Proc. Natl. Acad. Sci. USA
Vol. 84, pp. 6297–6301, September 1987
Neurobiology

Shifter circuits: A computational strategy for dynamic aspects of visual processing

(stereopsis/attention/motion analysis/vision/striate cortex)

C. H. ANDERSON*† AND D. C. VAN ESSEN‡§

*David Sarnoff Research Center, Princeton, NJ 08540; and ‡Division of Biology, 216-76, California Institute of Technology, Pasadena, CA 91125

Communicated by John J. Hopfield, May 11, 1987 (received for review March 17, 1987)

ABSTRACT **We propose a general strategy for dynamic control of information flow between arrays of neurons at different levels of the visual pathway, starting in the lateral geniculate nucleus and the geniculorecipient layers of cortical area V1. This strategy can be used for resolving computational problems arising in the domains of stereopsis, directed visual attention, and the perception of moving images. In each of these situations, some means of dynamically controlling how retinal outputs map onto higher-level targets is desirable—in order to achieve binocular fusion, to allow shifts of the focus of attention, and to prevent blurring of moving images. The proposed solution involves what we term "shifter circuits," which allow for dynamic shifts in the relative alignment of input and output arrays without loss of local spatial relationships. The shifts are produced in increments along a succession of relay stages that are linked by diverging excitatory inputs. The direction of shift is controlled at each stage by inhibitory neurons that selectively suppress appropriate sets of ascending inputs. The shifter hypothesis is consistent with available anatomical and physiological evidence on the organization of the primate visual pathway, and it offers a sensible explanation for a variety of otherwise puzzling facts, such as the plethora of cells in the geniculorecipient layers of V1.**

Information-processing systems, whether biological or electronic, should be designed for efficient and timely routing of the signals used for computation. In a standard digital computer, the routing of information to and from the central processor is inherently dynamic. With each computational cycle, the central processor selects its input data from a vast array of memory addresses and likewise sends the output to any desired address. Are there analogous switching processes that regulate information flow in the nervous system? That is, can the inputs used by a neuron for its computations be dynamically switched, or are they rigidly fixed? The possibility of dynamic switching processes in the visual system has been suggested in relation to several functionally distinct aspects of perception, including directed visual attention, stereopsis, and the compensation for motion blur (1–6).

In this report, we will show how a specific type of information-routing strategy, implemented by what we call a "shifter circuit," may provide a common mechanism underlying each of these seemingly disparate perceptual processes. The shifter circuit offers a general means of linking an array of lower-level neurons [such as retinal ganglion cells (RGCs)] to a higher-level processor in a manner that allows for dynamic shifts in the relative alignment of the two levels without the loss of local spatial relationships. We first consider the case of stereopsis, as it allows for the clearest formulation of both the computational problem and our proposed solution.

The Registration Problem in Stereopsis. In stereopsis, information about binocular disparities of images in the left and right eyes is used to make inferences about the depth of objects in the visual field. This requires having the two eyes properly converged, so that the two retinal images are in register. However, this mechanical alignment process, mediated by vergence eye movements, is imperfect; the misalignment is several minutes of arc under optimal conditions and can be an order of magnitude larger under more realistic conditions where head movements are allowed (7). Nonetheless, images are readily fused binocularly and are perceived to be stable in depth, and relative disparities of only a few seconds of arc are sufficient for stereoscopic depth discrimination (8). Thus, specialized neural mechanisms must exist to provide for binocular fusion, perceptual stability, and stereo discrimination in the face of binocular vergence fluctuations. Poggio and his colleagues (4, 8, 9) have demonstrated that cells in cortical area V1 (striate cortex) of alert monkeys show disparity tuning that is considerably sharper than the measured variability in binocular alignment; they accordingly have suggested that a dynamic neural alignment process occurs early in the primate visual pathway.

