A Computational Model of Perceptual Fill-in Following Retinal Degeneration

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McManus JN, Ullman S, Gilbert CD. A computational model of perceptual fill-in following retinal degeneration. J Neurophysiol 99: 2086–2100, 2008. First published January 16, 2008; doi:10.1152/jn.00871.2007. The ablation of afferent input results in the reorganization of sensory and motor cortices. In the primary visual cortex (V1), binocular retinal lesions deprive a corresponding cortical region [lesion projection zone (LPZ)] of visual input. Nevertheless, neurons in the LPZ regain responsiveness by shifting their receptive fields (RFs) outside the retinal lesions; this re-emergence of neural activity is paralleled by the perceptual completion of disrupted visual input in human subjects with retinal damage. To determine whether V1 reorganization can account for perceptual fill-in, we developed a neural network model that simulates the cortical remapping in V1. The model shows that RF shifts mediated by the plexus of spatial- and orientation-dependent horizontal connections in V1 can engender filling-in that is both robust and consistent with psychophysical reports of perceptual completion. Our model suggests that V1 reorganization may underlie perceptual fill-in, and it predicts spatial relationships between the original and remapped RFs that can be tested experimentally. More generally, it provides a general explanation for adaptive functional changes following CNS lesions, based on the recruitment of existing cortical connections that are involved in normal integrative mechanisms.

INTRODUCTION

The removal of afferent input to the motor, somatosensory, auditory, and visual cortices triggers reorganization in those areas, with concomitant changes in neural function and behavior. In particular, the primary visual cortex (V1) undergoes topographic reorganization after the removal of sensory input from the retina (Baker et al. 2005; Calford et al. 2000; Chino et al. 1995; Darian-Smith and Gilbert 1995; Das and Gilbert 1995; Gilbert et al. 1990; Heinen and Skavenski 1991; Kaas et al. 1990; Schmid et al. 1995). When focal binocular retinal lesions are made in the adult cat or macaque, the loss of sensory input silences a region of the lateral geniculate nucleus (LGN) while spurring reorganization in the corresponding cortical region, known as the lesion projection zone (LPZ) (Darian-Smith and Gilbert 1995). Neurons ≈4 mm from the boundary of the LPZ regain responsiveness by shifting their receptive fields (RFs) from inside the lesion, or scotoma, to the region of visual space immediately outside the scotoma perimeter (the perisocoma).

Interestingly, these profound changes in V1 topography and neural function are paralleled by equally impressive perceptual consequences. Human subjects with artificially induced retinal lesions or with macular degeneration do not receive visual input from the damaged portions of their retinas, yet their visual perception is continuous and complete (Burke 1999; Craik 1966; Gerrits and Timmerman 1969; Schuchard 1993, 1995; Zur and Ullman 2003). Rather than perceiving holes or blind spots in their vision, these individuals can often “see” portions of the visual scene that are masked by their retinal lesions. Moreover, this phenomenon—known as perceptual fill-in or perceptual completion—is not the result of passively ignoring the missing regions of the retinal image, but rather it is an active filling-in of the lost regions of the visual field (Zur and Ullman 2003). Individuals with retinal damage do not simply ignore the lesion-induced blind spots, because they really “see” objects at the locations corresponding to their lesions, and their percepts within the scotomata can even contain anomalies and distinguishing features.

Convincing evidence for the active nature of perceptual completion comes from psychophysical studies on subjects with age-related macular degeneration (AMD), a common disease among the elderly in which lesions progressively develop on the retinae (Zur and Ullman 2003). When subjects with advanced AMD are shown regular arrays of dots that are partially occluded by a very large retinal lesion (spanning 5–7° in radius), the subjects can actually count the number of dots that fall within the lesion. When the same subjects are shown two-dimensional gratings, the grating lines appear to run continuously through the scotoma, but the perceptually reconstructed lines in the scotoma appear blurrier and at lower contrast than the lines outside the scotoma. Furthermore, this nonuniformity in their perception depends on the attributes of the grating lines: subjects experience better perceptual fill-in as the spatial frequency of the gratings is increased. Under unfavorable conditions, advanced AMD subjects even report gaps in the perception of objects they can only partially fill-in. If AMD subjects have a continuous perception only because they ignore the holes in their vision, they should never even notice stimuli within their scotomata—much less be able to report distinguishing characteristics about them—and their perception should not depend on the attributes of the environment they ignore.

The evidence for an active mode of perceptual completion is clear, and the anatomical and functional changes during V1 reorganization are well described. What remains unclear, however, is how the physical changes in cortical structure and function engender perceptual completion. Nevertheless, it seems likely that the substrate for cortical reorganization (and thus perceptual fill-in) is intrinsic to the cortex. The degree of reorganization observed along the visual pathway antecedent to the cortex, most notably at the LGN, is much more limited than...
the cortical reorganization (Darian-Smith and Gilbert 1995; Eysel 1982). Since the lateral spread of geniculocortical afferents to V1 is insufficient to account for the extent of the observed reorganization, the next most likely substrate is the plexus of long-range horizontal connections in V1 (Calford et al. 2003; Darian-Smith and Gilbert 1995). V1 reorganization is accompanied by a sprouting of these horizontal axon collaterals from outside to inside the LPZ, whereby the preexisting pattern of connections is intensified and reinforced (Darian-Smith and Gilbert 1994). Moreover, intrinsic optical imaging in the cat shows that reorganization preserves the orientation column architecture in V1 (Das and Gilbert 1995). Since the horizontal projections preferentially link orientation columns with similar orientation preference (Bosking et al. 1997; Chisum et al. 2003; Gilbert and Wiesel 1989; Shmuel et al. 2005; Stettler et al. 2002), this preservation of orientation column structure further suggests a role for the horizontal connections in the remapping (Das and Gilbert 1995).

If the horizontal connections play an important role in cortical reorganization—and if the reorganization respects their preexisting pattern of connectivity—it is tempting to speculate that the reorganized cortex should retain its ability to perform the computations implemented by those connections. A prominent role that has been proposed for the horizontal connections in normal V1, based on evidence from electrophysiological, anatomical, and psychophysical studies, as well as from theoretical considerations, is contour integration (Kapadia et al. 1995, 2000; Li 1998; Li and Gilbert 2002; Li et al. 2006; Sigman et al. 2001; Stettler et al. 2002). Given the premise that the reorganized cortex should retain the functions implemented by the horizontal connections, and the idea that these connections mediate contour integration and contour saliency, the implication is that V1 reorganization may preserve the perceptual integrity of contours occluded by retinal damage.

Here, we show how the functional changes that accompany V1 reorganization can be linked to the perceptual changes reported by human subjects with retinal lesions. We developed a neural network model to simulate the recovered neural activity in the LPZ, and the perceptual fill-in that might result, if the cortical reorganization in V1 is mediated by a network of long-range horizontal connections that normally underlies contour integration. Our goal was to determine if the lateral connections in V1 can account for the robust perceptual completion reported by subjects with retinal damage.

