Biol. Cybernetics 21, 145–159 (1976)
© by Springer-Verlag 1976

On the Development of Feature Detectors in the Visual Cortex with Applications to Learning and Reaction-Diffusion Systems*

Stephen Grossberg
Department of Mathematics, Boston University, Boston, Mass., USA

Received: June 11, 1975

Abstract

Developmental mechanisms for tuning of visual cortex are derived from adult learning mechanisms: an adaptational property of shunting on-center off-surround networks that prevents saturation of parallel processed patterns at high input intensities, a contrast enhancement and short-term memory mechanism, and plastic synaptic strengths that compute a time average of presynaptic signals and postsynaptic activities and multiplicatively gate signals. The mechanisms can generate fields of feature detectors; e.g., line or picture detectors. A developing hierarchy of such fields can be synthesized in which successive critical developmental periods are triggered as a dynamic equilibrium is established between short-term memory and long-term memory at each stage. Shunting adaptation can account for some data on spatial frequency adaptation. Shunting network properties resemble properties of certain reaction-diffusion systems that have been used to model developmental data in various species; e.g., Hydra, *Xenopus* retina, slime molds. For example, positional information due to regulation in reaction-diffusion systems is analogous to constancies due to network adaptation, firing of a developmental gradient is analogous to contrast enhancement, and maintenance of a pattern of morphogens is analogous to short-term memory.

1. Introduction

Two recent theoretical papers discuss the development of specificity in primate visual cortex (Von der Malsburg, 1973; Pérez, Glass, and Shlaer, 1974). Both papers use analogous mechanisms to derive their results. One of these mechanisms (plasticity of cross-correlational synapses) has been mathematically proved capable of learning arbitrary acts in a wide variety of circumstances (Grossberg, 1967, 1970a, 1971a, b, 1972a–d, 1974). A second mechanism (on-center off-surround anatomy) is believed to exist in the adult sensory processing areas, such as retina (Kuffler, 1953; Werblin, 1971) and neocortex (Eccles, 1965; Phillips, 1959; Stefanis, 1969), of many species. A third mechanism (attenuation of small signals in a recurrent network) has recently been shown necessary to prevent amplification of noise in networks capable of short-term memory (Ellias and Grossberg, 1975; Grossberg, 1973; Grossberg and Levine, 1975). A fourth mechanism (conservation of total synaptic strength at each cell) can be replaced by an adaptational property of on-center off-surround networks undergoing shunting interactions (Grossberg, 1973). This paper therefore argues that the formal mechanisms which have been suggested for the *development* of specificity are also mechanisms that are needed for efficient *learning* in the mature organism. This theme has become increasingly popular since Wiesel and Hubel (1963) demonstrated that abnormal early visual experience can induce abnormal development of afferent connections to the visual cortex; cf., Mark (1974). Our synthesis of formal developmental and learning mechanisms clarifies the results of the Pérez *et al.* and Von der Malsburg computer studies by making available mathematical results that impose similar design constraints embodying natural properties of the adult learning process. It also shows that the conservation of total synaptic strength, which has also been used to model spatial frequency adaptation in the visual cortex (Wilson, 1975), can be generated as a *global* property of network interactions; in other words, there need not exist a conservation mechanism at each cell if the network has an on-center off-surround anatomy undergoing shunting interactions. Indeed, this global property, which make possible the emergence of selectively tuned feature detectors in the developmental models, also makes possible selective attention by these feature detectors to particular cues in models of adult sensory processing (Grossberg, 1975a). Moreover, the conservation law, as stated in the developmental models, is *not* compatible with rudimentary learning postulates, whereas the global property of network interactions is. The developmental mechanisms that select for frequently experienced features in the immature network also

* Supported in part by the Advanced Research Projects Agency (DAHC15-73-C-0320) administered by Computer Corporation America.

allow the activity of populations which represent infrequently experienced features to be totally suppressed, or masked, in the mature network. The latter populations exist because they might be needed occasionally to code the occurrance of infrequent features, but their existence need not interfere, say via noise effects, with the efficient coding of frequently experienced features.

A final observation relates the roles in development played by recurrent on-center off-surround shunting networks and by systems of reaction-diffusion equations in which activators and inhibitors interact (Gierer and Meinhardt, 1972: Meinhardt and Gierer, 1974). A striking formal analogy exists between these systems in which, for example, self-regulation in the reaction-diffusion system corresponds to total activity adaptation in the network, and maintenance of a pattern of morphogens in the reaction-diffusion system corresponds to short-term memory in the network. By comparison with the network, the reaction-diffusion system uses relatively slow and short-range diffusional mechanisms to establish its on-center off-surround interaction. The network mechanism is capable of "long-range order" that can be rapidly activated, and it can supplement slow electrotonic interactions with rapid wave-like regulation of activity patterns across its cells. In this formal sense, the network is a more sophisticated example of evolution than the reaction-diffusion mechanism. This observation supports a two-stage concept of the developmental process in sensory fields: first, the development of a recurrent on-center off-surround network under the guidance of a reaction-diffusion, or formally similar, mechanism that also establishes a map of positional information, as in the case of the amphibian retina (Gaze, 1970; Hunt and Jacobson, 1972, 1973a, b); and second, the development of selective tuning in network cells using the recurrent network interactions for guidance. Apart from tacitly assumed genetic commands and cross-correlational synapses, the only formal concepts that occur in this description are on-center off-surround interactions of one form or another.

2. Review of Developmental Network Models

Both models address the same issue: what mechanisms control the development of visual cortical cells that selectively respond to particular features in the visual environment, such as lines of prescribed orientation? The model of Von der Malsburg will first be reviewed (see Fig. 1). Von der Malsburg arranges his retinal and cortical cells in a hexagonal array. His retina contains 19 cells r_i that are each connected to

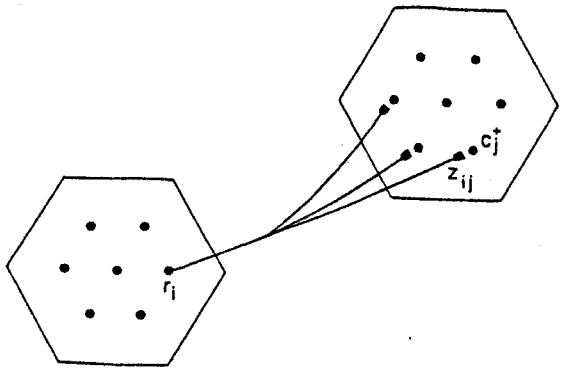

Fig. 1. Cross-correlational synapses from retina to cortex

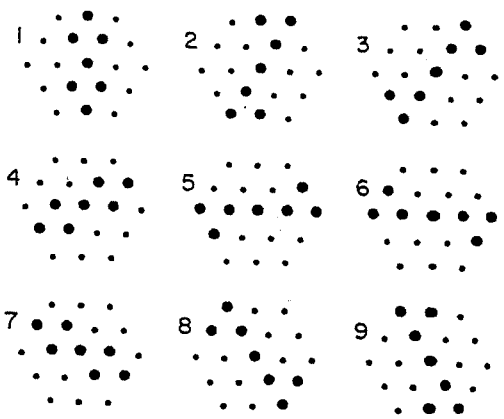

Fig. 2. The standard set of stimuli presented to the retina. Large and small dots represent active and non-active cells respectively (von der Malsburg, 1973)

169 excitatory cortical cells c_j^+ by a pathway with strength z_{ij}. Initially, for each fixed r_i, the strengths z_{ij} are randomly distributed across cortical cells c_j^+. The retina is sequentially presented with the patterns depicted in Fig. 2. If a given pattern excites cell r_i, then r_i emits a signal of unit strength into all of its retinocortical pathways. Otherwise no signal is emitted. Denoting the signal from r_i by $R_i(t)$, the total retinal signal to cell c_j^+ is $\Sigma_i R_i z_{ij}$, as in Grossberg (1971b). Denote the output signal from c_j^+ by $C_j(t)$. Four mechanisms are imposed on this general framework:

I. Learning at Cross-Correlational Synapses

The connection strength z_{ij} grows at a rate proportional to the product $R_i C_j$, as in Grossberg (1971 b).

II. Synaptic Conservation

The total connection strength $\Sigma_i z_{ij}$ at each cell c_j^+ is constant through time. Thus strengthening one connection z_{ij} automatically weakens other connections z_{kj}.

III. On-Center Off-Surround Interaction

The cortex is composed of a hexagonal array of cell pairs: an excitatory cell c_j^+ and an inhibitory cell c_j^- constitute a pair. Each active cell c_j^+ excites the immediately neighboring c_k^+ cells with strength p and the immediately neighboring c_k^- cells with strength r. Each active c_j^- cell inhibits its next-to-immediate c_k^+ neighbors with strength q. This arrangement simulates a recurrent on-center off-surround interaction pattern in which both excitatory and inhibitory connection strengths diminish as a function of distance.

