

Simulating a Lesion in a Basis Function Model of Spatial Representations: Comparison With Hemineglect

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The basis function theory of spatial representations explains how neurons in the parietal cortex can perform nonlinear transformations from sensory to motor coordinates. The authors present computer simulations showing that unilateral parietal lesions leading to a neuronal gradient in basis function maps can account for the behavior of patients with hemineglect, including (a) neglect in line cancellation and line bisection experiments; (b) neglect in multiple frames of reference simultaneously; (c) relative neglect, a form of what is sometime called object-centered neglect; and (d) neglect without optic ataxia. Contralateral neglect arises in the model because the lesion produces an imbalance in the salience of stimuli that is modulated by the orientation of the body in space. These results strongly support the basis function theory for spatial representations in humans and provide a computational model of hemineglect at the single-cell level.

A unilateral lesion of the parieto-occipital cortex in humans often produces hemineglect (Heilman, Watson, & Valenstein, 1985; Pouget & Driver, 1999; Vallar, 1998), a neurologic syndrome characterized by a conspicuous inability to react or respond to stimuli presented in the hemispace contralateral to the lesion. For example, when asked to mark off a set of line segments uniformly spread over a sheet of paper, patients typically fail to cross out the segments located in the contralesional space (Figure 6A).

Several theories have been proposed to account for neglect, falling into two major categories: attentional and representational neglect (see Vallar, 1998, for a review). According to the attentional theory, the lesion affects the mechanisms responsible for orientation toward the contralateral side of space (Kinsbourne, 1987; Posner, Walker, Friedrich, & Rafal, 1984). In contrast, the representational theory postulates that the spatial representation of the contralateral side of space is damaged or distorted (Bisiach & Luzzatti, 1978; Halligan & Marshall, 1991; Rizzolatti & Berti, 1990).

Strong evidence exists for both theories, which are only two extremes from a continuum of possibilities. The distinction be-

tween orienting mechanisms and the representations on which they act may not be straightforward, because the same neurons involved in spatial representations may also be involved in attention (Andersen, 1995; Cohen, Farah, Romero, & Servan-Schreiber, 1994; Gottlieb, Kusunoki, & Goldberg, 1998). Consequently, it is conceivable that the syndrome affects both components (Pouget & Driver, 2000).

One issue that has dominated research over the last few years within the representational theory of neglect concerns the frame of reference of the neglected hemispace. One possibility is that right parietal patients neglect any object located on the left side of their trunk regardless of their head or eye position. Conversely, they might neglect objects on the left side of their head even if these objects are located on the right side of their trunk (a situation occurring, for example, when the head is rotated 90° toward the right).

Numerous experiments have addressed this issue and have reached the conclusion that neglect always affects multiple frames of reference. Hence, neglect can be retinocentric, head centered, trunk centered, and environment centered (Behrmann & Moscovitch, 1994; Beschin, Cubelli, Salla, & Spinazola, 1997; Bisiach, Capitani, & Porta, 1985; Calvanio, Petrone, & Levine, 1987; Farah, Brunn, Wong, Wallace, & Carpenter, 1990; Karnath, Christ, & Hartje, 1993; Karnath, Schenkel, & Fischer, 1991; Ladavas, 1987; Ladavas, Pesce, & Provinciali, 1989; Moscovitch & Behrmann, 1994). Moreover, with the exception of one study (Beschlin et al., 1997), these frames of reference were found to be concomitantly affected in all patients and to a first approximation, independent of the task. Some patients also neglect the left side of objects whether they appear in the "good" or "bad" hemispace, a phenomenon often called object-centered neglect (Caramazza & Hillis, 1990; Driver & Halligan, 1991; Driver, Baylis, Goodrich, & Rafal, 1994; Tipper & Behrmann, 1996). These findings suggest that the spatial representations in the parietal cortex may encode

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the location of an object in multiple frames of reference simultaneously.

Only a few attempts have been made to reconcile these findings regarding multiple frames of reference with what is known about spatial representations at the cellular level in the parietal cortex (Anderson, 1996; Behrmann, Moscovitch, & Mozer, 1991; Monaghan & Shillcock, 1998; Mozer & Behrmann, 1990; Mozer, Halligan, & Marshall, 1997). These models, however, address only the retinocentric aspects of neglect and are limited to neglect dyslexia or line bisection experiments (an experiment in which patients are asked to judge the midpoint of a line).

The lack of modeling is perhaps related to the fact that, until recently, little was known about the response properties of cortical neurons involved in spatial representations and orienting mechanisms. Over the last decade, however, several studies using single-cell recording techniques in behaving monkeys have elucidated the properties of neurons in the parietal cortex (Andersen, Snyder, Bradley, & Xing, 1997; Colby, Duhamel, & Goldberg, 1995). Andersen (1995), in particular, provided a detailed account of how parietal cells integrate sensory and postural signals.

On the basis of these experimental results, we have developed a neuronal theory of spatial representations that relies on the mathematical concept of basis functions (Pouget & Sejnowski, 1994, 1995; see later discussion for a short summary of this approach). We reason that, if this framework provides a theory of spatial representations in the parietal cortex, it should also predict the behavior of hemineglect patients. Indeed, the basis function model was developed solely on the basis of computational principles and neurophysiological data. Consequently, neuropsychological data and, in particular, the behavior of hemineglect patients can be used as an independent test for this hypothesis.

Therefore, the main goal of this article is to show that a simulated unilateral lesion of a basis function representation can replicate a wide variety of behaviors that have been reported in neglect. By doing so, we mean to provide the first link between the behavior of hemineglect patients and its neural underpinnings.

Although our model is restricted to the parietal cortex, it can be readily generalized to other parts of the brain. For instance, neglect

has been reported after lesions of the prefrontal lobe and other subcortical structures (Heilman et al., 1985). It is conceivable that the basis function hypothesis could also account for these other forms of neglect. However, this question is difficult to address because few behavioral studies focus on these other forms of the syndrome and little is known about the response properties of the cells in these other areas. Therefore, our focus on parietal cortex is primarily dictated by the currently available data.

In this article, we first summarize the basis function theory and its neural basis. Second, we describe the network architecture along with the mechanisms used to assess the performance of the network in a variety of experimental tasks. Third, we compare the results obtained with the damaged network with the performance of left-neglect patients on several representative sets of experiments: (a) a simple detection and reaction time task; (b) the line cancellation and line bisection tasks used clinically; (c) tests for frames of reference; (d) conditions that elicit object-centered neglect; and (e) simple reaching to objects. Finally, we discuss the implications of this model for theories of neglect.

Basis Function Representation

Most neurons in the lateral and medial parietal areas (LIP, MIP) and in area 7a respond to visual stimuli flashed within their receptive fields (Andersen et al., 1997). As in the primary visual cortex, these neurons have retinotopic receptive fields; that is, they respond only when the stimulus impinges within a particular part of the retina. These receptive fields, however, are typically much larger than those found in V1; angular sizes range anywhere between a few degrees to a whole hemifield. They often have a characteristic bell-shaped profile, as illustrated in Figure 1A, although it is also common to find receptive fields with multiple peaks, which are best modeled by mixtures of Gaussians.

In addition to their sensory response, parietal neurons are also often selective to the position of the eyes in the orbit. This sensitivity can be revealed by mapping the retinal receptive field of a cell for various eye positions. These experiments have shown that eye position modulates the gain of the visually evoked re-

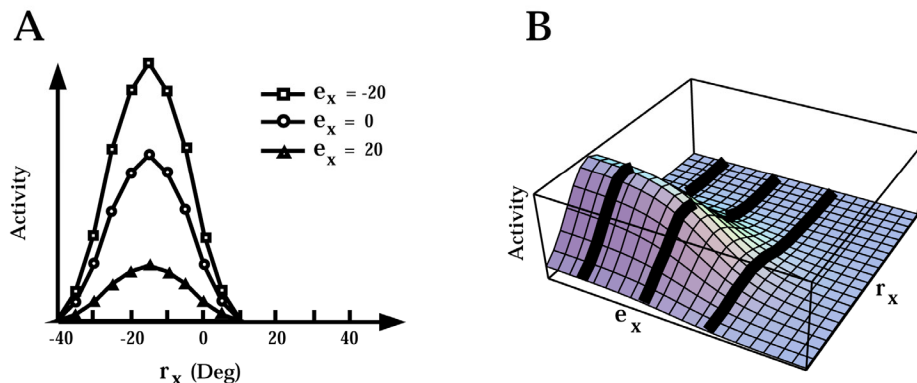


Figure 1. Gain field of a neuron in the monkey parietal cortex illustrated in two different graphical plots. Panel A: Idealized retinotopic visual receptive field of a typical parietal neuron for three different gaze angles (e_x). Note that eye position modulates the amplitude of the response but does not affect the retinotopic position of the receptive field (data from Andersen et al., 1985). Panel B: Three-dimensional plot showing the response functions of an idealized parietal neuron for all possible eye and retinotopic positions, e_x and r_x . The plot in Panel A was obtained by mapping the visual receptive field of this idealized parietal neuron for three different eye positions as indicated by the hold lines in Panel B. Deg = degree.

sponse. This effect is shown in Figure 1 A for an idealized parietal neuron. The shape and position of the receptive field in retinotopic coordinates are unaffected by eye position, but the amplitude, or gain, of the receptive field is dependent on where the animal fixates on the screen.

In most cases, the gain of the sensory response increases monotonically as the eye moves along a particular direction in space, which is specific to each cell (Andersen, Essick, & Siegel, 1985). For the example shown in Figure 1A, the gain increases as the eye moves further to the left.

In a previous study, we showed that these responses can be described as a Gaussian function of retinal location multiplied by a sigmoid function of eye position (Pouget & Sejnowski, 1997). In Figure 113, we show the response of an idealized parietal neuron whose response follows such a profile. A mapping of the visual receptive field of this idealized parietal neuron for three eye positions (as indicated by the bold lines in Figure 1B) can indeed account for the gain modulation of a retinotopic bell-shaped receptive field by eye position shown in Figure 1A.

The fact that the response of parietal neurons can be modeled as a product between a Gaussian and a sigmoid function suggests that parietal neurons compute basis functions of their inputs (Pouget & Sejnowski, 1995, 1997). Indeed, Gaussian and sigmoid functions are basis functions, and the set of functions obtained by taking the product of Gaussian and sigmoid functions is itself a basis set.

A complete set of basis functions has the property that any nonlinear function can be approximated by a linear combination of the basis functions (Poggio, 1990). Therefore, basis functions reduce the computation of nonlinear mappings to linear transformations, a simpler computation.

This computational property of basis functions has some important implications in the context of spatial representations. Spatial representations in the parietal cortex are thought to play a critical role in sensorimotor transformation, a perspective that led Goodale and Milner (1990) to call the dorsal pathway of the visual system the "how" pathway to emphasize its role in object manipulation as opposed to localization. If the parietal cortex is indeed combining sensory information with posture signals to compute motor commands, one would expect that the code found in the parietal cortex simplifies the computation of motor commands. This is precisely what a basis function representation can provide. Indeed, most motor commands, such as reaching for an object, are nonlinear functions of sensory and postural signals. As such, they can be approximated by a linear combination of basis functions of the sensory and postural signals. Consequently, parietal neurons can reduce nonlinear sensorimotor transformation to linear mapping if they compute basis functions of the sensory and postural signals.