**Methods**

**Qualitative description and general overview**

**THE PARADIGM.** To explore the relationship between V1 reorganization and perceptual fill-in, we modeled the neural and perceptual changes that might accompany retinal deterioration in human subjects. The retinal degeneration simulated in the model includes idealized features of the dry form of AMD: large, continuous areas of photoreceptor loss, or scotomata, and intervening regions of diffuse, “salt-and-pepper” photoreceptor loss (Fig. 1A). The input to the model is a stationary visual scene, projected onto the lesioned retina of a hypothetical subject who fixates on the center of the image (e.g., Fig. 4B). The output from the model shows what this individual might perceive when looking at the input image, if the reorganization in V1 is mediated by geometrically specific horizontal connections (e.g., Fig. 4, D and E). The model overlays the input image with an array of black blotches (representing the “blind spots” caused by retinal lesions), and it computes an image that allows the reader to perceive what a subject might “see” underneath the lesion-induced blind spots.1

Our simulations were carried out in two stages. In the first stage, we simulated an ensemble of complex cells, in a hypothetical retinal lesion subject, from the superficial layers of the V1 LPZ. Our model focuses on the properties of superficial layer complex cells because the experimental evidence indicates that these are among the cells principally involved in the cortical reorganization. The axonal sprouting thought to underlie cortical reorganization occurs predominantly among the horizontal collaterals of pyramidal cells in the superficial layers of V1 (Darian-Smith and Gilbert 1994). These cells tend to have complex RFs (Hubel and Wiesel 1968), they participate both pre- and postsynaptically in the plexus of horizontal connections, and their extrastriate projections constitute the output from V1 to the rest of the visual cortex.

In the second stage, we computed an image that demonstrates to the reader the visual perception we expect to emerge from the neural responses simulated during the first phase. The activity in the simulated LPZ signals the presence of continuous contours that pass through the retinal lesions, so the model generates its output image by filling-in the input image along these contours. This second stage of our model is intended to produce images that allow the reader to perceive the visual scene like a subject with the simulated retinal damage. (We refer to this process as “illustrating the perceptual fill-in.”) Since the visual cortex of the retinal lesion subjects undergoes a compensatory process of reorganization, the images computed by our model are not simply black, or even left blank, where the input has been ablated. Rather, they are actively filled-in by means of a neural network model of V1 reorganization and neuronal responses. These images of perceptual fill-in are designed to produce activity patterns in the visual cortex of healthy readers that resemble the patterns we expect in retinal lesion subjects when they view the visual scene. To the extent that the reader’s perception coincides with the reports from lesion subjects, our model explains the cortical reorganization and perceptual fill-in phenomena.

**Stage 1: simulation of neural activity**

The first stage of the model, the simulation of neuronal activity, is itself composed of two successive computations, which simulate neural activity in the retina/LGN and in V1. The functional recovery of neuronal activity begins, in a limited way, at the retina and LGN, but the truly extensive reorganization takes place in V1.

**RETINAL/LGN COMPUTATIONS.** In our model, the image underneath the diffuse photoreceptor loss—but not underneath the large retinal lesions—is recovered by retinal ganglion cells (RGCs) and LGN neurons. The RFs of these cells contain a mixture of responsive photoreceptors juxtaposed with destroyed receptors (Fig. 1B), and the visual signal transduced by the functional photoreceptors diffuses through the unresponsive region in each RF. The retinal image is relayed to the cortex, but each spot of diffuse retinal damage is filled-in with a Gaussian average of the image on the adjacent spots of healthy retina (Fig. 4C). This process is modeled on a very small spatial scale. It simulates mechanisms that are potentially mediated by a reweighting of local lateral interactions in the retina, although a reweighting of converging inputs onto RGCs and LGN neurons may

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1 Since the reader will tend to direct his or her gaze throughout the simulation images, we used the same spatial resolution (i.e., pixel size and image clarity) throughout each image, even though the density of the photoreceptor mosaic, and therefore the spatial resolution of our vision, decreases with retinal eccentricity. This simplifies the presentation and implementation of the model, allows the reader to make saccades when perceiving the results, and it partially mimics the real-life perception of a subject with retinal lesions, whereby spatial attention and saccadic eye movements typically compensate for differences in resolution across the image.
photoreceptor loss over 60% of the retina. Black pixels represent regions of photoreceptor loss [where \( M(x,y) = 0 \)]; white pixels correspond to responsive regions of the photoreceptor mosaic [where \( M(x,y) = 1 \)]. In this example, the black pixels cover a total of 76.6% of the simulated portion of the retina. The lesions, which simulate scotomata from an early-to-intermediate stage of AMD, cover 41.4% of the retina in this example. Wherever the 2 components of retinal deterioration overlap in \( M(x,y) \), the form of atrophy is considered to be geographic rather than diffuse. Effectively, the geographic lesions cover 41.4% of the image, and the diffuse photoreceptor loss covers 60% of the remaining retina, or 35.2%.) Here, the simulated portion of the retina is taken to span the central 15° of the visual field, each pixel spans 2.2 min of arc, and the average area of the geographic lesions is 1.2 square degrees of visual arc. There are 79 lesions that dot the central retina, distributed over an area of 225 square degrees of visual arc.

also play a role. Since the RGCs and the LGN never recover from large retinal lesions, the image relayed to the cortex still contains the large blind spots.

The signal transmitted to V1 is the partially recovered retinal image—rather than the spiking activity of explicitly modeled neurons from the retina and LGN—because the processing done by RGCs and LGN neurons is subsumed by the complex cells in our network.

V1 COMPUTATIONS. In V1, we simulated a network of complex cells interconnected by a pattern of spatial- and orientation-selective horizontal connections. The connectivity pattern, called the association field (AF, after Field et al. 1993) and denoted by \( K \) throughout this work, models the neural interactions mediated by the long-range horizontal connections. Consistent with the anatomical and physiological data reviewed in the Introduction, these connections underlie contour integration and contour saliency in the “healthy” regions of our model cortex, and they resuscitate neural activity in the simulated LPZ. All of the neurons in our network, both in the LPZ and in the surrounding cortical area (termed the peri-LPZ), receive lateral connections from presynaptic neurons, which send out their axon collaterals according to the geometric pattern specified by \( K \). The strength of the lateral connections between each pair of model neurons depends on the relative spatial positions of their RFs and on their preferred orientations (POs). We tested two geometric patterns of connectivity, both of which are consistent with the known anatomical and physiological data. The first of these is called the collinear AF, in which the strongest network connections are between cells with collinear RFs (collinear RFs lay along a straight line whose orientation matches the POs of the neurons; Fig. 2E). The second is the co-circular AF, whereby co-circular RFs enjoy the strongest connection strengths. Co-circular RFs lay along a circular arc that passes through both RFs, where the PO of each RF is tangent to the circular arc (Fig. 2E). See Technical details of the model and Fig. 2 for the mathematical and graphical descriptions of these alternative connectivity patterns.

Given the connectivity pattern \( K \), in conjunction with the classical energy model of the complex cell (Adelson and Bergen 1985; Dayan and Abbott 2001), we simulated the activity of a network of V1 superficial layer neurons. The model RFs were distributed over a grid of spatial positions at each pixel in the input image, with five RF sizes and eight POs at each location. (We evenly spaced the RF sizes between 0.2 and 1.0° and the POs between 0 and 157.5°.) All the cells in our model received a normalized combination of bottom-up and lateral inputs, where the relative weight of the two contributions was determined by the amount of retinal damage in each cell’s RF (see Technical details of the model). As in the real cortex, healthy neuronal responses were driven by bottom-up input from the retinal RF and modulated both by geometric lateral inputs and surround inhibition. On the other hand, neuronal responses in the LPZ were driven primarily or exclusively by lateral inputs from the adjacent region of normal cortex, where the synaptic weights between healthy presynaptic cells and the postsynaptic LPZ neurons were specified by the connection pattern \( K \). At stimulus onset, the stimulation of intact retinal loci seeded the cortical activity in the healthy cortex, which then coursed recurrently through the network \( K \) and engendered new activity in the otherwise silent LPZ.