IV. Attenuated Cortical Feedback

The signal $C_j(t)$ is definable as follows. Given any function $f_j(t)$, let

$$f_j^*(t) = \begin{cases} f_j(t) - \Theta_j & \text{if} \quad f_j(t) > \Theta_j \\ 0 & \text{otherwise.} \end{cases}$$

The number Θ_j is the signal threshold of f_j. Let $E_j(I_j)$ be the activity of the j^{th} excitatory (inhibitory) cortical cell. Then $C_j \equiv E_j^*$, where

$$E_j = \sum_{k \neq j} E_k^* p_{kj} - \sum_k I_k^* q_{kj} + \sum_k R_k z_{kj},$$

and the coefficients p_{kj} and q_{kj} equal p and q, respectively, or zero, depending on whether a given pair (c_k^+, c_k^-) of cortical cells excites, inhibits, or does not influence c_j^+.

A learning trial is defined by a single presentation of every input pattern depicted in Fig. 2, and in the prescribed order. After sufficiently many learning trials, Von der Malsburg finds that most of the cells c_j^+ fire preferentially in response to a line of a given orientation, and that nearby cells fire preferentially to lines of similar orientation (see Fig. 3).

The paper by Pérez, Glass, and Shlaer also uses the basic mechanisms (I) and (II). In fact, this paper reports that without (II), their results have not yet been generated. Instead of using a signal threshold, as in (III), they use a sigmoid, or S-shaped, signal function (see Fig. 4). As Fig. 4 illustrates, both signal functions attenuate small values of w and respond approximately linearly to intermediate values of w. Thus both models use positive feedback generated by the total input to c_j^+ to drive the learning at z_{ij}, and this feedback is attenuated at low total input levels.

Instead of using a recurrent on-center off-surround field in the cortex, Pérez *et al.* assume that their retino-cortical inputs are organized in an on-center off-surround fashion. This has a similar effect on the distribution of excitatory and inhibitory signals to the cortex. Given these assumptions, Pérez *et al.* study the

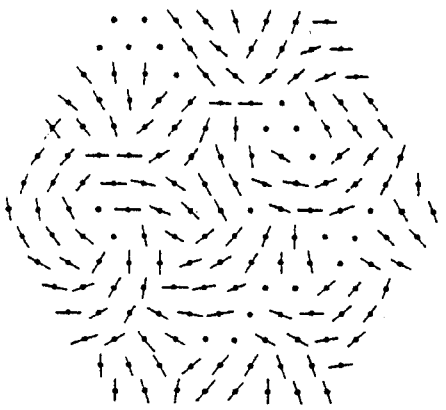

Fig. 3. View onto the cortex after 100 steps of learning. Each bar indicates the optimal orientation of a given c_m^+ cell. Dots without a bar are cells which never reacted to the standard set of stimuli (von der Malsburg, 1973)

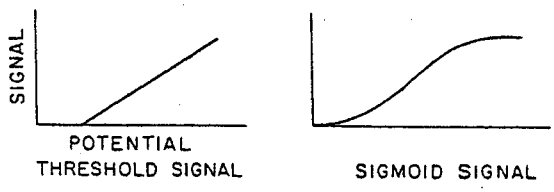

Fig. 4. Threshold and sigmoid signal functions

response of the cortex to various randomized series of straight line inputs to the retina. They also impose various constraints on the distribution of connection strengths z_{ij} at time $t = 0$; e.g., random, highly polarized in a given orientation, or intermediate. In all cases, after sufficiently many learning trials, cortical cells tend to respond most vigorously to lines of a fixed orientation, although the particular orientation that develops can depend on the initial distribution of connection strengths z_{ij}.

In summary, both models use cross-correlational synapses as their learning mechanism, a recurrent feedback within the cortex to drive the postsynaptic part of this mechanism, an on-center off-surround anatomy to regulate the input distribution, and a conservation law in the total synaptic strength. We first show that the conservation law can be replaced by network interactions.

3. Adaptation of Total Activity in Shunting On-Center Off-Surround Networks

A basic constraint on the design of sensory systems can be used to replace the conservation of total synaptic strength at each cell. This constraint describes the adaptation, or normalization, of total activity in a

sensory field. It is illustrated below by showing that the total activity of certain shunting on-center off-surround networks can be bounded above by a fixed constant value, independent of the number of cells in the network or the intensity of inputs to the network. Indeed, the total activity can even converge to a unique positive value as $t \to \infty$. This property is derived below from a thought experiment concerning the ability of certain systems to do parallel processing of input patterns in the presence of noise. Because of the generality of this problem it is not surprising that the resulting mechanism can be used to discuss pattern discrimination (Grossberg, 1970b); color and brightness constancies (Grossberg, 1972c); problems in decision making, short-term memory, and contrast enhancement (Grossberg, 1973); problems in reinforcement theory (Grossberg, 1972b); problems in attention and discrimination learning (Grossberg, 1975a); and problems in constructing position codes for motor control (Grossberg, 1973, 1975a). The normalization property was originally derived from a property of network learning mechanisms (Grossberg, 1970a, 1972d, 1974); namely, a network cell can learn the *relative* intensities of an activity pattern distributed across a given field of cells; i.e., a *spatial pattern* is the unit of long-term memory (LTM). In order to construct cells further downstream in the network that can discriminate during performance trials what has been learned from the field, it is necessary to normalize total field activity, so that the relative intensities can be unambiguously deciphered. In effect, this argument notes the futility of an evolutionary mechanism that produces discriminative cells whose data cannot be encoded in memory, and conversely. We will illustrate the normalization property using the simplest on-center off-surround networks whose interactions are of shunting, or passive membrane, type (Hodgkin, 1964; Sperling, 1970; Sperling and Sondhi, 1968).

The systems are formally derived as follows. Consider n cell populations v_i, $i=1, 2, \ldots, n$, whose responses $x_i(t)$ to inputs $I_i(t)$ are linear, decay linearly to an equilibrium value in the absence of inputs, and have a finite maximum. Let the equilibrium value equal zero, for convenience, and let the maximum value be B. Then

$$\dot{x}_i = -Ax_i + (B - x_i)I_i \tag{1}$$

with $0 \leqq x_i(0) \leqq B$. Let $x_i(t)$ be the average number of active sites, or average potential, of v_i at time t, and let B be the total number of excitable sites in v_i. Then term $(B-x_i)I_i$ in (1) says that inactive sites are activated at a rate jointly proportional to the number of inactive sites $(B-x_i)$ and the excitatory input size. This is a mass action law for exciting the inactive sites.

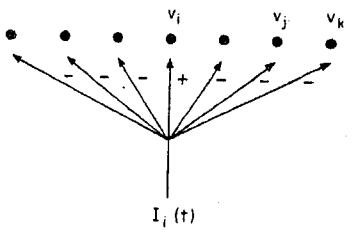

Fig. 5. Nonrecurrent on-center off-surround interactions

Two deficiencies of such a system are apparent. Let the inputs $I_i(t)$ have fixed relative sizes Θ_i; i.e., define $I_i(t) = \Theta_i I(t)$ where $\Theta_i \geqq 0$, $\sum_{k=1}^{n} \Theta_k = 1$, and $I(t)$ is the total input strength. The parameter Θ_i measures the relative importance of the feature coded by v_i in the entire pattern of input features. Can this system preserve a record of how important each feature in the pattern is as the total input $I(t) = I$ is parametrically increased? The answer is "no" because the equilibrium value $\dot{x}_i = 0$ of (1) is

$$x_i = \frac{B\Theta_i I}{A + \Theta_i I},$$

which converges to B as I becomes large. Thus the Θ_i values are obliterated because the responses x_i saturate as I increases. If noise exists in the system, then the Θ_i's are distorted by the noise if I is chosen too small. Both at small and large I values, such a system cannot process the relative sizes of a prescribed input pattern.

The problem of saturation at high background input intensities can be overcome without violating the linearity, equilibrium, or boundedness properties of the equations. Interactions between populations must, however, be introduced, if only because each Θ_i is defined in terms of all the inputs I_k, $k=1, 2, \ldots, n$. An on-center off-surround anatomy suffices, in which the off-surround acts as an automatic gain control of population response. Figure 5 illustrates this anatomy. In Fig. 5, if an input excites v_i, then it also inhibits all v_k, $k \neq i$. (The effects of spatially nonuniform inhibitory fields will be ignored for simplicity.) Equation (1) is changed to

$$\dot{x}_i = -Ax_i + (B - x_i)I_i - x_i \sum_{k \neq i} I_k. \tag{2}$$

The new term $-x_i \sum_{k \neq i} I_k$ says that excited sites become inhibited at a rate jointly proportional to the total number of excited sites and the total inhibitory input size. This is again a mass action law. The equilibrium value $\dot{x}_i = 0$ of (2) is

$$x_i = \Theta_i \frac{BI}{A + I}. \tag{3}$$

No matter how large I becomes, the activity x_i is proportional to Θ_i; no saturation occurs. The off-surround "adapts" the network to fluctuations in total input activity by automatically changing the gain at each population. Note also in (3) that the total equilibrium activity $x = \sum_{k=1}^{n} x_k$ is $BI(A+I)^{-1}$, since $\sum_{k=1}^{n} \Theta_k = 1$. The maximum total activity is B, and is thus independent of the number n of populations and the total input intensity I.