Because the experimental data are indeed consistent with the hypothesis that parietal neurons compute basis functions, it appears that parietal neurons simplify the computation of motor commands. Not all models of parietal cells have this computational property. For example, Goodman and Andersen (1990) as well as Mazzoni and Andersen (1995) proposed that parietal cells are linear (i.e., they simply add the retinal and eye position signals). If this is the case, computing a nonlinear motor command would require a nonlinear combination of the response of parietal neurons. Computing and learning these nonlinear transformations would require more neuronal resources than what is required when using basis function (Pouget & Sejnowski, 1995, 1997). In con-

trast, the hidden units of the Zipser and Andersen model (1988) for head-centered representations in parietal cortex have response properties closer to those of basis functions, and the basis function model can be considered a formalization of this previous model (for a detailed discussion, see Pouget & Sejnowski, 1997).

Interpreting the response of parietal neurons as basis functions has several important implications for our model of hemineglect. First, this interpretation entails that parietal neurons encode the location of objects in multiple frames of reference simultaneously. Indeed, one can recover simultaneously the position of an object in retinocentric and head-centered coordinates from the responses of a group of basis function neurons similar to the one shown in Figure 1B (Pouget & Sejnowski, 1995, 1997). The reason is that recovering the position of an object in head-centered coordinates requires a function to be computed that depends on the retinal position of the object and the current eye position, which can be approximated by a linear combination of the response of basis function units. The same is true for the retinal location of an object.

Second, if parietal neurons compute basis functions, they can contribute to the computation of several motor commands toward the same object. The reasons are the same as before. Moving the eyes and hand toward an object requires two distinct motor commands to be computed, each specified in the appropriate set of coordinates for its respective effector. Again, these commands are functions of the retinal position of the object and the current eye position, which can be approximated by linear combinations of basis functions. As shown in the next section, this property allows the same set of neurons to perform multiple spatial transformations in parallel (Figure 2A).

Although we have considered only basis functions of eye position and retinal location, this approach can be extended to other sensory and postural signals and to other parts of the brain where similar gain modulation has been reported (Bremmer, Ilg, Thiele, & Hoffmann, 1997; Boussaoud, Barth, & Wise, 1993; Brochier, Andersen, Snyder, & Goodman, 1995; Field & Olson, 1994; Guo & Li, 1997; Trotter, Celebrini, Stricanne, Thorpe, & Imbert, 1992). When generalized to other postural signals, such as vestibular inputs of head position, the resulting representation encodes simultaneously the retinal, head-centered, body-centered, and world-centered coordinates of objects.

This study explores the effects of lesioning a model of spatial representation in the parietal cortex that uses basis functions. It provides a bridge between our current understanding of spatial representations at the neurophysiological and neuropsychological levels.

Method

Network Architecture

The overall network architecture is shown in Figure 2A. The input contains two neuronal maps: a retina and a map-encoding eye position. The one-dimensional retina has 81 units, each of which responds in proportion to the image intensity at the corresponding retinal location. It was assumed to extend from -40° to 40° for a total range of 80° .

The 41 eye position units respond with a sigmoid function of eye position:

$$a_j^E = \frac{1}{1 + \exp(-(e - e_j)/\mu)} \quad (1)$$

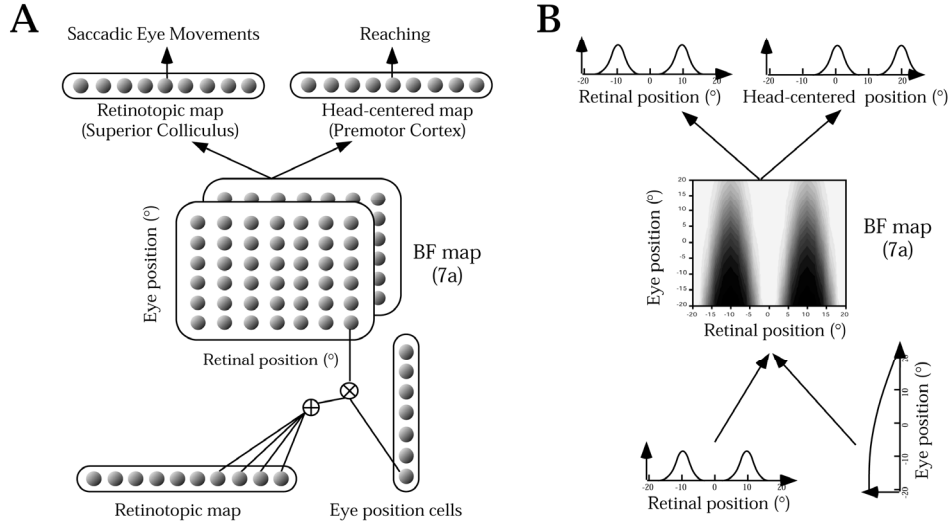


Figure 2. Feedforward network architecture for computing the positions of objects in several different reference frames simultaneously using a set of basis functions. Panel A: Each unit in the intermediate layers is a basis function with a Gaussian retinal receptive field modulated by a sigmoid function of eye position. This type of modulation is characteristic of responses from parietal neurons. Only two basis function (BF) maps are shown: one for the right and one for the left hemisphere. In the simulation, we used two maps in each hemisphere for a total of four maps. See text for details. Panel B: Pattern of activity for two visual stimuli presented at 10° and -10° on the retina with the eye pointing at 10° . Instead of showing the actual activities in the retina of the model (which would look like two delta functions in this case), we show the retinal activity convolved by the Gaussian weights (Equation 2). This is the actual retinal input that reaches the basis function units.

where e is the current eye position, μ is the slope of the sigmoid, and $\bar{e}_j$ is the abscissa of the inflection point of the sigmoid (the point at which the sigmoid reaches half of its maximum value). The slope, μ was set to 8° , a value for which the cell response saturates at roughly 30° on either side of the inflection point, leading to a dynamical range of 60° . The $\bar{e}_j$ points were systematically varied along the eye position map from -20° to $+20^\circ$ in increments of 1° . A second map (not shown) had units with similar response functions but opposite slope (-8° instead of 8°). This second map was introduced because eye position-sensitive neurons in the brain have both positive and negative slopes (Andersen, Asanuma, Essick, & Siegel, 1990; Andersen et al., 1985; Schlag-Rey & Schlag, 1984).

The intermediate layer contained four 41×41 basis function maps meant to model a population of neurons in the parietal cortex (only two are shown in Figure 2A). The 41×41 is not the number of units per map but the number of types of units per map. We later describe how we determined the number of units per type.

Two of these maps were assigned to the right hemisphere and the other two to the left hemisphere. These two groups of maps had identical patterns of connectivity from the input maps and differed only in the number of units per map (see next section). Both maps received inputs from the retina, but the first map received connections only from the first eye position map (slope $+8^\circ$), and the second map received connections only from the second eye position map (slope -8°). It would have been easier to use only one map per hemisphere, with neurons in the left map receiving connections from the eye position neurons with a positive slope and the reverse in the right hemisphere. This is, however, inconsistent with the data (Andersen et al., 1985, 1990): There are neurons with positive and negative slopes in each hemisphere, hence the need for two maps.

Within each map, a basis function unit located at position ij received one connection with a weight of 1 from one eye position unit located at position j on the eye position map (see Figure 2B). It also received a set of connections from 41 retinal units resulting in a Gaussian receptive field centered on position $\bar{r}_i$ on the retina. The retinal and eye position

were combined multiplicatively such that the overall the response of a unit was given by:

$$a_{ij} = a_j^E \sum_{k=-20}^{20} w_{ik} a_{i+k}^R, \quad (2)$$

where

$$w_{ik} = \exp\left(-\frac{(\bar{r}_i + \bar{r}_{i+k})^2}{2\sigma^2}\right). \quad (3)$$

a_{i+k}^R is the activity of a retinal unit at position $i+k$, and $\bar{r}_i$ is the center of the retinal receptive field on unit i (see Figure 2B).

As a result of the convergence between the retina and the basis function map (i.e., the sum over k in Equation 2), the basis function map is only 41 units wide along the retinal dimension compared with 81 for the retina. This entails that the centers of the retinal receptive field of the basis function units extend from -20° to 20° by increments of 1° compared with -40° to 40° for the retina.

The receptive field width, σ , was set to 5° for most experiments and 2° for experiments in which more than two objects were presented simultaneously. These values correspond to the narrowest receptive field widths observed in the parietal cortex (Andersen et al., 1985). Wider receptive fields did not change our results for a single object, but problems occurred when several objects were present at the same time. The equation used for the receptive fields (the retinal term in Equation 2) is linear; consequently, the network cannot distinguish different objects when they are separated by less than the width of the receptive field. There is evidence that the retinal receptive fields of parietal neurons are not linear (Constantinidis & Steinmetz, 1995; Gottlieb et al., 1998), but little is known about how multiple objects are handled by parietal cells. Linear receptive fields with small widths were adopted to avoid this issue. A model of area MST, which

projects to the parietal cortex, has been developed that can account for the partial segmentation of transparent and occluding moving objects (Zemel & Sejnowski, 1998). Similar preprocessing mechanisms may allow more complex scenes to be handled by a basis function representation in the parietal cortex.

In most simulations, the visual stimulus was modeled as a point with a fixed intensity. In this case, the pattern of activity on the retina was simply a Gaussian function centered on the retinal location of the stimulus. For a stimulus like an elongated bar, used in tasks such as the line bisection test, unit activity depended also on the spatial extent of the stimulus.

Finally, the network had two output maps: one in retinocentric coordinates and the other in head-centered coordinates. A similar retinotopic map is found in the superior colliculus (Sparks, 1991), and there are head-centered maps in the premotor cortex (Fogassi et al., 1992; Graziano, Yap, & Gross, 1994) and the ventral parietal area (Duhamel, Bremner, Bentsen, & Graf, 1997). These output layers received inputs from all the units in the four basis function maps and their activity was computed according to

$$o_i = \sum_{j=1}^4 \sum_{k=1}^{41} \sum_{l=1}^{41} w_{ijkl} n_{jkl} a_{jkl}, \quad (4)$$

where a_{jkl} is the activity of the basis function unit at position kl in the basis function map j and n_{jkl} is the number of basis function of a particular type (see *Hemispheric Distribution*).

A gradient descent procedure was used to find the appropriate sets of weights to obtain Gaussian retinotopic receptive fields in the retinotopic output map and Gaussian head-centered receptive fields in the head-centered output map. The existence of such weight matrices is guaranteed by the fact that the units form a complete set of basis functions, as demonstrated previously (Pouget & Sejnowski, 1995). Figure 2B shows a typical pattern of activity in the network when two objects were presented at -10° and 10° on the retina and the eyes were fixated at 10° . Note that the activity in the head-centered map peaked at 0° and 20° , which is indeed the head-centered location of these two stimuli (the head-centered location of an object is roughly equal to the sum of the retinal location and current eye position, see Westheimer, 1957, for details).

Hemispheric Distribution

We have assumed that maps in each hemisphere contain all possible types of basis functions, but the number of units for each type in each map was chosen to overrepresent the contralateral side of space. Hence, in the

left maps, a majority of units preferred right retinal positions and right eye positions: that is, there were more units whose activity increased with right eye position.

Note that a particular type of basis function is characterized by a pair of values $(\bar{r}_i, \bar{e}_j)$ that correspond to the peak position of the retinal receptive field and the position of the inflection point of the sigmoid function of eye position. The gradients were chosen to be linear functions of these variables. For example, in the left hemisphere, the number of units of each type was given by

$$N_{ij} = \lambda_r \bar{r}_i + \lambda_e \bar{e}_j + \epsilon_1, \quad (5)$$

for the map with units whose activity increases with greater rightward deviation of the eyes, and

$$N_{ij} = \lambda_r \bar{r}_i - \lambda_e \bar{e}_j + \epsilon_2, \quad (6)$$

for the map with units whose activity decreases with eye position. Symmetric gradients with respect to a vertical axis were used for the two maps in the right hemisphere. Therefore, the right and left maps together formed a uniform representation of space with the same number of units assigned to each spatial location. Figure 3 illustrates the gradients in the left and right maps for units having increasing activity with eye position.