Stage 2: illustration of perceptual fill-in

The simulated activity in the cortex constitutes a prediction of the image orientations underlying the retinal lesions, and the model uses this neuronal prediction to illustrate the perception of a human subject with a specific pattern of retinal deterioration (see Technical details of the model). We postulated that the perception of the occluded visual scene arises from the joint activity in the peri-LPZ and the LPZ and that it minimizes brightness discontinuities along the continuous features that are predicted to run through the lesions. Experimental evidence (Li et al. 2006) shows that a particular response property of V1 neurons—namely, contour facilitation—accounts for the perceptual phenomena of contour integration and contour saliency. Therefore our model assumes that the facilitation of LPZ neurons by contours that pass through retinal lesions should lead to the integration and saliency of those contours within the scotomata—that is, to their perceptual completion.

To compute the output image illustrating perceptual completion, the model predicts the orientation around each point in the lesioned retinal image. The predicted orientation is derived from a principal components analysis (PCA) of the neural responses corresponding to each point on the lesioned retina. The PCA deciphers the underlying image orientation encoded by the activity in the LPZ, and it yields a measure of the saliency of the encoded orientation (which can also be interpreted as a measure of how reliably the neural responses predict the occluded image orientation). The output from our model is the image that selectively minimizes the luminance difference along the directions of the predicted image orientations (see Technical details of the model).

Technical details of the model

THE PARADIGM. Let the visual input to the model, defined over a domain in \( \mathbb{N} \times \mathbb{N} \), be the grayscale image \( I(x,y) : [1,A] \times [1,B] \rightarrow \mathbb{R} \); let the simulated pattern of retinal deterioration be the mask \( M(x,y) : [1,A] \times [1,B] \rightarrow \{0,1\} \), which is zero (i.e., occluding) wherever the retinae have atrophied, and unity (i.e., transparent) wherever both retinae are healthy.
The simulated image on the retinae is then the input image overlain with the retinal degeneration (e.g., Fig. 4B), and it is given by

$$I^R(x,y) = I(x,y)M(x,y)$$  \hspace{1cm} (I)

We define the regions of retinal atrophy by the set

$$\Omega = \{(x,y)|M(x,y) = 0\} = S_0 \cup \bigcup_{i=1}^{k} S_i$$  \hspace{1cm} (2)

where $S_0$ is the set of all pixels $(x,y)$ that constitute the diffuse photoreceptor loss (Fig. 1B), $k$ is the number of geographic lesions on the simulated retinae, and $S_i$ is the set of all pixels within the $i$th scotoma.

The retinal damage in our model, which is defined by a single mask (rather than one for each eye), may be interpreted as either monocular or binocular. Under the binocular interpretation, any point $(x,y)$ in the mask refers to a pair of corresponding points on each retina. After a monocular lesion is made, there is a shift in ocular dominance in the LPZ so that the input from the healthy, normal eye either exclusively drives (Chino et al. 1992), or at least dominates (Calford et al. 2000), or the healthy input from corresponding points on the other retina, as if both retinae were undamaged.

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the retinal damage in only one eye and the hypothetical subject views the world through only the correspondingly lesioned eye. In either case, binocular interactions are ignored in our model.

RETAIL/LGN COMPUTATIONS. The visual signal, $I^{n1}$, transmitted from the retina to V1 is given by

$$I^{n1}(x,y) = \begin{cases} \hat{P}(x,y) & \text{for all } (x,y) \notin S_0 \\ \sum_{(i,j) \in R_{x,y}} G^{(i,j)}(i,j) \hat{P}(i,j) & \text{for all } (x,y) \in S_0 \end{cases}$$

(3)

where

$$G^{(i,j)}(i,j) = \frac{1}{2\pi \sigma^2} e^{-((i-x)^2 + (j-y)^2) / 2\sigma^2}$$

(4)

and

$$R_{x,y} = \{(a,b) | a \in \Omega \}$$

(5)

Here, $G^{(i,j)}(i,j)$ is a two-dimensional Gaussian centered at the masked point $(x,y)$, and $R_{x,y}$ is the set of all unmasked points surrounding $(x,y)$ within a radius of $\delta$ pixels. The radius over which the Gaussian operating, $\delta$, is set at the lower limit of the dimensions needed to fill-in the diffuse, salt-and-pepper atrophy. The parameter $\sigma$ is set so that $\sum_{i,j \in R_{x,y}} G^{(i,j)}(i,j) = 1$, where $P_{x,y}$ is the set of all pixels surrounding $(x,y)$ within a radius of $\delta$ pixels. This condition simply scales the width of the Gaussian to the size of the averaging disk $R_{x,y}$ (Our simulations were run with $\delta = 2$ pixels, corresponding to between 4.4 and 5.8 min of arc, and $\sigma = 0.83$). We refer to the image $I^{n1}$ as the partially filled-in image; this is the image that is filtered by the complex cells in our V1 network, as described, to regenerate.

Model V1 Neurons

Let $n^{x,y}_{\gamma,\eta}$ be the V1 neuron with the following stimulus response characteristics: RF center $(x,y)$, RF width $r$, and PO $\theta$. Then we compute $n^{x,y}_{\gamma,\eta}$, the depolarization elicited by the (partially filled-in) stimulus within the cell’s RF, using the relation

$$n^{x,y}_{\gamma,\eta} = (f^{x,y}_{\gamma,\eta})^2 + (h^{x,y}_{\gamma,\eta})^2$$

(6)

Equation 6 is an expression of the classical energy model of the complex cell, whereby the neural response is simulated by convolving the visual stimulus (in our case, $I^{n1}$) with two quadrature RF filters and summing the squared filter responses (Adelson and Bergen 1985; Dayan and Abbott 2001). The classical RF of each neuron $n^{x,y}_{\gamma,\eta}$ is modeled with two square Gabor filters centered at $(x,y)$ with RF width $r$ and orientation $\theta$; the variables $f^{x,y}_{\gamma,\eta}$ and $h^{x,y}_{\gamma,\eta}$ denote the point-by-point multiplication of each of these filters with the image falling within the RF. As a convention, we set the L2-norm of the (unmasked) Gabor filters to unity, and we map the pixel values in the image $I^{n1}$ into the interval [0,1]. The spatial substructure of the RF filters is set to match the characteristics of superficial layer V1 RFs, with the additional constraint that, wherever the RFs are partially or completely damaged by a retinal lesion, the masked subregions of the Gabor filters are made unresponsive (i.e., they are set to zero). The RFs of our model neurons densely cover the input image. Centered over each pixel in the input image, there are 40 RFs with five different sizes (ranging from 0.2 to 1.0° in width) and eight different POs (spaced evenly between 0 and 157.5°).

The Gabor filters simulate how neurons respond to vertical inputs from stimuli falling within the classical RF. They do not encompass the extraclassical RF surround, which arises from lateral activation through the horizontal connections, and so they do not mediate reorganization following retinal lesions. When the RF of a cell is partially ablated, the corresponding regions of the Gabor filters are also permanently ablated, so that the classical RF becomes less responsive. Moreover, it often happens that for any partially ablated Gabor filter, the retinal damage destroys, for instance, a larger portion of the filter’s inhibitory subregion than of its excitatory one. Wherever that occurs, the magnitude (or responsiveness) of the stronger subregion is scaled down so that the total responsiveness of the excitatory and inhibitory lobes is balanced. That is, filter regions that are directly masked are nullified, whereas unmasked regions of the same filter may be scaled down so that the total magnitude of the unmasked inhibitory lobes equals the unmasked excitatory lobes. (Otherwise, unbalanced RF filters would cause chronic neural activity in many cells at the border of the LPZ, a phenomenon that is not observed in the long-term reorganized cortex.)

