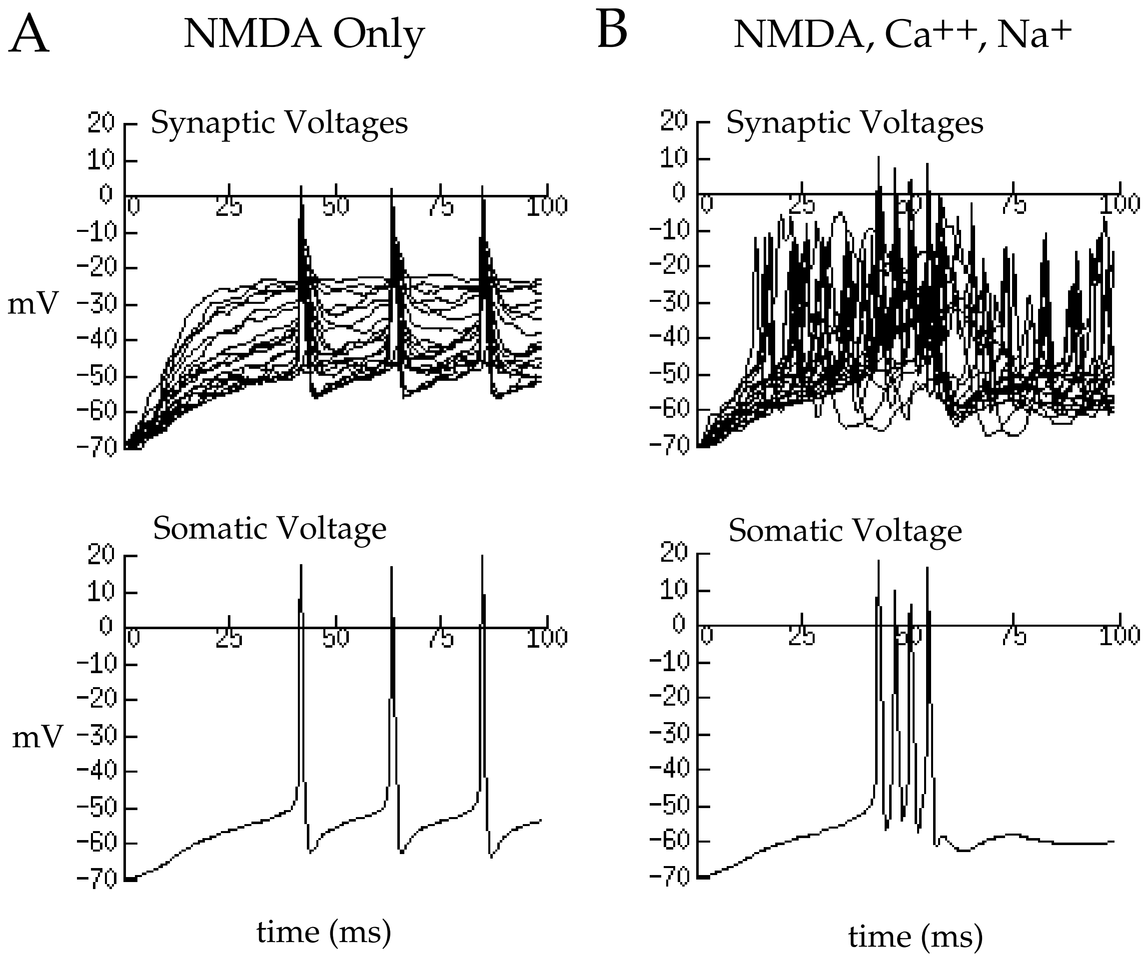


Figure 9: A. Dendritic and somatic voltage traces for clusters of size 5, in high-NMDA condition. Dendritic voltage traces are relatively placid (except when interrupted by somatic spiking). B. In contrast, with addition of slow and fast spiking conductances to the dendritic tree (condition as in fig. 7E), chaotic dendritic spiking ensues that is invisible in somatic voltage traces. In spite of their biophysical differences, both cases show strong cluster sensitivity. Bursting in somatic voltage trace in B is due to presence of slow-spiking channels in basal dendrites.