In both left maps, the number of units was proportional to $\bar{r}_i$, the position of the retinal receptive field for unit type i (λ_r was set to 1 in most simulations). This creates a gradient such that the left maps contained more neurons with their retinal receptive fields centered on the right hemiretina. This gradient is consistent with the results of electrophysiological studies that have demonstrated that most neurons in the left hemisphere in monkeys have their receptive fields in the right hemiretina (Andersen et al., 1990).

The gradient along the eye position axis is controlled by the parameters λ_e , ϵ_1 and ϵ_2 . This gradient is somewhat difficult to visualize because the eye position selectivity is characterized not by a preferred eye position but by an inflection point, $\bar{e}_j$. Moreover, there are neurons in the parietal lobe with positive and negative slopes (the parameter μ , in Equation 1) in each hemisphere (Andersen et al., 1985, 1990). Altogether, the gradients are arranged so that the summed activity of all the units in the left maps is greater when the eyes point to the right than when they point to the left and vice versa in the right map. In most simulations, we set to λ_e , ϵ_1 , and ϵ_2 to 0.5, 80, and 40. These values were selected because they lead to a salience gradient (defined in the *Target Processing* section) in the damaged network that is roughly linear with eye position. Other values would lead to similar quantitative results, but a linear salience gradient simplifies the description of the results.

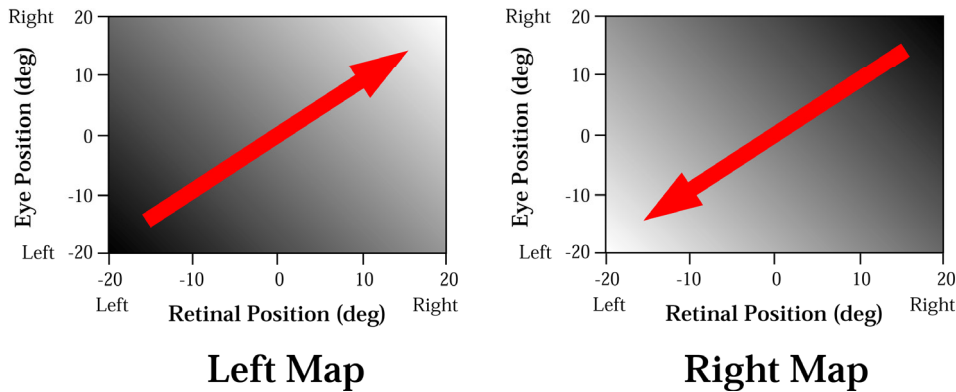


Figure 3. Neuronal gradients in left and right basis function maps for units whose activity increases with greater rightward deviation of the eyes. The right map contains more neurons for left retinal and left eye positions, whereas the left map has the opposite gradient. deg = degree.

Whether similar eye position gradients exist in the brains of monkeys or humans is still being investigated. In the cortex, Galletti and Battaglini (1989) reported that neurons in area V3a tend to prefer contralateral eye positions, as predicted by our model, but this was not statistically significant. No trend was reported in Andersen et al.'s (1990) study in parietal areas LIP and 7a, or the one by Bremmer et al. (1997) in areas MT and MST, but eye positions were never tested beyond $\pm 20^\circ$, in contrast with retinal positions, which were tested up to $\pm 70^\circ$. Evidence exists for such bias in subcortical structures such as the superior colliculus (Van Opstal, Hepp, Suzuki, & Henn, 1995) and the intralaminar nuclei of the thalamus, a structure that may provide the eye position signals for the parietal cortex (Schlag-Rey & Schlag, 1984). An imaging study in humans has also revealed a contralateral gradient for eye position signal in the human parietal lobe (DeSouza et al., 2000).

In the absence of adequate data concerning the profile of the gradient, linear gradients were chosen but the qualitative aspects of our results do not depend on this assumption. Other monotonic gradients would predict the same qualitative results as those reported here as long as the left hemisphere is more active than the right hemisphere for right retinal and right eye positions.

Lesion Model

The spatial representations in our network were symmetrical, and lesions of the left and right hemispheres were equivalent. Because most articles describe patients with right parietal lesions, we present results simulating a lesion of the right side. The lesion was modeled by deleting the right maps. The behavior of the network was then studied with the biased left maps under various experimental conditions.

We also simulated partial lesions by manipulating the profile of the neuronal gradients in the left maps. For example, steep profiles, having high values for the slopes λ_e and λ_r , in Equation 5, corresponded to severe lesions and, conversely, shallower profiles were equivalent to milder lesions.

As mentioned, neglect is observed predominantly after right parietal lesions. This is not addressed directly in the present model, but, as explained in the General Discussion section, our model can be easily modified to incorporate this hemispheric bias.

Other Sensory and Postural Signals

Our approach was extended to other combinations of sensory and postural signals by using the same types of representations used for the visual fields and the eye positions: Gaussian sensory receptive fields were multiplied by a sigmoid function of the postural signal, with contralateral gradients within the basis function maps.

Target Processing

Many experiments on patients with hemineglect investigated not only the spatial aspects of the deficit but other facets as well. Some experiments involved simple detection, whereas others used reaction time, memorization, drawing, verbal reports, and other cognitive functions. This study was mainly concerned with the spatial deficit in neglect, but some simulations required simple models of several cognitive processes such as target detection, selection, and memorization. Whenever possible, established models for these cognitive process were adapted for these purposes.

Unfortunately, most of these existing models are not based on distributed representations of the kind used in the present model. Models of stimulus selection, for instance, typically use local representations in which a stimulus is characterized by one number, usually the activity of a single unit (Burgess, 1995). In contrast, distributed patterns of activity occur in the basis function maps of the present model to represent one or several stimuli (see Figure 2B).

Therefore, we had first to reduce the dimensionality of our representation before an existing model of target selections could be used. One possibility is to sum the activity of all the units responding to a particular stimulus. Because it is not always easy to assign neurons to each stimulus, a variation of this idea was used: A stimulus salience, s_i , was defined as the sum of the activity of all the basis function units whose receptive fields were centered exactly on the retinal position of the stimulus, r_i :

$$s_i = \sum_{j=1, r_{0j}=r_i}^{41} N_{ij} a_{ij}. \quad (7)$$

This method is mathematically equivalent to defining the salience of the stimuli as the peak in the profile of activity in the superior colliculus output map. Qualitatively similar values can also be obtained from the profile of activation in the head-centered map, although peak values were in general quantitatively different in this map.

d'

In signal-detection theory, d' , a measure of the signal-to-noise ratio, is proportional to the amplitude of the signal generated by a stimulus—the stimulus salience in our model—and is inversely proportional to the amount of noise. The exact relationship between d' and salience depends on the noise model, but in all cases the larger the signal, the larger is d' . Consequently, d' and salience are qualitatively equivalent measures.

Target Sequential Selection

The model of the line cancellation task required a model for the temporal selection of stimuli. We used a selection mechanism similar to that used by Burgess (1995). This mechanism operated on the reduced salience representation described in the previous section. Thus, for each stimulus, the associated saliences were first computed using Equation 7, and then the selection mechanism was applied to the resulting values.

The selection process is initialized by computing the salience of all stimuli according to Equation 7. Then, three steps are executed every time increment:

1. Winner-take-all: The stimulus with the highest activity value was selected.
2. Inhibition of return: The activity of the selected stimulus was set to zero.
3. Recovery: the salience over time for stimulus i , $s_i(t)$, is updated according to

$$s_i(t+1) = s_i(t) + \tau(s_i - s_i(t)) + n(t), \quad (8)$$

where $n(t)$ is a white noise process with a normal distribution and s_i is the salience of stimulus i as defined in Equation 7.

The time increment was assumed to be 100 ms long, and these steps were repeated for the entire duration of the trial. The duration of a trial was different for each experiment.

The inhibition of return was essential to prevent the system from selecting the same stimulus repeatedly. However, because saliences were allowed to recover, the stimulus with the highest salience could compete for selection even if it had been previously selected. The probability that a stimulus would be selected among a group of stimuli depends on its relative salience and the rate of recovery, τ . When τ is high (0.6), only the stimuli with the two or three highest saliences were selected.

It is possible to implement a selection mechanism equivalent to the one described here by using lateral connections within the basis function map (Cohen et al., 1994). This implementation does not make an artificial distinction between the representation and the selection mechanism, as made here, and is more biologically plausible. This is indeed our view of the parietal cortex; we believe that the same cells contribute to attention and spatial representations. However, the model of Cohen et al. (1994)

required complex dynamics and computation-intensive simulations and would have produced the same results as the present model. Thus, the selection mechanism used here was motivated by practical considerations.

Reaction Time

In reaction time experiments, it was assumed that processing involved two sequential steps: target selection and target processing. The time needed for target selection was proportional to the number of iterations, n , required by the selection network to select the stimulus. The time required for target processing (including target recognition, naming, and response) was inversely proportional to stimulus salience, s_i . The total reaction time, expressed in milliseconds, was the sum of these components:

$$RT = 100 + 50n + \frac{1}{\rho s_i}. \quad (9)$$

The constant ρ was adjusted to obtain reaction times comparable to one reported in patients.

Visual Attention

A spotlight of attention was modeled as a 10% local increase in salience. This provided a competitive advantage to the targets in the spotlight.

Detection and Recognition Performance

The probability of correctly detecting a stimulus was taken to be a sigmoid function of the stimulus salience. This recognition model was based on signal-detection theory when assuming Gaussian noise of equal variance for signal and noise (Green & Swets, 1966). This model predicts that the rate of correct detection (hit rate) is equal to the integral of the probability distribution of the signal from the decision criterion to infinity. This integral can be approximated with the sigmoid function

$$p = \frac{0.5}{1 + \exp(-(s - s_0)/t)} + 0.5 \quad (10)$$

where the parameters t and s_0 , which are the slope and threshold of the sigmoid, were tuned to match human experimental data (see *Simulations: Frames of Reference* subsection). Recognition was not explicitly modeled in the network in the sense that the basis function units had no particular selectivity for the shapes of objects. Although this is a major simplification of the actual computation performed in the cortex, it is sufficient for the purpose of this model.

Line Bisection

In line bisection experiments, individuals are asked to judge the midpoint of a line segment. In some experiments, the orientation of the line is varied from trial to trial. Simulating these experiments required that we use a 41 X 41 two-dimensional retina as opposed to the one-dimensional retina used in all other simulations. Furthermore, we assumed that the eye position of the network was fixed during this test. This allowed us to ignore the eye position signal in the basis function maps, whose sizes were, therefore, kept at 41 X 41. In such maps, each basis function unit is indexed by a horizontal and vertical retinal location, $\vec{r}_k^x$ and $\vec{r}_l^y$ which we denote as the vector $\vec{r}_{kl}$. The network estimated the midpoint of a line, m , by computing the center of mass of the activity in the network induced by the line:

$$\vec{m} = \frac{\sum_{j=1}^4 \sum_{k=1}^{41} \sum_{l=1}^{41} a_{jkl} \vec{r}_{kl}}{\sum_{j=1}^4 \sum_{k=1}^{41} \sum_{l=1}^{41} a_{jkl}}, \quad (11)$$

where $\{a_{jkl}\}$ are the activities of the units in response to the line segment. The first sum (over j) is a sum over the four basis function maps (or over the remaining two maps in the damaged network).

