CHAPTER 2
PARALLEL PROCESSING OF MOVEMENT AND ERROR SIGNALS

2.1. Sensory-Motor Coordinates: Hemifield Gradients

A neural signal can take on behavioral meaning when it occurs in a network topography that is linked to behavioral consequences. In problems about visually-evoked eye movements, network topographies mediate between the distinct peripheral organizations of retina and extraocular muscles. Many of these topographic features are prewired to create a computational substrate whereby saccades can be initiated, so that saccadic errors can be generated and used to improve future saccadic accuracy. We will begin our discussion by considering the simplest sensory-motor map capable of initiating saccades. Then we will refine this map in several ways to incorporate increasingly sophisticated constraints governing different aspects of saccadic learning.

In the simplest example, lights hit the retina, where they are encoded in retinal coordinates. Eventually some of these lights can selectively activate the six extraocular muscles. These muscles are organized into three agonist-antagonist pairs (Figure 1.2). Thus, even in the simplest examples, a transformation from retinal coordinates to muscle coordinates is required. For definiteness, consider how a retinal signal could influence the pair of muscles controlling horizontal movements, the lateral and medial recti. A simple map that mediates between a two-dimensional retinal array and an agonist-antagonist muscle pair is depicted in Figure 2.1. The "retina" in Figure 2.1 maps topographically into a structure that is subdivided into a right and a left hemifield. A gradient of connections exists from each point of this hemifield map to the muscle pair, such that more eccentric retinal points cause more asymmetric muscle contractions. In particular, more eccentric points in the right hemifield excite the right muscle more and inhibit the left muscle more in a push-pull fashion (van Gisbergen, Robinson, and Gielen, 1981). Edwards (1980) showed evidence for a gradient of projections from superior colliculus of the cat to the oculomotor area. Horseradish peroxidase was injected into the abducens area in order to find its inputs. The results showed that along the rostral-caudal dimension of the superior colliculus the incidence of abducens directed cells gradually increased at successively more caudal levels. More caudal levels of superior colliculus represent more peripheral parts of the visual field.

If each of the three pairs of muscles derives signals from a hemifield map, then each pair determines a different hemifield axis. Figure 2.2 depicts the simplest realization of this idea: a six-sectored map that we call a *sensory-motor sector map*. Such a sector map could be prewired using relatively simple developmental mechanisms: for example, three hemifield gradients of morphogens, in much the same way as tectal coordinates are established (Hunt and Jacobson, 1972, 1973a, 1973b).

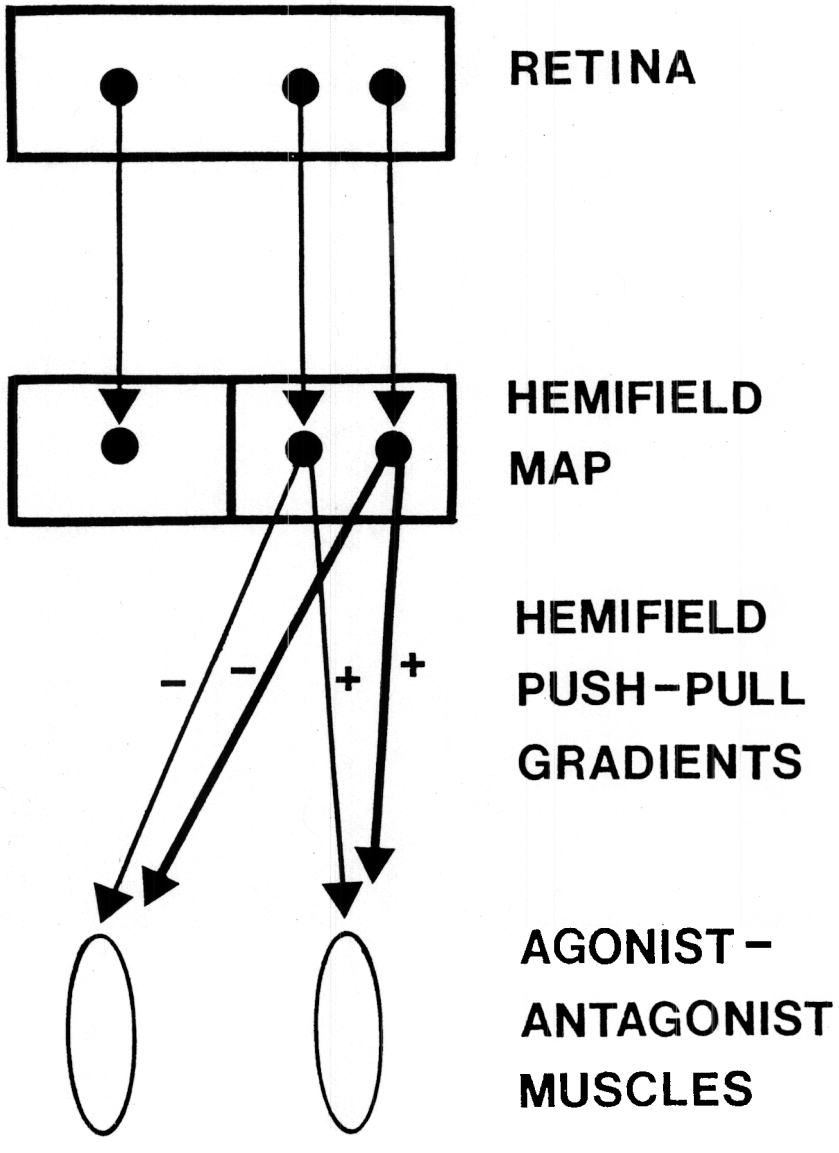

Figure 2.1. A scheme for mapping retinal position signals into agonist-antagonist muscle commands: First retinal positions are mapped topographically into a hemifield map. The output signals from the hemifield map increasingly favor one muscle of the pair as a function of retinal eccentricity.

Such a direct link from retina to muscles does not, of course, control saccadic motions *in vivo*. The ensuing theoretical argument shows how intermediate processing stages can be derived from constraints on adaptive saccadic learning and performance. In the complete model, the final stage of the six-sectored map plays the role of the oculomotor nuclei.

2.2. Choice of Fixation Light: Network Competition

A visual scene contains many possible fixation points. One of these points is chosen for fixation from the many possible candidates. Within the computational framework defined in Figure 2.1, such a choice mechanism needs to transform a broad array of lights on the retina into a relatively localized activation of the sector map. The sector map can then, in turn, preferentially contract some muscles more than others. Since broadly distributed lights on the retina activate many pathways to the sector map, a competitive interaction exists between the retina and the sector map that converts a broadly distributed input pattern into a more narrowly focused activity pattern. Section 2.6 describes how to design a competitive network capable of making such a choice across spatially distributed alternatives.

2.3. Correcting Fixation Errors: Competition Precedes Storage in Sensory Short Term Memory

A focal activation of the sector map can elicit eye movements towards the chosen light, but the direction and length of these movements may be inaccurate. We assume that genetic mechanisms are unable to precisely prewire the correct coordination of eye separation and size, muscle inertia, neural signal strengths, and other relevant eye movement parameters. Since several parameters contribute to each motion, the only meaningful test of the collective effect of these parameters is whether or not they generate an accurate fixation. Consequently, we base our saccadic error scheme on the location of the chosen light on the retina after an eye movement takes place. This simple idea imposes several design constraints. To discuss these constraints, we call the chosen light before movement the *first light* and the chosen light after movement the *second light*. This terminology emphasizes the fact that the system does not *a priori* know whether these two lights are due to the same light source.

