the letter "E" is defined as target and the "X" as a distractor. Hence, the IT pool corresponding to the identification of the target letter "E" is reinforced positively by an external attentional object specific bias $I_i^{IT,A} = 0.07$ (i corresponds to the "E" pool). Four conditions are analysed: 1) target in the ipsilesional hemifield (TI1); 2) target in the ipsilesional hemifield and distractor in the contralesional hemifield (TI2); 3) target in the contralesional hemifield (TC1); 4) target in the contralesional hemifield and distractor in the ipsilesional hemifields (TC2). Figure 7.a shows the input display for the cases TI2 and TC2. The reaction time for target localization is as usually achieved when the PP polarization $P = A_T^{PP} - A_D^{PP}$ reaches a certain threshold $\theta = 0.05$. The reaction times for the four conditions are plotted in Figure 7.b. The computational simulation results agree with the experimental performance observed by neglect patients in visual search tasks of Eglin et al. (1989). Target detection on the contralesional hemifield is strongly delayed by the presence of a distractor in the opposite hemifield.

Figure 8 shows the evolution of the underlying dynamics by plotting the maximum activities of target assemblies and the distractor assemblies in the module PP for the TC2 and TI2 conditions.

When the target is in the neglected contralesional hemifield (TC2), the activity of the PP neuronal pools corresponding to the location of the distractor in the opposite undamaged hemifield is very high and therefore the attentional spatial competition between target and distractor pools is delayed. This delay is even augmented by the slowness of the intrinsically impaired PP pools associated with the target. In contrast, when the target is in the undamaged ipsilesional hemifield (TI2), the activity of the PP neuronal pools corresponding to the location of the distractor in the neglected hemifield, is much lower than in the TC2 condition because of the slowness of the impaired pools on the respective right PP hemisphere. This effect facilitates the attentional spatial competition between target and distractor pools. The fast increasing target activity supports even more the polarization in the PP module in favour of the target location. The pure unilateral damage in the PP module unbalances the dynamical competition so that it is more difficult to disengage spatial attention from stimuli in the undamaged ipsilesional hemifield.

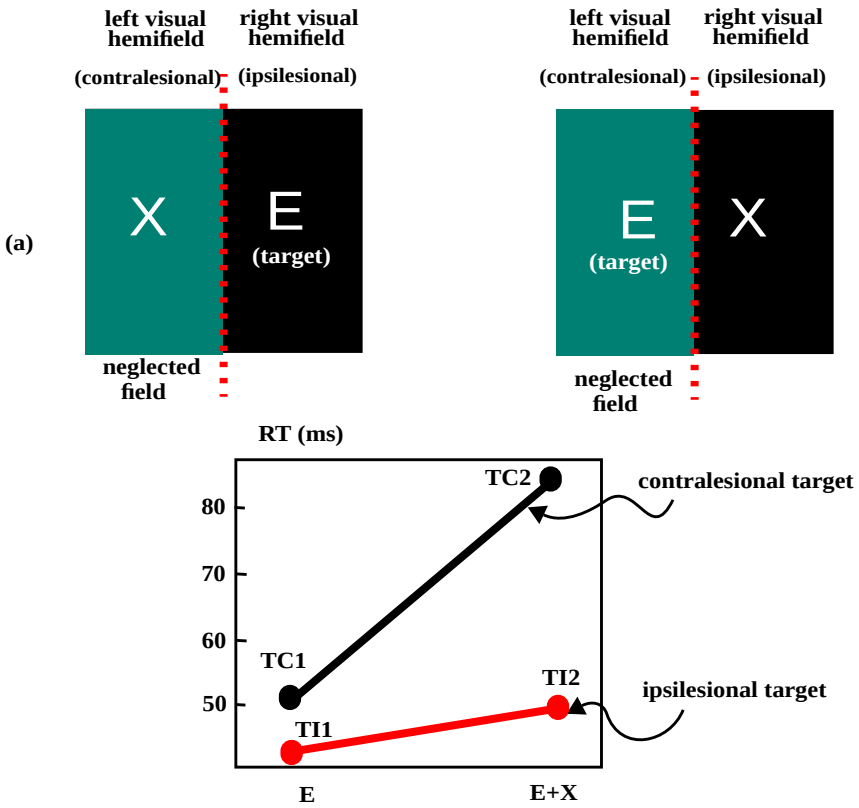

Figure 7. Extinction-like effect observed in patients with unilateral neglect in the context of visual search experiments. Four conditions are analysed: 1) target in the ipsilesional hemifield (TI1); 2) target in the ipsilesional hemifield and distractor in the contralesional hemifield (TI2); 3) target in the contralesional hemifield (TC1); 4) target in the contralesional hemifield and distractor in the ipsilesional hemifields (TC2). (a) Input display for the cases TI2 and TC2; (b) Reaction times for the four conditions.

