Chapter 5

Accessory Circuits

In principle, the synaptic matrix and the retinoid system can perform important cognitive operations in the networks of the brain. For these neuronal modules to function effectively, however, given the normal range of variation in stimulus properties and the constantly shifting relationships between an individual and significant objects within the immediate environment, the brain's basic modules must be augmented by appropriate accessory circuits. This chapter describes a number of such accessory mechanisms and shows how they work with the synaptic matrix and the retinoid registers.

Dendrodendritic Gradients of Excitation

The simulation tests of the synaptic matrix described in chapter 3 assumed a discrete point-to-point projection of excitation going from each active cell in the afferent bundle to its corresponding cell in the mosaic array within a retinotopic coordinate frame (see, for example, figure 3.2). The problem with this kind of strict channeling of excitation is that it results in brittle performance. An object that was learned in one retinotopic position could be abruptly mismatched against its synaptic profile on a test of recognition if its retinal location deviated even slightly from the position in which it was originally learned. Performance would be more robust if the synaptic representation on filter cells were composed of a gradient of transfer weights (ϕ) with peak values corresponding to the input coordinates of the originally learned stimulus and having progressivly decreasing ϕ values at neighboring points.

Illustrated at the top of figure 5.1 is a retinotopically organized neuronal array with dendrodendritic synapses between each of its neighboring cells. This structure is assumed to serve during learning as a local diffusion (gradient) layer between the mosaic cell array (M) and the ungraded pattern input to M from the afferent bundle.

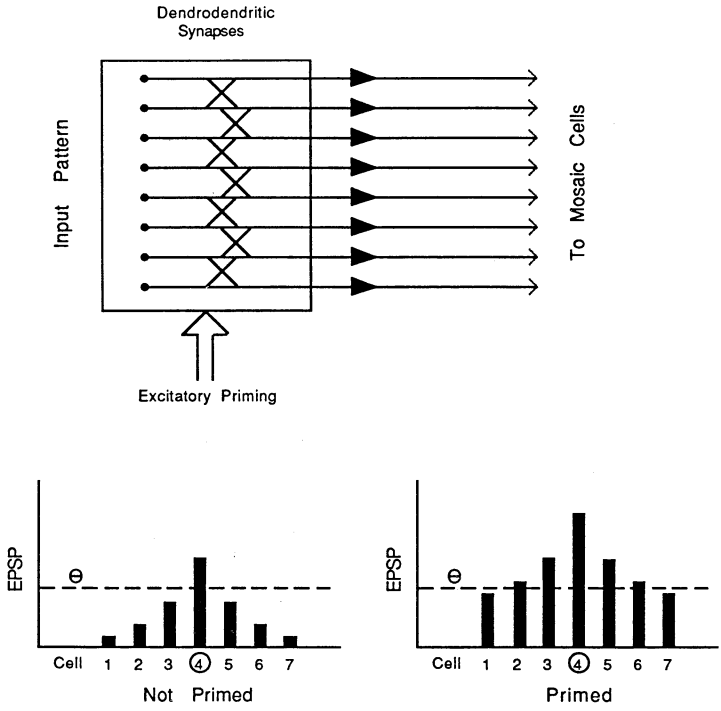

Figure 5.1
Top: Dendrodendritic diffusion layer. Retinotopically organized cells between retinal output and mosaic cells create a gradient of EPSP around primary stimulus pattern by dendrodendritic excitation to neighboring cells. *Bottom:* Cell 4 is discharged directly by retinal stimulation. Priming of cells in diffusion layer raises total EPSP in each cell so that EPSP in cells close to cell 4 exceeds threshold (θ) and evokes a gradient of discharge over these neighboring cells.

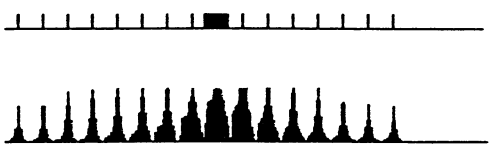

Figure 5.2
Distribution of synaptic transfer weights (ϕ) on filter cells in detection matrix. *Top:* Without dendrodendritic diffusion. *Bottom:* With diffusion. Each point on the dendritic line represents a discrete synaptic location.