A. *Short Term Memory of the First Light*

In order to correct the command due to the first light using information about the position of the second light, the system needs to store an internal marker of the position of the first light until the second light is registered. We call this storage process *short term memory* (STM). Since the command to be stored represents a sensory activation, we call this example of STM *sensory short term memory* (SSTM) to distinguish it from *motor short term memory* (MSTM) processes that will also be needed. Section 2.6 shows how a chosen light position can be stored in STM.

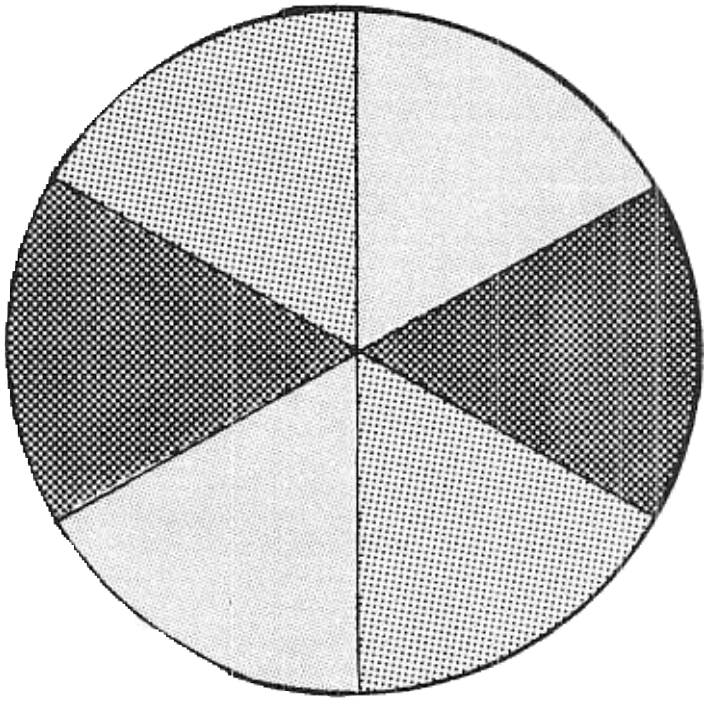

Figure 2.2. A sector map: Each wedge of the circle represents a region that maps preferentially into one of the six extraocular muscles. Pairs of wedges with similar hatching correspond to agonist-antagonist muscle pairs.

B. *Competition Stage Precedes Sensory Short Term Memory Stage*

Another way to say that the first light's position is chosen and stored in SSTM is to say that no other light position can be stored in SSTM while the first light's position is stored there. In particular, the second light cannot be stored in SSTM until the first light is no longer stored there. However, the second light must act as an error signal while the command corresponding to the first light is still being stored. Otherwise there would be no internal trace of which pathways the error signal should correct.

After the movement terminates, many lights will again activate the retina. In order to even define a second light, a competitive process needs to choose a light from among the many retinal lights. This choice process occurs, moreover, while the command corresponding to the first light is still stored in SSTM. Thus the competitive process that chooses among retinal lights, whether the first light or the second light, occurs prior to the stage that stores a light in SSTM (Figure 2.3).

2.4. Parallel Processing of Movement and Error Signals

Activation of a retinal position by a light can elicit signals in two functionally distinct pathways, a movement command pathway and an error signal pathway. To understand why this is so, note that each retinal position can be activated by either the first light or the second light of some saccade. When a retinal position is activated by the second light in a saccade, it can generate an error signal. This error signal is elicited at a stage *subsequent* to the competition that chooses the second light (Figure 2.4). The error signal is elicited at a stage *prior* to the SSTM stage that stores the first light in SSTM, since this stage blocks storage of other lights in SSTM until after the second light error signal is registered.

A second light error signal alters the strength of the conditioned pathway that is activated by the SSTM representation of the first light (Figure 2.4). The role of this learning process is to improve the ability of the first light to elicit correct saccades on future performance trials. Thus the conditioned pathway that is activated by the first light is a source of saccadic movement signals.

We can now draw another conclusion by invoking the fact that every retinal position can be activated by either the first light or the second light of some saccade. This fact implies that the second light provides a source of movement signals for the next saccade, and the first light provides a source of error signals for the previous saccade.

In summary, in order to correct previous errors before helping to generate the next movement, each retinal position gives rise to an error signal pathway as well as a pathway that activates a positional map at the SSTM stage. The SSTM stage, in turn, activates a conditioned pathway which can be altered by these error signals.

Figure 2.4 expands the network to include the new processing stages that are needed to implement these functional requirements. Figure 2.4

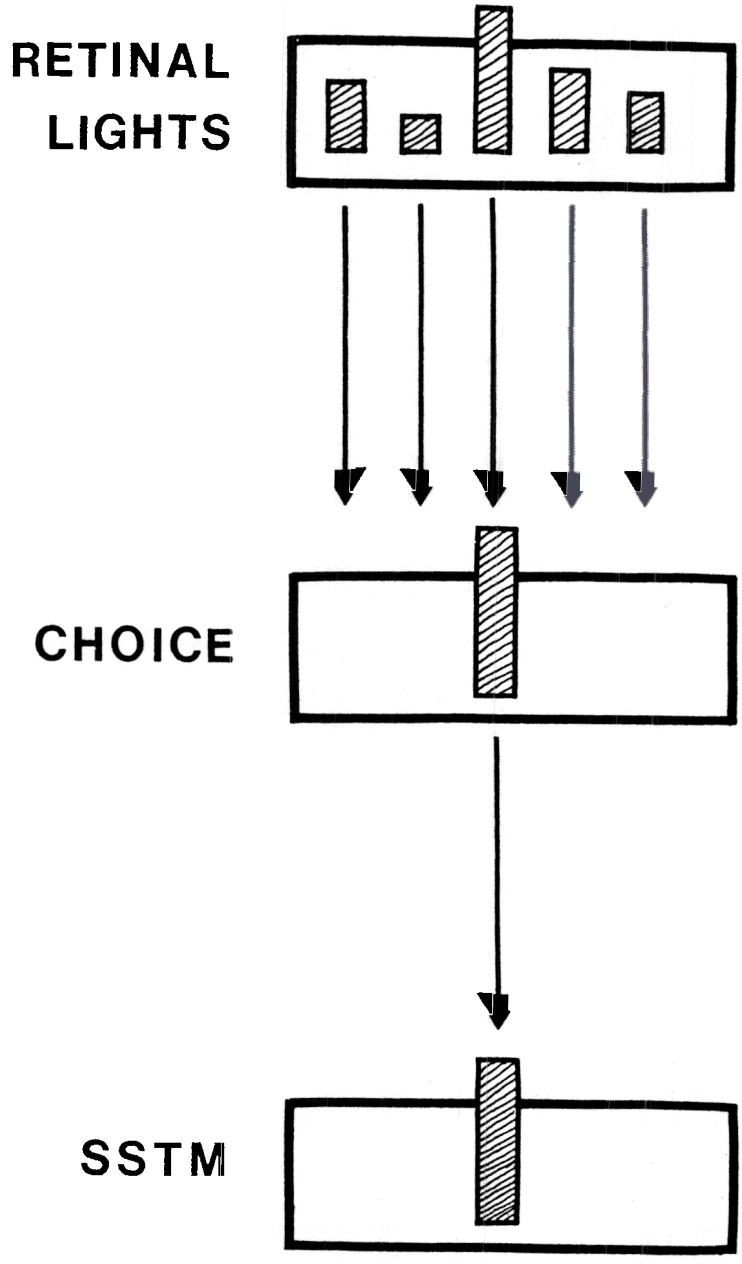

Figure 2.3. A choice among retinal lights occurs before the chosen light is stored in sensory short term memory (SSTM).

includes an unconditioned movement pathway as well as a conditioned movement pathway. The unconditioned pathway enables saccades to be generated by lights even before learning occurs. The error signals that are registered due to these saccades alter the strengths of the conditioned movement pathways. As a result, the total movement signal, which consists of an unconditioned and a conditioned component, generates more accurate movements than the unconditioned component alone. Figure 2.4 thus illustrates an example of a one-to-many analysis of a sensory input that occurs prior to a many-to-one synthesis of a movement (Section 1.4).