An efficient way to compensate for misregistration of the eyes would be to shift the monocular image representations prior to the first stage of binocular integration. An outline of this strategy is illustrated schematically for a one-dimensional situation in Fig. 1. The processing sequence begins with two sets of RGCs, one from each eye. Each RGC layer projects by way of a shifter circuit to an intermediate layer, designated the registration stage, where the inputs are still monocular but have been adjusted for optimal binocular alignment. The shifting process should respect several major constraints. (*i*) Each cell in the registration stage is, at a given moment, excited by only a single RGC. (*ii*) Local ordering of inputs is preserved, so that the inputs from each eye activate a contiguous set of cells at the registration stage. (*iii*) The relative alignment between the two image representations is under the control of a visually driven dynamic shifting process, which in this case has compensated precisely for the misalignment of the illustrated luminance patterns in the two eyes.

Once registration is achieved, the two monocular input arrays project to the binocular integration stage, where disparity-tuned neurons can be generated by way of a stereo algorithm that need not be specified here. Thus, the introduction of a dynamic shifting process does not solve the well-known "correspondence problem" in stereopsis (8), but it offers a major computational simplification by greatly

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. §1734 solely to indicate this fact.

Abbreviations: RGC, retinal ganglion cell; LGN, lateral geniculate nucleus.

†Present address: Jet Propulsion Laboratory, Mailstop 198-330, 4800 Oak Grove Drive, Pasadena, CA 91109.
§To whom reprint requests should be addressed.

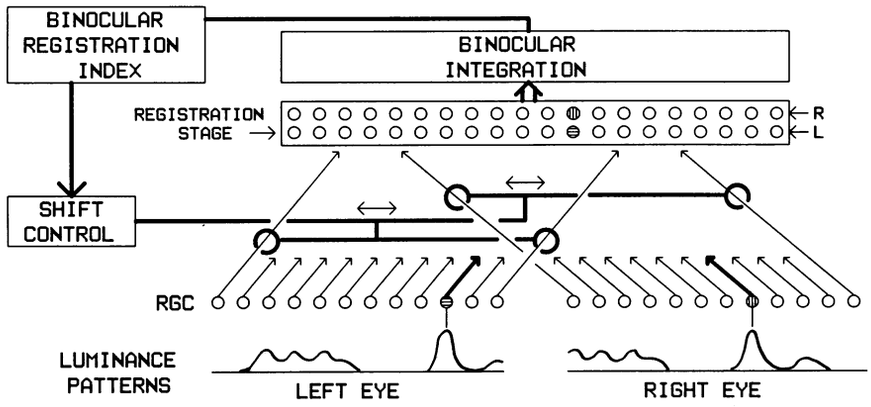

FIG. 1. Schematic diagram of how a shifting process could provide binocular registration at the cortical level (V1) despite misregistration of luminance patterns for the two eyes. Note that the sharp luminance peak, which activates noncorresponding RGCs (hatched circles), maps onto corresponding cells at the registration stage. R, right; L, left.

reducing the range over which false correspondences need to be tested.

A Basic Shifter Circuit. An elementary shifter circuit capable of producing dynamic realignments is illustrated in Fig. 2. For simplicity, it is shown only for one dimension and for a small, monocular input array. At the core of the circuit is a hierarchy of cell layers (four in A; three in B) connected by ascending, symmetrically diverging excitatory connections. Switching of information flow is mediated by a set of laterally directed inhibitory pathways that selectively suppress particular sets of inputs.

In the ascending component of the pathway (Fig. 2*A*) each cell in level 1 connects to two target cells in level 2: one a half-step to the left and the other a half-step to the right. Each cell in level 2 in turn connects to two target cells in level 3, each one a full step to the right or left. Finally, between levels 3 and 4 each cell bifurcates and contacts cells two full steps to the right or left. As a result, any one cell in level 1 has potential access to eight different cells in level 4.

Information flow through each level is regulated by inhibitory switches that selectively suppress one of the two inputs to each cell (Fig. 2*B*). The scheme illustrated here presumes that individual dendrites operate as discrete functional units, with each dendrite receiving excitatory input from a single lower-order cell and an inhibitory connection from a single inhibitory neuron. The inhibitory input is presumed to act as a spatially restricted shunt that can "veto" excitatory inputs arising more distally on the same dendrite (10).