Learning is assumed to occur only with a sufficient level of general activation (arousal) to the synaptic matrix. Similarly, in the gradient layer, the general activation normally associated with learning is required to raise the profile of EPSP transfer by dendrodendritic contacts so that the neighboring cells surrounding a primary afferent input target can reach discharge threshold, though their spike frequency will be less than that of the primary target cell (figure 5.1, bottom). When this occurs, mosaic cells neighboring the primary stimulus targets will discharge accordingly, and this activity will be reflected in the distribution of synaptic transfer weights on the filter cell that is modified during learning.

An example of the effect on synaptic transfer weights (ϕ) in a filter cell when a dendrodendritic gradient layer intervenes between the sensory input tract and the mosaic array is shown by the comparative simulation results in figure 5.2. In this case, a visual stimulus (a cross) was presented to a 16 $\times$ 16 cell retina to be learned in the synaptic matrix. The screen printout at the top of figure 5.2 presents the distribution profile of ϕ on the modified filter cell after learning without a dendrodendritic gradient layer. The bottom of the figure shows the distribution profile of ϕ on the filter cell when the model incorporated a dendrodendritic gradient layer. The underlying models and the stimulus for both simulations were identical except for the inclusion of a gradient layer in one. An arbitrary activation transfer coefficient of 0.6 was set for dendrodendritic excitation among neighboring cells in the gradient layer, which was incorporated in the simulation producing the profile shown at the bottom of figure 5.2. Because instances of a moderately close mismatch between stimulus coordinates and coordinates of maximum synaptic efficacy intersect elevated (though reduced) filter cell transfer weights in the latter case, it can be seen that such mismatches will be less critical for pattern recognition when there is a ϕ gradient around each primary synaptic locus.

Retinal-Afferent Organization

If the neuronal models proposed here are to operate effectively as processors of events in visual space, the retinotopic coordinates of the retinal receptor field must be conserved through the afferent channels and in the mechanisms for central processing. Although there are a number of different coordinate representations that one might choose for retinotopic indexing, I have found a ring-ray representation to be more useful and efficient than any other that I have considered. In this representational scheme, receptor cells in the retina (and their associated ganglion cell projections) are indexed with respect to the central foveal axis in terms of their locations on imaginary concentric rings (i) centering on the axis, and imaginary rays (j) projecting from the axis and intersecting all rings. Concentric rings (i) are indexed from the smallest ($i = 1$) to the largest ($i = n$). Rays (j) are indexed in clockwise sequence from the twelve o'clock position ($j = 1$) through one rotation to the last position before twelve o'clock ($j = n$).

In correspondence with the decreasing density of retinal receptors, the indexing scheme proposed here assumes that in terms of visual field coordinates, the spacing between concentric rings increases progressively as rings become larger and the distance between points of intersection of adjacent rays on the successively larger rings increases. This principle of ring-ray organization of the retina is illustrated in figure 5.3.

Afferent-Field Aperture

The ring-ray organization of the retina lends itself to central nervous system control of the effective processing aperture for input from the afferent visual field. Inhibitory neurons that synapse with selected ring groups of mosaic array cells can constrict the diameter of the visual window around the foveal axis. Conversely, termination of the activity of these inhibitory neurons can result in expansion of the effective visual window (figure 5.4). This afferent control circuit modulates the field of view and allows either aperture widening or dynamic masking and cropping of images before they are conveyed to the synaptic matrix for learning or recognition (Trehub 1977). Control of the afferent field aperture plays an integral part in the processes of focal attention and scene analysis.

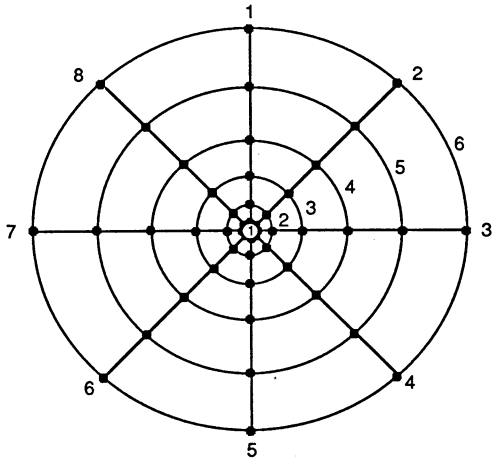

Figure 5.3
Ring-ray organization of retina. Each receptor and its afferent projection is identified by its coordinate in terms of the intersection of its *i*th ring on its *j*th ray.