2.5. Why Does a Saccade Generator Exist?

In order for an SSTM stage to work well, there must also exist a saccade generator that converts the spatially coded signal within a light-activated retinal map into a temporally coded signal that determines how long and in what direction the eye will move (Keller, 1974; van Gisbergen, Robinson, and Gielen, 1981). To understand why such a spatial-to-temporal conversion is necessary, recall that the SSTM stage activates a conditioned pathway until *after* the eye comes to rest, so that this pathway remains active long enough to sample the second light error signal. The second light error signal cannot, however, be initiated until the eye stops moving. The network needs to convert the sustained output signal from the SSTM stage into a phasic movement signal whose duration is less than that of the SSTM output signal itself. Otherwise the sustained SSTM output signal would keep the eye moving until some muscles maximally contract. The onset of the movement signal that is activated by output from the SSTM stage thus initiates a process that eventually inhibits the movement signal before the SSTM output itself shuts off. The mechanism that initiates, maintains, and terminates the movement signal is called the *saccade generator* (SG).

This argument is important because it shows that the existence and properties of an SG, which have been analysed in the literature entirely from the viewpoint of saccadic performance, take on additional meaning when they are analysed from the perspective of saccadic learning. Indeed, this learning argument is the only one we know which suggests why a saccade generator *must* exist, rather than some other type of mechanism for moving the eyes.

An analysis of SG properties from the viewpoint of saccadic learning places strong constraints on the design of the SG. Two of the more obvious constraints are the following ones. The error signal acts at a stage prior to the SG so that both the unconditioned and conditioned movement pathways can input to the SG. The SG must be designed so that learned changes in the strength of the conditionable pathway can improve saccadic foveation. In particular, changes in the amplitude of the signals in the conditioned pathways must cause changes in the SG output that improve the accuracy of saccadic length and direction. A network synthesis of such an SG circuit will be described in Chapter 7.

To illustrate how our approach supplements earlier work about the SG,

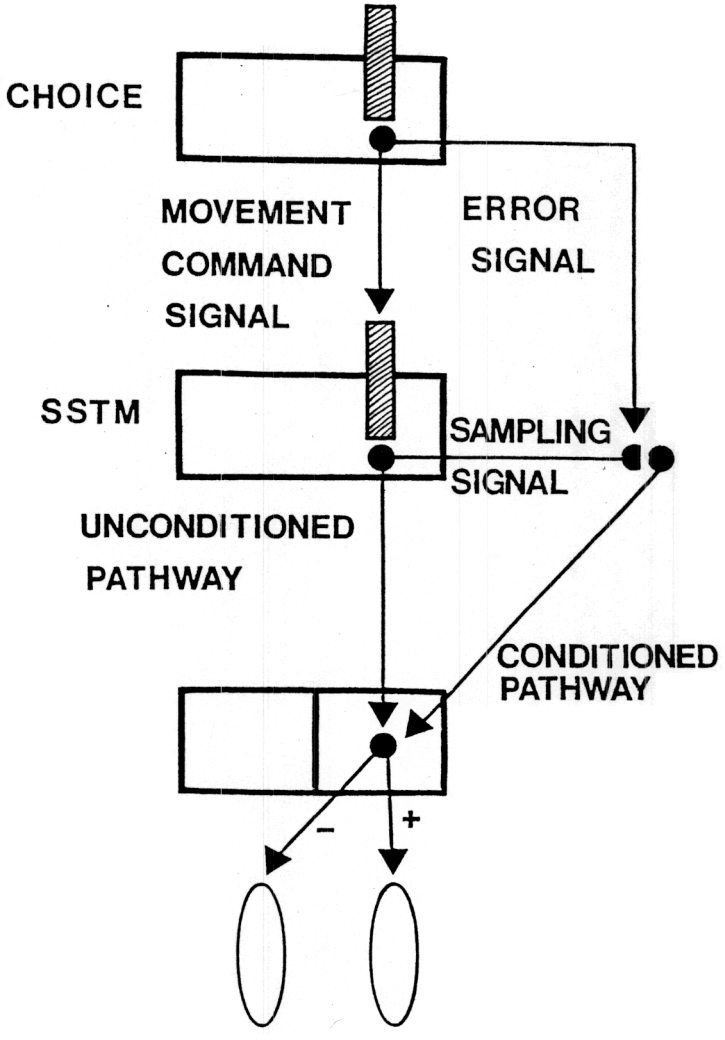

Figure 2.4. The representation of the chosen first light gives rise to an unconditioned movement signal and a conditioned movement signal. The unconditioned signal causes movements that are corrected by the conditioned movement signal via learning. The conditioned pathway carries sampling signals whose strength can be altered by second-light mediated error signals. These sampling signals give rise to the conditioned movement signal. The representation of the first light must be stored until after the end of the saccade, so that the second-light mediated error signal can act.

consider the following quotation from Robinson (1981, p.1302) concerning his important model of the SG: "The output of the neural integrator...is a copy of instantaneous eye position. If higher centers, which selected the target, made available a signal representing the location of the target with respect to the head, the difference between the target and eye signals would be the neurally encoded motor error...If this signal drove the burst neurons, their firing rate would drive the eyes at a high velocity until the error was zero, and at this time the burst would stop. This hypothesis guarantees that all bursts automatically have the correct size for each saccade, because the burst always continues until the eye is on target." For further discussion of Robinson's model, see the summary of Keller (1981, p.57).

We concur with Robinson's concept of an SG in which a difference signal terminates output from burst neurons. Our analysis of this system differs, however, from that of Robinson. This is because, without further mechanisms, a difference signal does not "automatically have the correct size for each saccade." It only achieves the correct size for each saccade after several major problems of calibration are solved. For example: How does the neural network know how a prescribed instantaneous burst activity will be translated into instantaneous eye movement velocity? What if the eye muscle contracts too slowly or too quickly? How does burst activity compensate for possible nonlinearities in muscle response? Each motoneuron cell may have a different threshold and slope of reaction to a given input (Robinson, 1970; Schiller, 1970). How is the total output from a population of such bursters calibrated to produce a linear total response from target muscles? If this is not accomplished, how can "output of the neural integrator" be used to provide "a copy of instantaneous eye position"? In a similar vein, how does the system determine that it has reached the desired target by using a computation wherein a target signal that is calibrated in "higher centers" numerically equals the output from a "neural integrator" in a different part of the brain? Unless these problems of calibration are solved, such a difference computation will not yield the desired behavior. In Robinson's model, it is assumed that these parameters have been correctly chosen. Our theory shows how the model can calibrate its own parameters using only locally computable qualities.