The interpretation of (3) as an adaptational effect is supported by various evidence. In the mudpuppy retina, for example, Werblin (1971) has shown that adaptation occurs at the bipolar cell level due to signals from the lateral inhibitory horizontal layer (off-surround), which is of broad spatial extent. Both cell types receive inputs from peripheral light sources (on-center). Moreover, Werblin and Dowling (1969) showed that the ratio of center-to-surround illumination essentially determines the bipolar cell response – cf., (3) – and that this response can be sustained given a sustained input, which also argues for a shunting rather than a subtractive mechanism (Grossberg, 1970b, p. 321). When such a network mechanism is appropriately attached to color-sensitive cone receptors, various familiar psychophysical properties formally emerge (Grossberg, 1972c; Koenderink *et al.*, 1972).

4. Adaptation Instead of Synaptic Conservation

The synaptic conservation law can be replaced by a retinal (or other sensory field's) adaptational mechanism as follows. The conservation law says that if one retinocortical connection strength z_{ij} is strengthened, then other connection strengths z_{kj} are weakened. Strength z_{ij} will grow, other things equal, if the signal from r_i to the cortex increases. This signal can only increase if the potential at r_i increases. Since the total potential of the retina is bounded, independent of which pattern impinges on it, the potentials of other r_k must decrease, thereby decreasing their retinocortical signals, and their connection strengths z_{kj}.

The synaptic conservation law has deficiencies which are not shared by the adaptational mechanism. In (3), adaptation does not distort the relative importance of signals from different retinal loci to the cortex. Since the synaptic strengths z_{ij} grow at a rate proportional to these signals, no distortion in relative learning rates necessarily follows from an adaptational mechanism. This is not true of the synaptic conservation law. For example, consider a classical conditioning experiment in which a sensory cue S_1 elicits a response pattern R, and cue S_2 is paired with S_1 to learn the pattern R (see Fig. 6).

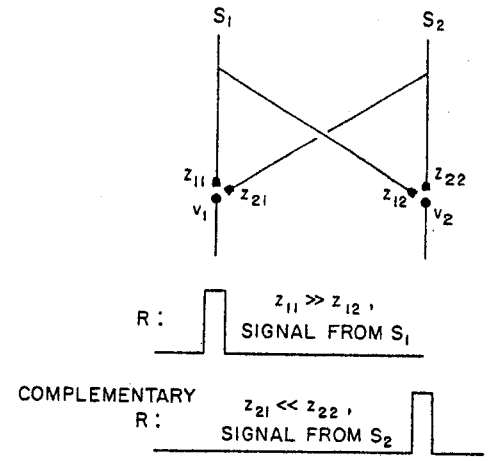

Fig. 6. Conservating of synaptic strength prevents two stimuli from learning the same pattern

Suppose that the pattern R consists of high activation of v_1 and (approximately) zero activation of v_2. This can be caused by a signal from S_1 if z_{11} is much larger than z_{12}. But then, by synaptic conservation, z_{21} is much larger than z_{22}, so that before learning occurs, S_2 controls the *complementary* pattern to R at v_1 and v_2. As S_2 and S_1 are paired, any learning of R by S_2 forces forgetting of R by S_1. Thus, if synaptic conservation were to exist in the mature network as well as the developing network, it would make classical conditioning impossible. It therefore seems that synaptic conservation, in addition to being unnecessary, is also sometimes undesirable. In any case, von der Malsburg (p. 88) introduced the concept primarily to keep the synaptic strengths bounded, but boundedness can be readily achieved without this property (Grossberg, 1970a).

A physical process in which synaptic conservation holds is one wherein a fixed quantity in each cell is continually redistributed among the cell's synapses until saturation occurs. A process in which retinal adaptation occurs allows new synaptic specialization and growth to occur without directly depleting old synapses. A weakening of old synapses can *indirectly* be caused by the adaptation of total retinal output, but this is not a constraint on the learning mechanism *per se*.

5. Contrast Enhancement and Short-Term Memory in Recurrent Networks

In many situations, the anatomy cannot be feedforward, or nonrecurrent, as in Eq. (2). For example, in instrumental conditioning experiments, a mechanism is needed to store internal representations of cues

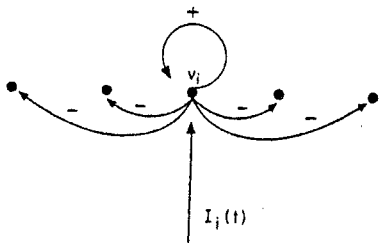

Fig. 7. Recurrent on-center off-surround interactions

in short-term memory (STM) thousands of times longer than passively decaying cells potentials can, until later rewards transfer appropriate indices of the stored representations into long-term memory (LTM). See Grossberg (1971a; 1972a, b; 1975a) for the development of a relevant reinforcement theory. We now summarize results which show that STM storage can occur if signals feed back into the networks. For the reasons cited in Section 3, the network is again chosen to have an on-center off-surround anatomy, but its signals feed back as in Fig. 7. The nonrecurrent system (2) is replaced by the recurrent system

$$\dot{x}_i = -Ax_i + (B - x_i)[f(x_i) + I_i] - x_i[\textstyle\sum_{k \neq i} f(x_k) + J_i]. \quad (4)$$

The function $f(w)$ is the average feedback signal produced by an average activity level w, and functions I_i and J_i are excitatory and inhibitory inputs, respectively. Thus term $(B - x_i)f(x_i)$ in (4) describes the rate of self-activation in v_i due to an average activity level x_i in v_i, whereas term $-x_i f(x_k)$ describes inhibition of active v_i sites by a feedback signal from v_k. A fundamental difficulty must be overcome by this system. It must be capable of storing behaviorally important patterns in STM using its feedback signals, but it must be prevented from amplifying noise via these signals. The functions $f(w)$ can be chosen to accomplish this. Grossberg (1973) proves that a linear signal function (Fig. 8a) or a slower-than-linear signal function (Fig. 8b) both amplify noise. A faster-than-linear signal function (Fig. 8c) can suppress noise, thereby approaching the problem of losing pattern weights Θ_i in noise if the input is small. Unfortunately, this signal function suppresses *all* but the maximal population activity level(s). Figure 9a illustrates this "choice making" property of the network. Such dramatic contrast enhancement is not desirable if graded patterns of activity are desired in STM. A faster-than-linear signal function at small activity levels that becomes linear at high levels (Fig. 8d) overcomes this problem. It suppresses all activity, including noise, that falls below a prescribed threshold level; it contrast enhances and stores in STM all

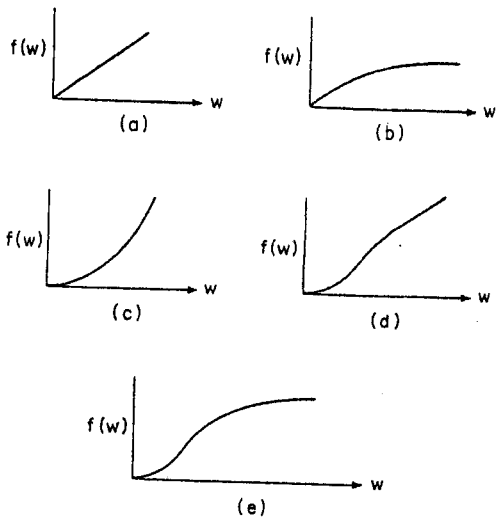

Fig. 8a—e. A classification of some possible signal functions

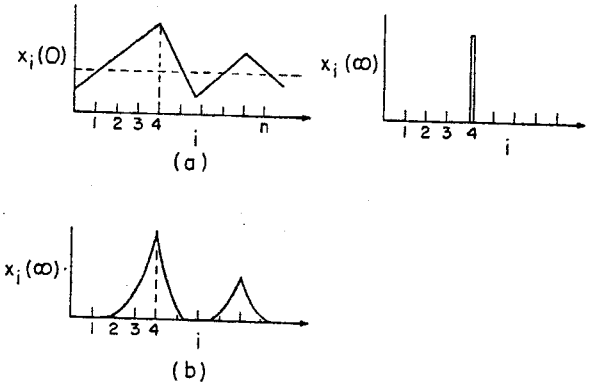

Fig. 9a and b. Contrast enhancing properties of two signal functions

activity levels that fall above the threshold (Fig. 9b). A sigmoid signal function (Fig. 8e) also has this property, given a robust choice of network parameters. Such a signal function has been reported in various experiments (Creutzfeldt *et al.*, 1964; Kernell, 1965a, b; Rall, 1955).

Does the recurrent network adapt the total activity $x = \sum_{k=1}^{n} x_k$ if (say) $f(w)$ is sigmoid? Define $g(w) \equiv w^{-1} f(w)$, and let all inputs I_i and J_i in (4) be shut off to study STM *per se* (consider Fig. 10). If the initial total activity $x(0) < E_1$, then all population activities are suppressed; that is, $x(\infty) = 0$. If $x(0) > E_1$, then $x(t)$ approaches E_2, which is defined by the equation $(B - E_2)g(E_2) = A$; the total activity always approaches E_2 if the network reverberates a pattern in STM. Thus, the recurrent network normalizes total activity in STM. If the inputs in (4) are delivered by an on-center

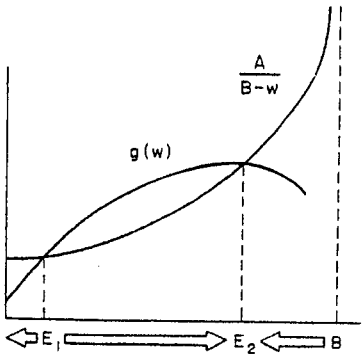

Fig. 10. Normalization of total field activity due to a sigmoid signal function

off-surround anatomy (that is, $J_i = \sum_{k \neq i} I_k$), then again the total activity is bounded by B even if an arbitrarily large input is kept on.