Errors were defined as the projection onto the line of the vector difference between actual middle, m_0 and the estimated middle, m :

$$e = (\vec{m}_0 - \vec{m}) \cdot \vec{P}, \quad (12)$$

where $\vec{P}$ is a vector of unit length collinear with the line.

Simulations

Simulations: d' and Reaction Time

First, the salience of a single stimulus was measured as a function of its retinal location and eye position. Because d' and reaction times are, respectively, proportional and inversely proportional to salience in the model (see Method section), these results can be compared with d' and reaction time experiments on patients with left hemineglect.

Results

Figure 4A shows the salience obtained in the model for a single stimulus as a function of its retinal location ($[-20^\circ, 20^\circ]$) and eye position ($[-20^\circ, 20^\circ]$) after a right parietal lesion. The graph shows that salience is roughly proportional to the retinal locations and right eye positions such that it is maximum for right retinal locations and right eye positions.

This gradient implies that d' increases in the model for right retinal and right eye positions because d' is proportional to salience in the model. The opposite relationship holds for reaction time, which is inversely proportional to salience.

A similar increase of d' and decrease of reaction time for right retinal locations has been reported in human patients (Jha, Kingstone, & Mangun, 2000; Ladavas, Petronio, & Umiltà, 1990). As shown in Figure 4B, d' was larger for stimuli appearing on the right side of the retina while reaction times were faster. Preliminary results by Behrmann et al. (personal communication, May 2000) indicate that reaction times for saccadic eye movement also decrease as the starting position of the eyes is moved further to the right. To our knowledge, the effect of a similar manipulation on d' has not been tested, but the model makes the clear prediction that d' should increase for a fixed retinal location when the eyes are deviated to the right.

Discussion

The lesion introduced a contralateral bias in the basis function map such that more neurons were dedicated to rightward positions along both the retinal and eye position axes. The salience gradient directly reflected this underlying neuronal gradient. The fact that human patients show a pattern of d' consistent with this salience gradient along the retinal axis suggests that a parietal lesion may also produce a similar neuronal gradient.

Indirect evidence for the existence of the retinal gradient also comes from studies of exploratory eye movements, either in the dark (Hornak, 1992; Karnath & Fetter, 1995; Karnath, Fetter, & Dichgans, 1996) or in the light (Behrmann, Watt, Black, & Barton, 1997). Such studies revealed that the number and duration of fixations in neglect patients increase linearly for rightward posi-

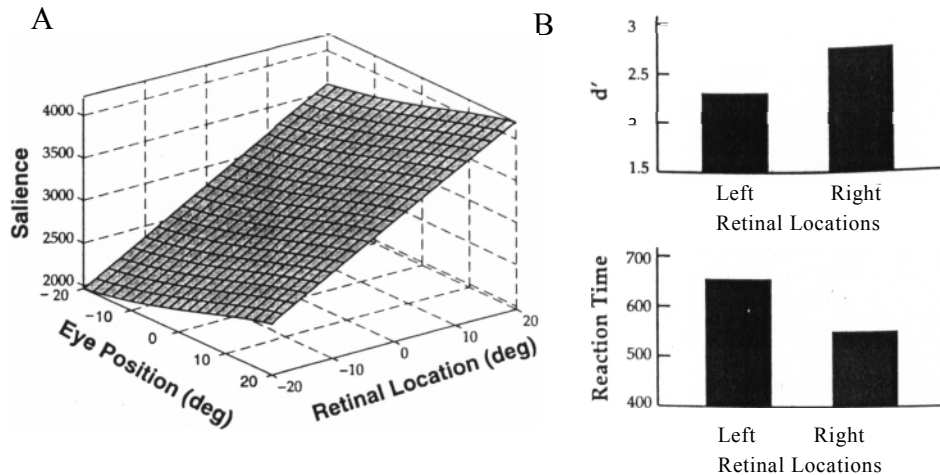


Figure 4. Panel A: Dependence of saliency on retinal location and eye position in a model with a right parietal lesion. Panel B: d' (top) and reaction time (bottom) for two retinal locations in a right parietal patient (data from Ladavas et al., 1990). The signal-to-noise ratio d' is larger and reaction time shorter, for right retinal location, as predicted by the model. deg = degree.

tions as the gradient hypothesis would predict. Likewise, there is indirect evidence in favor of a gradient along the eye position axis. Kooistra and Heilman (1989) reported a patient who appears to be left hemianopic when the eyes fixate straight ahead but does not show any deficit when the eyes are deviated to the right. This could be an extreme case of an increase in saliency for left retinal location as a function of eye position.

The saliency gradient underlies most of the results shown in the other experiments simulated here. Because the attentional mechanism in the model relied on winner-take-all, the gradient conferred an advantage to any stimulus located further toward the right, where right was defined in retinal coordinates or in terms of eye position.

The saliency does not shift suddenly from low to high values when crossing the midline of the retina or when the eye goes from left to right. Instead, saliency variations are smooth and continuous, a property that contrasts with the way the model performs in the line cancellation task described in the next section.

Simulations: Classic Clinical Tests

A second series of simulations explored the network behavior in the line bisection and line cancellation tests, which are commonly used to diagnose hemineglect. Both simulations used a 81×81 two-dimensional retina in the input, resulting in basis functions maps that are 41×41 along the retinal axis (as opposed to 41×1 in other simulations).

Results

Line bisection. In the line bisection task, the damaged network estimated the midpoint of the line to the right of the actual midpoint. This error was proportional to the length of the line, as shown in Figure 5B. In contrast, the performance of an intact network was always centered regardless of the length of the line (not shown).

The constant of proportionality between the error and the length of the line depended on the severity of the lesion (the slope of the neuronal gradient). The curves shown on the right graph in Figure 5B shows the performance of three networks with different lesions. The curve with the highest slope corresponds to the most severe lesion (the steepest neuronal gradient).

The bisection error was also found to vary according to the cosine of the orientation of the line (Figure 5C) and the amplitude of maximum error was dependent on the slope of the contralateral neuronal gradient (not shown).

The relation between bisection error and line orientation also depended on the orientation of the retinal neuronal gradient. In most of the simulations, there was a horizontal gradient (0°), but there is little reason to expect that this gradient should be perfectly horizontal for all patients. Therefore, we tested three different networks with retinal gradient of 0° , 45° , and 135° , respectively, corresponding to the three different curves in the right graph of Figure 5C. The error curve had the same cosine shape in all cases, but their phases differed by an amount equal to the orientation of the retinal neuronal gradient. Thus, the error was maximal when the orientation of the bar was lined up with the retinal neuronal gradient.

Human performance on the line bisection experiments shows similar relationships (Burnett-Stuart, Halligan, & Marshall, 1991; Halligan & Marshall, 1989). The error is proportional to the length of the line (Figure 5B) and follows a cosine curve as a function of line orientation (Figure 5C). The slope of the curve on the error versus length graph also varies with the patient, perhaps reflecting differences in the severity of the lesion. Likewise, the phase of the cosine relationship between error and line orientation varies across subjects, possibly because of differences in the orientation of the retinal gradient.

There is, however, one aspect of the human performance that was not captured by the model. Halligan and Marshall (1989) reported that the sign of the error was reversed for very short lines,

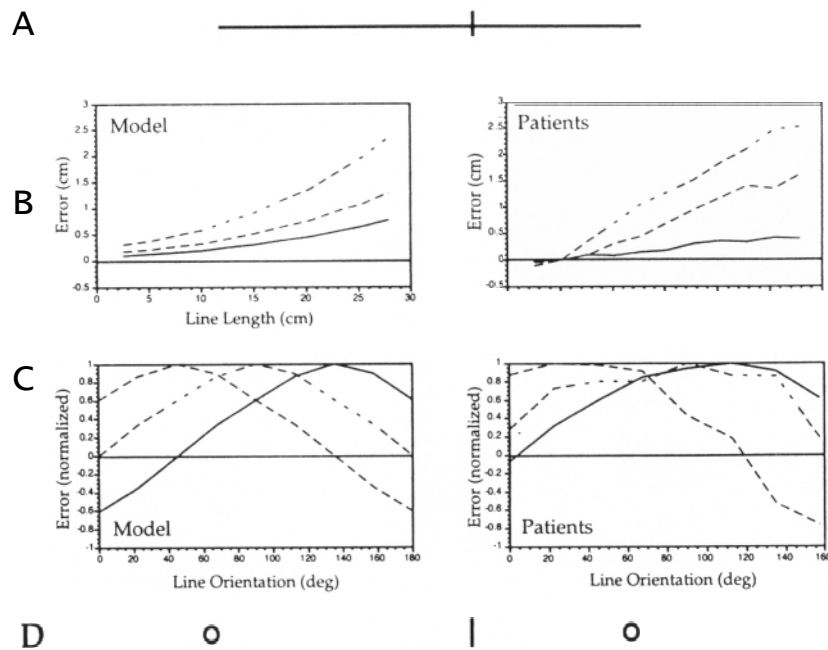


Figure 5. Simulation of a line bisection experiment. Panel A: The network misjudged the midpoint too far to the right. Panel B: Magnitude of the bisection error as a function of the line length. The error grew in proportion to the line length in both the model (left) and neglect patients (right; data from Marshall & Halligan, 1989). Different curves correspond to different lesions in the network and different patients for the experimental data. Panel C: Magnitude of the bisection error as a function of the line orientation. The error follows a cosine of the line orientation in both the model (left) and the neglect patients (right; data from Bumett-Stuart et al., 1991). As before, different lines correspond to different orientations of the retinal gradient in the network and different patients for the experimental data. Panel D: Simulation of the Bisiach et al. (1994) experiment. The network placed the left end of the imaginary bar further away from the check mark than the right end. deg = degree.

such that left-neglect patients tended to perceive the middle of short lines too far to the left. Error reversals were not observed in the model (but see next discussion).

We also simulated an experiment by Bisiach, Rusconi, Peretti, & Vallar (1994) in which patients were asked to indicate the endpoints of an imaginary line segment given its midpoint. To simulate this experiment, the model simply moved the two endpoints until the estimated midpoint (according to Equation 11) corresponded to that provided by the experimenter. The results of a typical simulation are shown in Figure 5D. The network always chose to place the right endpoint closer to the midpoint than the left endpoint, a result consistent with the behavior of the patients.

Line cancellation. In the line cancellation task, patients are asked to cross out a set of line segments shown on a sheet of paper. This experiment was simulated in the network by presenting an array of bars and monitoring the sequence of bars that were chosen by the selection mechanism over time. The results did not depend on presentation times longer than 30 s, and results shown below were for a presentation time of 40 s.

A typical pattern of line crossing for the network after the lesion is shown in Figure 6A. As in patients with hemineglect, the damaged network missed the line segments located on the left side of the display. In contrast, the intact network crossed out all the lines (not shown).

The network was also tested after a lesion that introduces an oblique retinal gradient, as in the line bisection experiment. In

Figure 6B, the gradient decreased from the top right retinal position to the bottom left. The network crossed out only the lines located on the top right half of the display. Similar oblique patterns of line cancellation have been reported in neglect patients (Mark & Heilman, 1997).

The damaged network-with a horizontal gradient-had a strong tendency to begin by checking the lines on the right and then moving toward the left. At some point, the network kept selecting lines that had already been crossed out and failed to detect the lines on the far left regardless of how long the display was presented.