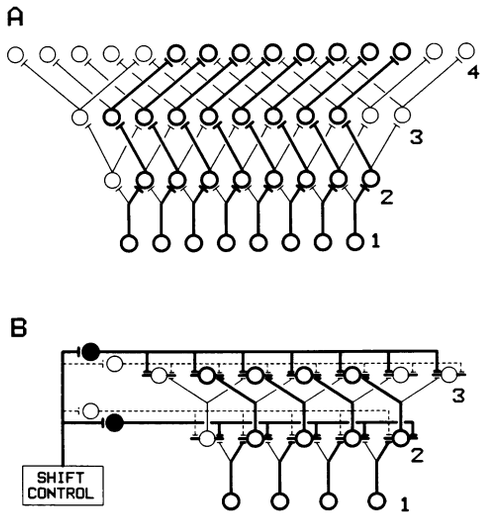

FIG. 2. A simple shifter circuit. (*A*) Ascending components for a four-level circuit with eight cells at the bottom. Cells at each level bifurcate and contact a pair of target cells at the next level. (*B*) A complete shifter circuit for a three-level circuit starting with four cells. Specific dendritic innervation patterns are shown for both ascending inputs and inhibitory neurons involved in shift control. Heavy lines in *A* and *B* represent an activity pattern involving successive shifts to the right, left, and (in *A*) again to the right.

Coordinated shifts occur at each level because of the geometry of the ascending connections and of the inhibitory cell connections. At each level, the left branch of the ascending axon contacts the right dendrite of a higher-order cell, while the right branch contacts the left dendrite of a different higher-order cell. At all levels, the inhibitory neurons follow a simple rule: each one either contacts exclusively right-side dendrites or exclusively left-side dendrites. Thus, depending on which inhibitory neurons are active (thick lines and filled cell bodies) and which are inactive (dashed lines and open cell bodies), information at each stage will be directed to the left or to the right, but it will automatically be kept in register for the whole array. Linkages involving noncontiguous cells are excluded as long as one and only one of the inhibitory neurons is active out of each pair.

Shifter circuits can be readily extended to two dimensions. One straightforward way to accomplish this is to have four connections from one cell to its targets at the next level, which suffices to ensure complete coverage of all possible shifts. The number of stages needed is no different for two dimensions than for one—namely, $\log_2 k$ for a shifting range of k cells.

Descending Control. In the circuit of Fig. 2*B*, the degree of shifting at any given moment is specified by the pattern of activity in the inhibitory neurons at each level. For stereopsis, changes in the direction and magnitude of the shift for each eye could be provided by a visually driven feedback system that involves some measure of the degree of alignment of the two monocular activity patterns over a restricted part of the visual field (represented by the "binocular registration index" in Fig. 1). This could account for psychophysical evidence that the depth at which an object is perceived can be modulated dramatically by the disparity cues provided by nearby features in the visual field (11). The binocular registration index might simply be based on the relative balance of activity among neurons tuned for "near" and "far" disparities (9), with binocular alignment driven in one direction if the activity of near cells exceeded that of far cells and in the opposite direction if the balance of activity were reversed. Alternatively, the binocular registration index might be based upon the pooled activity of "tuned excitatory" cells (9) that respond optimally to objects on or near the

perceptual horopter. In this case, the shifters could operate by keeping the binocular registration index maximized for a given set of visual inputs. Simple versions of either type of control mechanisms should not be difficult to implement. The circuitry needed to attain and maintain binocular registration in realistic operating circumstances is likely to be rather complex, though, since the system must be capable of adjusting rapidly to a dynamic visual environment, must perform smoothly over a range of spatial scales and over the entire visual field, and may also be linked to the motor system for vergence eye movements.