6. Recurrent Feedback in Learning

The recurrent on-center off-surround network can enhance significant inputs and store them in STM until learning mechanisms, such as cross-correlational synapses, can encode them in LTM. Variants of such a mechanism are used by both Pérez *et al.* and Von der Malsburg in their developmental models. Both authors state that the cortical influence on z_{ij} growth is due to a feedback signal driven by the total input to c_j^+, after the signal is attenuated at small activity levels by a sigmoid or threshold cut-off. Section 5 shows that such a signal function is needed to prevent amplification of noise in a recurrent on-center off-surround shunting network. Once again a proposed developmental mechanism is subsumed by a basic constraint on the adult learning process.

The two models for development of specificity can therefore be replaced by a model in which:

1. the retina has an on-center off-surround anatomy that is capable of adaptation;

2. the cortex has a recurrent on-center off-surround anatomy undergoing shunting interactions with a sigmoid-like signal function; and

3. excitatory cross-correlational synapses join the retina to the cortex, perhaps indirectly.

It cannot be too strongly emphasized that properties (1) and (2) are consequences of basic constraints on the parallel processing of patterns in the presence of noise, and are therefore very robust. Moreover, as remarked in Section 3, these constraints are themselves motivated by the fact that the unit of LTM is a spatial pattern. Property (3) follows from an analysis of the simplest properties of classical conditioning (Grossberg, 1967, 1974). Thus all the properties of this developmental model are derivable from postulates about the adult learning process.

7. Developmental Hierarchy and Critical Periods

Another important property of the model is that it can be used to generate a *hierarchy* of selectively tuned networks; compare the visual cortices 17, 18, and 19 in which there exist cells exhibiting ever more selective response criteria (Hubel and Wiesel, 1962). This hierarchical property follows from the fact that the "cortex" in our model also adapts its total activity, as Section 5 describes. Hence the cortex can be used as the "retina" for a second stage in the hierarchy; its outputs will be the inputs to the next "cortical" structure, and its axons will also possess cross-correlational synapses. A central problem for such a model is: how are the critical periods of developmental plasticity in successive anatomical levels regulated so that the coded meaning of outputs from one stage to the next does not change from moment to moment? If the critical period at a given level did not terminate, then massive shifts in the coded meaning of cell activity patterns at this level would destroy the coded meaning of all later stages of population response.

Rudimentary analogs of successive critical periods are already present in the model as it stands. This fact does not deny the possible existence of other critical period mechanisms, but rather illustrates that several mechanisms can operate together to achieve the same end. Understanding why successive critical periods can arise in the model, given a hierarchical anatomy, amounts to understanding the stability of the developmental mechanisms at each level in the hierarchy. Imagine that every level in the hierarchy initially has randomly distributed synaptic strengths. If all synaptic strengths were equal, then no learning could occur, because all cells in a given level of the hierarchy, not including the retina, would always receive the same inputs. The developmental mechanism detects small initial differences in the responses of cortical populations to a given retinal input pattern. If the total retinal input to a particular cortical population is sufficiently large, it can generate positive feedback to its on-center and negative feedback to its off-surround. The recurrent network hereby converts small differences into large differences via its contrast enhancement property. The positive feedback strengthens the retinocortical synapses to this population and the negative feedback weakens the synapses to suppressed

populations. On a later learning trial, the same retinal pattern will generate a larger input to the preferred population because its synaptic strengths are greater than before, and a weaker input to other populations because their synaptic strengths are weaker than before. The recurrent on-center off-surround thereupon enhances this difference once again, thereby strengthening even further the retinocortical synapses that feed the preferred population. Thus the short-term memory reverberation acts to enhance and stabilize the activity pattern that is coded in long-term memory by the synaptic strengths.

Before this process of developmental tuning occurs, there exist at least three reasons why the cortical response to an input pattern can be sluggish despite the existence of viable connections; cf., Hubel and Wiesel (1962). The inputs might be too small to trigger contrast enhancement via the field's recurrent interactions; this can occur either because the initial z_{ij} connections are too small or because the inputs are not supplemented by a source of nonspecific arousal; cf., Grossberg (1973). Third, a given input pattern at the retina will create activity patterns that are distributed rather uniformly across the populations in each cortical region of the hierarchy. Consequently, the off-surrounds of these cortices will not enhance a particular population, but will rather tend to suppress all population responses. A given cortical stage will send large signals to the next cortical stage only after the synapses that feed it have been enhanced and stabilized by its recurrent on-center off-surround interactions. Only after the next stage begins to receive these large signals can it enhance and stabilize the synapses that feed it. Successive stages of critical periods are hereby generated as a dynamic equilibrium is set up between short-term memory and long-term memory at successive stages of the hierarchy.

The requirement that synaptic strengths be initially distributed in a random fashion can be weakened. Figure 1 can be interpreted to depict a small retinal region initially projecting to a small cortical region. Given this interpretation, different retinal regions initially project to their own cortical regions. After learning trials, the cortex will then contain cells that respond to lines of given orientation *in prescribed retinal areas*. In other words, the cortical map "covers" the retina with "vector fields". We will say that the initial retinocortical map is *coarse* when a given retinal area initially connects rather randomly to a given cortical area. Development then tunes the map to yield a covering by vector fields. If the initial retinocortical map was defined in a precise point-to-point fashion, then no such covering could evolve by the mechanism of this paper. Coarsening the initial map and then tuning it, in two stages, is not equivalent to establishing a point-to-point map in one stage. If the initial maps between all stages of the hierarchy are coarse, then ever more complex discriminations can be made by cortical cells as one ascends the hierarchy. For example, a given population in the second cortical stage might fire maximally when it receives converging signals from the first stage that are elicited by a line of fixed orientation moving in a certain direction across the retina. If each of the maps were initially point-to-point, this property of ever more complex coding as the hierarchy is ascended would be lost.

8. Development of Spatial Pattern Discrimination and Pattern Classification

The developmental mechanism can be used to synthesize fields of cortical cells that respond selectively to graded two-dimensional patterns, or pictures, on the retina. In fact, a cortical field with M cells can learn to classify all retinal inputs into M distinguishable classes. The same mechanism can be applied when a given cortical field sends signals to the next cortical field in a hierarchy. A graded two-dimensional pattern on the first cortical field will selectively fire a cell, or cells, in the second cortical field. In this way, cells capable of firing selectively to complex classes, or "gestalts", of retinal patterns can be generated. Below we sketch the main ideas needed to understand how this happens.

Let the initial values of the synaptic strengths from a retina with cells r_i to a cortex with cells c_j^+ be denoted by $z_{ij}(0)$. Let a spatial pattern received by the retina be characterized by inputs $I_i(t)=\Theta_i I(t)$ to each cell r_i, where Θ_i is the fixed relative intensity of the spatial pattern and $I(t)$ is the total pattern intensity at time t; thus $\Theta_i \geqq 0$ and $\Sigma_k \Theta_k = 1$. Because the retina adapts its total activity, we can suppose that the response of r_i after adaptation is Θ_i. (This assumption ignores scaling constants or contrast enhancement effects without loss of the main idea.) Then the total retinocortical signal to c_j^+ at time $t=0$ is $\Sigma_k \Theta_k z_{kj}(0)$. First we study how cortical tuning occurs in response to a single pattern $\Theta=(\Theta_1, \Theta_2, \ldots, \Theta_n)$ presented several times. Then we study what happens when several different patterns are presented to the retina.

Let c_m^+ be the cortical cell that receives the largest total input $\Sigma_k \Theta_k z_{km}(0)$ when pattern Θ first excites the retina. Positive feedback from c_m^+ to itself will therefore be generated, and negative feedback will inhibit other cortical populations via the off-surround of c_m^+. Since

the total cortical activity in STM is normalized, the activity of c_m^+ in STM can be approximated by a constant value whenever c_m^+ receives the largest total retinocortical input on a given learning trial. This fact leads to the main question: if c_m^+ initially receives the largest retinocortical signal in response to pattern Θ, will c_m^+ also receive the largest retinocortical signal on later retinal exposures to Θ?

The answer is "yes" because of the following facts that will immediately be justified. If the total signal $\Sigma_k \Theta_k z_{km}(t)$ to c_m^+ at time $t=0$ exceeds the total input to any other cortical cell at this time, then $\Sigma_k \Theta_k z_{km}(t)$ grows on successive learning trials as other total signals decrease. In fact, learning trials tend to maximize $\Sigma_k \Theta_k z_{km}(t)$ over all possible choices of $z_{km}(t)$.