Novelty Detection

During the earliest years of human maturation, learning is probably usually passive and nonselective. It is assumed that this kind of early learning depends on a high level of generalized arousal (corticipetal discharge diffusely distributed by the reticular activating system), which lowers thresholds in the synaptic matrix so that filter cells are sporadically fired and their transfer weights (ϕ) modified by whatever stimuli the alert child happens to encounter. In the more mature individual, however, it is more likely that stimuli must be salient before threshold priming and subsequent changes in ϕ occur in the synaptic matrix. It is assumed that whatever is relevant to the needs of the individual and also novel (not previously learned) should be learned. By what physical process might the novelty of the input be determined? In this model, classification time—the interval between the presentation of a stimulus and the firing of a class cell—is taken as the basis for determining the novelty of a stimulus.

Figure 5.5 shows a neuronal mechanism for detecting novelty. At the detection of an input in a context relevant to current motivation, a novelty test cell is discharged at an activity level (spike rate) sufficient to fire its target novelty cell after the elapse of a standard period of time. During this latency interval, the novelty cell integrates EPSP from the novelty test cell. An inhibitory neuron ($-$), which serves to reset the novelty cell, receives its input from the axons of all class

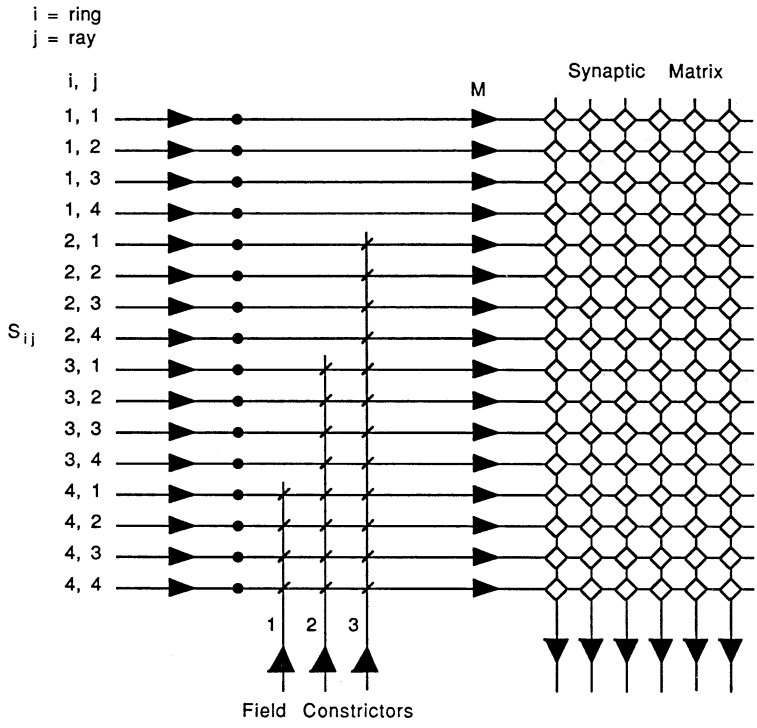

Figure 5.4
Mechanism for constricting effective visual field. Discharge of constrictor cell 1 blocks input from ring 4 (outer ring). Discharge of constrictor 3 blocks input from rings 4, 3, and 2, restricting input to ring 1, the innermost ring of receptors and afferents.