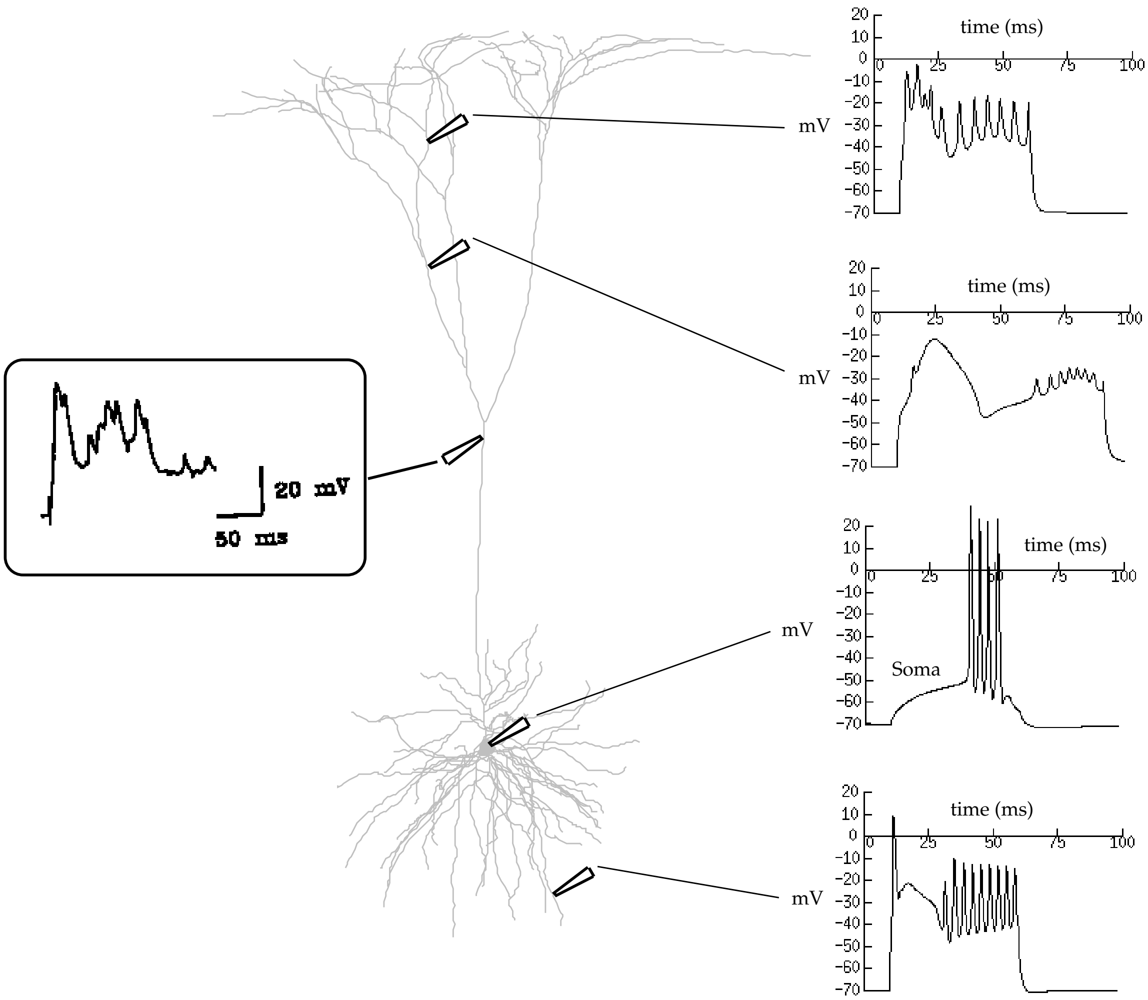
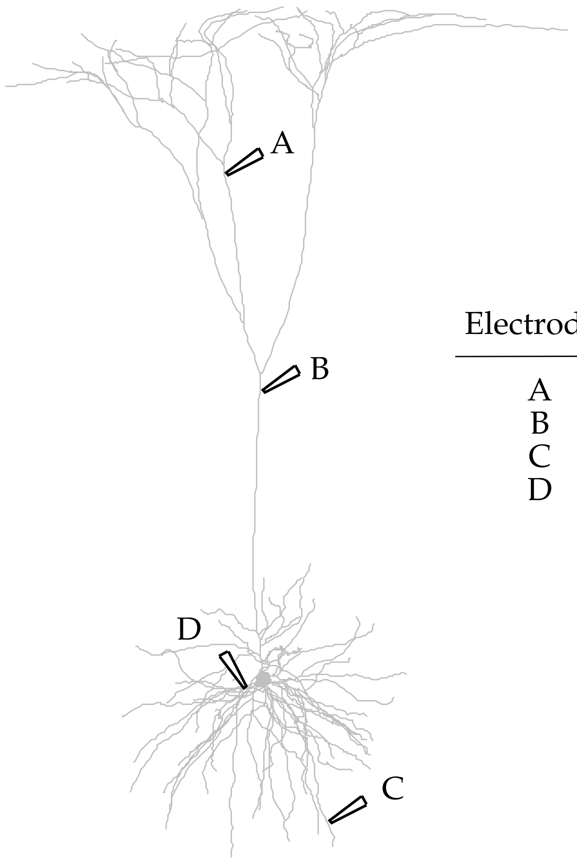