Let $m^{x,y}_{\gamma,\eta}$ be the fraction of the RF of neuron $n^{x,y}_{\gamma,\eta}$ that has been ablated by retinal damage. Additionally, let $G_{1,x,y}$ and $G_{2,x,y}$ be the Gabor filters of the partially masked neuron $n^{x,y}_{\gamma,\eta}$, and let $G_{1,x,y}$ and $G_{2,x,y}$ denote the unmasked versions of these same Gabor filters, which would be revealed if we were only to remove the retinal damage overlying the RF. Then $\sum_{i,j}(G_{1,x,y}(i,j) - G_{1,x,y}(i,j))$ denotes the sum over the absolute value of the unmasked Gabor filters $G_{1,x,y}$, and we define the masked fraction of the RF as follows

$$m^{x,y}_{\gamma,\eta} = \sum_{i,j}|G_{1,x,y}(i,j) - G_{1,x,y}(i,j)| + \sum_{i,j}|G_{2,x,y}(i,j) - G_{2,x,y}(i,j)|$$

(7)

MODEL V1 CONNECTIVITY. The V1 neurons in our model are interconnected by a plexus of long-range horizontal connections. The lateral connectivity is based on a vector field that represents the strongest excitatory interactions in the network. Each presynaptic cell is associated with a vector field that describes the neurons that receive the strongest excitatory connections from that cell. For instance, let $\bar{F}_{x,y}$ be the vector field of a presynaptic neuron $(n^{x,y}_{\gamma,\eta})$ with RF center $(x,y)$ and PO $\theta$. The direction of the field $\bar{F}_{x,y}$ at each point $(i,j)$ specifies the PO of the neuron with RF center $(i,j)$ that receives the strongest excitatory connection from the presynaptic cell. All of the neurons at $(i,j)$, it is the one whose PO makes it co-circular with the presynaptic cell that receives the strongest connection. The field magnitude at $(i,j)$, in turn, is the connection strength between these two co-circular cells. We therefore call $\bar{F}_{x,y}$ the co-circular vector field, since it specifies that the strongest excitatory connections in the network are between co-circular neurons (see Fig. 2, A and D, for a graphical depiction of this field). The mathematical description of the field, which was modified from Guy and Medioni (1996), is given by

$$\bar{F}_{x,y}(i,j) = \begin{cases} \exp(-A_1(x'y')^2) \left[ \frac{x'}{|x'|} \right] & \text{if } y' = 0 \\ \frac{1}{r_{\text{aug}}} \exp(-A_1(x'y')^2 - B_1(\arctan(|y'|/|x'|)))^2 \times \left[ \frac{x'}{|x'|} \left( \sqrt{R^2 - x'^2 + z} \right) \right] & \text{if } y' \neq 0 \end{cases}$$

(8)

where

$$(x',y') = (i - x, y - j)$$

(9)
The parameters in the equation are as follows: $A_1 = 1.2$ deg of arc subtended at the eye, $B_1 = 5.7$; $A_3 = 24.1$ deg of arc subtended at the eye; $B_3 = 2.85$; and $C = 50$. For simplicity, we express $\tilde{F}_{x,y,0}$ in a rotated coordinate system whose $x$-axis is collinear with the PO of the presynaptic neuron. The exponential terms to the left of the vectors in Eq. 8 determine the magnitude of the field at each position $(i,j)$ (see Fig. 2D). Beside the exponential terms are the unit vectors that determine the co-circular geometry of the field, with a collinear bias near the origin given by $z$. This correction term $z$ favors iso-orientation facilitation over co-orientation at very small spatial scales, in agreement with psychophysical measurements of contour saliency and with the statistics of natural scenes (Field et al. 1993; Geisler et al. 2001; Sigman et al. 2001).

In some of our simulations, to test the effect of reshaping the AF geometry through top-down interactions, we substituted this co-circular vector field with a unidirectional one (see Fig. 2C).

$$\tilde{F}_{x,y,0}(i,j) = \exp(-A_1(\gamma x^2 - B_1(\arctan(|y|x^2))))[x|/x| \ 0]^T$$

expressed here with the same notation as the co-circular field above. We call $\tilde{F}_{x,y,0}$ the “collinear field”; it specifies that the strongest excitatory connections are between neurons that share the same orientation preference.

Given the vector field of maximally excitatory connections made by each presynaptic cell, we can now specify all of the connections in the network as a function of their deviation from co-circularity (or collinearity, depending on which vector field is used as the basis for the network connectivity). Let $DF_{x,y,0}(i,j,\alpha)$ be the angular difference (in radians) between the vector field direction at $(i,j)$ and the orientation preference $\alpha$. Additionally, let $\tilde{F}_{x,y,0}(i,j)$ be the field obtained by rotating each vector in $\tilde{F}_{x,y,0}$ by 90° (Fig. 2B). Then $K_{x,y,0}(i,j,\alpha)$, which denotes the sign and magnitude of the connection between a presynaptic neuron with RF center $(x,y)$ and PO $\theta$ and a postsynaptic neuron at $(i,j)$ with PO $\alpha$, is given by

$$K_{x,y,0}(i,j,\alpha) = \frac{\|\tilde{F}_{x,y,0}(i,j)\exp(-D(\tilde{F}_{x,y,0}(i,j,\alpha))^{\alpha/15})}{\|\tilde{F}_{x,y,0}(i,j)\exp(-D(\tilde{F}_{x,y,0}(i,j,\alpha))^{\alpha/15})}$$

If the geometric relationship between the pre- and postsynaptic neurons is close to co-circular [i.e., if $D(\tilde{F}_{x,y,0}(i,j,\alpha)) \leq \pi/4$], then the presynaptic neuron makes an excitatory connection onto the postsynaptic cell, with a strength that falls off exponentially with the angular deviation from co-circularity. On the other hand, if the relationship between the two neurons is very different from co-circularity [$D(\tilde{F}_{x,y,0}(i,j,\alpha)) > \pi/4$], the connection between the two neurons is modeled as an (implicitly dissociative) inhibitory connection whose strength rises exponentially with the angular deviation from co-circularity. The number 0.15 in Eq. 14 was chosen so that the connection strength between cells was very weak (~2% of the vector field strength) when the postsynaptic PO differs by $45^\circ$ ($\pi/4$ radians) from the field direction.