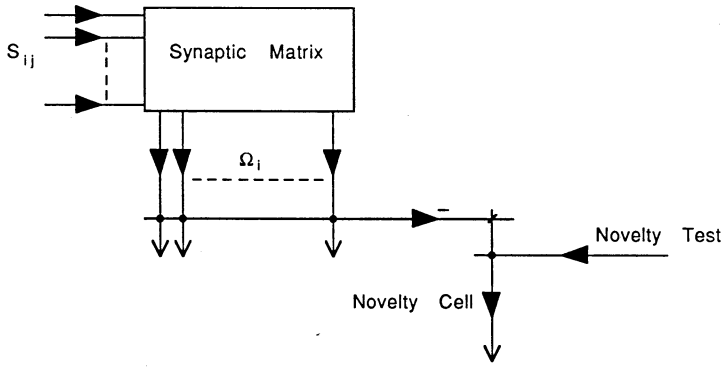

Figure 5.5
Schematic of novelty detection circuit.

cells (Ω) in the synaptic matrix. If a stimulus has been recognized (a class cell discharges) before the elapse of the criterion period, the novelty cell will be reset before it reaches spike threshold, and the stimulus will be treated as a familiar one. If an input pattern has not been learned, no class cell will fire within the criterion period, and the novelty cell (not having received a resetting pulse) will reach threshold and fire, signaling that the current stimulus is novel (Trehub 1977). In this case, it is assumed that the firing of the novelty cell commands an increment of diffuse excitation to the synaptic matrix, thus lowering the thresholds of filter cells (f) so that a previously unmodified f will fire and become tuned to the novel stimulus in accordance with the learning formula.

Transformations of Size and Angular Orientation

An object with a particular size and angular orientation at the time it is learned can usually be recognized later as the same object despite wide variations in its projected retinal size and orientation. The neuronal mechanisms described below provide a means for internally reorganizing the retinotopic projections of visual patterns that undergo such transformations.

Shown in figure 5.6 is a network in which the mosaic cells of a synaptic matrix project retinotopically through two series-connected intermatrix neuronal layers onto a second detection matrix. The dendrite of each cell in the first intermatrix layer receives an excitatory input from each of two decoupler cells, one activated by an initiate zoom command the other by an initiate rotation command. The axon of each cell in the first layer projects in excitatory synapse to its coordinate cell in the second layer and also sends off two excitatory collaterals—one that contacts its retinotopically coordinate cell in the size transformer mechanism and the other that contacts its coordinate cell in the rotation transformer. The dendrite of each cell in the second layer receives, in addition, excitatory inputs from its coordinate cells in the size transformer and the rotation transformer. Finally, the axon of each second layer cell projects to form an adaptive synapse with all filter cells in the second detection matrix.

Size Transformer

The spatial transformation mechanism located above the synaptic matrix in figure 5.6 operates to expand (or contract) an initial activation pattern represented retinotopically on the mosaic cell array. Inputs from the intermatrix cells that represent the initial pattern do not