We finish this chapter by illustrating how on-center off-surround feedback interactions among cells which obey membrane equations can be designed to make choices, to store them in STM, and to respond to attentional and motivational signals.

2.6. Competitive Choice and Storage in Short Term Memory

This section reviews basic properties of networks of neurons which obey membrane equations and interact via on-center off-surround anatomies, or related cooperative-competitive anatomies. Then we focus the discussion to consider properties of primary interest such as competitive choice, STM storage, and attentional or motivational modulation. The mathematical theory of such networks began to be developed in Gross-

berg (1973) and has since undergone rapid development. See, for example, Carpenter and Grossberg (1983, 1984, 1985a, 1985b), Cohen and Grossberg (1983, 1984a, 1984b, 1985), Grossberg (1980, 1981, 1983), Grossberg and Mingolla (1985a, 1985b), and Grossberg and Stone (1985a, 1985b) for some recent contributions.

A. *Shunting Interactions*

A membrane equation is an equation of the form

$$C\frac{\partial V}{\partial t} = (V^+ - V)g^+ + (V^- - V)g^- + (V^p - V)g^p.$$

In equation (2.1), $V(t)$ is the cell's variable voltage. Parameter C is a constant capacitance. The constants V^+, V^-, and V^p are excitatory, inhibitory, and passive saturation points, respectively. Often V^+ and V^- are associated with Na^+, K^+, and Cl^- channels, respectively (Hodgkin, 1964; Katz, 1966). The terms g^+, g^-, and g^p are conductances that can vary through time as a function of input signals. Due to the multiplicative relationship between conductances and voltages in (2.1), a membrane equation is also said to describe a *shunting* interaction. In the next paragraphs, we show how on-center off-surround interactions among cells obeying such shunting interactions can be derived from functional considerations. Then we will indicate how desirable functional properties, such as competitive choice and STM storage, can be achieved by networks of this type whose parameters are appropriately chosen.

B. *Ratio Processing and Normalization of Spatial Patterns by Shunting On-Center Off-Surround Networks*

Let $x_i(t)$ be the activity, or potential, of the ith cell (population) v_i in a field F of cells $v_1, v_2, \ldots, v_n$. Suppose that each v_i has B excitable sites of which $B - x_i$ are unexcited. Let an input pattern $(I_1, I_2, \ldots, I_n)$ perturb F in such a way that I_i excites v_i's unexcited sites by mass action. Also let excitation x_i spontaneously decay at a constant rate A. Then the net rate $\frac{d}{dt}x_i$ at which sites v_i are activated is

$$\frac{d}{dt}x_i = -Ax_i + (B - x_i)I_i,$$

$i = 1, 2, \ldots, n$.

This law is inadequate because all the activities x_i can saturate at their maximal values B in response to an intensely activated input pattern. To see this, we define a *spatial pattern* to be an input pattern whose relative activities θ_i are constant through time. Then each $I_i(t) = \theta_i I(t)$, where the ratio θ_i is the constant "reflectance" of the input pattern at v_i and $I(t)$ is the total, and possibly variable, background intensity. The convention that $\sum_{i=1}^n \theta_i = 1$ implies that $I(t) = \sum_{i=1}^n I_i(t)$. Choose a constant background intensity $I(t) = I$ and let the activities equilibrate to their respective

inputs. The equilibrium activities of (2.2) are found by setting $\frac{d}{dt}x_i = 0$. We find

$$x_i = \frac{B\theta_i I}{A + \theta_i I}. \tag{2.3}$$

Now set the background intensity I at progressively higher levels without changing the reflectances θ_i. Then each x_i saturates at B no matter how differently the $\theta_i (> 0)$ are chosen.

This saturation problem can be solved by letting lateral inhibitory inputs shut off some sites as excitatory inputs turn on other sites in a feedforward competitive anatomy (Figure 2.5a). In the simplest version of this idea, (2.2) is replaced by

$$\frac{d}{dt}x_i = -Ax_i + (B - x_i)I_i - x_i \sum_{k \neq i} I_k,$$

$i = 1, 2, \ldots, n$. The new term $-x_i \sum_{k \neq i} I_k$ says that the lateral inhibitory inputs $\sum_{k \neq i} I_k$ shut off the active sites x_i by mass action. In response to a sustained spatial pattern $I_i = \theta_i I$, the equilibrium activities of (2.4) are

$$x_i = \theta_i \frac{BI}{A + I}.$$

By (2.5), each x_i is proportional to θ_i no matter how large the total input I is chosen. The background activity I is factored into the Weber-law modulation term $BI(A + I)^{-1}$, which approaches the constant B as I increases. Thus (2.5) shows that system (2.4) can accurately process the reflectances θ_i no matter how large the total input I is chosen. This property is due to the multiplication, or shunting, of x_i by lateral inhibitory signals in (2.4).

The total coefficient of x_i in (2.4) is called the *gain* of x_i. Thus the saturation problem is solved by automatic gain control due to lateral inhibition. System (2.4) describes the simplest example of a feedforward shunting on-center off-surround network.

System (2.4) also possesses a *normalization* property. The total activity $x = \sum_{i=1}^{n} x_i$ satisfies the equation

$$x = \frac{BI}{A + I}$$

because $\sum_{i=1}^{n} \theta_i = 1$. By (2.6), given a fixed total input I, the total activity x is independent of the number of active cells. Shunting competitive networks hereby tend to conserve their total activity. In shunting on-center off-surround feedback networks, the normalization property provides a

dynamical explanation of why short term memory is a limited capacity process.

C. *Featural Noise Suppression: Adaptation Level and Pattern Matching*

In (2.4), activity x_i can fluctuate between 0 and B. *In vivo*, inhibition can often hyperpolarize x_i below its passive equilibrium point 0. To fully understand competitive dynamics requires that we classify other biologically relevant competitive designs than the simplest example (2.4). Hyperpolarization is possible in the following generalization of (2.4):

$$\frac{d}{dt}x_i = -Ax_i + (B - x_i)I_i - (x_i + C)\sum_{k \neq i} I_k,$$

where $-C \leq 0 \leq B$. If $C > 0$ in (2.7), then x_i can be hyperpolarized by inhibitory inputs to any negative value between 0 and $-C$. In response to a sustained spatial pattern $I_i = \theta_i I$, the equilibrium activities of (2.7) are

$$x_i = \frac{(B + C)I}{A + I}\left(\theta_i - \frac{C}{B + C}\right).$$

By (2.8), $x_i > 0$ only if $\theta_i > C/B + C$. Since output signals are generated only by depolarized, or positive, values of x_i. the term $C(B + C)^{-1}$ is called the *adaptation level* of the network. Raising the adaptation level makes output signals harder to generate.

The special choice $B = (n - 1)C$ illustrates how the adaptation level works in its simplest form. Then $C(B + C)^{-1} = 1/n$. In response to any uniform spatial pattern $I_i = 1/n$. Then (2.8) implies that all $x_i = 0$ no matter how large I is chosen. This property is called *featural noise suppression*, or the suppression of zero spatial frequency patterns. Due to this property, the network suppresses input patterns that do not energetically favor any cellular feature detectors.