Figure 10: Channel distribution of fig. 7E consisted of 1) high-NMDA synapses, 2) slow-spiking channels in the distal configuration, and 3) fast-spiking channels in the branch-point configuration. When simulated current was injected intradendritically at four sites (in separate experiments), resulting traces showed burst of fast spikes at cell body, and complex superpositions of fast and slow spikes at three dendritic loci. Inset: real intradendritic recording trace from apical dendrite of a layer 3 neocortical pyramids cell (from Amitai et al. 1993).



Electrode	Distance from Soma (um)	Peak Voltage Attenuation		
		Fast Spike	Slow Spike	Steady State
A	600	360	83	27
B	370	30	6	3
C	200	220	55	22
D	15	10	3.2	1.1

Figure 11: Table of attenuation of fast spikes, slow spikes, and steady-state inputs for four dendritic loci. Dendritic locus was stimulated; attenuation shown was ratio of peak local voltage response to peak somatic voltage response. In spiking cases, a 40 μm patch of fast or slow spiking membrane was inserted at the point of stimulation in order to generate a local action potential. For both fast and slow spikes, peak conductances were set as follows: $\overline{g}_{\mathrm{Na}} = 0.2\mathrm{S/cm}^2$ and $\overline{g}_{\mathrm{K}} = 0.1\mathrm{S/cm}^2$. When these same tests were rerun with membrane parameters $R_m = 21,000\Omega\mathrm{cm}^2$, $C_m = 1.0\mu F/\mathrm{cm}^2$, $R_a = 306\Omega\mathrm{cm}$, taken from Stratford *et al.* 1989, the attenuation values were somewhat increased for fast spikes, and somewhat reduced for steady state inputs.

In the basal tree, the situation is quite different. The cell body acts as a large current sink, isolating voltage fluctuations in one basal dendrite from all of the others. An individual basal dendrite can therefore spike freely without igniting the other basal dendrites. This is made possible by the strong attenuation of fast dendritic voltage peaks when propagated to the soma and beyond (fig. 11). Only when a dendritic spike happens to push the soma over spiking threshold can it have a more global effect within the basal arbor. In general, the stellate structure of the basal dendritic tree is electrotonically better suited than the branched apical tree to permit numerous simultaneous but independent local processing operations, such as that which underlies cluster-sensitivity.