Neuroanatomical Substrates. For reasons of computational simplicity and efficiency, shifting related to stereopsis should take place before binocular convergence and before extensive feature analysis has taken place. This clearly points to the lateral geniculate nucleus (LGN) and layers 4C and 4A of area V1, because cells at these levels are monocularly driven and have small, center-surround receptive fields that reportedly differ little from those of RGCs (12, 13). Fig. 3 illustrates schematically some of the major routes of information flow into and out of these layers as determined from connectivity studies in the macaque monkey (12, 15, 16). There is a basic segregation into magnocellular and parvocellular streams, which originate as separate cell populations at the RGC level and continue as largely independent pathways through the LGN and V1. The magnocellular stream includes the P-alpha class of ganglion cells, the magnocellular layers of the LGN, and layers 4Ca and 4B of V1. Layer 4B is the first stage at which cells are binocularly driven and show a high incidence of selectivity for binocular disparity, orientation, and direction (9, 12, 13). The parvocellular stream includes the P-beta ganglion cells, the parvocellular LGN layers, and four stages in V1: lower 4Cb, upper 4Cb, layer 4A, and finally the supragranular layers (2 and 3), where binocularity and other integrative properties first appear. The larger number of relays associated with the parvocellular stream may be related to the smaller size of receptive fields (17), which would necessitate more stages of shifting to achieve a given absolute displacement.

Layer 6 of V1 provides massive feedback to all of the relay stages in the LGN and in the layer 4 complex (16, 18). At each of these stages there are also numerous γ-aminobutyric acid-containing, presumably inhibitory interneurons (18, 19). Thus, the essential elements needed for assembling a shifter circuit like that in Fig. 2 exist in an appropriate general configuration. The absolute numbers of cells are also more than adequate, as indicated in Fig. 3 by the number of cells at each level, in millions of neurons per hemisphere (14). Indeed, shifter circuits provide a sensible explanation for what has been a puzzling overabundance of cells in layers previously regarded as simple relay stages.

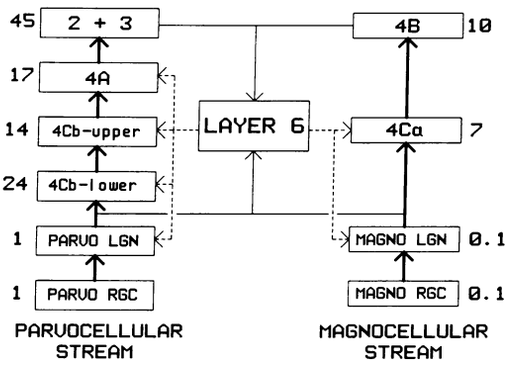

FIG. 3. Connectivity patterns in the parvocellular and magnocellular processing streams of the visual pathway in the macaque monkey. Solid lines indicate major ascending pathways; dashed lines indicate feedback. Not shown are identified ascending connections that skip one or more levels, such as that from the LGN directly to layer 4A. Numbers next to each box represent millions of cells per hemisphere (data from ref. 14). Terminology for their sublayers has been revised to match that of ref. 13.

A biological shifter circuit might have a rather different configuration than the simple binary configuration of excitatory inputs and outputs shown in Fig. 2, while nonetheless conforming to the same basic design principles. Irrespective of the detailed connectivity pattern, the dynamic range over which shifts can occur is constrained by the aggregate horizontal spread of interconnections, summed over successive stages between input and binocular registration stages. To compensate for binocular misalignments of $\pm 0.2°$, the approximate range suggested by eye-movement measurements (4, 7), relative shifts of ± 3 mm across the cortical surface (± 1.5 mm per eye) would be needed in the foveal region, where the cortical magnification factor is 15 mm/degree (20). For the magnocellular system, the extent of individual geniculocortical afferent fibers is about 1 mm, while the intrinsic cortical connections spread about 1 mm in each direction in layer 4Ca and even further (± 4–5 mm) in layer 4B (15, 16). Thus, the magnocellular registration stage might be represented in either layer 4Ca or layer 4B. The parvocellular system is more restricted in its spread within layers 4Cb and 4A (± 0.5 mm), whereas there is a divergence of ± 1 mm in the projections to layer 3 (15, 16). Unless the parvocellular system operates over a narrower dynamic range, its registration stage would apparently be constrained to lie in layer 3 rather than layer 4A.