The featural noise suppression property implies a pattern matching property. For example, let two input patterns $J^* = (J_1, J_2, \ldots, J_n)$ and $K^* = (K_1, K_2, \ldots, K_n)$ add their inputs $I_i = J_i + K_i$ to generate a total input pattern $I^* = (I_1, I_2, \ldots, I_n)$ to the network. If the two patterns J^* and K^* are mismatched so that their peaks and troughs are spatially out-of-phase, then I^* will tend to be approximately uniform and will be suppressed by the adaptation level. By contrast, if J^* and K^* have the same reflectances, say $J_i = \theta_i J$ and $K_i = \theta_i K$, then (2.7) implies that

$$x_i = \frac{(B + C)(J + K)}{A + J + K}\left(\theta_i - \frac{C}{B + C}\right).$$

By (2.9), the network energetically amplifies its response to matched patterns via Weber-law modulation. This type of energetic amplification due

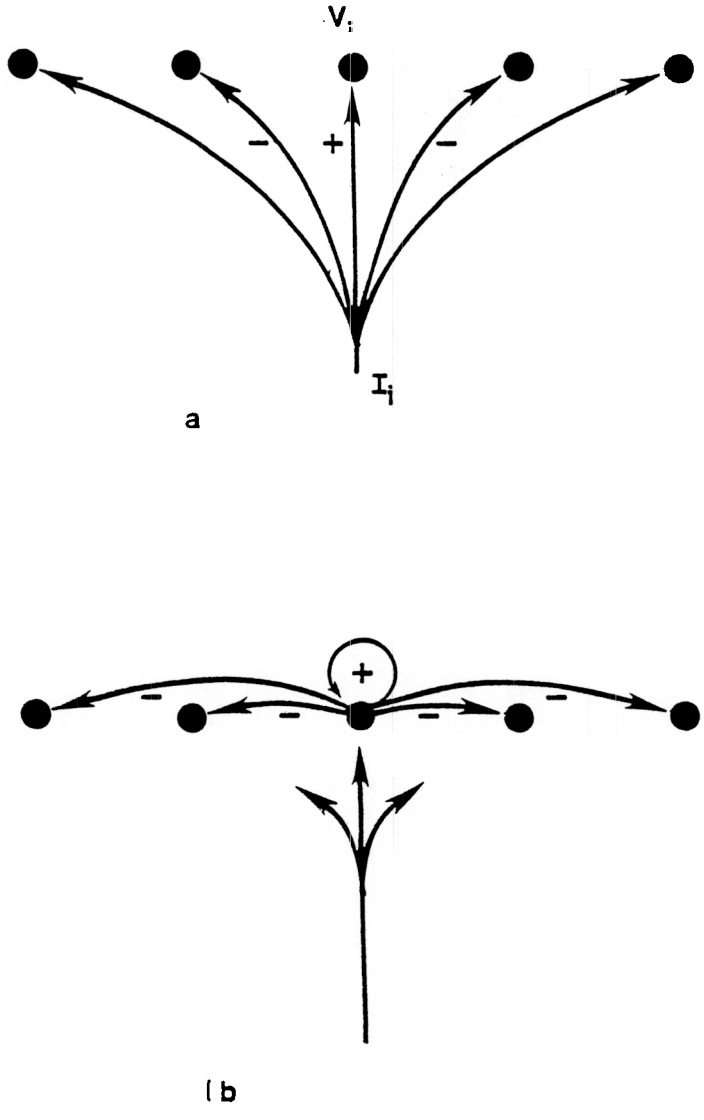

Figure 2.5. Two types of on-center off-surround networks: (a) A feedforward network in which the input pathways define the on-center off-surround interactions; (b) A feedback network in which interneurons define the on-center off-surround interactions.

to matching is different from the suppressive matching that occurs when a target position equals a present position (Section 2.6).

D. *Receptive Fields, Spatial Frequencies, and Edges*

In more general feedforward shunting networks, the above properties hold in a modified form. Consider for example the class of feedforward networks

$$\frac{d}{dt}x_i = -Ax_i + (B - x_i)\sum_{k=1}^{n} I_k C_{ki} - (x_i + D)\sum_{k=1}^{n} I_k E_{ki}, \tag{2.10}$$

$i = 1, 2, \ldots, n$. In (2.10), the coefficients C_{ki} and E_{ki} describe the fall-off with the distance between cells v_k and v_i of the excitatory and inhibitory influences, respectively, of input I_k on cell v_i. In response to a sustained spatial pattern $I_i = \theta_i I$, the equilibrium activities of (2.10) are

$$x_i = \frac{F_i I}{A + G_i I} \tag{2.11}$$

where

$$F_i = \sum_{k=1}^{n} \theta_k (BC_{ki} - DE_{ki}) \tag{2.12}$$

and

$$G_i = \sum_{k=1}^{n} \theta_k (C_{ki} + E_{ki}). \tag{2.13}$$

The featural noise suppression property is implied by the inequalities

$$B\sum_{k=1}^{n} C_{ki} \leq D\sum_{k=1}^{n} E_{ki} \tag{2.14}$$

since then, by (2.11) and (2.12), all $x_i \leq 0$ in response to a uniform pattern $\theta_i = 1/n$ no matter how large the total input I is chosen.

When the featural noise suppression property holds in a distance-dependent network, the network can, in addition to the other properties cited above, detect edges and other spatially nonuniform gradients in input patterns for the following reason. Inputs are suppressed by all the cells across whose receptive fields the input pattern looks approximately uniform, no matter how intense the input pattern is near these cells. In particular, a rectangular input pattern is suppressed both outside and inside the pattern by this mechanism. Only those cells can respond which occur near input regions where the input intensity changes across space at a rate that is no coarser than the receptive fields.

The responding cells compute input reflectances or relative contrast differences in their vicinity using the ratios that occur in equation (2.11).

Equation (2.11) generalizes the reflectance processing properties of (2.5) and (2.8). The breadth of the edge reflects both the rate of change of the input pattern and of the structural scales of the network. Larger structural scales cause broader edges, other things being equal, and thus make it easier to match a pair of partially out-of-phase edges. Thus both relative contrasts and spatial scaling properties are encoded within the edges extracted by (2.11). Equation (2.11) generalizes the familiar difference-of-Gaussian receptive field model that is broadly used in analyses of spatial vision (Blakemore, Carpenter, and Georgeson, 1970; Ellias and Grossberg, 1975; Enroth-Cugell and Robson, 1966; Levine and Grossberg, 1976; Rodieck and Stone, 1965; Wilson and Bergen, 1979).