Figure 6A shows the probability for a line to be crossed out as a function of its position in the display for a horizontal retinal gradient. There was a sharp break in the probability function such that lines on the right of this break have a probability of 1 of being selected; whereas lines on the left or the break have a probability of zero (see Figure 6C. A similar sharp transition has been reported in patients (Marshall & Halligan, 1989).

The sharp break in the probability of selection contrasts with the smooth and monotonic profile of the neuronal gradient. Whereas the sharp boundary in the pattern of line crossing may suggest that the model "sees" only one half of the display, the linear profile of the neuronal gradient shows that this is not the case. The sharp break is mostly a consequence of the selection process.

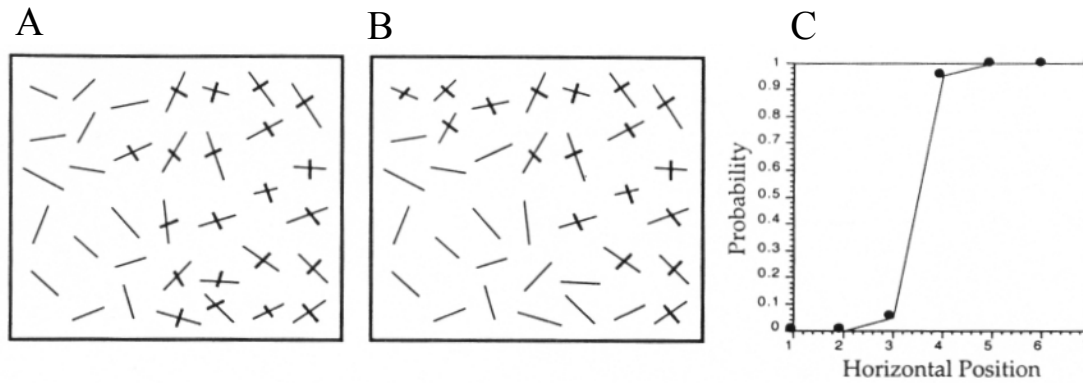


Figure 6. Simulations of a damaged network on the line cancellation experiment, Panel A: Typical pattern of cancellations, The network missed the lines on the left side similar to the pattern exhibited by left neglect patients. Panel B: Results for a simulation with an oblique retinal gradient to the upper right, The network missed the lines in the bottom left half of the display, Panel C: Probability of line crossing as a function of position on the display for a horizontal retinal gradient as in Panel A, The steep step function explains why the network behaves as if it were completely blind to the bars located on the left side of the display.

Discussion

The damaged network and parietal patients showed similar patterns of performance in the line bisection and the line cancellation tests. In both cases, the mistakes made by the network stemmed primarily from the neuronal gradient, which conferred an advantage to the right side of the stimuli (line bisection) or to the right side of the display (line cancellation).

In the line bisection task, the midpoint was estimated by computing the center of mass of the activity induced by the stimulus (see Nielsen, Intriligator, & Barton, 1999, for a similar approach). Because there was more activity induced by the right side of the line than the left side, the network was biased toward the right. When the line was short, the left-right imbalance was small and, consequently, the deviation small. As the line length increased, the imbalance became stronger, leading to larger error.

For short lines, the network continued to make mistakes toward the right; even when the line was reduced to a point, the network tended to locate the midpoint slightly to the right of the point. This was an unavoidable consequence of using large retinal receptive fields, which blurred the image of the point such that the slightest imbalance in the neuronal gradient led to a misjudgment of its position.

This behavior is not consistent with the report of Halligan and Marshall (1989), who found that for some patients the bisection error reverses from the left to the right for very short lines. We do not think, however, that this failure to replicate the error reversal is a serious problem for our main hypothesis: namely, that the error is due to a read-out mechanism operating on a graded representation. Models that have successfully simulated the error reversal rely on the idea that a parietal lesion leads to a spatial gradient, just like our model. To account for the reversal, they either adjusted the profile of the gradient (by reversing the slope of the gradient in the central part of the map; Anderson, 1996) or added constant leftward bias in the estimation of the midpoint of the line (Mozier et al., 1997). These explanations can be easily implemented in our network as well.

The cosine relationship found between error and line orientation is related to the one-dimensional nature of the retinal gradient. For

a given lesion, the retinal gradient in the model was chosen to be along one particular axis. Lining up the test line with this axis led to the largest error. When the test line and the retinal gradient formed an angle, the error depended on the projection of the line onto the axis of the lesion. Simple geometry shows that the projection is proportional to the cosine of the angle, which explains why the error followed a cosine of the angle in the model and in patients.

The dependency between error and line length or line orientation has been explained by previous models (Burnett-Stuart et al., 1991; Mozer et al., 1997). In particular, Mozer et al. (1997) also relied on a monotonically graded lesion. That model, however, depended on a dynamic attentional mechanism implemented with a recurrent network. The present model demonstrates that there is no need to invoke this mechanism to account for the behavior of the patients in these experiments.

In the line cancellation test, the performance of the network was the result of both the gradient of the lesion and the competition mechanism used in the selection mechanism. Because the bars on the right were associated with higher activities, they consistently won the competition to the detriment of the bars on the left. As a consequence, the network started by selecting the furthest bar on the right and moved toward the bars on the left because of inhibition of return. Eventually, the previously inhibited bars recovered and won the competition again, preventing the network from selecting the leftmost bars. The point at which the network stopped selecting bars toward the left was dependent on the exact recovery rate and the total number of items in the display.

In the simulations, the eyes were fixated on the center of the display throughout the task, which is unrealistic because patients are typically allowed to move their eyes. We did not introduce eye movements in our simulation because saccades raise a difficult problem: namely, how does a patient memorize which lines have been canceled? The mechanism we used, the inhibition of return in retinocentric coordinates, works only when the eyes are fixed. because there is a stable mapping between the position of the bars in space and their position on the retina.

Note, however, that our results do not rely on the memory being in retinocentric coordinates. The critical assumptions are that the probability that an item will be selected at any given time is (a) proportional to its salience relative to the competing item (which is unaffected by eye position because eye position only modulates the DC component of the saliencies and, accordingly, does not affect relative saliencies) and (b) reduced if the item has been recently selected. Inhibition of return followed by a recovery is only one way to achieve the latter, but we do not claim that this is the solution implemented in the brain. We emphasize that these two assumptions are the key components of our model of line cancellation. Any experimental data questioning these assumptions would also challenge our model.

As it is, our model uses the same neural substrate to model line bisection and line cancellation. Therefore, we predict that the performance on these two tasks should be correlated. In particular, the direction of the neuronal gradient should affect both tasks. For example, a patient who shows the largest error for a line oriented at 2 o'clock should also tend to cancel out lines in the upper right quadrant with maximum neglect in the lower left quadrant. To our knowledge, this prediction has not been tested.

There are reports of patients with double dissociations, such as neglect in line cancellation but not bisection and vice versa (Halligan & Marshall, 1992). This suggests that there may exist neural substrates used preferentially in one task or the other. Note, however, that double dissociations do not argue against the existence of a common stage in addition to the specialized representations. It is, therefore, possible that our prediction will be verified even though some patients show a double dissociation.

In both line bisection and line cancellation, the source of the deficit for the network is not related to changes in the basic components of the selection process, the winner-take-all and the inhibition of return. Neither is it due to a basic inability to orient toward the left side of space. The network can clearly orient toward left targets presented in isolation, and its performance in the Bisiach et al. (1994) variation of the line bisection experiments (Figure 5D) is also incompatible with an orientation problem. Rather, the problem stems from the gradient introduced by the lesion in the basis function map. In the case of the line bisection, this gradient decreases the salience of the left side of the bar relative to the right side and, in the case of the line cancellation, it biases the competition in favor of the rightmost element.

In that sense, our model is more compatible with the representational rather than the attentional theory of hemineglect. However, as we point out in the general discussion, a description of neglect in representational terms is not consistent with what we know of the parietal lobe. Parietal neurons are clearly involved in attention and spatial representations (Andersen, 1995; Gottlieb et al., 1998), making it difficult to distinguish between the two functions. We did distinguish between these functions in our simulations; in fact, we used distinct algorithms as explained in the Methods section. We did so, however, only for convenience: to speed up our simulations. We could just as well have used a recurrent network in which the competition would have been mediated by lateral connections between basis function units, and this is what we believe exists in the parietal cortex (Cohen et al., 1994).

At this point, we emphasize that the models we have proposed for line cancellation and line bisection were not meant to be

precise models of these tasks in humans. Our goal was not to model line cancellation and bisection per se but rather to establish that a graded lesion on a basis function map is consistent with the behavior of the patients. As we have seen, the interaction between the gradient and a selection process is sufficient to replicate the main features of the patients' behavior, and these are the main components of the model. Details, such as the use of a center of mass for line bisection, should not be taken literally; further experimental work is needed before these details can be sorted out.

Simulations: Frames of Reference

Numerous experiments have attempted to determine the frames of reference affected in hemineglect (Behrmann & Moscovitch, 1994; Bisiach et al., 1985; Calvanio et al., 1987; Farah et al., 1990; Karnath et al., 1991, 1993; Ladavas, 1987; Ladavas et al., 1989). Most of these studies first tested patients in a control situation, in which all frames of reference were superposed, and in a test condition, in which they were decoupled by asking the subjects to change posture. The network was tested on an experiment performed by Karnath et al. (1993), which reported results that are representative of those found in the other experiments.

This experiment involved recognizing the shape of an object presented 7° to the right or the left of the fixation point. Three conditions were tested (see Figure 7A). In the control (Condition 2), left-neglect patients were seated with their eyes, head, and trunk lined up and facing straight ahead. In the other two conditions, the trunk was rotated 15° to either the right (Condition 1) or the left (Condition 3). Because a trunk rotation of 15° to the right is the same as a head rotation of 15° to the left, another way to think about these three experimental conditions is that the individual's head was turned progressively more to the right (i.e., toward the "good" side of space) from Condition 1 to Condition 3.

Note that the retinal position of the stimuli is the same in all conditions, because the eyes do not move with respect to the stimuli. In contrast, the trunk-centered location of the stimuli varies. Interestingly, the right stimulus in Condition 1 and the left stimulus in Condition 2 share the same trunk-centered location (angle α in Figure 7A). The same holds for the right stimulus in Condition 2 and the left stimulus in Condition 3 (angle β).

The performance of the left-neglect patients should be different under different hypotheses for the frame of reference. If neglect is retinocentric (i.e., the patient's performance is determined by the retinocentric location of the stimuli), the performance of the patient should be the same in all conditions (Figure 7B). If, instead, neglect is trunk centered, then performance should follow the trend illustrated in Figure 7C. In particular, the right stimulus in Condition 1 and the left stimulus in Condition 2 should have the same performance, as should the right stimulus in Condition 2 and the left stimulus in Condition 3 (as indicated by the dotted lines).

The results are intermediate between these different possibilities shown in Figure 8A. Thus, neglect appears to affect a mixture of frames of reference, in this case trunk centered and retinocentric. A similar conclusion was reached in all the other experiments (Behrmann & Moscovitch, 1994; Bisiach et al., 1985; Calvanio et al., 1987; Farah et al., 1990; Karnath et al., 1991, 1993; Ladavas, 1987; Ladavas et al., 1989). Neglect was never confined to a single frame of reference but always involved a mixture of frames of reference.