Motion Compensation. The world is perceived as stable and unblurred despite the fact that images on the retina are in constant motion, even during periods of careful fixation (6, 7). We suggest that shifter circuits may play a key role in preventing motion blur. The proposed strategy is to introduce a compensatory cortical shift whose velocity is equal but opposite to the locally measured retinal velocity field. As illustrated in Fig. 4, this would transform a coherently moving retinal image (shown at discrete times, t_1 and t_2) into a physically stabilized image representation at the level identified as the "stabilization stage," analogous to the registration stage for stereopsis. As with stereopsis, it makes sense to carry out this stabilization prior to detailed form analysis, again implicating the layer 4 complex of V1 as the primary site for neural implementation.

The maximal range of motion-compensating shifts is indicated schematically in Fig. 4 by the dashed lines between the ganglion cells and the stabilization stage. The pattern on the right indicates the number of cells at the stabilization stage accessible to a particular ganglion cell (the divergence range). Equivalently, the convergence range is represented on the left as the number of ganglion cells having access to a

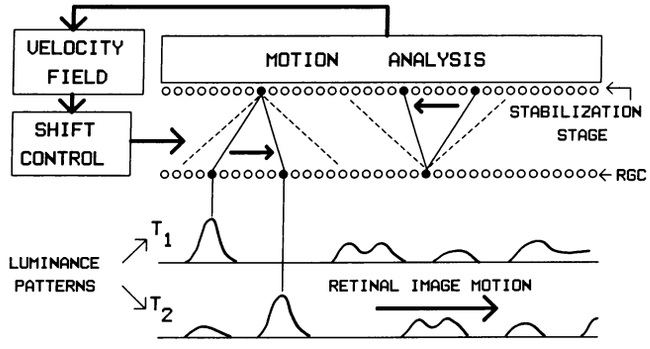

FIG. 4. A strategy for motion compensation. A velocity field measurement feeds into the shift control mechanism, thereby compensating for retinal image motion and producing a stationary activity pattern at the cortical stabilization stage.

particular cortical neuron at the top of the cascade. When the limit of the shifter is reached, the circuit should be quickly reset. Such a sawtooth shifting process would translate smooth, continuous retinal image motion into a succession of stationary cortical images that jump in saltatory fashion across the cortical surface. The trade-off between space and time implied by this scheme could account for spatiotemporal interpolation processes reported psychophysically (21).

Exposure times of at least 100 msec are needed to suppress motion blur (6). This might signify a lower bound on the duration of the "frames" associated with the shifting process. At 3°/sec, the speed at which Vernier hyperacuity begins to decline (22), the shifting process would need to operate over a cortical distance of 4–5 mm per 100 msec in the foveal representation. This is within the range of the horizontal spread of connections already noted for the magnocellular stream.

Scaling and Blurring. At any given eccentricity, we are able to analyze patterns over a wide range of spatial scales. One efficient strategy for obtaining this range is to create a "pyramid" consisting of multiple spatial representations, each of which is coarser and more blurred than its predecessor (23). The shifter circuit of Fig. 2 can be generalized to provide such a multiresolution representation of the visual world. For example, silencing of all inhibitory inputs at any one level would automatically lead to a 2-fold spatial blurring of the information that reaches each successive stage. This could be done in a dynamic fashion (by relaxing the constraint that one and only one inhibitory neuron be active at each level) or in hard-wired fashion (by setting up separate shifter circuits for each spatial scale). The latter scheme would have the attractive feature that information over a range of scales would be simultaneously available for subsequent form, texture, and motion analysis.

Directed Visual Attention. At a given moment we are capable of concentrating our attention on a small portion of the visual field, known as the focus of attention (1, 24). Within this region, intricate details of the visual world can be closely scrutinized, while information streaming in from the rest of the visual world is attended to a lesser degree, if at all. The focus of attention can be shifted rapidly (and without eye movements) to any part of the visual field, and it can also be scaled to different size ranges (24).