Visibility of Dendritic Spikes at Soma

In a related observation, distal dendritic spiking is likely to be essentially invisible in somatic recordings in cells of this or similar construction. The smooth somatic voltage trace in fig. 9B, interrupted only by somatic action potentials, supports this conclusion. Additional control experiments were carried out to confirm that individual dendritic spikes are in fact strongly filtered in somatic recordings due to their large high-frequency components. Figure 11 shows the peak voltage attenuation at the soma for inputs to four dendritic loci. Each dendritic locus consisted of a 40 μm patch of membrane that was either entirely passive, or contained either a fast or a slow spiking conductance complex. Both spiking complexes had modified Hodgkin-Huxley kinetics; the fast complex generated spikes approximately 1 ms in width at half height, the slow complex generated spikes approximately 6 ms in width. Both fast and slow spikes were roughly 100 mV in height locally. The rest of the cell was passive, including the soma. A small current was injected into each dendritic compartment and the local peak voltage response was compared to the peak somatic voltage response.

The table in fig. 11 shows the voltage attenuation for fast spikes, slow spikes, and steady state current injections for each of the four dendritic loci. For fast spikes, the peak voltage attenuation was never less than a factor of 10, which occured when the spiking membrane patch was only 15 μm from the cell body (A). For a spiking patch at the top of the main apical trunk (B), the attenuation factor for fast spikes was 30, giving a peak somatic depolarization of only 3 mV in height. A fast spike on the tip of a basal dendrite (C), 200 μm from the cell body, is attenuated by a factor of 220, giving a peak somatic depolarization of only 0.5 mV. Attenutation of spike height is still greater for a more distal apical locus (D), where a 360 fold attentuation leads to somatic deflections of only 0.25 mV. Slow-spike and steady-state attenuation were much less for all four loci; steady state voltage attenuation values varied from 1.1 for locus A to 27 for locus D.

These results suggest that an absence of clear, miniature action potentials in somatic voltage traces should not be taken as evidence for dendritic passivity. Rather, superpositions of fast and slow spikes at multiple dendritic sites are compatible with relatively smooth voltage traces at the cell body. This is consistent with the failure to see complex spikes of the kind seen intradendritically when recording from the cell body (Amitai *et al.* 1993).

Bursting

For some distributions of spiking conductances in the dendritic tree, it was observed that somatic spikes occured exclusively in bursts (figs. 9). This effect was found to be due to the presence of a slow-spiking membrane in the basal dendrites in both the "distal" and "branch point" conditions. Under either of these conditions, whenever the soma reached firing threshold and generated an action potential, all of the basal slow-spiking conductances were quasi-synchronously activated. The 10-20 ms cycle time of these slow spiking conductances, open *en masse* across the basal arbor, contributed a significant flow of current into the soma which stimuluated a train of action potentials there. As the basal dendrites became hyperpolarized by their outward current component, the spike train at the cell body was terminated. Similar origins of bursting behavior has been previously noted (Traub & Llinas 1979; Traub 1982; Rhodes & Gray 1992). The presence or absence of bursting behavior in layer 5 pyramidal cells (Connors & Gutnick 1990) could thus potentially be explained by the presence or absence of slow voltage-dependent depolarizing conductances in the basal dendrites and/or main apical trunk.

Predictions

On the basis of the simulation experiments presented in this paper, it seems a merited prediction that the dendrites of neocortical pyramidal cells are selectively responsive to spatially inhomogeneous patterns of synaptic activation. This prediction assumes only a strong action of excitatory voltage-dependent membrane mechanisms in their dendrites, for which there is good evidence (e.g. Fox *et al.* 1990; Miller *et al.* 1989; Amitai *et al.* 1993). Given the relative insensitivity of the simulation results presented here (and in Mel 1992a) to manipulation of biophysical membrane parameters, synaptic waveforms, type, distribution, or kinetics of excitatory synaptic and membrane conductances, number of active synapses, frequency of synaptic activation, or details of dendritic morphology, it seems unlikely that the phenomenon is an artifact of some aspect of the compartmental modeling approach. In fact, the validity of the results would seem to hold essentially as well for other types of vertebrate CNS neurons, including hippocampal pyramidal cells, and cerebellar purkinje cells.