E. *Short Term Memory, Feedback Competitive Networks, and Nonlinear Cross-Correlation*

Short term memory (STM) storage of input patterns is possible in networks possessing positive and negative feedback pathways (Figure 2.5b). In order to prevent saturation due to positive feedback signalling, the positive feedback signals are balanced by competitive, or lateral inhibitory, feedback signals that automatically change the network's gain, just as in feedforward competitive networks. The feedback competitive analog of the feedforward competitive system (2.10) is

$$\begin{aligned}\frac{d}{dt}x_i = -Ax_i + (B - x_i)[I_i + \sum_{k=1}^{n} f_k(x_k)C_{ki}] \\ - (x_i + D)[J_i + \sum_{k=1}^{n} g_k(x_k)E_{ki}],\end{aligned} \tag{2.15}$$

$i = 1, 2, \ldots, n$. In (2.15), I_i is the excitatory input to v_i, J_i is the inhibitory input to v_i, $f_k(x_k)C_{ki}$ is the positive feedback signal from v_k to v_i, and $g_k(x_k)E_{ki}$ is the negative feedback signal from v_k to v_i. Each input term I_i or J_i may itself be a weighted average of distributed inputs from a prior stage of processing. When C_{ki} and E_{ki} are functions of intercellular distances, then the excitatory and inhibitory interaction terms $f_i(x^*) = \sum_{k=1}^{n} f_k(x_k)C_{ki}$ and $g_i(x^*) = \sum_{k=1}^{n} g_k(x_k)E_{ki}$ in (2.15), where $x^* = (x_1, x_2, \ldots, x_n)$, define nonlinear cross-correlations

$$x^* \to (f_1(x^*), f_2(x^*), \ldots, f_n(x^*)) \tag{2.16}$$

and

$$x^* \to (g_1(x^*), g_2(x^*), \ldots, g_n(x^*)) \tag{2.17}$$

of the STM activities x^*. Thus the concepts of feedback signalling and of nonlinear cross-correlation are the same in a distance-dependent network.

F. *Signal Noise Suppression and Nonlinear Signals*

The transformations (2.16) and (2.17) must define *nonlinear* cross-correlators due to a mathematical property of the networks (2.15). The positive feedback signals can amplify small activities into large activities

(signals amplify "noise") unless the signal functions $f_k(x_k)$ and $g_k(x_k)$ are nonlinear functions of the STM activities x_k (Grossberg, 1973). Nonlinearity *per se* is not sufficient to prevent this from happening, since a nonlinear signal function such as $f_k(w) = \alpha w(\beta + w)^{-1}$ can cause a pathological STM response in which all STM activities are amplified to equal asymptotes no matter how different, and small, were their initial activities. To avoid such pathologies, the positive feedback signals $f_k(x_k)$ need to be faster-than-linear functions of x_k, such as powers $f_k(x_k) = \alpha x_k^n$ with $n > 1$, at small values of the activities x_k. Sigmoid, or S-shaped, functions of activity are the simplest physically plausible signal functions that solve the signal noise suppression problem (Grossberg, 1973).

G. *Dynamic Control of Network Sensitivity: Quenching Threshold and Attentional Gain Control*

When sigmoid feedback signals are used in a feedback competitive network such as (2.15), the network possesses a parameter that is called the *quenching threshold* (QT): STM activities that start out less than the QT tend to be suppressed, whereas the pattern of STM activities that initially exceeds the QT tends to be contrast enhanced through time (Figure 2.6). The QT is not just the manifest threshold of a signal function. The QT is a parameter that depends on the global structure of the network. For example, consider the following special case of (2.15):

$$\frac{d}{dt}x_i = -Ax_i + (B - x_i)f(x_i) - x_i \sum_{k \neq i} f(x_k),$$

$i = 1, 2, \ldots, n$. In (2.18), all inputs are shut off and the competitive interaction $\sum_{k \neq i}^{n} f(x_k)$ describes long-range lateral inhibition, just like the term $\sum_{k \neq i}^{n} I_k$ in the feedforward network (2.4). Suppose that the feedback signal function $f(w)$ satisfies

$$f(w) = Cwg(w)$$

where $C \geq 0$, $g(w)$ is increasing if $0 \leq w \leq x^{(1)}$, and $g(w) = 1$ if $x^{(1)} \leq w \leq B$. Thus $f(w)$ is faster-than-linear for $0 \leq w \leq x^{(1)}$, linear for $x^{(1)} \leq w \leq B$, and attains a maximum value of BC at $w = B$ within the activity interval from 0 to B. The values of $f(w)$ at activities $w \geq B$ do not affect network dynamics because each $x_i \leq B$ in (2.18). It was proved in Grossberg (1973, pp.238–242) that the QT of this network is

$$QT = \frac{x^{(1)}}{B - AC^{-1}}. \tag{2.20}$$

By (2.20), the QT is not the manifest threshold of $f(w)$, which occurs where $g(w)$ is increasing. The QT depends on the transition activity $x^{(1)}$ where $f(w)$ changes from faster-than-linear, upon the overall slope C of the

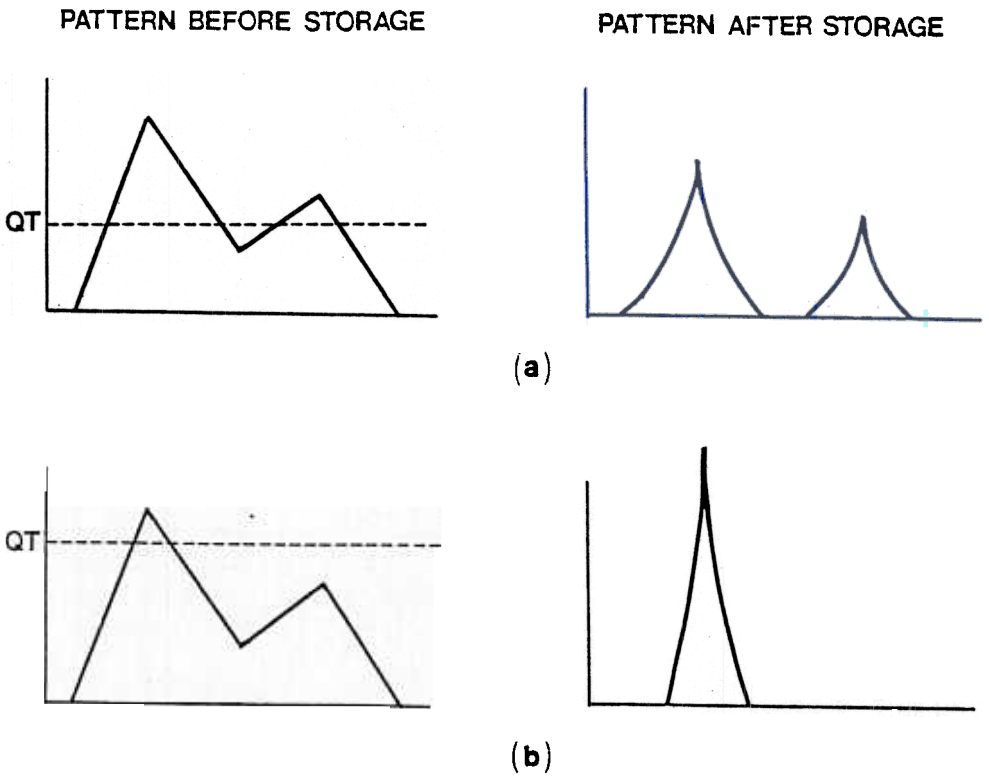

Figure 2.6. The quenching threshold (QT). In Figures 2.6a and 2.6b, the same input pattern is differently transformed and stored in short term memory due to different settings of the network QT.

signal function in the physiological range, upon the number B of excitable sites in each population, and upon the STM decay rate A. Equation (2.20) shows that an increase in C causes a decrease in the QT. Consequently, increasing a shunting signal C that nonspecifically gates all the network's feedback signals facilitates STM storage. Equation (2.20) also shows how using a linear signal function destabilizes network dynamics. If $f(w)$ is linear in (2.19), then $x^{(1)} = 0$. By (2.20), the QT $= 0$. Hence any positive network activity, no matter how small, will be amplified by a linear signal function. From the inception of the theory, it was realized that any of several network parameters can cause the QT to become pathologically small, thereby destabilizing network dynamics and leading to network "seizures" or "hallucinations" (Grossberg, 1973; Traub and Wong, 1983).