What types of neural circuitry could mediate shifts in the focus of attention? Despite our lack of knowledge about the location, number, and internal organization of the high-level centers responsible for attentive scrutiny, the problem can nonetheless be analyzed profitably as a general question of how to route information from any restricted portion of the visual field to an arbitrary high-level center without inordinate loss of detailed spatial relationships. This is similar in several respects to the registration problems already discussed in the stereo and motion domains, suggesting that shifter circuits might again prove useful. However, the attentional system needs the capacity for shifts corresponding to very large physical displacements, with inputs funneled into the highest level from anywhere on the retina but arising from only a small region at a given time. To achieve this goal efficiently, we propose an attentional shifting process that is divided into two major phases, as illustrated schematically and for one dimension in Fig. 5.

In the first phase, the objective is to bring information from the desired attentional focus, whose retinal location is arbitrary, into alignment with a set of discrete blocks, or modules, that are situated at some intermediate processing stage (Fig. 5*A*). This alignment can be achieved by a series of "microshifts," similar to those postulated for stereo and motion (*cf.* Fig. 2), which map the attentional focus onto the nearest cortical module. In the second phase, the objective is to link the appropriate module to a higher center, of which

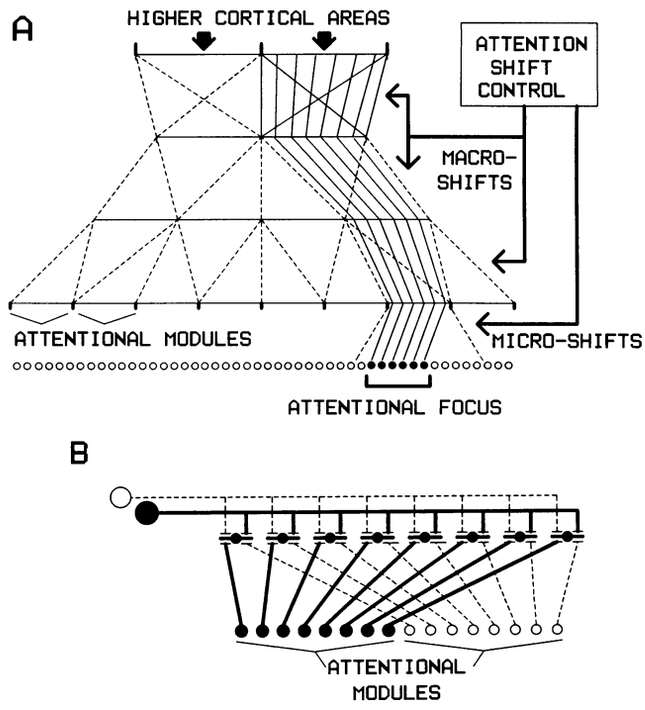

FIG. 5. (*A*) A strategy for shifting the focus of attention. A sequence of microshifts (*cf.* Fig. 2) aligns the attentional focus with the nearest "attentional module"; a series of macroshifts directs inputs from that module to the appropriate higher center. (*B*) Details of the macroshifting circuitry, which allows a higher module to select inputs from either of a pair of lower modules.

there are two shown at the top of the pyramid. This can be achieved by a series of "macroshifts," in which each module at a higher stage dynamically switches between a pair of modules at the next lower stage, using circuitry of the type illustrated in Fig. 5*B*. In this scheme, each cell in the upper module receives an excitatory input onto its left dendrite from the corresponding cell in the left module and an excitatory input onto its right dendrite from the corresponding cell in the right module. As with the microshifts, switching is controlled by a pair of inhibitory neurons, one that shunts all of the left dendrites of cells in the upper module and another that shunts all of the right dendrites in the module. The number of modules at each stage decreases by a factor of 2 (4 for an analogous two-dimensional scheme). This might continue until it reached a single "attention center" at the highest level. Alternatively, there might be divergence near the top, so that there is more than one center at the highest processing level (e.g., two in Fig. 5*A*).