Why do learning trials cause $\Sigma_k \Theta_k z_{km}(t)$ to increase? Suppose for definiteness that

$$\dot{z}_{im} = -a z_{im} + R_i C_m, \quad a > 0, \tag{5}$$

where R_i is the signal from r_i to c_m^+ and C_m is the feedback signal from c_m^+ to itself. Signal R_i is proportional to Θ_i, and C_m is approximately the same on all learning trials where c_m^+ receives the largest retinocortical signal. Equation (5) therefore implies that each z_{im} *monotonically* approaches a value proportional to Θ_i on each such learning trial. Let z_{im} approach Θ_i to avoid tedious details about scaling. Indeed, the scaling and adaptation assumptions allow us to approximate (5) on learning trials (i.e., when the retina is adapted and cortical STM is active) by

$$\dot{z}_{im} = -z_{im} + \Theta_i.$$

Consequently, on learning trials the vector $z^{(m)} = (z_{1m}, z_{2m}, \ldots, z_{nm})$ obeys the differential equation

$$\frac{d}{dt}(\Theta \cdot z^{(m)}) = -\Theta \cdot z^{(m)} + |\Theta|^2, \tag{6}$$

where $|\Theta|^2 = \Sigma_k \Theta_k^2$ and $\Theta \cdot z^{(m)} = \Sigma_k \Theta_k z_{km}$. Furthermore, letting $|z^{(m)}| = (\Sigma_k z_{km}^2)^{1/2}$ denote the Euclidean length of $z^{(m)}$, and $\varrho_m = (\Theta \cdot z^{(m)})|\Theta|^{-1}|z^{(m)}|^{-1}$ denote the cosine of the angle between the vectors Θ and $z^{(m)}$, it is readily proved that

$$\frac{d}{dt}|z^{(m)}| = |z^{(m)}|^{-1}[\Theta \cdot z^{(m)} - |z^{(m)}|^2] \tag{7}$$

and

$$\frac{d}{dt}\varrho_m = |\Theta|\,|z^{(m)}|^{-1}(1-\varrho_m^2). \tag{8}$$

Consequently, if initially $|\Theta|^2 \geqq \Theta \cdot z^{(m)}(0)$, then by presenting pattern Θ to the retina sufficiently often, the angle between Θ and $z^{(m)}(t)$ monotonically decreases to zero, by (8), while $\Theta \cdot z^{(m)}(t)$ monotonically increases to $|\Theta|^2$, by (6), and $|z^{(m)}|$ approaches $|\Theta|$ with at most one oscillation, by (7). In effect, as time goes on, the learning process maximizes the inner product $\Theta \cdot z^{(m)}$ over all possible choices of $z^{(m)}$ such that $|z^{(m)}| \leqq |\Theta|$. Simultaneously, other cortical responses $C_j(t), j \neq m$, will be suppressed by the off-surround of c_m^+; their coefficients $z_{ij}(t)$ will therefore tend to decrease along with their retinocortical signals $\Sigma_k \Theta_k z_{kj}(t)$.

What happens if several different spatial patterns $\Theta^{(m)} = (\Theta_1^{(m)}, \Theta_2^{(m)}, \ldots, \Theta_n^{(m)})$ can all perturb the retina at various times? How do we prevent changes in the z_{ij}'s due to one pattern from contradicting changes in the z_{ij}'s due to a different pattern? The recurrent on-center off-surround cortical network does this for us; its contrast enhancement, or choice making, property acts as a sampling device that prevents contradictions from occurring. To see how this works, let M patterns $\Theta^{(m)}$, $m = 1, 2, \ldots, M$, be chosen such that $\Theta^{(m)} \cdot z^{(m)}(0)$ is larger than any other retinocortical input $\Theta^{(m)} \cdot z^{(j)}(0)$, $j \neq m$, before learning occurs. Let $\Theta^{(1)}$ be the first pattern to perturb the retina, for definiteness. Cell c_1^+ thereupon receives the largest retinocortical input. Its off-surround then inhibits all c_m^+, $m \neq 1$, so that none of the synaptic strengths $z_{km}(t)$, $m \neq 1$, can learn when $\Theta^{(1)}$ is presented. Learning on this trial makes $z^{(1)}(t)$ more parallel to $\Theta^{(1)}$ as t increases. Consequently, if a different pattern, say $\Theta^{(2)}$, perturbs the retina on the next learning trial, then it will excite c_2^+ more than any other cortical cell: it cannot excite c_1^+ because the coefficients $z^{(1)}(t)$ are more parallel to $\Theta^{(1)}$ than before; and it cannot excite any c_m^+, $m \neq 1, 2$, because the c_m^+ coefficients $z^{(m)}(t)$ have at worst decayed a little without changing their relative sizes. In response to $\Theta^{(2)}$, c_2^+ inhibits all other cortical cells c_m^+, $m \neq 2$. Consequently none of the c_m^+ coefficients $z_{im}(t)$, $m \neq 2$, can learn; learning makes the coefficients $z^{(2)}(t)$ become more parallel to $\Theta^{(2)}$ as t increases. The same occurs on all learning trials. By inhibiting the postsynaptic part of the learning mechanism in all but the preferred cortical population, the on-center off-surround network samples one vector $z^{(m)}(t)$ of trainable coefficients at any time. In this way, the cortex can learn to classify M patterns if it contains M cells.

Further mathematical details concerning the process will be described elsewhere (Grossberg, 1975b). Here it suffices to note that the choice of initial vectors $z^{(m)}(0)$, $m = 1, 2, \ldots, M$, influences what spatial patterns can be classified by the cortex. For example, given any M mutually *nonparallel* vectors $z^{(m)}(0)$, any set of M spatial patterns $\Theta^{(m)}$, $m = 1, 2, \ldots, M$, can be classified by the cortex if $\Theta^{(m)} \cdot z^{(m)}(0) > \Theta^{(m)} \cdot z^{(j)}(0)$ for all $j \neq m$.

In fact, essentially *any* set of spatial patterns can be classified by the cortex in the sense that all spatial patterns in the set will ultimately fire a definite c_m^+. Moreover, most of the spatial patterns which are more parallel initially to $z^{(m)}(0)$ than to any other vector $z^{(j)}(0)$ will all ultimately fire c_m^+, and many will do so more vigorously than they did early in the model's development; in other words, c_m^+ asymptotically codes this class of patterns. For example, suppose that the initial vectors $z^{(m)}(0)$ are few in number. This can happen either because the cortex has few cells, or because many initial vectors are parallel. Using these initial vectors, the cortex can classify a large number of retinal patterns into a small number of easily distinguishable classes. No learning occurs if all $z_{ij}(0)$ are equal because then all initial vectors are identical. If the number of initial nonparallel vectors are many in number and point in many different directions, then the cortex can classify a large number of retinal patterns into a large number of closely related classes. In short, the choice of initial vectors greatly influences the ultimate classification of spatial patterns, and an essentially arbitrary classification is made possible by a proper choice of initial vectors.

Given the importance of initial vector choice to the ultimate discriminations made by a cortex, this choice is presumably not left to chance. Developmental mechanisms operating before the cortical tuning stage presumably determine the statistical rules whereby the values $z_{ij}(0)$ are generated. These rules will then govern which patterns a given cortex will try to classify. Perhaps gradients that establish positional information (Wolpert, 1969) across a retinal or cortical field influence these rules; cf., Sections 11 and 12. This idea extrapolates from data and hypotheses which suggest that positional gradients guide the development of connections between certain pairs of neural fields; for example, the development of point-to-point retinotectal connections in *Xenopus* (Gaze, 1970; Hunt and Jacobson, 1972, 1973a, b; Wolpert, 1969). If this extrapolation is correct, then the developmental control mechanisms that distinguish point-to-point maps from tunable coarse maps would differ only in the precision of their positional codes. An interesting classification problem is suggested by this distinction: to what extent can all developmental maps be classified into point-to-point or tunable coarse maps? Given that a map is tunable, at what stage of development does tuning take place? It is, for example, conceivable that tuning can be driven by endogeneously active patterns rather than external inputs in certain cases.

Other insights about tuning mechanisms now readily suggest themselves. For example, if a given cortical cell c_m^+ does not inhibit all cells c_j^+, $j \neq m$, but rather excites some nearby cells ("distributed on-center"), then a given retinal pattern $\Theta^{(m)}$ can excite a generalization gradient of activity across a collection of cells c_j^+ that code similar patterns; cf., Ellias and Grossberg (1975, Sections 14 and 15). Interactions from cortex to cortex can then organize these generalization gradients into remarkably complex pattern classes ("grandmother cells"?). Grossberg (1975b) analyses this situation in greater detail.