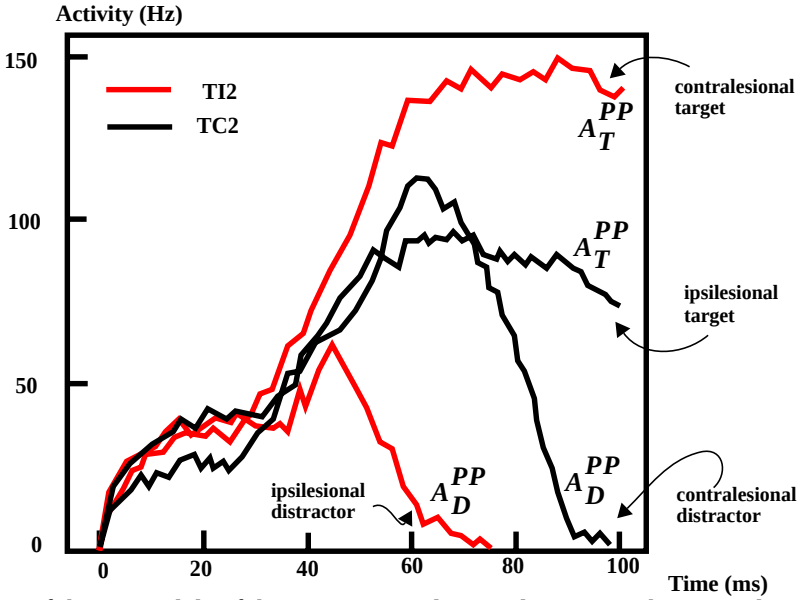

Figure 8. Dynamical evolution of the PP module of the computational cortical system with pure unilateral damage of the PP module during a visual search task. The curves correspond to the maximum activities of target assemblies and the distractor assemblies in the module PP for the TC2 (target in the contralesional hemifield and distractor in the ipsilesional hemifields) and TI2 (target in the ipsilesional hemifield and distractor in the contralesional hemifield) conditions.

6. Effect of Top-down Knowledge about Objects on Neglect

6.1 Computational Predictions

The influence of top-down knowledge about objects that have to be recognized can easily be studied in the framework of our computational model. We analyse the effect of object specific external top-down bias coming from the prefrontal area v46 and impinging on IT neuronal pools during the recognition phase. In concrete, we compare the dynamical evolution of the spatial attention activity in the module PP during the recognition of a previously learned object when a top-down reinforcement from prefrontal area v46 on the IT module is considered or is not take into account, respectively.

We use an input display given by a pixelized 203×265 image showing a specific woman face of the Carnegie Mellon Database. The V1 hypercolumns cover the entire image uniformly. They are distributed in 100×130 locations and each hypercolumn is sensitive to three spatial frequencies and to eight different orientations (i.e., $K = 3$ and $L = 8$). The PP module contains pools corresponding to each possible spatial location, i.e. to each of the 100×130 pixels, and a common inhibitory pool. An IT neuronal pool was trained for recognition of the specific face during a learning phase. We consider a pure unilateral damage of the right hemisphere of the PP module in order to simulate the behaviour of left neglect patients. As in Section 3, we symbolize with a red point the pixels at locations that correspond to a PP neuronal pool which has an activity larger than a certain threshold $\vartheta = 0.08$. These points can be interpreted as the scan path of the fixation locations during the perception of the object. During the recognition phase two conditions were analysed: 1) top-down object specific knowledge is used; 2) no top-down knowledge is used. The first condition is simulated by reinforcing positively with an external positive top-down bias $I_i^{IT,A} = 0.07$ the IT pool associated with the recognition of the specific face. The second condition is simulated by cancelling the external top-down bias impinging on IT. Figure 9 shows the simulated scan path of the fixation locations. The effect of top-down knowledge about the object causes an increase in the activity in the PP module even in the damaged right hemisphere. Hence, much more fixations appear in the ipsilesional visual field and even some fixations in the contralesional (left) visual hemifield can be observed. The pool activity in the damaged PP hemisphere is reinforced by the stronger activity in the corresponding right hemisphere in V1 module which are strongly stimulated by the feedbacks coming from the positively top-down reinforced IT pool. The mutually coupled intercortical dynamic predicts that top-down knowledge of the object to be recognized compensates slightly the abnormal scan path of fixations typically observed in visual neglect.

(a) neglect **(b) top-down effect on neglect**

Figure 9. Oculomotor pattern during the scanning of a face: (a) without top-down effect; (b) with top-down effect (computational simulation results). The effect of top-down knowledge about the object causes an increase in the activity in the PP module even in the damaged right hemisphere. Hence, much more fixations appear in the ipsilesional visual field and even some fixations in the contralesional (left) visual hemifield can be observed.

6.2 Experimental Results: Top-down Effect on Saccadic Patterns by a Neglect Patient

We present in this section, first experimental results obtained in collaboration with the Psychological Department of Birmingham University (England) that support the model predictions of last section. We analysed the influence of object knowledge on the saccadic patterns in a patient (MB) with mild left neglect.