The focus of attention has been estimated to cover 3–10 degrees^2 when centered at parafoveal eccentricities (3–5 degrees) in humans (25). An equivalent size in the macaque monkey would correspond to 7000–15,000 RGCs from each eye, which is roughly 1% of the total ganglion cell population (17). Based on known cortical magnification factors (20, 26), an attentional focus of this size would map onto a region 5–6 mm in diameter for V1, much greater than the dimensions of ocular dominance stripes (12), but onto a slightly smaller region in area V2, comparable to a full cycle of the stripe pattern revealed by cytochrome oxidase histochemistry (27). Thus, the compartmental organization of V2 might conceivably be linked to the proposed modularity of the attention-shifting system.

Physiological Tests for Shifter Circuits. The shifter hypothesis leads to a set of specific predictions about the dynamics of receptive field locations for cells at particular stages of the visual pathway. The most novel proposition is that cells in

layers 4C and 4A of V1, and possibly also in the LGN, should undergo outright receptive-field shifts when driven by stimulus patterns that provide an appropriate reference frame based on stereo, motion, or attentional cues. The range over which shifts occur should increase at successive stages; beyond layer 4C, the shifts should be many times the size of RGC receptive fields.

Previous studies (28, 29) involving precise mapping of V1 receptive fields in anesthetized, paralyzed monkeys indicate that receptive fields are, under some circumstances, stable to within a few minutes of arc. On the other hand, there are also hints that dynamic receptive-field shifts may indeed occur (29, 35). We do not consider the existing evidence to be compelling, either for or against the shifter hypothesis. In particular, we note that anesthesia might seriously disrupt cortical feedback control circuitry, thereby reducing or eliminating any dynamic shifting and possibly also increasing receptive-field sizes, by way of the blurring process described in an earlier section. A more decisive test would be to record simultaneously from cells at different levels of the putative shifter circuit in alert animals, so that a low-level cell could serve as a reference for eye position; the experiment should also use a visual stimulation paradigm that would modulate the shifting process over its full range and in a controlled fashion.

If shifter circuits are indeed present in the early visual pathway, traditional notions of neuronal receptive-field boundaries as static entities must be substantially modified. Receptive fields are typically plotted using a simple stimulus moving on a blank background. In the absence of a well-defined reference frame, the mapping stimulus itself might tend to drive the shifting mechanism and thereby induce the receptive field to move dynamically along with the stimulus. If so, the classical receptive field would be considerably larger than the actual receptive-field size at any given instant (see Fig. 4).

In extrastriate cortex, evidence for dynamic modulation of receptive-field size by attentional cues has been presented for area V4 (30) and for inferotemporal cortex (31) in monkeys. These findings are compatible with several possible mechanisms for selective attention (2, 3) and thus do not on their own constitute strong evidence for the shifter hypothesis. Also, we have not dealt with the full complexity of attentive mechanisms, such as the ability to shift attention along nonspatial dimensions (e.g., for color). Thus, the shifter hypothesis is arguably more speculative and less fully developed for attention than for stereopsis and motion. Nonetheless, it emphasizes that a relatively simple strategy may apply to a variety of dynamic routing problems in vision. Shifter circuits might also be relevant to other systems and processes, such as the rapid adjustment of visual and auditory maps in the primate superior colliculus (32) and the much slower modulation of sensory maps in the somatosensory cortex after peripheral nerve lesions (33). More generally, the use of shunting inhibition to modulate a restricted subset of the connections onto a given neuron offers a powerful strategy that can be exploited for tasks of computation *per se* (10, 34), as well as for the routing strategies discussed in the present report.

We thank Eric Mjolsness and many other colleagues for valuable discussions, ideas, and suggestions. This work was supported in part by contract N00014-85K-0068 from the Office of Naval Research to D.C.V.E.