It is nonetheless useful to enumerate five conditions that could invalidate the prediction of cluster-sensitivity in neocortical dendrites. These are: 1) passive dendrites, or at minimum a general paucity of excitatory voltage-dependent inward currents in comparison to voltage-dependent outward currents in a dendritic tree, 2) extremely high-resistance spine necks that prevent effective voltage communication among neighboring synapses, 3) peak synaptic conductance values of such a magnitude that any single postsynaptic event saturates the synaptic driving force, making cooperativity among neighboring synapses irrelevant, 4) a radically different cell morphology, such as a long, single unbranched dendrite, or a cell so small as to be essentially isopotential, or 5) malign network effects, such as a highly disruptive role for local circuit inhibition. Two additional conditions could render dendritic cluster-sensitivity functionally irrelevant. These are, 1) a very high average level of synaptic activation of pyramidal cells, such that cell outputs are saturated and hence insensitive to modest changes in the effectiveness of the synaptic stimulus pattern, and 2) a high degree of variability in the average level of synaptic activation of pyramidal cells, such that changes in cell response due to differences in the spatial configuration of active synapses are occluded.

Direct experimental verification of dendritic cluster-sensitivity may require the ability to image voltage and/or calcium concentration over a large portion of a dendritic tree during normal synaptic activation of the cell. The standard experimental method for "orthodromic" stimulation, i.e. via a shock to an afferent synaptic pathway, is inappropriate in this case since it affords no control over the fine spatial distribution of activated synapses on a post-synaptic dendrite. Even when laminar specificity of orthodromic stimulation is achievable, it would remain inadequate since afferents projecting to the same dendritic lamina may actually make synaptic contacts on electrotonically remote regions of a dendritic arbor. Confocal microscopy with intracellular voltage-sensitive dyes in vivo, though beyond the current state of the art, seems the most likely experimental venue for direct and full verification of this hypothesis, or any other like it.

Functional Significance of Dendritic Cluster Sensitivity

The functional significance of dendritic cluster sensitivity was first demonstrated in Mel (1992a), where it was shown that the nonlinear input-ouput behavior of an NMDA-rich dendritic tree could provide a capacity for nonlinear pattern discrimination. In a subsequent study, it has been estimated that a 5 x 5 mm slab of neocortex has the capacity to represent on the order of 100,000 input-output pattern associations with high accuracy (Mel, 1993 in preparation). We consider here a related computation provided directly by the biophysics of cluster-sensitive dendrites: a *correlation* operation. It is shown that nonlinear *disparity selectivity* in a binocular visual cell, for example, could arise purely through an intradendritic correlation of inputs from left and right-eye receptive fields.

The underlying idea is that groups of frequently coactivated afferent axons represent significant higher-order "features" in an input stream. These features may be encoded as groups of neighboring synaptic contacts onto a cluster-sensitive dendritic tree (see Brown et al. 1991 for discussion of related ideas). In the case of stereopsis, a significant higher-order feature is any pair of afferents from corresponding locations in the left and right-eye receptive fields, which have a high probability of being coactivated during normal visual behavior. A zero-disparity visual stimulus would thus, 1) contain many of these higher-order "correspondence" features, 2) activate many clustered pairs of synapses in an appropriately configured dendritic tree, and hence, 3) produce a much stronger cell response than a non-corresponding stereo stimulus. This idea is illustrated schematically in figure 12. The global response of such a cell is nonlinear since the response to a matched stereo pair is greater than the response to an unmatched stereo pair of the same stimulus intensity. Such a preference for stimuli at fixed disparity across the receptive field is a characteristic of many binocularly drivable complex cells in the primate visual system (Ohzawa *et al.* 1990).