9. Population Size or Tuning Influences Feature Choice: A Two-Stage Model

Various data show that there *is* a critical period, beginning at about 23 days of age and extending up to four months, during which experimental manipulations can alter the functional properties of the cat's visual system. Pérez *et al.* (1974) review some of this data. For example, if patterned stimulation is excluded from one eye during this period, the animal is functionally blind in that eye. Physiologically, most of the cells in area 17 respond only to the eye that did receive patterned inputs, and the binocular connections present at birth are apparently lost (Hubel and Wiesel, 1970; Wiesel and Hubel, 1963, 1965). If the eyes are alternately occluded during maturation, the response properties of the cortical cells are normal, but binocularity is lost (Hubel and Wiesel, 1965). If kittens are raised viewing elongated patterns of one orientation, then the distribution of orientations of receptive fields in cortical cells is strongly biased towards that orientation, and discrimination of patterns at right angles to this orientation suffers (Blakemore and Cooper, 1970; Hirsch and Spinelli, 1970; Mansfield, 1974). After the critical period is over, it seems that even extreme manipulations of the visual environment have no permanent effect (Hubel and Wiesel, 1970; Wiesel and Hubel, 1963, 1965). This lability of early cortical structure permits a fine-tuning of cortical response profiles in a way that is compatible with the organism's early experience. As noted in Section 7, however, if the critical period did not terminate, then massive shifts in the coding properties of the cortex could occur throughout the cat's life. This would destroy all spatially, albeit statistically, coded *hierarchical* properties of learned organization that use the cortex as a source of inputs, since the coded meaning of these inputs would be continually changing.

A main effect of the critical period is to produce a network of interacting populations, in which some populations – such as the one's that receive afferent input in a deprivation experiment performed during the critical period – have greater weight, or different tuning

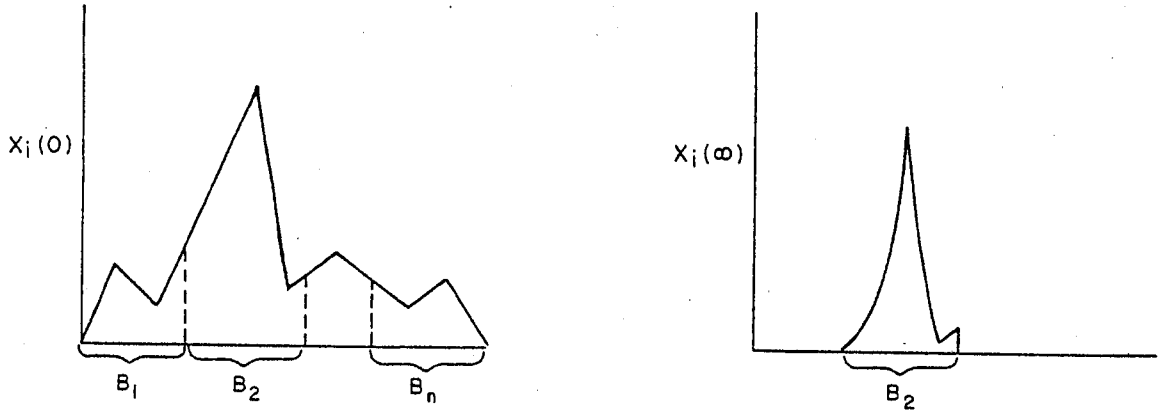

Fig. 11. Suppression in STM of populations with larger numbers B_i of sites by a subfield with large initial activity

curves, than others. A system that captures some of these effects is

$$\dot{x}_i = -Ax_i + (B_i - x_i)[f(x_i) + I_i] - x_i[\textstyle\sum_{k \neq i} f(x_k) + J_i], \quad (8)$$

where B_i is largest in populations that have the largest number of sites coded to respond to the i^{th} feature, or equivalently

$$\dot{x}_i = -Ax_i + (B - x_i)[f(C_i x_i) + I_i] - x_i[\textstyle\sum_{k \neq i} f(C_k x_k) + J_i], \quad (9)$$

where the parameters C_i represent a simple form of nonuniform tuning across populations. Grossberg and Levine (1975) have analysed how these networks work. Below we summarize the main result of this work to show how the on-center off-surround mechanism, which enhances population coding in the model during development, also enhances STM storage in preferentially coded populations during adult performance. For example, if the signal function is chosen as in Fig. 8d, then the features in a given input pattern can trigger a complicated tug-of-war in the network in which three feature properties struggle to determine which features will be stored in STM. Developmentally selected features compete with features that are attended to, or salient. The physical parameters of each feature, such as input energy or density, compete also. The results show how these different parameters struggle until a consensus is established leading to STM storage of those features which have the strongest balance of energetic, attentional, and developmental factors in a given time frame. Speaking mathematically, this tug-of-war occurs between the activities x_i of populations having the largest number of excitable sites B_i and the activities of populations having the largest initial activities $x_j(0)$ due to widespread occurrence of their feature(s) in a given input display. Other things equal, the features with maximal B_i win in STM, and all others are suppressed; but a feature with a larger B_i can be overcome in STM by a feature that has a larger $x_j(0)$ in the display. See Fig. 11, where populations are labelled so that $B_1 \leqq B_2 \leqq \ldots \leqq B_n$.

These results show that certain features can have their activities *totally* masked by more salient features during many experiments, but can be activated by an appropriate display. This mechanism surmounts the following problem: how can infrequently occurring features be represented within the network without increasing the noise in the system due to the mere existence of many infrequently used populations? The on-center off-surround interaction, together with the nonuniformly distributed population weights B_i, or tuning parameters C_i, can totally suppress the activity of these unused populations when a frequently occurring population is excited. Nonetheless, when the infrequent features do occur, they can be stored in STM because of the tug-of-war. Ellias and Grossberg (1975) and Levine and Grossberg (1975) extend these observations to various cases in which the on-center off-surround interaction strengths decrease with distance.

10. Spatial Frequency Adaptation

Synaptic conservation has been used to formally explain other data as well. Here we summarize a related example and indicate how naturally adaptation can replace synaptic conservation.

Wilson (1975) has proposed a neural model to explain various data about spatial frequency adaptation to sine wave gratings, square wave gratings, tilted gratings, and single bars. In his model, signals are feedforward from retina to cortex, and are distributed in an on-center off-surround interaction pattern whose connection strengths decrease monotonically with distance. Wilson uses modifiable synaptic weights as

his mechanism of adaptation. Only the inhibitory synapses of the model are modifiable: their changes are determined by a product of presynaptic signal size and (net excitatory) postsynaptic potential. Thus if the net postsynaptic potential of a given cell is large, then the inhibitory synaptic strengths of active synapses impinging on the cell get stronger, and tend to inhibit the potential more vigorously. This negative feedback mechanism produces good fits to various experiments on adaptation, and is compatible with the idea that excitatory retinocortical synapses, which establish what feature detectors will be coded by the cortex, become stable after the critical period. Wilson is also led to consider a synaptic conservation law: the total inhibitory synaptic strength impinging on each excitatory neuron is constant through time. This mechanism correctly predicts that elevation of perceptual threshold should be greater at higher spatial frequencies of the adapting grating, and it overcomes the otherwise unduly great depression of the modulation transfer function at all frequencies below 3 cycle/degree, given an adapting spatial frequency of 3 cycles/degree. A retinal adaptation mechanism can replace the synaptic conservation law as a formal basis for these cortical adaptational effects.

11. Comparison with Reaction-Diffusion Systems

The remaining sections describe a remarkable similarity in structure and properties between reverberating shunting networks and certain reaction-diffusion systems that have succeeded in modelling developmental phenomena not only in such neural structures as *Xenopus* retina, but also in Hydra, slime molds, etc. (Gierer and Meinhardt, 1972; Meinhardt and Gierer, 1974). The success of a unified class of models at describing development in such diverse organisms suggests that there exist cytoplasmic mechanisms of genetic expression that are commonly shared by many species. Such a universality of developmental control mechanisms is also suggested by the universality of the genetic code (Watson, 1970), and is being actively studied on an intracellular level (Dickson *et al.*, 1975; Holliday and Pugh, 1975). Wolpert (1969) argued for developmental universality by exhibiting formal control principles that seem to be shared by many embryonic structures. The fact that reverberating shunting networks also embody such principles is striking in at least three respects. First, it sharpens the appealing idea that an adult organism is a later stage, or stages, in a continuous process of development that uses the same organizational principles at all stages. Second, it broaches the possibility

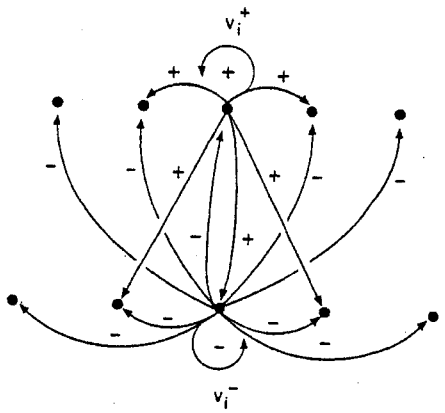

Fig. 12. Unlumped recurrent on-center off-surround network

that shunting networks are used, in one or another form, as a developmental mechanism in the immature organism. And third, the rigorous mathematical results about shunting networks illuminate computer results about reaction-diffusion systems, and suggest particular reactions that have desirable formal properties.