Equation (2.20) illustrates that the network's sensitivity can be modulated by dynamical factors. If the nonspecific gain C is chosen very small, for example, then the QT may be so large as to desensitize the network to all inputs. By contrast, a large choice of C can render the network sensitive to its input by decreasing the QT. A nonspecific form of attentional gain control can thus modulate the network's sensitivity to its inputs by controlling the size of the QT through time.

The QT property is not "built into" the network. It is a *mathematical* consequence of using shunting on-center off-surround feedback networks, and was considered surprising when it was first discovered by Grossberg (1973). Thus the network design which prevents saturation by automatically adjusting its gains in response to variable input loads (Section 2.6B), and prevents amplification and STM storage of network noise by using a proper signal function (Section 2.6F) is already prepared to respond adaptively to nonspecific attentional gain control signals. This type of attentional processing will be used to discuss how attention modulates STM storage of target positions within the posterior parietal lobe (Chapters 4 and 11).

H. *Competitive Choice*

A sigmoid signal function $f(w)$ is composed of a faster-than-linear part at small activity values w, a slower-than-linear part at large activity values w, and an approximately linear part at intermediate activity values w (Figure 2.7). Each of these activity regions transforms input patterns in a different way before storing them as STM activity patterns. If network activities remain within the faster-than-linear range of a sigmoid signal function, then the network is capable of making a competitive choice by contrast-enhancing the input pattern via the network's feedback pathways until only the population with the largest initial activity has positive activity.

A competitive choice can be accomplished in either of two ways: structurally or dynamically. In the structural solution, network parameters are chosen so that all STM activities remain within the faster-than-linear range under all circumstances. In the dynamical solution, a nonspecific attentional gain control signal shifts either STM activities into the faster-than-linear range, or shifts interaction parameters such as inhibitory in-

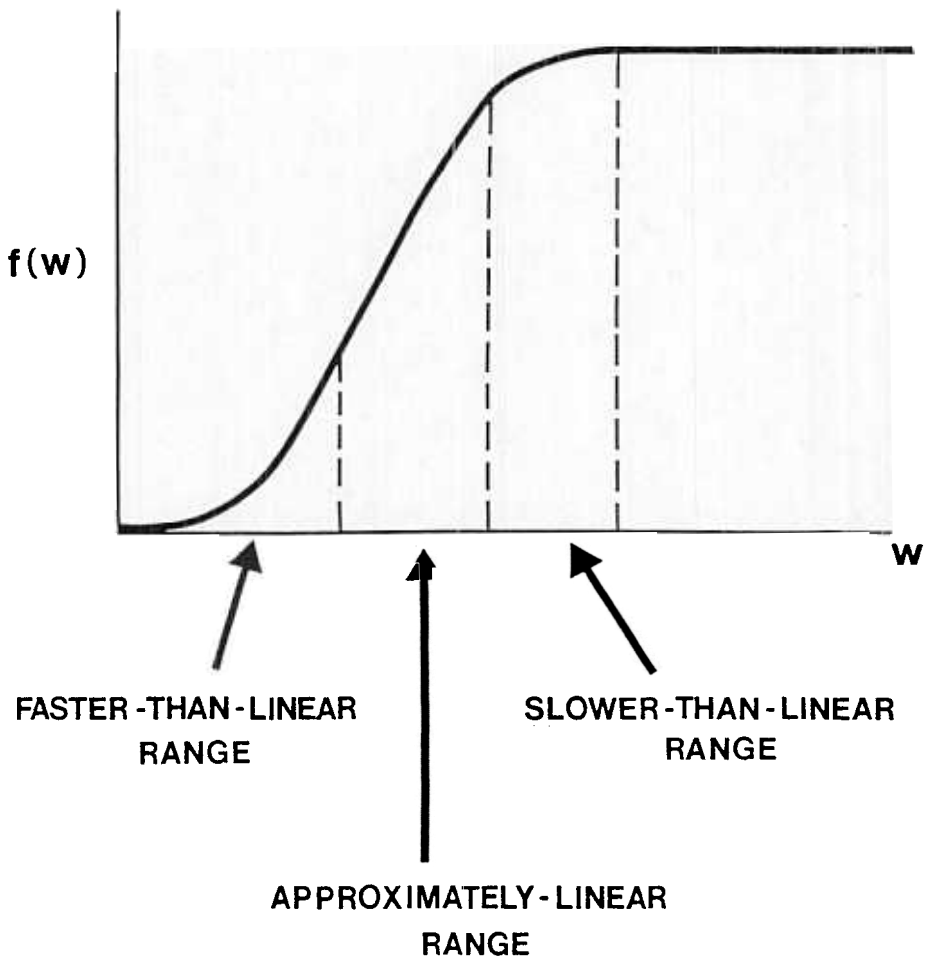

Figure 2.7. A sigmoid signal function: The faster-than-linear part tends to contrast-enhance activity patterns; the linear part tends to preserve activity patterns; the slower-than-linear part tends to uniformize activity patterns, as in Table 2.1. Taken together, these tendencies define the quenching threshold.

teraction strengths into the faster-than-linear range until the choice is made.

A more complete understanding of how the signal function determines the stored STM pattern can be achieved by considering the following special case of (2.15):

$$\frac{d}{dt}x_i = -Ax_i + (B - x_i)[I_i + f(x_i)] - x_i[J_i + \sum_{k \neq i} f(x_k)], \qquad (2.21)$$

$i = 1, 2, \ldots, n$. Network (2.21) is just (2.18) with the inputs I_i and J_i left on. Network (2.21) strips away all extraneous factors to focus on the following issue. After an input pattern $(I_1, I_2, \ldots, I_n, J_1, J_2, \ldots, J_n)$ delivered before time $t = 0$ establishes an initial pattern $(x_1(0), x_2(0), \ldots, x_n(0))$ in the network's activities, how does feedback signalling within the network transform the initial pattern before it is stored in STM? This problem was solved in Grossberg (1973).

Table 2.1 summarizes the main features of the solution. The function $g(w) = w^{-1}f(w)$ is graphed in Table 2.1 because the property that determines the pattern transformation is whether $g(w)$ is an increasing, constant, or decreasing function at prescribed activities w. For example, a linear $f(w) = aw$ determines a constant $g(w) = a$; a slower-than-linear $f(w) = aw(b + w)^{-1}$ determines a decreasing $g(w) = a(b + w)^{-1}$; a faster-than-linear $f(w) = aw^n$, $n > 1$, determines an increasing $g(w) = aw^{n-1}$; and a sigmoid signal function $f(w) = aw^2(b+w^2)^{-1}$ determines a concave $g(w) = aw(b + w^2)^{-1}$. Both linear and slower-than-linear signal functions amplify noise, and are therefore unsatisfactory. Faster-than-linear signal functions, such as power laws with powers greater than one, or threshold rules, suppress noise so vigorously that they make a choice. Table 2.1 shows that sigmoid signal functions determine a QT by mixing together properties of the other types of signal functions.