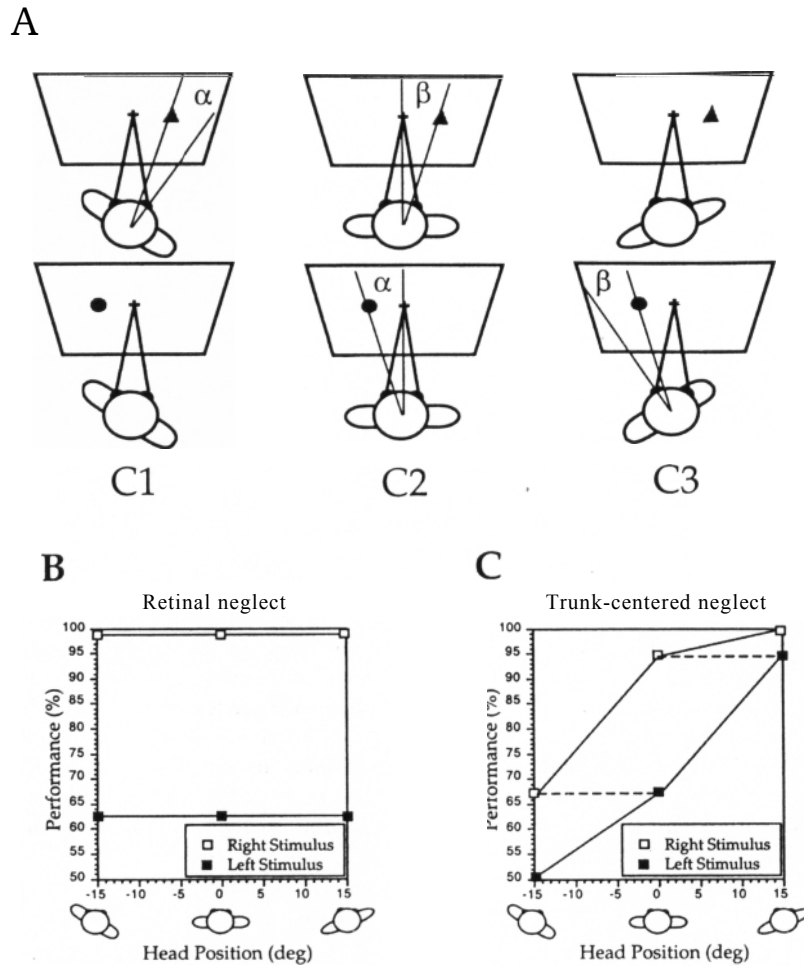


Figure 7. Predictions of experiments designed to test for the frame of reference in hemineglect patients. Panel A: Three different conditions tested in the Karnath et al. experiment (1993). Note that the right stimulus in C1 has the same trunk-centered position as the left stimulus in C2 (angle α). The same is true for the right stimulus in C2 and the left stimulus in C3 (angle β). Panels B and C: Predicted performance in the Karnath et al. (1993) experiment for retinocentric (Panel B) and trunk-centered (Panel C) neglect. The dashed lines indicate the conditions in which performance should be identical because these stimuli share the same trunk-centered positions. deg = degree.

The Karnath et al. (1993) experiment was simulated with a version of the network shown in Figure 2A in which head position was substituted for eye position. The performance of the network was determined using Equation 10 in the Method section. Neurons with gain field for head position in the parietal cortex have been reported by Brothie et al. (1995).

Results

Figure 8B shows the results of the model in the Karnath et al. (1993) experiment, in which the reported performance was proportional to the stimulus salience (see Methods section). As observed in patients, the results were intermediate between retinocentric and trunk-centered neglect.

In particular, performance on the left stimulus improved as the head was rotated toward the right but did not match the performance for the right stimulus in the first condition. The perfor-

mances of the model and the patients were qualitatively, but not quantitatively, similar. These quantitative differences could have been reduced by tuning the parameters of the model and, in particular, the profile of the neuronal gradients in the basis function maps.

Discussion

In these simulations, the behavior of the model simply reflected the salience gradient introduced by the lesion, because performance was proportional to stimulus salience. Performance on the right stimulus was always better than for the left stimulus because the salience of the right stimulus was always greater than the salience of the left stimulus (Figure 4A). Performance on the right and left stimuli increased across conditions because of the salience gradient along the head position axis (shown as the eye position axis in Figure 4, because head position had been substituted for eye

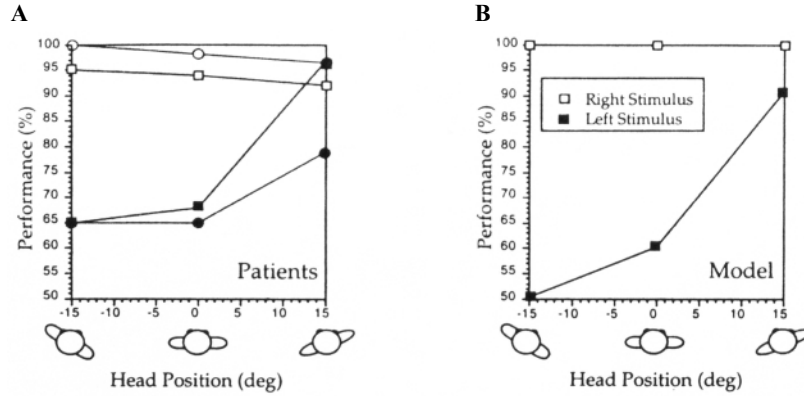


Figure 8. Comparison between the performance of (Panel A) two hemineglect patients in Karnath et al, (1993; squares and circles) and (Panel B) the network for the right (open symbols) and left (closed symbols) stimuli. The same trends were observed for both the patients and the network: The overall pattern was intermediate between trunk-centered and retinocentric neglect. deg = degree.

position in these simulations). As the head turned further toward the right, the salencies of the right and left stimuli increased, leading to better performances.

Exactly how much performance improved for a given head rotation or retinal translation of the stimulus toward the right depended on the slope of the salience gradient. As shown in Figure 5A, the gradient was roughly proportional to retinal and head position:

$$s_i \approx c_r r_i + c_h h_i. \quad (13)$$

One important feature of this gradient is that the slope along the retinal axis was greater than the slope along the head position axis (i.e., $c_r > c_h$). It follows that given two stimuli with the same trunk-centered position, $t1 = r1 + h1 = r2 + h2 = t2$, the stimulus with the largest (i.e., rightmost) retinal location will have the highest salience. If, for example, $r_1 < r_2$, then

$$s_1 \approx c_r r_1 + c_h h_1 < s_2 \approx c_r r_2 + c_h h_2. \quad (14)$$

This explains why the performance of the right stimulus in Condition 2 does not match the performance of the left stimulus in Condition 3, even though they share the same head-centered location (the same holds for the right stimulus in Condition 1 and the left stimulus in Condition 3). Consequently, the overall performance pattern is intermediate between trunk centered and retinocentric.

If, after a lesion, the retinal and eye position gradients were equal (i.e., $c_r = c_h$), the deficit would appear to be purely trunk centered. Therefore, it is possible to obtain pure trunk-centered neglect or, more generally, a deficit confined to one frame of reference using our basis function model. However, the extent and severity of a lesion are highly variable among patients, and the corresponding c_r and c_h are likely to vary as well. Therefore, the chances that $c_r = c_h$ are small, making it unlikely that an individual would show only trunk-centered neglect.

Similar principles can be used to account for the behavior of patients in all the other experiments designed to uncover frame of reference (Behrmann & Moscovitch, 1994; Bisiach et al., 1985;

Calvanio et al., 1987; Farah et al., 1990; Karnath et al., 1991, 1993; Ladavas, 1987; Ladavas et al., 1989).

Simulations: Relative and Object-Based Neglect

Several experiments suggested that neglect may not be confined to a particular hemispace, but may be relative in the sense that, when several stimuli appear simultaneously, the stimuli on the left of the set of stimuli will be neglected whether they appear in the right or left visual field (Arguin & Bub, 1993a, b; Kinsbourne, 1987).

The issue of relative neglect is closely linked to what has been called object-centered neglect, a term that has been applied to a variety of experiments in which the left side of objects is neglected regardless of the position of the objects in the visual field (Arguin & Bub, 1993a; Driver & Halligan, 1991; Halligan & Marshall, 1994). If, indeed, neglect is relative, patients should neglect the leftmost part of an object regardless of the position of the object in space.

There are many studies of object-based neglect, but not all of them can be simulated without implementing additional mechanisms such as drawing or visual segmentation. The experiment by Arguin and Bub (1993a), however, demonstrates object-based neglect in a way that is amenable to simulation with the current model.

Figure 9A illustrates the temporal sequence of screens that were presented to a patient with right parietal damage on a single trial (Arguin & Bub, 1993a). The patient was first asked to fixate in the center of the screen. Then four visual shapes appeared, one cross and three circles. These were replaced 60 ms later by four letters appearing at the same locations as the shapes. The task was to name the letter at the location in space previously occupied by the cross.

Several conditions were tested, but only three are relevant for our purpose (see Figure 9B). In conditions 1 and 2, the relative position of the target with respect to the distractors was varied while keeping constant the target eccentricity at 10° to the left. In the first condition the target was to the left of the distractors,

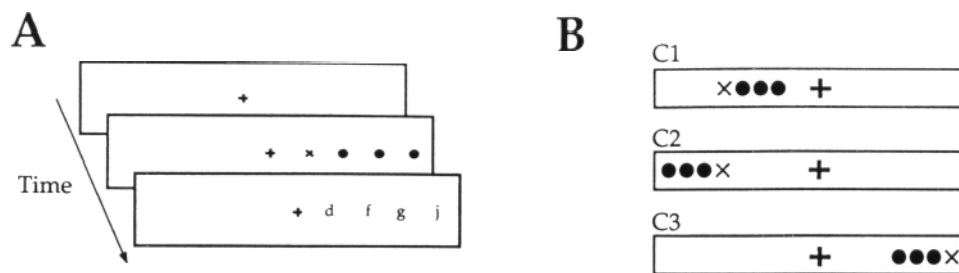


Figure 9. Description of the Arguin and Bub (1993a) experiment. Panel A: Temporal sequence of screens presented to the subject on each trial. The task was to name the letter appearing at the same location as the cross. Panel B: Three of the conditions tested. Target on the left of the distractors (top); target on the right of the distractors at the same retinal location as in C1 (middle); target on the right of the distractors and on the right hemiretina (bottom).

whereas in Condition 2 the target was to the right. In Condition 3 the target was still on the right of the distractors, but the entire display was presented on the right hemiretina with the target 10° to the right.

The results of their experiments are shown in Figure 10A. Naming times—the time it took for an individual to name the letter appearing at the same location as the cross—were briefer in Condition 2 than in Condition 1 and even faster in Condition 3.

Because the target had the same retinal position in Conditions 1 and 2, the decrease in naming time was related to the change in the relative position of the targets with respect to the distractors. Hence, the task was easier when the target appeared on the right of the distractors than when it appeared on the left.

The fact that naming time decreased further in Condition 3 suggests that neglect is also retinocentric. Thus, the deficit appears to affect multiple frames of reference simultaneously, consistent with the experiments described previously.

Result

The experiments in Arguin and Bub (1993a) were simulated using the damaged network model. The letters were modeled as simple points of light on the retina, because no letter recognition mechanism was implemented in the network. On any given trial,

the four letters were presented to the network and their corresponding saliencies were determined. Next, the salience of the letter appearing at the same location as the cross was increased by 10% to simulate spatial priming. Finally, the selection process was run, and the number of iterations required by the selection mechanism to find the target letter was used to compute the reaction times (see Methods section) (Equation 9).