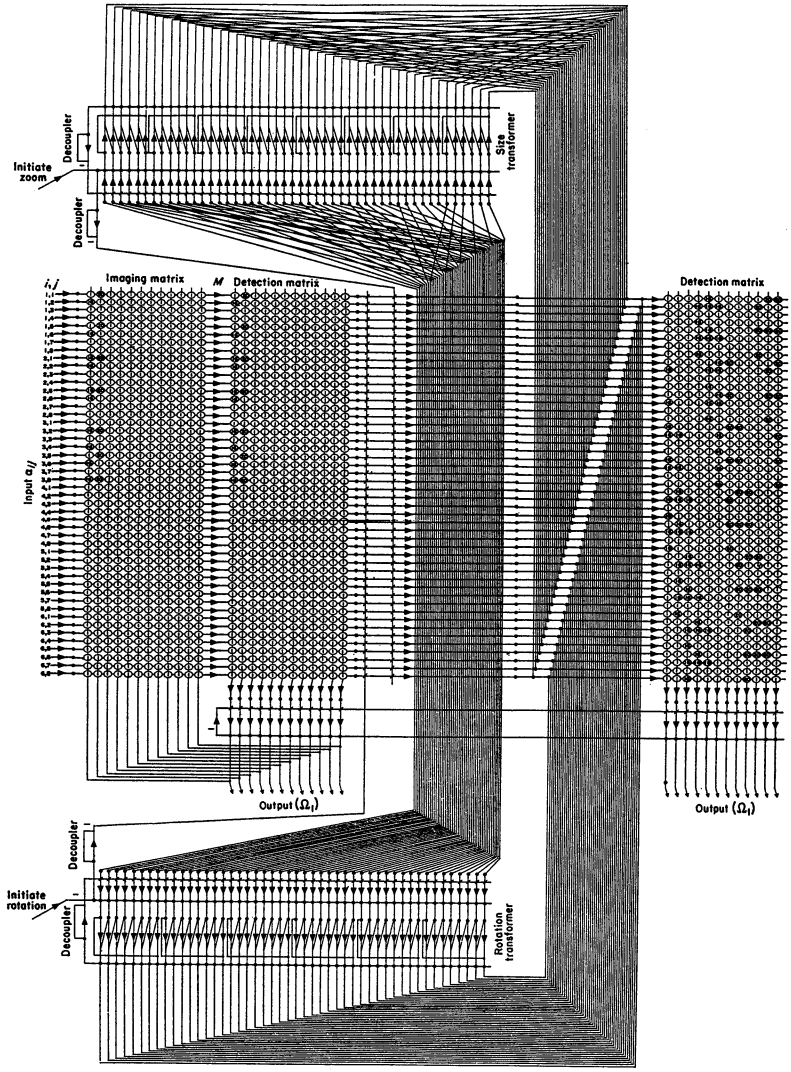

Figure 5.6
Synaptic matrices and neuronal circuits for transformation of size and angular orientation of input patterns. Values in detection matrix 1 represent synaptic transfer weights (ϕ) associated with the veridical input of two learned patterns (T and Z). Values in detection matrix 2 represent ϕ associated with all possible retinal sizes of the two patterns. Source: Trehub 1977. Copyright Academic Press (London) Ltd. Reproduced by permission.

activate the size transformer until the thresholds of neurons in the transformer are lowered by a concurrent pulse from the initiate zoom cell. When this occurs, the pattern of discharge that represents the current retinal image is evoked within the size transformer, and other input to the transformer is blocked by the inhibitory action of two decoupler cells. One decoupler line, activated immediately by input from the initiate zoom cell, inhibits the first array of intermatrix neurons. The other decoupler, activated by the output of any cell(s) in the size transformer, inhibits the array of interneurons that link the size transformer to the axons that have provided its initial pattern of excitation (the image to be transformed). This initial pattern may be considered a prototypical image, which can then be neuronally re-represented for learning or recognition at all the size variations that are within the capacity of the size transformer.

The action of the size transformer can be considered similar to that of a zoom lens. Given a retina with an output of 48 ganglion cells (as in figure 5.6) organized into six rings and eight rays, the size transformer has a corresponding functional organization. When an initial input pattern is presented and the size transformer is activated, the transformer sends this pattern to the second array of intermatrix cells and then to the second detection matrix. However, when any cell on a given transformer ring is fired, it in turn fires the cell in the next-larger ring on its own ray (see figure 5.6). Reversal of the direction of ring-to-ring excitation in an otherwise similar-size transformer produces successive contraction in the projected size of any mosaic cell pattern. Thus, the original pattern is successively enlarged or contracted by the appropriate retinotopic increment in a succession of projections to the second detection matrix, where it can be learned and subsequently recognized over a wide range of sizes (see Larsen and Bundesen 1978).

Rotation Transformer

The spatial transformation mechanism located below the synaptic matrix in figure 5.6 operates to rotate an initial pattern of excitation represented retinotopically on the mosaic cell array. As in the case of the size transformer, inputs from the intermatrix neurons that carry a given pattern of stimulation do not activate the rotation transformer until the thresholds of neurons in the transformer array are lowered by a concurrent pulse from an enabling command cell. In this case, the activating pulse is given by the initiate rotation cell. When such a pulse occurs, thresholds are lowered, and the pattern of discharge representing the current retinal image is evoked in the

rotation transformer. At the same time, additional input to the transformer is blocked by the inhibitory output of two decoupler cells (as in the case of the size transformer). The pattern "captured" in the rotation transformer can then be neuronally re-represented for learning or recognition at all rotation variations within the capacity of the transformer.