I. *Attentional Biasing and Competitive Masking*

A suitably designed shunting on-center off-surround feedback network is also capable of biasing its stored STM in response to spatially focussed attentional or developmental factors (Grossberg, 1981; Grossberg and Levine, 1975). Such a spatially delimited attentional bias is not the same process as nonspecific attentional gain control, and it may coexist with attentional gain control (Grossberg, 1978a, 1982b). Both types of mechanism will be used to describe how attention modulates storage of target positions by the posterior parietal lobes (Chapters 4 and 11).

To distinguish focal attentional biasing from nonspecific attentional gain control, we call the focal process competitive *masking*. To illustrate the main properties of masking, we use the simplest possible example. A more sophisticated example, in which masking enables the network to respond in a context-sensitive way to a temporally evolving speech stream, is described in Cohen and Grossberg (1985) and Grossberg (1985c).

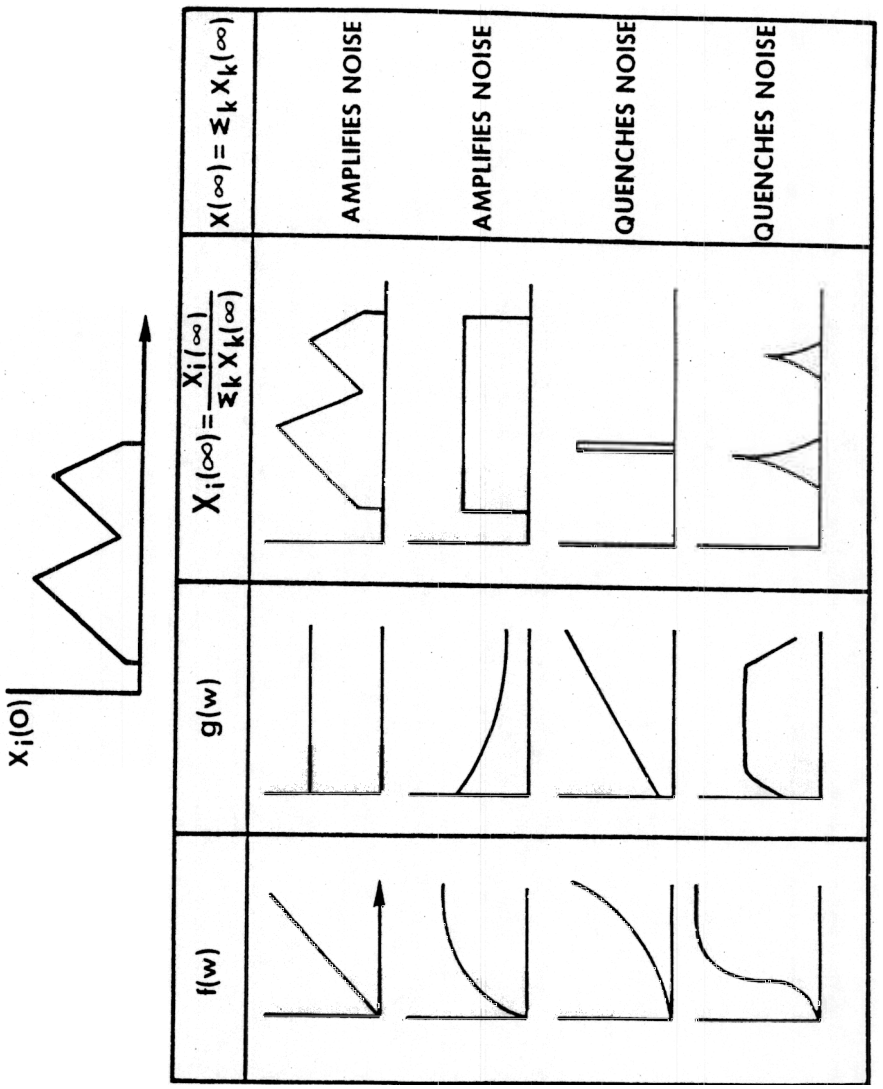

Table 2.1. Influence of signal function $f(w)$ on input pattern transformation and short term memory storage.

Masking occurs in systems

$$\frac{d}{dt}x_i = -Ax_i + (B_i - x_i)f(x_i) - x_i \sum_{k \neq i} f(x_k) \tag{2.22}$$

in which populations v_i can have different numbers B_i of excitable sites, or equivalently in systems

$$\frac{d}{dt}x_i = -Ax_i + (B - x_i)f(C_i x_i) - x_i \sum_{k \neq i} f(C_k x_k) \tag{2.23}$$

whose population activities or signals are differentially amplified by shunting factors C_i. System (2.23) can be formally transformed into system (2.22) by a change of variables. Despite this formal equivalence, the physical interpretations of these systems may differ. In (2.22), the different numbers of sites can be interpreted as a developmental bias in which certain input features are coded by more sites B_i than others. In (2.23), the differential amplification of population signals can be interpreted as an attentional shunt that gates all the feedback interneurons, both excitatory and inhibitory, of each population v_i using its own shunting parameter C_i. Such a shunt may, for example, be controlled by a learned incentive motivational signal from a midbrain reinforcement center (Grossberg, 1982b).

If both developmental and attentional biases occur, as in

$$\frac{d}{dt}x_i = -Ax_i + (B_i - x_i)f(C_i x_i) - x_i \sum_{k \neq i} f(C_k x_k), \tag{2.24}$$

then masking is controlled by the relative sizes of the products B_1C_1, B_2C_2, ..., B_nC_n. For definiteness, label the cells so that

$$B_1C_1 \geq B_2C_2 \geq \ldots \geq B_nC_n. \tag{2.25}$$

We now show that the nature of the masking depends upon the choice of signal function $f(w)$.

To start, let the signal function be linear, say $f(w) = Ew$. Then a masking phenomenon occurs such that $x_i(\infty) = 0$ if $B_iC_i < B_1C_1$, whereas

$$\frac{x_i(\infty)}{x_j(\infty)} = \frac{x_i(0)}{x_j(0)} \tag{2.26}$$

for all i and j such that $B_1C_1 = B_iC_i = B_jC_j$. By (2.26), the activity pattern across the subfield of populations v_i with maximal parameters $B_iC_i = B_1C_1$ is faithfully stored, but all other population activities

are competitively masked. This type of masking is inadequate in a sensory processor, because the salience of a feature in an external display, as measured by a large initial $x_i(0)$ value, cannot overcome internal biases $B_iC_i < B_1C_1$ even if $x_i(0)$ is much larger than $x_1(0)$.

This problem is overcome if a sigmoid signal function $f(w)$ is used. Then a tug-of-war occurs between cue salience $x_i(0)$, developmental biases B_i, and attentional shunts C_i to determine which population activities will be stored in STM (Grossberg and Levine, 1975). Superimposed upon this masking bias is the usual contrast-enhancement that a sigmoid signal function can elicit. Thus the same nonlinear signal function that suppresses noise and contrast-enhances STM activities exceeding the QT automatically generates the type of masking bias that can successfully refocus attention in response to incentive motivational signals.

In summary, the ubiquity in the brain of the shunting on-center off-surround network design can be better appreciated from the mathematical fact that variations of this design imply constellations of formal properties which solve a large number of important functional problems.