To make this comparison, we note that system (4) is special in several respects. For example, it is *lumped*; that is, the potentials of its inhibitory cells can be expressed in terms of the potentials of its excitatory cells, because the inhibitory cell response to excitatory inputs is rapid. This is not always true. An example of an unlumped system is given by

$$\dot{x}_i = -A_i x_i + (B_i - C_i x_i)\left[\sum_{k=1}^{n} f(x_k) D_{ki} + I_i\right] - x_i\left[\sum_{k=1}^{n} g(y_k) E_{ki} + J_i\right] \tag{10}$$

and

$$\dot{y}_i = -\hat{A}_i y_i + (\hat{B}_i - \hat{C}_i y_i)\left[\sum_{k=1}^{n} \hat{f}(x_k) \hat{D}_{ki} + \hat{I}_i\right] - y_i\left[\sum_{k=1}^{n} \hat{g}(y_k) \hat{E}_{ki} + \hat{J}_i\right]. \tag{11}$$

Here x_i is the average potential of the i^{th} excitatory population v_i^+, and y_i is the average potential of the i^{th} inhibitory population v_i^- (see Fig. 12). In (10) [in (11)] x_k can excite $x_i(y_i)$ if $D_{ki}(\hat{D}_{ki})$ is positive, and y_k can inhibit $x_i(y_i)$ if $E_{ki}(\hat{E}_{ki})$ is positive. The coefficients D_{ki} determine the on-center of v_i^+, and the coefficients E_{ki} determine the off-surround of v_i^-. In addition, the coefficients $\hat{D}_{ki}$ describe the spread of excitatory-to-inhibitory cell signals, and the coefficients $\hat{E}_{ki}$ describe the spread of disinhibitory signals. These coefficients are often chosen to be decreasing functions of the distance between the k^{th} and i^{th} populations, say at a Gaussian rate, as in Fig. 13. The papers by Ellias and Grossberg (1975) and Levine and Grossberg (1975) study various such systems.

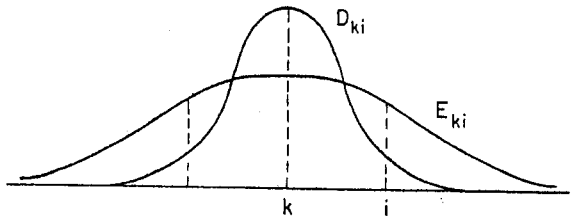

Fig. 13. Gaussian reduction of connection strength as a function of distance

Gierer and Meinhardt (1972) introduce a class of reaction-diffusion systems in which the concentrations of activators $a(x, t)$ and of inhibitors $h(x, t)$ at various positions x control development through time t. Two examples of such systems are defined (in their notation, pp. 33, 34) by

$$\frac{\partial a}{\partial t} = -\mu a + c\varrho a^r h^{-s} + D_a \frac{\partial^2 a}{\partial x^2} + \varrho_0 \varrho\,, \tag{12}$$

$$\frac{\partial h}{\partial t} = -\nu h + c'\varrho' a' h^{-u} + D_h \frac{\partial^2 a}{\partial x^2}\,, \tag{13}$$

and

$$\frac{\partial a}{\partial t} = -\mu a + \frac{c\varrho a^2}{h(1+\kappa a^2)} + D_a \frac{\partial^2 a}{\partial x^2} + \varrho_0 \varrho\,, \tag{14}$$

$$\frac{\partial h}{\partial t} = -\nu h + c'\varrho' a^2 + D_h \frac{\partial^2 h}{\partial x^2}\,. \tag{15}$$

These authors are concerned with conditions under which a slight peak of activator concentration will lead to further increases of $a(x, t)$ at that position. They want these small initial concentration differences to yield large final concentration differences which are thereupon self-maintaining. At a region of peak activator concentration, a new developmental stage is triggered, for example head formation in the Hydra. This proposal is strikingly similar to the network developmental mechanism described in Section 7. There small differences in the pattern of retinocortical signals are converted into large differences by the contrast enhancement mechanism. These large differences are then maintained in STM, and trigger slow developmental changes in retinocortical connection strengths.

A term by term comparison of (12) with (10) and of (13) with (11) is revealing; cf., Table 1. Just as Gierer and Meinhardt (1972) make a distinction between morphogen source density [e.g., $\varrho(x)$] and morphogen concentration, we distinguish input intensity (e.g., I_i) and population activity. They introduce activators $a(x, t)$ and inhibitors $h(x, t)$, whereas we need excitatory activities $x_i(t)$ and inhibitory activities $y_i(t)$. They call the mechanism whereby small differences become large

Table 1

Reaction-diffusion	Shunting reverberating net
activator $a(x, t)$	excitatory activity $x_i(t)$
inhibitor $h(x, t)$	inhibitory activity $y_i(t)$
morphagen source density	inputs
firing of morphagen gradient	contrast enhancement
maintenance of morphagen gradient	short-term memory
power or sigmoid signal functions	power or sigmoid signal functions
on-center off-surround interactions via diffusion	on-center off-surround interactions via electronic propagation or signals
self-stabilizing distributions of morphogens if inhibitors equilibrate rapidly	short-term memory pattern if inhibitors equilibrate rapidly
periodic pulses if inhibitors equilibrate slowly	periodic pulses if inhibitors equilibrate slowly
regulation	adaptation

differences "firing" of a gradient. For systems (12) and (13), they show that firing occurs only if $st(u+1)^{-1} > r-1 > 0$; in particular $r > 1$. A signal function of the form $f(w) = w^r$, $r > 1$, will create contrast enhancement in a shunting network. In both kinds of systems, exponential decay of concentrations or activities occur; e.g., the first terms of the right hand sides of (12) and (13). In both systems, activators (excitatory cells) can excite both themselves and inhibitors, whereas inhibitors can inhibit activators and possibly, but not necessarily [e.g., $u=0$ in (13)] themselves. In both systems, mutual interactions between activators and inhibitors at different locations can occur; in (12)–(13) this occurs via the diffusional terms $D_a \frac{\partial^2 a}{\partial x^2}$ and $D_h \frac{\partial^2 h}{\partial x^2}$, whereas in the networks it occurs via electrotonic or wave-like signals. The diffusional coefficients are chosen to make inhibitor concentration spread within a wider area than activator concentration. This constraint simulates an on-center off-surround field in the reaction-diffusion systems. The diffused inhibitor thereupon inhibits activators at other positions, much as the coefficients $\hat{D}_{ki}$ and E_{ki} in (10) spread inhibitory signals across an expanse of excitatory cells. If inhibitor concentration equilibrates rapidly, say because ν is large, then one finds self-stabilizing distributions of morphogens (Meinhardt and Gierer, 1974). Analogously, a limiting pattern in STM is typically found in lumped shunting networks (Grossberg, 1973; Grossberg and Levine, 1975; Levine and Grossberg, 1975). If inhibitor equilibrates slowly, then periodic pulses of

activation can be obtained, and have been used to discuss aggregating cellular slime moulds and related phenomena (Meinhardt and Gierer, 1974). Analogously, in unlumped networks, periodic pulses of activity can be obtained (Ellias and Grossberg, 1975). The analogy becomes even more suggestive when we realize that the partial *derivatives* in the diffusional terms of a cellular reaction-diffusion system can be replaced by partial *differences* of concentrations in contiguous cells, and this system is formally a network.

12. The Analogy between Regulation and Adaptation

An important property in reaction-diffusion systems is *regulation*. This property supplies "positional information" to cells in the field so that they can create the same developmental pattern independent of total field size, thereby solving the so-called "French Flag" or "Football Field" problem (Gaze, 1970; Wolpert, 1969); for example, keep the region of activation proportional in size to the total size of the developing field (Gierer and Meinhardt, 1972, pp. 32, 34, 35). Gierer and Meinhardt (1972, p. 34) remark that this amounts to a "normalization" of the pattern in subsections of the cellular array. System (14)–(15) has approximately this property. Note in (14) that a sigmoid signal function $f(a)=a^2(1+\kappa a^2)^{-1}$ replaces the power law a^r of (12).

In shunting networks, *regulation* is replaced by *adaptation*, as illustrated by (3). Adaptation also describes a normalization property that makes maximal network response independent of the total number of cells or the input intensity. In STM, the normalization property is stronger because total activity can converge to a unique limit point as $t\to\infty$. The adaptational mechanism preserves a record of the relative magnitude of activation at each position, just as positional information provides individual cells with indices of their relative position in a cellular array. Indeed, the adaptational property can be used to construct a "position code" for guiding the motion of motor systems (e.g., eyes or arms) to fixed target positions even if the target luminance varies widely at these positions (Grossberg, 1973, 1975a).

The analogy between regulation and adaptation becomes particularly suggestive when we consider neural structures in which both kinds of mechanisms might simultaneously be operative. For example, ingenious experiments (e.g., Gaze, 1970; Hunt and Jacobson, 1972, 1973a, b) have been done on amphibia to study how the retina and optic tectum establish positional information that guides the growth of a continuous mapping of the retinal surface onto the tectal surface by means of optic nerve fibers. Suppose as in Section 3 that the adaptational mechanism of such a retina is due to a shunting on-center off-surround network. Then ablation of some retinal cells can renormalize excitatory neuronal activity through adaptation, even as a reaction-diffusion among network cells – supposing with Meinhardt and Gierer (1974) that it exists – could renormalize its activator concentrations through regulation. Such examples naturally lead one to ask whether adaptation plays an organizing role in the adult organism like the one regulation plays in the developing organism, or whether, moreover, the two mechanisms ever act together? The various uses of adaptation cited in Section 3 support this conjecture for the adult organism; the use of adaptation in the model of development of cortical tuning bridges the gap between the adult and the developing organism; and the possible simultaneity of both mechanisms in the infant raises the possibility that network properties regulate as well as adapt.