SIMULATION OF CORTICAL ACTIVITY. We denote the total membrane depolarization of neuron $n_{x,y}^{\text{po}}$ at time $t$ by $S_{x,y}^{\text{po}}$, and the instantaneous firing rate, obtained by passing the depolarization through a static nonlinearity, as $g(S_{x,y}^{\text{po}})$. The activation $S_{x,y}^{\text{po}}$ arises from the depolarization ($n_{x,y}^{\text{po}}$) induced by the RF stimulus, and it is modulated by the sum of excitatory and inhibitory inputs from the association field $K$. Besides the vertical inputs to the RF, each cell $n_{x,y}^{\text{po}}$ receives postsynaptic potentials from the network of horizontal collaterals extending from the presynaptic cells $n_{x,y}^{\text{po}}$ in the peri-LPZ. The sign and strength of the synapse between each pre- and postsynaptic cell pair is given by the product of a scaling factor $\gamma_1$ and the connection $K_{x,y,0}(x,y,\theta)$. Therefore at time $t$, the synaptic potentials from the horizontal connections in postsynaptic cell $n_{x,y}^{\text{po}}$ are given by the firing rates $g(S_{x,y}^{\text{po}}(\cdot))$ of the presynaptic cells, multiplied by the term $\gamma_1 K_{x,y,0}(x,y,\theta)$ (see Eq. 15). The effect of these potentials on the postsynaptic cell is gated by a toggle-like term (given by $\{G_{x,y,0}(1-m_{x,y}^{\text{po}}) + ([f_x^{\text{po}} + (h_x^{\text{po}})^2])G_{x,y,0}(m_{x,y}^{\text{po}}))\}$), which tends to multiply the potentials either by the responses of the (normalized) postsynaptic RF filters or by a constant factor $\xi$, depending on the degree of RF degeneration, $m_{x,y}^{\text{po}}$. This term recapitulates experimentally observed phenomena in the normal cortex and in the LPZ, where the efficacy of the horizontal inputs is either modulated by coincident activation from within the RF (as in the normal cortex, Hirsch and Gilbert 1991) or else is facilitated to suprathreshold levels when the vertical input is silenced (as in the LPZ, Calvert et al. 2003; Darian-Smith and Gilbert 1994). The term mediates a drastic and switch-like change in neuronal response properties after cortical reorganization. As the amount of retinal deterioration in a neuron’s classical RF increases, the neuron rapidly becomes highly responsive to lateral inputs from neighboring cortical cells, regardless of the visual input within its classical RF.

In addition to the excitation and inhibition mediated by the lateral connections, the simulated cortical activity is subject to a gain control mechanism mediated by surround inhibition. The firing of the model neurons is inhibited by the overall cortical activity as a function of the distance between the inhibited RFs and the RFs of the active cells in the surrounding cortex.

The total membrane depolarization, $S_{x,y}^{\text{po}}$, is therefore computed via the recurrence relation

$$n_{x,y}^{\text{po}} + \{G_{x,y,0}(1-m_{x,y}^{\text{po}}) + ([f_x^{\text{po}} + (h_x^{\text{po}})^2])G_{x,y,0}(m_{x,y}^{\text{po}}))\} \times \gamma_1 K_{x,y,0}(x,y,\theta) \sum_{i,j} g(S_{i,j}^{\text{po}})K_{i,j,0}(x,y,\theta)$$

$$S_{x,y}^{\text{po}} = \frac{1 + \gamma_2 \sum_{i,j} g(S_{i,j}^{\text{po}})K_{i,j,0}(x,y,\theta)}{1 + \gamma_2 \sum_{i,j} K_{i,j,0}(x,y,\theta)}$$

The membrane depolarization $S_{x,y}^{\text{po}}$ at the initial time point (right after stimulus onset) is the classical response $n_{x,y}^{\text{po}}$, and the membrane potentials converge to a steady state in $t \sim 10$ iterations of network activity. The static nonlinearity $g(x)$ that converts the membrane depolarization to an instantaneous firing rate is given by

$$g(x) = \begin{cases} 0 & \text{for } x < 0 \\ x & \text{for } 0 \leq x \leq x_{\text{th}} \\ x_{\text{th}} & \text{for } x > x_{\text{th}} \end{cases}$$

In Eq. 15, $f_x^{\text{po}}$ and $h_x^{\text{po}}$ are the convolution responses obtained from the Gabor filters of neuron $n_{x,y}^{\text{po}}$, except that each filter is normalized so that its L2-norm equals 1. $L_{x,y}$ is the set of all spatial positions from which neurons in the peri-LPZ laterally contact the cells with RF center $(x,y)$; $N_{x,y}$ is the set of all points in the visible image within $2^\circ$.

Like many neural network models, we used a piecewise linear function to convert membrane depolarizations into instantaneous firing rates. We chose the simplest and most straightforward model, in which the neuronal firing rate is a linear function of the membrane potential, the minimum firing rate is 0 Hz, and the maximum firing rate is a finite number. The size of our network precludes continuous-time simulations of spike trains. Therefore we took the common approach of simulating our network with instantaneous firing rates that were updated at discrete iterations of network activity.
of the point \((x,y)\); \(\Phi\) is the set of neuronal orientation preferences in the model, \([0, 22.5, \ldots, 157.5°]\); \(d(x,y,i,j)\) is the spatial distance between the points \((x,y)\) and \((i,j)\) in degrees of arc subtended at the eye; \(b = 1\) degree of arc subtended at the eye; and \(G(m)\) is a logistic function that modulates the efficacy of horizontal connections as a function of the occluded fraction of the postsynaptic RFs

\[
G(m) = \frac{1}{1 + (1/p_1 - 1) \exp(-p_2(1 - m))} - p_1
\]

The function \(G(m)\) is a one-dimensional logistic curve that decreases from 1 to 0 as the variable \(m\) increases from 0 to 1. Its parameters, \(p_1\) and \(p_2\), are set to satisfy two constraints: 1) that \(G(m)\) maps its domain into the range \([0,1]\); and 2) that the midpoint of the function lies at the coordinate \(m = x\) [i.e., so that \(G(x) = 0.5\)].

The parameters referenced above are \(x_{th} = 20\); \(\xi = 10\); \(\gamma_1 = 0.001\); \(\gamma_2 = 0.002\); and \(p = 0.75\). For neurons in the peri-LPZ, \(L_{xy} = \nabla_{xy}\). For cells in the LPZ (i.e., all neurons in the set \(\{n^2\};(x,y) \in \bigcup_{r} S_i\}), \(L_{xy}\) is the set of all pericortical positions within 1° of the scotoma border, excluding all points \((i,j)\) such that the cortical distance between the neurons representing the points \((x,y)\) and \((i,j)\) exceeds 3.5 mm, the radial extent of the lateral projections (Stettler et al. 2002). Note that the magnitude and spatial extent of the contextual interactions in the real cortex are strongly dependent on the characteristics of the visual stimulus (Kapadia et al. 2000; Li et al. 2006). For instance, the modulatory effect of the lateral interactions in normal cortex is essentially nil for simple stimuli stimulated at high contrast. Therefore whenever the model input \(I(x,y)\) is specified as a black-and-white artificial image (as in Figs. 3 and 5), we use \(\gamma_1 = \gamma_2 = 0\) for neurons in the peri-LPZ, and for LPZ neurons we reduce the spatial radius in \(L_{xy}\) from 1 to 0.5°.

In the numerator of Eq. 15, the term on the right represents the lateral inputs to neuron \(n_{xy}^2\), modulated postsynaptically by the term \(\xi G_{1-p}(1 - m_{xy}^2) + [(\hat{f}_{xy}^2) + (h_{xy}^2)]G(m_{xy}^2)\), where the gating term \(\xi G_{1-p}(1 - m_{xy}^2) + [(\hat{f}_{xy}^2) + (h_{xy}^2)]G(m_{xy}^2)\) which describes the efficacy of the lateral connections as a function of the retinal damage \(m_{xy}^2\), is the mathematical expression of cortical reorganization in the model. It is here that the preexisting scaffold of horizontal connections is converted from a modulatory force in the normal cortex—whereby the connections modulate neural responses to stimuli in the RF center—to a driving force that engenders suprathreshold activity in the LPZ.