MB suffered a stroke when aged 53 years. A CT scan revealed a right hemisphere lesion affecting the sylvian fissure, the inferior frontal and superior temporal gyri, the inferior parietal lobe, the caudate and lenticular nuclei, and the insula. There was no field defect on confrontation testing. However, MB initially showed unilateral neglect on a variety of clinical standard tests including: drawing from memory, copying, line bisection, and star cancellation. On the star cancellation task from Behavioural Inattention Test, MB omitted stars from the left-most third of the sheet. She also showed a consistent right-sided bias in line bisection (twelve 6 cm lines were all biased to the right of centre, with inaccuracy varying from 0.3 to 1.6 cm from the true centre). Apart from neglect there are few other signs of cognitive deficits. MB was well oriented time and space; she showed no problems with speaking or with speech comprehension and no clinically-apparent memory deficit. The present tests started 6 months after her lesion and continued over a 6-month period, during which MB's deficit was stable.

In this single-case study, four objects are used: "car", "lorry", "ship" and "cargo ship". Each of the objects appeared at two different locations in the images. Figure 10 shows 4 examples of the images which were used in the experiment. The different locations were chosen so that the axis marked as broken line in Figure 10 was always aligned with the centre line of the screen. Noise was applied to each image, so that the images imitate the "Mooney" pictures (see Figure 11 for an example). The noise was set to a level which made it difficult for MB to recognize the objects without any additional information.

The images were shown in two blocks to MB. In the first block MB had no knowledge about the possible objects hidden in the noise and she was asked to guess what objects could be hidden. Before the second block, the objects without noise were shown to MB and the names of the objects were pointed out to her. She was also informed that the object might appear at different locations. After that, the same images were shown to her in a different order. This time she was asked to name the object she could identify in the image. We recorded the eye movement of MB during the experiment.

Figure 10. Illustration of the four objects shown to patient MB. For the analysis of the results the location of the fixations in the objects were categorized into two different areas (right and left).

Figure 12 shows the results of the experiment. In order to analyse the eye movements, the area of the four objects was separated into two different sections: right and left (see Figure 10). The frequency of the fixations for the areas for each image

was computed and the average of frequencies for each object at each position was determined. This was done separately for block one and two. In the first block, before learning, none of the objects was recognized.

Figure 11. Example of Mooney stimulus (car).

After learning, 10 out of 16 were correctly identified. The frequency of fixations in the left part of the object increases after learning according to the model predictions. ANOVA with objects (car, lorry, ship, and cargo ship) as random factor and location of the object as fixed factor was applied.

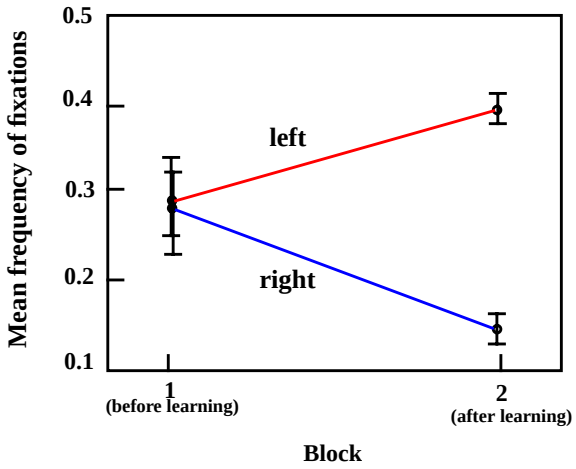

Figure 12. Mean frequency of fixations on the left and right side of the different objects during the first block before learning and second block after learning. The frequency of fixations on the side of the object that lies on the contralesional neglected hemifield increases if the object is known.

The block factor (before and after learning) had a significant influence on the frequency of fixations on the left side ($F(1,47)=6.87$, $p<0.05$) and on the right side ($F(1,47)=4.37$, $p<0.05$). Figure 13 shows a typical example of the scan path of fixations before and after learning (for comparison with the predictions of the theoretical model see Figure 9).

In conclusion, the experimental data confirm the theoretical predictions of our neurodynamical model, namely that the frequency of fixations on the side of the object that lies on the contralesional neglected hemifield increases if the object is known. This after learning effect validates the reinforcing influence on the neural activity of the top-down feedback interactions. Interactions coming from the inferotemporal cortex through the what-path into the primary visual cortex, are further transmitted through the where-path to the posterior parietal cortex. As a consequence, top-down interactions enhance the neural activity even in the damaged posterior parietal hemifield. This enhancement of neural activity can be interpreted as more deployment of spatial attention on the associated contralesional region that consequently attracts more saccades than before learning.

(a) Block1: before learning **(a) Block2: after learning**

Figure 13. Example of alteration of scan path due to the learning effect (left first block before learning and right second block after learning). Some fixations appear in the contralesional (left) visual hemifield after learning.

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