To test this idea in practice, a developmentally-inspired learning procedure called "clusteron learning" (Mel 1992b) was used here to sort the

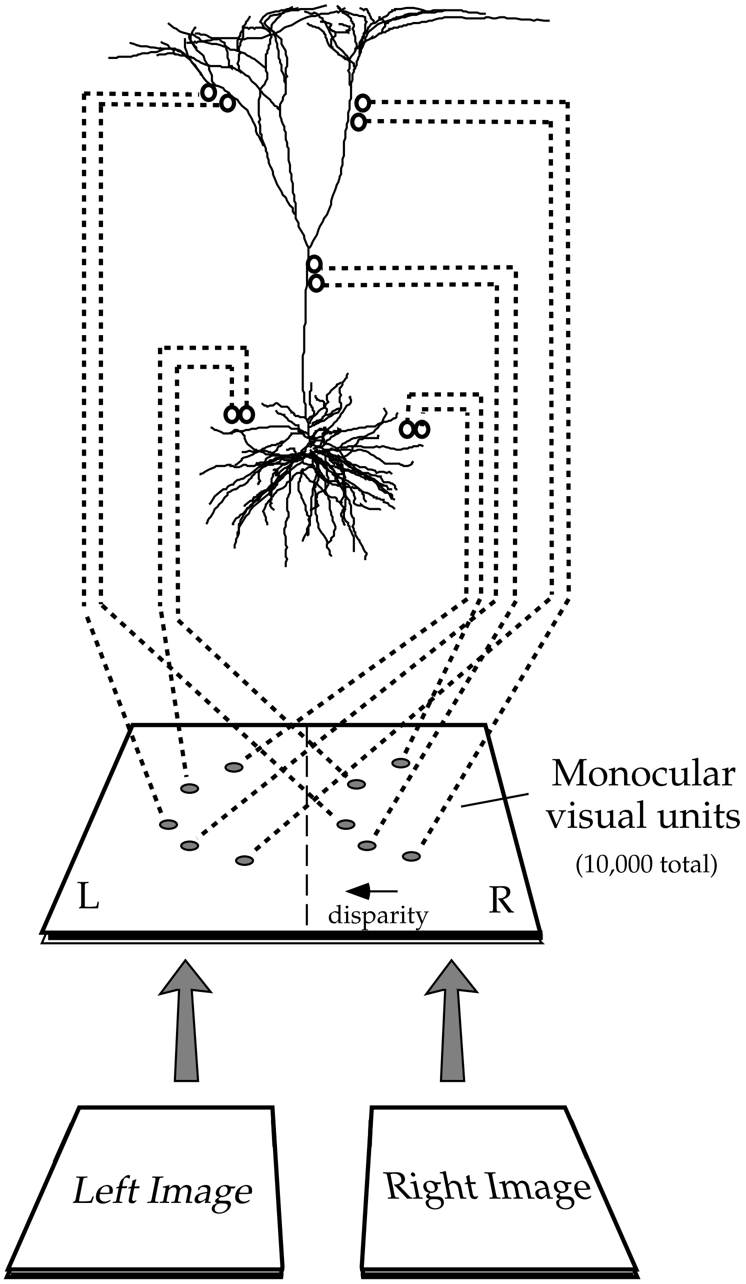


Figure 12: Implementation of "tuned excitatory" disparity-selective binocular cell in a schematized visual cortex. Visual input to left and right eyes drives a layer of left and right monocular units. Axons from corresponding left and right units, which are strongly correlated during normal vision, are assumed to make synaptic connections onto neighboring patches of the binocular dendritic tree. Zero-disparity binocular stimuli thus tend to activate synapses in pairs, a relatively effective (i.e. clustered) stimulus condition. Non-matching inputs to the left and right receptive fields activate synapses diffusely, and are thus relatively ineffective stimuli.