ILLUSTRATION OF PERCEPTUAL FILL-IN. For each point \((x,y) \in \bigcup_{r} S_i\), the converged firing rates \(g(S^{x,y}_{R};\theta)\) can be assembled into five covariance matrices (after Guy and Medioni 1996), one for each RF size. Let \(v_0\) be the unit vector whose direction matches the orientation preference \(\theta\) and let \((v_{0x}, v_{0y})\) be the \(x\)- and \(y\)-components of the vector, respectively. Then the covariance matrix for RF size \(r\) at the scotoma point \((x,y)\) is

\[
C_{xy} = \begin{bmatrix}
\sum_{m \in \Phi} \left(\hat{v}_{0x}(m_{xy}^2)^2 + \hat{v}_{0y}^2(m_{xy}^2)\right) & \sum_{m \in \Phi} \left(\hat{v}_{0x}(m_{xy}^2)^2 + \hat{v}_{0y}^2(m_{xy}^2)\right) \\
\sum_{m \in \Phi} \left(\hat{v}_{0x}(m_{xy}^2)^2 + \hat{v}_{0y}^2(m_{xy}^2)\right) & \sum_{m \in \Phi} \left(\hat{v}_{0x}^2(m_{xy}^2)^2 + \hat{v}_{0y}^2(m_{xy}^2)\right)
\end{bmatrix}
\]

where \(r\) can be any of the five RF sizes simulated in the network (i.e., \(r \in \{0.2, 0.4, 0.6, 0.8, 1.0°\}\)). The singular value decomposition (SVD) of the covariance matrix for each RF size \(r\) yields the two eigenvectors \((\hat{p} \text{ and } \hat{p}^\perp)\) of the matrix, together with their corresponding eigenvalues \((\lambda_1 \text{ and } \lambda_2)\)

\[
\text{SVD}(C_{xy}) = \begin{bmatrix}
\hat{p}^T & \hat{p}^\perp
\end{bmatrix}
\begin{bmatrix}
\lambda_1 & 0 \\
0 & \lambda_2
\end{bmatrix}
\begin{bmatrix}
\hat{p} & \hat{p}^\perp
\end{bmatrix}
\]

For each lesioned point \((x,y)\), we chose the covariance matrix (and its associated RF size \(r\) that possesses the largest difference between its two eigenvalues. The predicted orientation at \((x,y)\) was then \(\hat{p}\), the eigenvector from this chosen matrix with the largest corresponding eigenvalue, and our measure of the predicted saliency around \((x,y)\) was just the difference between the two eigenvalues of the matrix, \(\lambda_1 - \lambda_2\) (after Guy and Medioni 1996). (Alternatively, if all the responses from neurons with different RF sizes were merged into one covariance matrix \(C_{xy}\), and the orientation and saliency in the image were predicted from that matrix, the resultant predictions were quantitatively similar.) The products of the unit eigenvectors \(\hat{p}_{xy}\) and the saliencies \((\lambda_1 - \lambda_2)\) corresponding to each point \((x,y)\) on the lesioned retinae, constitute a vector field of predicted orientations and saliencies everywhere in the occluded retinal image (Fig. 3F).

We computed our illustrations of perceptual fill-in via a function minimization that was inspired by, and closely related to, an anisotropic diffusion process (Perona and Malik 1990). Let the illustrated perception, defined over a domain in \(\mathbf{N} \times \mathbf{N}\), be \(P(x,y) : [1,1] \times [1,1] \rightarrow \mathbf{R}\), with \(P(x,y) = I^{xy}(x,y)\) for all \((x,y) \in \bigcup_{r} S_i\). We find the image \(P\) that minimizes the function

\[
F = \sum_{(x,y) \in S_i} \frac{1}{T^2} \left[ C_{N^y}^{xy}(\Delta_{N^y}^{xy} + \Delta_{N^x}^{xy}) + C_{N^w}^{xy}(\Delta_{N^w}^{xy} + \Delta_{N^w}^{xy}) - C_{N^y}^{xy}(\Delta_{N^y}^{xy} + \Delta_{N^x}^{xy}) + \frac{1}{T^2} \left( C_{N^x}^{xy}(\Delta_{N^w}^{xy} + \Delta_{N^w}^{xy}) + C_{N^w}^{xy}(\Delta_{N^w}^{xy} + \Delta_{N^w}^{xy}) \right) \right]
\]

where

\[
C_{N^y}^{xy} = \exp(q(\lambda_1 - \lambda_2)\hat{p}_{xy} \cdot [0 1]^T)^2
\]

\[
C_{N^w}^{xy} = \exp(q(\lambda_1 - \lambda_2)\hat{p}_{xy} \cdot [1/\sqrt{2} 1/\sqrt{2}]^T)^2
\]

\[
C_{N^x}^{xy} = \exp(q(\lambda_1 - \lambda_2)\hat{p}_{xy} \cdot [1 0]^T)^2
\]

\[
C_{N^w}^{xy} = \exp(q(\lambda_1 - \lambda_2)\hat{p}_{xy} \cdot [-1/\sqrt{2} 1/\sqrt{2}]^T)^2
\]

Note that the gating term

\[
\xi G_{1-p}(1 - m_{xy}^2) + [(\hat{f}_{xy}^2) + (h_{xy}^2)]G(m_{xy}^2)
\]
A MODEL OF PERCEPTUAL FILL-IN

The average angular discrepancy (i.e., image reconstruction error) in the perceptual completion of the circle is $D = 5.1^\circ$. See RESULTS for a description of this average angular difference, $D$. (In the figures that follow, the image reconstruction errors for each simulation are reported parenthetically as $D = x^\circ$). $D$: the saliency map computed from the firing rates in the LPZ (bright white indicates high saliency; dark gray indicates low saliency). The lobes of high saliency indicate where contour facilitation within the LPZ signals the presence of a continuous contour passing through the lesions. $E$: the (normalized) vector field of predicted orientations $\hat{p}_{x,y}$ at each position underneath the indicated scotoma. $F$: the vector field of predicted orientations and saliencies at each position in the indicated scotoma. The magnitudes of the vectors signal the saliency $(\lambda_1 - \lambda_2)_{x,y}$ corresponding to the predicted orientation at each scotoma point $(x,y)$. The perceptual illustration in $C$ was obtained by filling-in the lesions along the vector fields in each scotoma. See text in METHODS accompanying Eqs. 18 and 19 for a description of how the predicted orientations and their corresponding saliencies are derived from the neural responses in the LPZ.

$\Delta_{\text{N}} = P(x, y - 1) - P(x, y)$
$\Delta_{\text{NE}} = P(x + 1, y - 1) - P(x, y)$
$\Delta_{\text{E}} = P(x + 1, y) - P(x, y)$
$\Delta_{\text{NE}} = P(x + 1, y + 1) - P(x, y)$
$\Delta_{\text{N}} = P(x, y + 1) - P(x, y)$
$\Delta_{\text{NE}} = P(x - 1, y + 1) - P(x, y)$
$\Delta_{\text{E}} = P(x - 1, y) - P(x, y)$
$\Delta_{\text{NE}} = P(x - 1, y - 1) - P(x, y)$

$T^{x,y} = 2 (C_N^{x,y} + C_{\text{NE}}^{x,y} + C_E^{x,y} + C_{\text{NW}}^{x,y})$

$F$ is the average of the local brightness differences in the image $P(x,y)$ along four directions (north-south, northeast-southwest, east-west, northwest-southeast), weighted by a set of four coefficients ($C_N, C_{\text{NE}}, C_E, C_{\text{NW}}$) that measure how well each direction matches $\hat{p}_{x,y}$, the predicted orientation in the underlying image. The minimization of this function selectively reduces differences in the image brightness along the predicted contours in the scotoma, thereby filling-in the image along those contours. (Note that this minimization is not intended to explicitly simulate the physiological activity of any neural network. It is merely intended to show the perceptual consequences of the cortical reorganization.)