1. Julesz, B. (1984) in *Dynamic Aspects of Neocortical Function* (Wiley, New York), pp. 585–612.
2. Crick, F. (1984) *Proc. Natl. Acad. Sci. USA* **81,** 4586–4590.
3. Koch, C. & Ullman, S. (1985) *Hum. Neurobiol.* **4,** 219–227.
4. Motter, B. & Poggio, G. (1984) *Exp. Brain Res.* **54,** 304–314.
5. Barlow, H. B. (1981) *Proc. R. Soc. London Ser. B* **212,** 1–36.
6. Burr, D. C. & Ross, J. (1986) *Trends NeuroSci.* **9,** 304–307.
7. Steinman, R. M., Cushman, W. B. & Martins, A. J. (1982) *Hum. Neurobiol.* **1,** 97–109.
8. Poggio, G. & Poggio, T. (1984) *Annu. Rev. Neurosci.* **7,** 379–412.
9. Poggio, G. & Talbot, W. H. (1981) *J. Physiol.* **315,** 469–492.
10. Koch, C., Poggio, T. & Torre, V. (1983) *Proc. Natl. Acad. Sci. USA* **80,** 2799–2802.
11. Mitchison, G. J. & McKee, S. P. (1985) *Nature (London)* **315,** 402–404.
12. Hubel, D. H. & Wiesel, T. N. (1977) *Proc. R. Soc. London Ser. B* **198,** 1–59.
13. Blasdel, G. G. & Fitzpatrick, D. J. (1984) *J. Neurosci.* **4,** 880–895.
14. O'Kusky, J. & Colonnier, M. (1982) *J. Comp. Neurol.* **210,** 278–290.
15. Fitzpatrick, D. S., Lund, J. S. & Blasdel, G. G. (1985) *J. Neurosci.* **5,** 3329–3349.
16. Blasdel, G. G., Lund, J. S. & Fitzpatrick, D. (1985) *J. Neurosci.* **5,** 3350–3369.
17. Perry, N. H. & Cowey, A. (1985) *Vision Res.* **25,** 1795–1810.
18. Sherman, S. M. & Koch, C. (1986) *Exp. Brain Res.* **63,** 1–20.
19. Hendrickson, A. E., Hunt, S. P. & Wu, J.-Y. (1981) *Nature (London)* **292,** 605–607.
20. Van Essen, D. C., Newsome, W. T. & Maunsell, J. H. R. (1984) *Vision Res.* **24,** 429–448.
21. Barlow, H. B. (1979) *Nature (London)* **279,** 189–190.
22. Westheimer, G. & McKee, S. P. (1975) *J. Opt. Soc. Am.* **65,** 847–850.
23. Rosenfeld, A. (1984) *Multiresolution Image Processing and Analysis* (Springer, New York).
24. Bergen, J. R. & Julesz, B. (1983) *Nature (London)* **303,** 696–698.
25. Sagi, D. & Julesz, B. (1985) *Nature (London)* **321,** 693–695.
26. Gattass, R., Gross, C. G. & Sandell, J. H. (1981) *J. Comp. Neurol.* **201,** 519–539.
27. Tootell, R. B. H., Silverman, M. S., DeValois, R. G. & Jacobs, G. H. (1983) *Science* **220,** 737–739.
28. DeValois, R. L., Albrecht, D. G. & Thorell, L. G. (1982) *Vision Res.* **22,** 545–559.
29. Parker, A. & Hawken, M. (1985) *J. Opt. Soc. Am.* **2,** 1101–1114.
30. Moran, J. & Desimone, R. (1985) *Science* **229,** 782–784.
31. Richmond, B. J., Wurtz, R. H. & Sato, T. (1983) *J. Neurophysiol.* **50,** 1415–1432.
32. Jay, M. F. & Sparks, D. L. (1984) *Nature (London)* **309,** 345–347.
33. Kaas, J. H., Merzenich, M. M. & Killackey, H. P. (1983) *Annu. Rev. Neurosci.* **6,** 325–350.
34. Anderson, C. H. & Abrahams, E. (1987) *Proceedings of the First IEEE International Conference on Neural Networks*, in press.
35. Motter, B. C. & Poggio, G. F. (1982) *Soc. Neurosci. Abstr.* **8,** 707.