The results in Figure 10B indicate that, for both the model and the patient, the reaction times decrease systematically from Condition 1 to Condition 2 to Condition 3.

Discussion

The decrease in reaction time across conditions in the model leads to the conclusion that the reference frame for neglect has both object-centered and retinocentric components, as observed in humans.

The object-centered component was unexpected, because there were no explicit object-centered representations in the network model. By explicit object-centered representation, we mean a representation in which neurons have a response field (receptive field or motor field) that is invariant in object-centered coordinates. This would imply that when an object is moved or rotated, the receptive field moves and rotates with the object. Instead, our

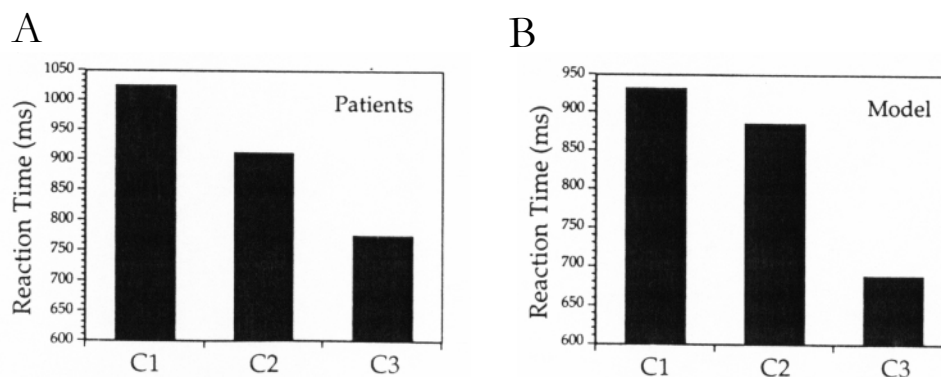


Figure 10. Naming times for (Panel A) left-neglect patient A.B. (Arguin & Bub, 1993a) and (Panel B) the damaged network. Both the patient and model show the same trend: progressively briefer reaction times from Condition 1 to Condition 2 to Condition 3. The decrease from Condition 1 to 2 suggests that neglect is object based, whereas the further decrease in Condition 3 indicates a retinal component.

network contains neurons with retinotopic receptive fields in which the gain is modulated by posture signals. The object-centered effect emerged as a result of the interaction between the selection mechanism and the smooth monotonic retinotopic gradient. In Condition 1, the salience of the distractors was higher than that of the target because of the retinal gradient. Consequently, the selection mechanism required several iterations before it could reliably select the target. In Condition 2, the target had the same salience as in the previous display, but the salience was now higher than the saliences of the distractors and the selection mechanism typically selected the target on the first iteration.

The retinocentric effect can be traced to the second term in Equation 9. The reaction time decreased further because the absolute salience of the stimulus had increased when the target was moved further to the right on the retina.

This result demonstrates that object-based neglect after a lesion of the parietal cortex does not necessarily imply that there is an *explicit* object-based representation. One may argue that the representation in our network is nevertheless implicitly object-centered. We believe that this would be a misleading terminology in this particular case. Our network reproduced the object-based effect of Arguin and Bub (1993a), because it neglects whatever has the smallest relative salience. Because the gradient decreases toward the left, it is always the leftmost object or leftmost subpart of an object or a perceptual group that ends up being neglected. Note, however, that when we say leftmost we mean left with respect to an *egocentric* horizontal axis, *not* with respect to an object-centered axis. For instance, rotating the object does not rotate the pattern of neglect; neglect is still observed along a left-right axis defined with respect to the viewer (see Farah et al., 1990). Perhaps a better term for this deficit is "egocentric relative neglect" (Driver & Pouget, 2000; Pouget & Driver, 1999), or "relative neglect" for short.

Other aspects of neglect could be explained by the same principles if the basis function map did not simply reflect the retinal image but represented instead a preprocessed version. Assume, for example, that at any given moment the parietal cortex can only represent the attended object (see Gottlieb et al., 1998, for neurophysiological evidence). Then the model predicts that a neglect patient would neglect the left side of an attended object—the one that has been segmented and selected—regardless of its position in space. This is indeed consistent with what has been reported by patients (Driver, Baylis, & Rafal, 1992; Halligan & Marshall, 1994; Pavlovskaya, Glass, Soroker, Blum, & Groswasser, 1997). Thus, the model can account for the interaction that has been reported between scene segmentation and neglect (Driver & Pouget, 2000). Note, however, that this form of object-centered neglect is, once again, not neglect of the left side of the object in object-centered coordinates but neglect of the leftmost side of the object along a horizontal axis defined in egocentric coordinates. Therefore, this is another example of "relative neglect."

There are, however, a few experiments that cannot be categorized as "relative neglect" and, as such, cannot be modeled with the basis function network described here. Hence, Tipper and Berhmann (1996) described an experiment in which neglect patients were shown two circles, one on the left and one on the right. The circles were sometimes linked by a bar, forming a barbell-like object. They explored whether priming can be defined in object-centered coordinates and found that, when the right circle was

primed, followed by a 180° rotation, which brought the right circle to the location of the left circle and vice versa; the priming stayed with the right circle (now on the left), but only if the two circles were linked by the bar.

This result can be easily explained with explicit object-centered representations, although this is not necessary. Mozer (1999) found that the results of Tipper and Berhmann (1996) can also be reproduced in a model of word recognition (Mozer & Behrmann, 1990), which does not contain neurons with object-centered receptive fields. Instead, the model relies on a retinotopic attentional map whose dynamics are influenced by whether the visual stimulus forms one or multiple objects. Our model does not contain an equivalent mechanism, but it would be straightforward to implement it in the basis function maps.

An experiment by Driver et al. (1994) provides another example of a result that cannot be explained by relative neglect. Driver et al. used rotated objects that have a natural symmetry axis. They found that patients neglect the side of the object relative to the object-centered axis rather than the position on the retina. This form of neglect is difficult to account for without invoking some form of axis-based or object-centered representations. However, this representation does not have to be explicit. Thus, a straightforward extension of our basis function framework, consistent with neurophysiological data from Olson and Gettner (1995) as well as Breznien, Sabes, and Andersen (1998), can explain the qualitative properties of axis-based neglect (see Deneve & Pouget, 1998; Pouget, Deneve, & Sejnowski, 1999). This network contains basis function neurons with retinotopic receptive fields whose gain is modulated by the properties of the object of interest, such as its orientation on the retina. The resulting network can perform object-centered saccades as in Olson and Gettner's experiment, and a graded lesion can reproduce axis-based neglect as found by Driver et al. (1994), despite the absence of neurons in the model with response fields that are invariant in object-centered coordinates, as would be found in an explicit object-centered representation.

Therefore, there are several advantages of the basis function representation over an explicit object-centered representation: (a) It is computationally efficient (i.e., it can be easily used to perform an object-centered task such as moving the eyes to a particular side of an object) and (b) it is consistent with neurophysiological and neuropsychological data.

Note that the axis-based neglect in Driver et al. (1994) is also exhibited in Mozer's network, even though this particular network does not perform any object-centered task (Mozer & Behrmann, 1990). This raises the possibility that an object-centered representation may not be needed to explain the results in Driver et al. (1994).

To summarize, many of the results that have been interpreted in terms of object-centered neglect (Arguin & Bub, 1993a; Driver et al., 1992; Driver & Halligan, 1991; Halligan & Marshall, 1994; Humphreys, Olson, Romani, & Riddoch, 1996; Pavlovskaya et al., 1997; Vallar, 1998; Walker, 1995; Young, Hellawell, & Welsh, 1992) can be explained in terms of relative neglect, a behavior that emerges naturally in our damaged network and that does not require explicit object-centered representations. In a few cases, it appears that the behavior of the patients is consistent with a lesion to an object-centered representation (as in Driver et al., 1994), but, even then, the object-centered representation does not have to be

explicit. It could involve basis function neurons, a possibility that is supported by neurophysiological data (Olson & Gettner, 1995; Breznen, Sabes, & Andersen, 1998). This does not imply that there are no such explicit object-centered representations in the brain as assumed in other models of visual perception (Hinton, 1981; Olshausen, Anderson, & Van Essen, 1995) and neglect (Ho, Behrmann, & Plaut, 1995; Rao & Ballard, 1997), but it demonstrates that this is not a requirement.

Simulations: Reaching and Optic Ataxia

Optic ataxia is an impairment in reaching toward visual targets (Perenin, 1997). When asked to trigger a ballistic movement of their hand to reach for an object, patients with optic ataxia tend to miss the target by 10° or more toward the ipsilesional side (Perenin, 1997).

Interestingly, optic ataxia is not always associated with hemineglect; conversely, patients with hemineglect do not generally suffer from optic ataxia (Perenin, 1997). It seems paradoxical that neglect could spare reaching if, as we have argued, neglect is the result of a lesion of the spatial representations used in sensorimotor coordination.

To test whether our network suffers from optic ataxia after a unilateral lesion, we computed the position of the hill in the head-centered map (the one that is assumed to project to the reaching system) in a damaged network for a single visual target. The position of the hill was determined with a center of mass estimator. We found that the damaged network misses the target by 1.03° to the right of the object, that is, 1.03° toward the ipsilesional side. This indicates that the network suffers from a small-reaching impairment, but the amplitude of the error is small compared with what typically is reported for patients with optic ataxia (10° or more; Perenin, 1997). Therefore, the damaged network does not suffer from optic ataxia.

The small amplitude of the bias is simply a reflection of the shallow slope of the gradient. A steeper gradient would lead to larger biases, but a steeper gradient cannot be maintained over a large extent of the visual field; eventually, the number of cells saturates at either zero or the maximum number found in normal individuals. Therefore, a steep gradient would predict that neglect is observed only over a small extent of the visual field, in which the slope of the gradient is different from 0. This is not consistent with what has been reported in neglect patients: that is, their deficit generalizes over a large extent of the visual field (Heilman et al., 1985).

To our knowledge, it is not known whether hemineglect patients also exhibit a small ipsilesional bias when reaching for an object. Such a small bias would indeed be difficult to detect in a clinical setting. The error is smaller than many objects and small enough to be corrected on the fly if the hand is visible to the patient. Furthermore, patients might be able to adapt quickly to the constant bias just as normal individuals can adapt to visual prisms. Therefore, further experiments are needed to determine whether our model is fully consistent with the behavior of the patients in a reaching task.

General Discussion

We have explored the consequences of introducing a monotonic contralateral gradient in a basis function model of the parietal

cortex. Simulations of several experiments performed on humans with parietal cortex lesions led to outcomes similar to those that accompany hemineglect. Therefore, this model provides a first step toward a neuronal theory of neglect and further supports the basis function hypothesis for the function of the intact parietal cortex.

The gradient hypothesis was previously suggested by Kinsbourne (1987; see also Driver et al., 1994) and was used in a computational model of neglect (Behrmann et al., 1991; Mozer & Behrmann, 1990; Mozer et al., 1997). These previous studies, however, considered only a gradient along the retinal axis. The model presented here generalizes the gradient hypothesis to postural signals and makes three major new contributions: (a) The model demonstrates that the gradient hypothesis can account for a wider range of experiments than previously suggested; (b) a new perspective is provided on the frame of reference in the parietal cortex by showing how the lesion of one representation can affect multiple frames of reference; and (c) a link is made for the first time between the well-established gain-modulated responses of parietal neurons and the complex behavioral deficits observed in human patients with lesions of the parietal cortex.