The superscripts in the preceding equations represent pixel coordinates, whereas the subscripts represent image directions. For instance, $NE$ denotes the northeast (top right) direction, so $\Delta_{\text{NE}}$ is the difference in the illustrated perception between the pixel $(x,y)$ and the pixel immediately above it and to the right. For every scotoma point, the image is selectively filled-in along the directions with the largest coefficients. The coefficient $C_X^{x,y}$, corresponding to direction $X$ (e.g., $X = \text{NE}$) at scotoma point $(x,y)$ is determined by the product of three quantities: 1) the predicted saliency at $(x,y)$; 2) the angular agreement between the predicted orientation at $(x,y)$ and the direction $X$; and 3) the parameter $q$—which controls the direction specificity of the perceptual fill-in. Large values of the parameter $q$ highlight differences in the coefficients between different directions. They therefore engender a sharp form of fill-in, whereby the image is filled-in along the directions that match the predicted orientations in the image. On the other hand, small values of $q$ produce the opposite effect: they yield a diffuse, blurry perception, whereby the brightness differences in the image are minimized along all directions at once. We set $q$ to be relatively large ($q \in \{10^4, 10^5\}$) to produce a highly directional mode of fill-in.

RESULTS

Our simulations show that cortical reorganization mediated by geometric horizontal connections can explain the perceptual fill-in phenomenon. Even in the face of severe retinal deterioration, the simulated activity in the LPZ can engender the robust perceptual completion of disrupted visual input. Figures 3–7 show several input images, their
The precise shape of the AF can noticeably influence the perceptual completion of the visual scene. In Fig. 4, we show how the perceptual fill-in of a city landscape differs when the neural responses in the LPZ are driven through either the co-circular or collinear AF. Since the geometry of the collinear field corresponds more closely to the buildings in the image than does the co-circular field, it leads to a slightly more accurate completion of the skyscraper at the left of the image (Fig. 4). A co-circular field, on the other hand, can outperform the collinear geometry when curvature and junctions between intersecting lines are prominent features in the visual scene (Fig. 5). More generally, these results suggest how top-down modulation of the contextual interactions in early visual cortical areas may be manifested in perceptual completion. Object expectation for an observer viewing a visual scene like the one depicted in Fig. 4 could conceivably lead to a dynamic, top-down reshaping of the AF to match the predominantly collinear geometry of the environment. Similarly, when looking at a face like Einstein’s in Fig. 6, a higher-level face representation might reshape early contextual interactions to match expected facial characteristics. Although top-down influences on the AF are outside the scope of this model, there is some compelling psychophysical evidence showing the importance of expectation in perceptual fill-in among patients with retinal damage (Schuchard 1995).
Figure 8 offers a comparison between the perceptual fill-in obtained from our model and the image reconstructions generated by filling-in the scotomata with a nondirectional process. While our model uses the simulated LPZ activity to fill-in the lesions along specific directions, the alternative method fills-in each scotoma pixel through isotropic diffusion. In isotropic diffusion, the image along the outside edges of the lesions "flows" nondirectionally into the scotomata, so each pixel is filled-in with an average of the pixel values in the image surrounding the lesions. (The isotropic diffusion process is equivalent to performing the function minimization described in METHODS, but with the parameter $q$ set to zero.) The comparisons in Fig. 8 clearly show the subjective disadvantage of a perceptual completion mechanism that does not use prior knowledge of the statistical correlations in images (see DISCUSSION). The isotropic diffusion produces results that are characterized exclusively by a hazy, diffuse-looking form of fill-in. The presence of the retinal lesions is much more evident in the image reconstructions generated by the nondirectional method than in the perceptual fill-in from the model. This discrepancy is greatest for scenes that are characterized by a set of salient contours and that lack textured surfaces. While the nondirectional filling-in process can produce reasonable reconstructions of certain images, it cannot capture many of the basic psychophysical observations reported by subjects with mild or intermediate retinal degeneration, particularly the completion of smooth contours and the inconspicuousness of the lesions.

We also analyzed the simulated ensemble activity in the LPZ quantitatively. We computed a measure of the angular difference between the image orientations underneath the retinal lesions and the orientations predicted by the model

$$D = \frac{\sum_{(x,y)} \Psi_{x,y} \min\left(\left|\theta_{x,y} - \theta_{x,y}^o\right|, \pi - \left|\theta_{x,y} - \theta_{x,y}^o\right|\right)}{\sum_{(x,y)} \Psi_{x,y}}$$

where $\theta_{x,y}^o$ is the actual orientation in the image around each "missing" image point $(x,y)$; $\Psi_{x,y}$ is a measure of the contrast along that orientation in the underlying image; $\theta_{x,y}^o$ is the corresponding neuronal prediction for the orientation around $(x,y)$; and $0 \leq \theta_{x,y}^o, \theta_{x,y} \leq \pi$. ($\Psi_{x,y}$ and $\theta_{x,y}$ are the "oriented energy" and the "dominant orientation" obtained from the method of Freeman and Adelson 1991.) $D$, then, is the average of the angular difference between the real and predicted image orientations, weighted by the contrast of the actual orientation.
in the underlying image. This measure gauges how well the neural activity in the LPZ, despite the absence of visual input, signals the orientation of contours that are present in the underlying image. The average value of $D$, taken over all the simulation results shown here, is $15.6^\circ$, with a SD of $7.4^\circ$ and a range from $5.1$ to $25.9^\circ$. That is significantly better than the expectation from random activity in the LPZ, which is $45^\circ$. The specific value of $D$ for each simulation result is presented in the corresponding figure captions. Quantitative differences in the value of $D$ resulting from whether the simulations were run with co-circular or collinear connections tended to be small, although subtle differences in the illustrated perceptual fill-in can be observed (see Figs. 4 and 5). (The two connectivity patterns engender similar network behavior because collinearity is a special case of co-circularity, occurring when the radius of circular curvature is infinite, and because the strongest interactions in both fields lay along the collinear axis.) We also found that the angular discrepancy between the real and predicted image orientations tended to be quite small wherever the underlying image contained a salient contour but larger elsewhere (data not shown). Not surprisingly, the image reconstructions were more accurate (the value of $D$ was smaller) for the artificial images consisting of only salient contours (like the images in Figs. 3 and 5) than for the photographic images.

The perceptual fill-in is mediated by a network of connections that normally underlies contour integration and contour saliency, so the fill-in mechanism specializes in the completion of lines and edges rather than surfaces and textures.

Both methods of evaluation—the subjective evaluation of the simulated perceptual fill-in and the quantitative analysis of LPZ activity—indicate that the neural activity in the LPZ can accurately describe a retinal image composed of clearly delineated contours, despite severe damage encompassing $75\%$ of the simulated retina.

**DISCUSSION**

We used AMD as a model to show how reorganization in V1 may engender perceptual fill-in, but the work described here pertains more to the general linkages between cortical reorganization and functional recovery than to AMD per se. Here, we have shown that cortical reorganization via the sprouting of V1 horizontal connections can engender perceptual fill-in by co-opting a preexisting set of connections ordinarily involved in the integration of local stimulus features into globally salient contours. The model makes the novel prediction that, if the horizontal connections really underlie perceptual fill-in, the reorganized RFs should shift along the directions of the geo-

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**FIG. 6.** The Einstein photograph. Clockwise, from top to bottom: input image (16° in width), retinal image, illustrated perception, and visual signal transmitted to V1 ($D = 25.9^\circ$).
metric association field (either along collinear or circular directions). These results may represent a more general phenomenon, whereby the connectivity and functional architecture that subserve normal integrative mechanisms are advantageously recruited during cortical reorganization and functional recovery following CNS lesions.