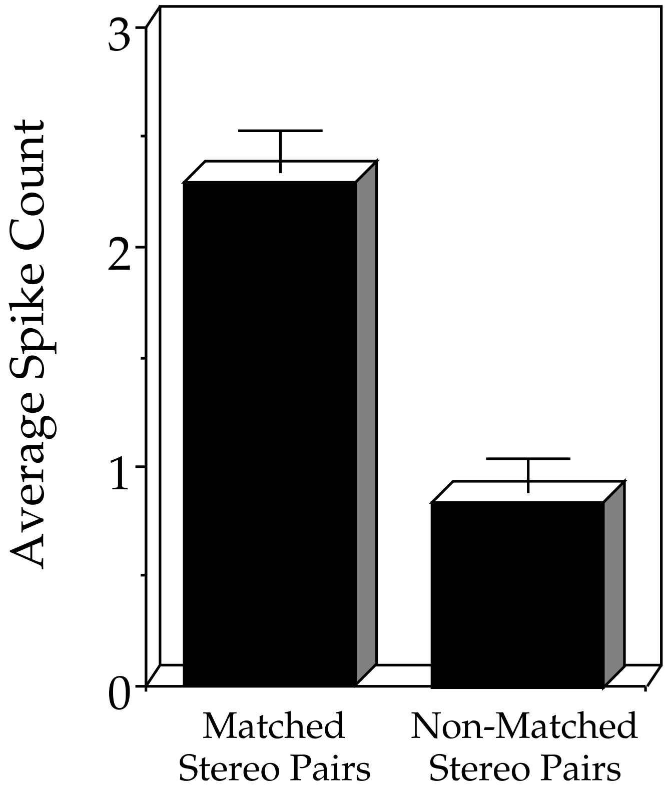


Figure 13: Each pair of stereo test stimuli, whether 1) matched zero-disparity or 2) non-matched, activated a total of exactly 100 synapses on a modeled dendritic tree configured biophysically as in fig. 7E. Test patterns could thus differ only in the spatial distribution of the 100 activated synapses. Average cell responses (in spikes/100 ms) were approximately three times greater for stimulus pairs at zero disparity than for other image pairs.

afferent fiber projection onto our modeled pyramidal cell; after learning, corresponding inputs from simulated left and right-eye receptive fields gave rise to neighboring synaptic connections with high probability. As suggested by the schematized "visual system" configuration of fig. 12, zero-disparity stereo pairs gave rise to relatively clustered patterns of synaptic activation within the dendritic tree. To assess the degree of nonlinear response selectivity possible under these circumstances, test stimuli were delivered to the modeled pyramidal cell that consisted of either 1) randomly generated matched stereo pairs consisting of the same stimulus to left and right eye receptive fields, or 2) randomly generated non-matched stereo pairs. In all test cases, exactly 100 synapses were activated, 50 from each receptive field. The dendritic tree was biophysically configured as for the run of fig. 7E. Average cell responses are shown in fig. 13. Zero-disparity pairs generated responses about three times greater than those due to non-corresponding stimulus pairs. Since the average *amount* of afferent drive to the cell was equivalent for both categories of test stimuli, the cell's strong preference for zero-disparity pairs was necessarily based solely on the spatial *ordering* of afferent synaptic contacts.

This demonstration must be interpreted with caution, as it concerns the input-output behavior of a single model pyramidal cell isolated from the cortical network in which it normally functions. Nor can the schematic of fig. 12 be presently defended as a realistic model for the anatomical substrate of disparity-selective binocular cell responses. The demonstration is nonetheless of interest in that the ability of the cluster-sensitive neuron to represent the second-order nonlinearity associated with binocular disparity selectivity—entirely internal to its dendritic tree—is highly suggestive of a *possible* neurobiological basis for this type of nonlinear cell response. It is interesting to note that the sum-of-products type of computation evidenced here has been proposed in various forms as a crucial nonlinear operation in other types of visual cell responses, including responses to illusory contours (Peterhans & von der Heydt 1989), responses to periodic gratings (von der Heydt et al. 1991), and velocity-tuned cell responses (Nowlan & Sejnowski 1992). The intriguing possibility of low-order polynomial computation within the dendrites of neocortical pyramidal cells may thus be worthy of further consideration.

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