Main Assumptions

The main goal of our study was to demonstrate that the behavior of neglect patients is consistent with two fundamental assumptions: (a) neuronal spatial representations rely on basis functions and (b) a unilateral lesion introduces a smooth gradient in the representation.

We do not claim that we have provided a detailed neuronal model of hemineglect. This would require all aspects of our model to be consistent with the behavioral and neurophysiological data. This is clearly not the case. For example, we have assumed that estimating the middle of a line is the result of a center of mass calculation. Yet there is no neuronal or behavioral evidence for or against the use of a center of mass in this perceptual judgment, and only further research can resolve this issue. However, it was important to establish that the behavior of patients in a line bisection experiment can be reconciled with our basis function hypothesis without making implausible assumptions or invoking unlikely mechanisms. If this were not so, we would be forced to reconsider our main assumptions, which was the motivation for the simulations of the line bisection task.

Is Neglect a Representational or Attentional Deficit?

There has been long, ongoing debate about whether neglect is a representational or an attentional deficit (Vallar, 1998). The representational theory assumes that neglect is due to a degraded spatial representation for the contralesional hemifield, whereas the attentional theory invokes a deficit in the mechanisms for orienting toward the contralateral side of space, so that a left-neglect patient would have difficulty orienting toward the left of the current gaze position (Kinsbourne, 1987; Posner et al., 1984).

According to these definitions, the model developed here appears to be more consistent with the representational theory, because the simulated lesion affected the representation while leaving intact the selection mechanism. However, we distinguished between selection and representation merely because it simplified

the simulations, not because we think that there are distinct neural structures devoted to each of these functions. Indeed it is becoming clear that the same parietal neurons involved in spatial representations are also the ones responsible for the neural control of attention (Andersen, 1995; Gottlieb et al., 1998). Consequently, a lesion of the parietal cortex will necessarily affect both the representation of space and the spatial control of attention. Therefore, the distinction between attentional and representational neglect is moot at this point (see also Pouget & Driver, 2000).

A more interactive type of model could have been used, such as the model in Cohen et al. (1994) that used competition between items implemented with lateral inhibitory connections. This is probably closer to what occurs in the cortex. However, this type of interactive model would have required more computation to achieve qualitatively similar results.

With regard to the representational theory of neglect, several proponents of this idea argued that parietal lesions affect spatial perception, by either contracting the contralesional hemispace (Halligan & Marshall, 1991) or rotating the perceived body axis toward the ipsilesional side (Karnath, Sievering, & Fetter, 1994). We have proposed instead that the lesion introduces a contralateral gradient of diminishing salience, and *as a result*, the damaged network behaves as though there was either a contraction of contralesional hemispace or a shift in body axis. In other words, we suggest that the *cause* of neglect is the salience gradient and that the contraction of the contralesional hemispace (Halligan & Marshall, 1991) or the rotation of the perceived body axis toward the ipsilesional side follows as a *consequence*.

Hemispheric Asymmetry

Left neglect is much more common than right neglect (Heilman et al., 1985). The reason for this asymmetry is unclear, but the common explanation depends on an asymmetry in the hemispheric representations resulting from the language specialization of the left hemisphere. Thus, although the left hemisphere may represent only the right hemifield, the right parietal cortex appears to represent both hemifields in some patients (Kinsbourne, 1987; Vallar, 1998).

This suggests that, contrary to what we have assumed in the model, the gradients in the right and left representations are not simply mirror images. Instead, the right hemisphere may have a shallower contralateral gradient and the left hemisphere a steeper gradient.

The model can be altered straightforwardly to incorporate this asymmetry. The only requirement would be that in the intact network the total number of units representing each region is approximately the same in order to ensure that the output of the network does not have a spatial bias.

Predictions of the Model

Because the model spans several levels, it makes both cellular and neuropsychological predictions. At the cellular level, the model predicts that gain-modulated cells of the type found in areas 7a, 7b, VIP, and LIP in monkeys (Andersen et al., 1990; Field & Olson, 1994) should be found in cortical areas typically found to be damaged in neglect patients. The model also predicts that the representation in the left hemisphere has a strong prefer-

ence for the right hemispace. Hence, most cells should have their sensory receptive field in the right hemiretina or on the right side of the head for tactile cells or the right side of the head for auditory cells. Furthermore, the majority of the neurons should also respond most strongly when the eyes or head is turned toward the right.

A gradient in distribution of sensory receptive fields has already been found in the parietal cortex of monkeys (Andersen et al., 1990), and an imaging experiment in humans also supports the existence of a contralateral hemispheric bias for eye position in the parietal lobe (DeSouza et al., 2000). The postural gradient, however, needs to be confirmed with neurophysiological studies in monkeys (Andersen et al., 1990; Bremner et al., 1997; Galletti & Battaglini, 1989; Schlag-Rey & Schlag, 1984; Van Opstal et al., 1995).

Lesion experiments in monkeys suggest that, contrary to what has been widely assumed, area 7 in the monkey may not be the homologue of the inferior parietal areas 39 and 40 in humans, the brain regions that are typically lesioned in the neglect syndrome (Watson, Valenstein, Day, & Heilman, 1994). Instead, it appears that the areas in the superior temporal sulcus (STS) of the monkey cortex are homologous to areas 39 and 40 in humans. If confirmed, the model predicts that the responses of cells in the STS should have gain fields to integrate sensory and postural signals, as in the parietal cortex of monkeys.

At the neuropsychological level, the network model makes detailed predictions for specific experiments. The most general prediction is that neglect should never be confined to a single frame of reference in any patient. A report by Moscovitch and Behrmann (1994) is particularly interesting in this respect. They tested left-neglect patients for tactile extinction between the right and left sides of the wrist with the palm pointing either up or down. They found that the extinguished side was the left side of the wrist in both conditions, in which left was defined with respect to the patient's body, suggesting that tactile neglect is centered on the trunk.

Further tests have revealed, however, that tactile neglect is not purely trunk centered. In another experiment (di Pellegrino, Ladavas, & Farne, 1997; Aglioti, Smania, & Peru, 1999), individuals were tested for neglect between the right and left wrists with the arms in their resting position or with the arms crossed, such that the right wrist was on the left side of the trunk and the left wrist was on the right side of the trunk. Trunk-centered neglect predicts that the left wrist would be neglected in the first condition and the right wrist in the second. Instead, it was found that the left wrist was always neglected, although less so in the arm-crossed conditions (Aglioti et al., 1999). This later result is more consistent with our basis function approach.

Temporary Recovery

Injection of cold water into the left ear, which stimulates the semicircular canals in a way that is equivalent to a rightward head rotation, leads to a temporary recovery of the neglect syndrome in left-hemineglect patients (Cappa, Sterzi, Vallar, & Bisiach, 1987; Rubens, 1985). In a similar fashion, neglect can be ameliorated by optokinetic stimulation, a global motion of the visual background. Leftward motion reduces the deficit, whereas rightward motion worsens neglect (Pizzamiglio, Frasca, Guariglia, Incoccia, & Antonucci, 1990; Vallar, Guariglia, Magnotti, & Pizzamiglio, 1995).

This is consistent with the effects of vestibular stimulation, because under normal conditions a head rotation to the right induces a leftward optic flow.

Both of these forms of sensory stimulation lead to a leftward nystagmus, which may be responsible for the temporary recovery (Rubens, 1985). The results of the model and recent neurophysiological findings suggest a different interpretation. Snyder, Grieve, Brothie, and Andersen (1998) reported neurons in the parietal cortex whose retinotopic receptive fields are gain modulated by vestibular inputs. Other neurons in the parietal lobe are also sensitive to combinations of vestibular signals, optic flow, and visual inputs (Bremmer, Duhamel, Ben Harried, & Graf, 1997). These vestibular and optic flow inputs are postural signals that may play a role in forming basis functions in parietal neurons similar to that of the eye position in the model. If there is a hemispheric contralateral gradient for these vestibular signals and an ipsilateral gradient for the optic flow, the model predicts that a rightward head rotation in the dark or a leftward optic flow should lead to an improvement of left neglect. This is analogous to the way a rotation of the head toward the right improves the performance of stimuli of the left side in the Karnath et al. experiment (see Figure 7A). Similar principles can explain the partial recovery observed after unilateral stimulation of posterior neck muscles (Karnath et al., 1993, 1996).

Therefore, according to the explanation offered here, vestibular recovery is an exaggerated form of the improvement observed in patients whenever the eye, head, or whole body turns toward the right. As a general rule, any postural change toward the ipsilesional side or any experimental procedure that leads the cortex to infer such a postural change should lead to a reduction of neglect.

Modularity in Basis Function Networks

It is theoretically possible to integrate all the sensory and postural signals into a single unified basis function map. However, the number of neurons that would be required in such a map grows exponentially with the number of signals being integrated, making this scheme impractical for more than a few dimensions. The existence of a unitary spatial representation is also unlikely because the parietal lobe contains several cortical areas with distinct cellular populations (Goldberg, Colby, & Duhamel, 1990; Snyder, Batista, & Andersen, 1997). Furthermore, double dissociations (Humphreys et al., 1996) and the large variations in the deficits among individuals also argue in favor of multiple modules, each integrating a restricted number of sensory and postural signals.

It is possible that, in each of these modules, neurons compute basis functions of the locally available inputs and, as a result, encode the locations of objects simultaneously in all the frames of reference achievable from this restricted set of inputs. A modular scheme simplifies coordination and communication, because many modules are likely to share at least one frame of reference. The result is a mosaic of highly interconnected modules, each containing three to four partially overlapping reference frames.

Even though lesions in this system would rarely be confined to a single frame of reference, it would still be possible for an individual to show visual neglect without tactile neglect (Beschin, Cazzani, Cubelli, Salla, & Spinazolla, 1996; Cubelli, Nichelli, Bonito, De Tanti, & Inzaghi, 1991), and in general, one would

expect to see great variation among patients depending on the exact combination of damaged modules.

Alternative Models

Several authors proposed that the representation of space in the parietal cortex might use a vectorial code rather than basis functions. According to this hypothesis, the gain-modulated neurons found in the parietal cortex perform vector addition of the sensory and postural signals. Hence, neurons integrating retinal and eye position encode the locations of objects in head-centered coordinates, and neurons also integrating head position encode the location of objects in trunk-centered coordinates (Goodman & Andersen, 1990; Mazzoni & Andersen, 1995; Touretzky, Redish, & Wan, 1993).

One critical feature of a vectorial code is that one particular neuron encodes the location of an object in one frame of reference only. Nevertheless, several frames of reference may coexist by having different subpopulations of neurons integrating different combinations of sensory and postural signals.

One possibility is that these neuronal subpopulations are used to control different behaviors. Hence, retinotopic neurons might be involved in the control of saccadic eye movements, whereas bodycentered neurons could have a role in reaching. In this case, a parietal lesion should affect several frames of reference, but the affected reference frames would be dependent on particular behaviors, such as retinocentric for eye movements and bodycentered for reaching. These associations are not found in parietal patients (Farah et al., 1990; Karnath et al., 1991, 1993). It appears that neglect generally affects multiple frames of reference regardless of the task.

It is also possible that every neuron contributes to many behaviors; consequently, a lesion affects different behaviors in a similar way. There is no existing network model that uses a shared vectorial representation for coordinating multiple behaviors. In contrast, this can be achieved naturally with basis functions as a consequence of their computational properties (Pouget & Sejnowski, 1995).

To conclude, our basis function model of spatial representations can account for a wide range of experimental results regarding spatial neglect in patients with parietal lesions. This demonstrates that the symptomology of neglect is consistent with what is currently known of the response properties and hemispheric distribution of parietal neurons.

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