Our model is based on known patterns of connectivity in the visual cortex. The mathematical descriptions of the model neurons and their interconnections, presented in methods, are derived from a substantial body of experimental work in the normal and reorganized cortex, including anatomical, electrophysiological, optical, psychophysical, and statistical studies (Adelson and Bergen 1985; Bosking et al. 1997; Chisum et al. 2003; Darian-Smith and Gilbert 1994, 1995; Das and Gilbert 1995; Geisler et al. 2001; Gilbert and Wiesel 1989; Hirsch and Gilbert 1991; Kapadia et al. 2000; Sceniak et al. 2001; Sigman et al. 2001; Stettler et al. 2002; Zur and Ullman 2003). The model is also inspired by a range of work in the theoretical literature on intermediate level vision and image reconstruction (Ballester et al. 2001; Guy and Medioni 1996; Parent and Zucker 1989; Perona and Malik 1990; Sha’ashua and Ullman 1988; Williams and Jacobs 1997).

With its strong experimental and theoretical foundations, our model accounts for many psychophysical observations reported by human subjects with retinal damage. The geometric relationships between the interconnected neurons in our model offer a parsimonious explanation for the robust perceptual completion of lines across retinal lesions. Furthermore, the limited lateral extent of the long-range horizontal connections is consistent with the observation that lines are perceptually completed across scotomata spanning a few degrees or less, but lines interrupted by larger scotomata spanning 10° or more are often perceived with gaps (Craik 1966; Gerrits and Timmerman 1969; Schuchard 1993, 1995; Zur and Ullman 2003). Although our model does not address the perceptual fill-in of textures, it does reproduce the general perceptual features described by individuals with retinal damage. Human visual perception is likely determined by neuronal activity throughout the whole visual cortex, but our model simulates specific components of perceptual fill-in that may arise from a specific set of neurons in V1. The model predicts that perceptual fill-in of the lines and edges that form the structure of visual scenes is mediated by the horizontal connections in V1, and that the fill-in of other visual attributes (like texture) involves similar neuronal networks.

The fundamental basis of the model, and the value of cortical reorganization in V1, derives from the long-range statistical correlations that characterize natural images. In natural visual scenes, locally oriented image regions tend to have a co-circular geometric relationship: given an orientation $\alpha$ around point $(x,y)$, the most probable orientation $\theta_p$ around any point $(i,j)$ is co-circular to the orientation $\alpha$ at $(i,y)$ (Fig. 2E) (Geisler et al. 2001; Sigman et al. 2001). The conditional probability of observing $\theta_p$ at $(i,j)$ is strongest when $\theta_p$ is collinear with the orientation at the given point $(x,y)$, but the conditional probability decreases as the curvature of the tangent circle connecting the two points increases and as the distance between the two points increases. This character of
natural scenes, combined with anatomical and psychophysical evidence (Field et al. 1993; Geisler et al. 2001; Sigman et al. 2001), provides the basis for the geometry of the association field used by the model. In a network exposed to natural images, neurons with co-circular RFs will tend to fire in coincidence more often than other neurons, in accordance with natural statistics. The principle of Hebbian plasticity, in turn, implies that neurons with co-circular RFs should have the strongest excitatory connection strengths and that excitatory connection strengths between co-circular RFs should fall off according to distance and radius of curvature. The result is that the neurons in the LPZ are, in some sense, optimal predictors of the image orientations underlying the scotomata, since they are activated by connections which reflect the most likely stimulus configurations. However, the usefulness of the cortical reorganization also depends on the perceptual interpretation of the recovered LPZ activity. The model cells represent “labeled lines” for specific regions of visual space, and although their input structures (their RFs) change during cortical reorganization, the interpretation of their outputs by subsequent cortical stages does not. The activity of the LPZ neurons, which are really responding to stimuli falling outside the scotoma, is interpreted as a reflection of the visual scene falling within the original RF positions.

Still, the assertion that topographic reorganization occurs in V1 is not without controversy (Smirnakis et al. 2005; but see Calford et al. 2005). In opposition to a large body of electrophysiological and intrinsic imaging evidence for the reorganization in adult V1 (Calford et al. 2000; Chino et al. 1992, 1995; Das and Gilbert 1995; Gilbert et al. 1990; Kaas et al. 1990), Smirnakis et al. (2005) have put forward functional MRI (fMRI) data suggesting that the LPZ border, as delineated by the BOLD signal, does not contract between the first 2–3 h following the retinal lesions and the ensuing 18–30 wk. An explanation for the apparent lack of reorganization in their study comes from the nature of the BOLD signal used in fMRI studies (for a detailed discussion, see the rebuttal in Calford et al. 2005). The BOLD signal reflects cortical inputs, including subthreshold activation, rather than outputs, as represented by spiking activity; and measuring cortical inputs can lead to a false determination of the LPZ boundary. Interestingly, the electrode recordings reported by Smirnakis et al. from the cortex outside their BOLD-defined LPZ yielded a pattern of clustered, highly-overlapping RF positions known to be signature of cortical reorganization in the LPZ, potentially because the recordings were actually from reorganized cortex that the authors mistook for normal cortex. In any case, although we speculate that the network involved in cortical reorganization is implemented in V1, the model does not require that the remapping occurs in V1.

The critical prediction of the model is only that the reorganization occurs in cortical areas where the association field is represented.
In general, any laterally connected network whose connections have been pruned by experience-dependent (Hebbian) learning to extract some stimulus correlation may be able to optimally estimate its characteristic operation when its input has been ablated—given that the network responds by a process of axonal sprouting and synaptogenesis that maintains the initial rules of connectivity (Das and Gilbert 1995). The model presented in this paper suggests a mode of reorganization that has an adaptive value in recovering visual perception, and it might represent a more general role of functional architecture—one that encodes information about the statistical structure of sensory input that is normally used to interpret the sensory environment, but which becomes beneficial in restoring functionality to disrupted cortical regions.

Our model establishes a tentative link between major components of the perceptual fill-in phenomenon and a specific network of laterally connected neurons in V1. Correspondingly, it makes the experimentally verifiable prediction that RFs in the LPZ should shift according to a co-circular AF. If neurons in the LPZ are activated through horizontal connections from the peri-LPZ, the post-lesion LPZ neurons will inherit the orientation preferences of the presynaptic cells that most effectively activate them. Moreover, if the horizontal connections tend to link co-circular or collinear neurons, any LPZ neuron that maintains its original preferred orientation should be activated by collinear cells from the peri-LPZ. Any LPZ neuron that changes its preferred orientation after the lesion should be activated by co-circular cells from the peri-LPZ. Therefore if a particular cell maintains the same preferred orientation before and after the retinal lesion, the cell’s RF should shift along the (linear) axis that runs through the original RF and that is parallel to the preferred orientation. For any cell that changes its preferred orientation after the lesion, its new RF should shift along a circular arc to a position that brings into co-circular alignment with the original RF position and preferred orientation. Testing these predictions promises to yield a better understanding not only of cortical reorganization, but of the precise functional relationships between laterally interconnected cortical neurons in general.

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