The above remarks indicate that a dictionary of parallels between shunting network and reaction-diffusion systems can not only suggest and illuminate formal properties of each, but can also ultimately shed more light on how both types of systems contribute separately and together to the developmental process. Networks such as (10) and (11) in which mass action laws hold among the excited and unexcited sites of excitatory and inhibitory processes can, in principle, exist in nonneural structures. As we noted in Section 11, the partial *derivatives* in the diffusional terms of cellular reaction-diffusion system can be represented by partial *differences* of concentrations in contiguous cells, and this system is formally a network. How widespread are such network systems in developing organisms?

References

Blakemore, C., Cooper, G. F.: Development of the brain depends on the visual environment. Nature (Lond.) **228**, 477–478 (1970)

Creutzfeldt, O. D., Lux, H. D., Nacimiento, A. C.: Intracelluläre Reizung corticaler Nervenzellen. Pflügers Arch. ges. Physiol. **281**, 129–151 (1964)

Dickson, R. C., Abelson, J., Barnes, W. M., Reznikoff, W. S.: Genetic regulation: the lac control region. Science **187**, 27–35 (1975)

Eccles, J. C.: In: Kimble, G. A. (Ed.): Anatomy of memory, p. 57. Science and Behavior Books, Inc. 1965

Ellias, S. A., Grossberg, S.: Pattern formation, contrast control, and oscillations in the short-term memory of shunting on-center off-surround networks. Biol. Cybernetics **20**, 69–98 (1975)

Gaze, R. M.: The formation of nerve connections. London-New York: Academic Press 1970

Gierer, A., Meinhardt, H.: A theory of biological pattern formation. Kybernetik **12**, 30—39 (1972)

Grossberg, S.: Nonlinear difference-differential equations in prediction and learning theory. Proc. nat. Acad. Sci. (Wash.) **58**, 1329—1334 (1967)

Grossberg, S.: Some networks that can learn, remember, and reproduce any number of complicated space-time patterns. II. Stud. appl. Math. **49**, 135—166 (1970a)

Grossberg, S.: Neural pattern discrimination. J. theor. Biol. **27**, 291—337 (1970b)

Grossberg, S.: On the dynamics of operant conditioning. J. theor. Biol. **33**, 225—255 (1971a)

Grossberg, S.: Pavlovian pattern learning by nonlinear neural networks. Proc. nat. Acad. Sci. (Wash.) **68**, 828—831 (1971b)

Grossberg, S.: A neural theory of punishment and avoidance. I. Qualitative theory. Math. Biosci. **15**, 39—67 (1972a)

Grossberg, S.: A neural theory of punishment and avoidance. II. Quantitative theory. Math. Biosci. **15**, 253—285 (1972b)

Grossberg, S.: Neural expectation: cerebellar and retinal analogs of cells fired by learnable or unlearned pattern classes. Kybernetik **10**, 49—57 (1972c)

Grossberg, S.: Pattern learning by functional-differential neural networks with arbitrary path weights. In: Schmitt, K. (Ed.): Delay and functional-differential equations and their applications, pp. 121—160. New York: Academic Press 1972d

Grossberg, S.: Contour enhancement, short-term memory, and constancies in reverberating neural networks. Stud. appl. Math. **52**, 213—257 (1973)

Grossberg, S.: Classical and instrumental learning by neural networks. In: Rosen, R., Snell, F. (Eds.): Progress in theoretical biology, pp. 51—141. New York: Academic Press 1974

Grossberg, S.: A neural model of attention, reinforcement, and discrimination learning. In: Pfeiffer, C.C. (Ed.). Int. Rev. Neurobiol. **18** (in press) (1975a)

Grossberg, S.: Adaptive pattern classification and universal recoding: parallel development of neural feature detectors (in preparation) (1975b)

Grossberg, S., Levine, D.S.: Some developmental and attentional biases in the contrast enhancement and short-term memory of recurrent neural networks. J. theor. Biol. (in press) (1975)

Hirsch, H.V.B., Spinelli, D.N.: Visual experience modifies distribution of horizontally and vertically oriented receptive fields in cats. Science **168**, 869—871 (1970)

Hodgkin, A.L.: The conduction of the nervous impulse. Springfield, Ill.: C.C. Thomas 1964

Holliday, R., Pugh, J.E.: DNA modification mechanisms and gene activity during development. Science **187**, 226—232 (1975)

Hubel, D.H., Wiesel, T.N.: Receptive fields, binocular interaction, and functional architecture in the cat's visual cortex. J. Physiol. (Lond.) **160**, 106—154 (1962)

Hubel, D.H., Wiesel, T.N.: Binocular interaction in striate cortex of kittens reared with artificial squint. J. Neurophysiol. **28**, 1041—1059 (1965)

Hubel, D.H., Wiesel, T.N.: The period of susceptibility to the physiological effects of unilateral eye closure in kittens. J. Physiol. (Lond.) **206**, 419—436 (1970)

Hunt, R.K., Jacobson, M.: Development and stability of positional information in *Xenopus* retinal ganglion cells. Proc. nat. Acad. Sci. (Wash.) **69**, 780—783 (1972)

Hunt, R.K., Jacobson, M.: Specification of positional information in retinal ganglion cells of *Xenopus*: assay of the unspecified state. Proc. nat. Acad. Sci. (Wash.) **70**, 507—511 (1973a)

Hunt, R.K., Jacobson, M.: Neuronal locus specificity: altered pattern of spatial development in fused fragments of embryonic *Xenopus* eyes. Science **180**, 509 (1973b)

Kernell, D.: The adaptation and the relation between discharge frequency and current strength of cat lumbosacral motoneurones stimulated by long-lasting injected currents. Acta physiol. scand. **65**, 65—73 (1965a)

Kernell, D.: High-frequency repetitive firing of cat lumbosacral motoneurones stimulated by long-lasting injected currents. Acta physiol. scand. **65**, 74—85 (1965b)

Koenderink, J.J., van de Grind, W.A., Bouman, M.A.: Opponent color coding: a mechanistic model and a new metric for color space. Kybernetik **10**, 78—98 (1972)

Kuffer, S.W.: Discharge patterns and functional organization of the mammalian retina. J. Neurophysiol. **16**, 37—68 (1953)

Levine, D.S., Grossberg, S.: Visual illusions in neural networks: Line neutralization, tilt aftereffect, and angle expansion. Submitted for publication

Mansfield, R.J.W.: Neural basis of orientation perception in primate vision. Science **186**, 1133—1135 (1974)

Mark, R.: Memory and nerve cell connections. New York: Clarendon Oxford University Press 1974

Meinhardt, H., Gierer, A.: Applications of a theory of biological pattern formation based on lateral inhibition. J. Cell Sci. **15**, 321—346 (1974)

Pérez, R., Glass, L., Shlaer, R.: Development of specificity in the cat's visual cortex. J. math. Biol. (in press) (1974)

Phillips, G.C.: Actions of antidromic pyramidal volleys on single betz cells in the cat. Quart. J. exp. Physiol. **44**, 1—25 (1959)

Rall, W.: Experimental monosynaptic input-output relations in the mammalian spinal cord. J. Cell Comp. Physiol. **46**, 413—437 (1955)

Sperling, G.: Model of visual adaptation and contrast detection. Percept. Psychophys. **8**, 143—157 (1970)

Sperling, G., Sondhi, M.M.: Model for visual luminance discrimination and flicker detection. J. Optic. Soc. Amer. **58**, 1133—1145 (1968)

Stefanis, C.: Interneuronal mechanisms in the cortex. In: Brazier, M. (Ed.): The interneuron, pp. 497—526. Los Angeles: University of Calif. Press 1969

Von der Malsburg, C.: Self-organization of orientation sensitive cells in the striate cortex. Kybernetik **14**, 85—100 (1973)

Watson, J.D.: Molecular biology of the gene (2nd ed.). New York: Benjamin 1970

Werblin, F.S.: Adaptation in a vertebrate retina: intracellular recording in *Necturus*. J. Neurophysiol. **34**, 228—241 (1971)

Werblin, F.S., Dowling, J.E.: Organization of the retina of the mudpuppy, *Necturus maculosus*. II. Intracellular recording. J. Neurophysiol. **32**, 339—355 (1969)

Wiesel, T.N., Hubel, D.H.: Single-cell responses in striate cortex of kittens deprived of vision in one eye. J. Neurophysiol. **26**, 1003—1017 (1963)

Wiesel, T.N., Hubel, D.H.: Comparison of the effects of unilateral and bilateral eye closure on cortical unit responses in kittens. J. Neurophysiol. **28**, 1029—1040 (1965)

Wilson, H.: A synaptic model for spatial frequency adaptation. J. theor. Biol. **49** (in press) (1975)

Wolpert, L.: Positional information and the spatial pattern of cellular differentiation. J. theor. Biol. **25**, 1—47 (1969)

Prof. Stephen Grossberg
Boston University
Dept. of Mathematics
Boston, Mass. 02215, USA