When an initial input pattern is presented and the rotation transformer is activated, the transformer projects this pattern of retinotopic excitation to the second array of intermatrix cells and then to the second detection matrix. However, when any cell on a given transformer ray is fired, it in turn fires the cell that is in the next clockwise ray on its own ring (see figure 5.6). Reversal of the direction of ray-to-ray excitation in an otherwise similar rotation transformer produces successive counterclockwise rotation in the projection of any mosaic cell pattern. Thus, the original pattern is rotated in a clockwise or counterclockwise direction in a succession of projections to the second detection matrix, where it can be learned and recognized over a wide range of angular orientations (see Shepard and Cooper 1982).

The neuronal transformer circuits can accept a single visual pattern and re-afferent the same pattern through the full range of size and orientation transformations that are consistent with the structural constraints and resolution of the attached retina and transformer mechanisms. Since the transforms are projected to a synaptic matrix, each can be stored in memory by the filter cells in accordance with the learning formula (equation 2.3). Thus, the presence of a single stimulus can result in the learning of not only the given stimulus but also that stimulus in a variety of size and orientation manifestations. In order to avoid interference between transformer outputs, it is assumed that the size and rotation transformers are connected in reciprocal inhibition and that only one kind of transformation is performed at any given instant.

The ability to recognize an object despite changes in size and orientation does not require filter cells to be tuned to all possible combinations of size and orientation. It is assumed that filter cells are normally tuned to a range of size transformations of any given object so that on any fortuitous encounter, the object can be quickly recognized despite variations in retinal size. However, where the trade-off of recognition time for storage space in the brain is a reasonable one, filter cells might be tuned to only a few orientation variants. If many retinal sizes of an object are represented in the detection matrix, any veridical orientation later encountered can be rotated internally until its re-afferented orientation conforms to that of its appropriate size

representation in the detection matrix. Moreover, given the spatial gradients of ϕ distributions on filter cells (see figure 5.2), exact matches of size and orientation are not required for adequate recognition performance.

Projections from the 3-D Retinoid: Size Constancy

The primate visual system has a large number of separate structures, cortical and subcortical, devoted to the processing of visual information (Livingstone and Hubel 1988). Some nonhuman species have at least 10 to 15 identifiable cortical areas that are responsive to visual stimulation (Van Essen 1985), and it is likely that the human brain has even more anatomically discrete structures, operating in parallel or hierarchical fashion, that represent and process information from the visual world. This abundance of visual processing centers suggests that a single complex stimulus is normally analyzed and represented in the brain in a number of different ways. The capacity of the retinoid model to represent and process concurrently multiple aspects of a current visual stimulus is consistent with these findings.

The perceived constancy in the size of an object over a range of observer-object distance despite large variations in the retinal size of the object (Graham 1951) does not seem to depend on a process of iterative size adjustment like that performed by the size transformer. As an object moves from a position close to an observer to a more distant position, its projected image on the retina becomes progressively smaller as its visual angle decreases (see figure 5.7 and top of figure 5.8). Within the 3-D retinoid as well, the size of the autaptic cell representation of the object decreases accordingly (figure 5.7), yet it is judged to be of constant size. The scheme of axonal projections from principal cells in the 3-D retinoid to a mosaic cell array (bottom of figure 5.8) provides a physical basis for the perception of size constancy. This model assumes that in the central receptive field, there is a structure of 3-D retinoid cell to 2-D mosaic cell connectivity that, in effect, normalizes (roughly) the projected size of an excitation pattern in the 3-D retinoid as a function of the particular Z-plane that the pattern occupies. Principal cells in the nearest 3-D Z-plane are mapped to their corresponding retinotopic coordinates in the mosaic cell array, while cells in more distant Z-planes diverge to project to increasingly more eccentric coordinates on the mosaic cell surface in accordance with the visual distance they represent. Thus, retinal images that become smaller as a function of increasing object distance are magnified in compensatory fashion as they are mapped onto the space represented by the mosaic cell array. (However, the actual

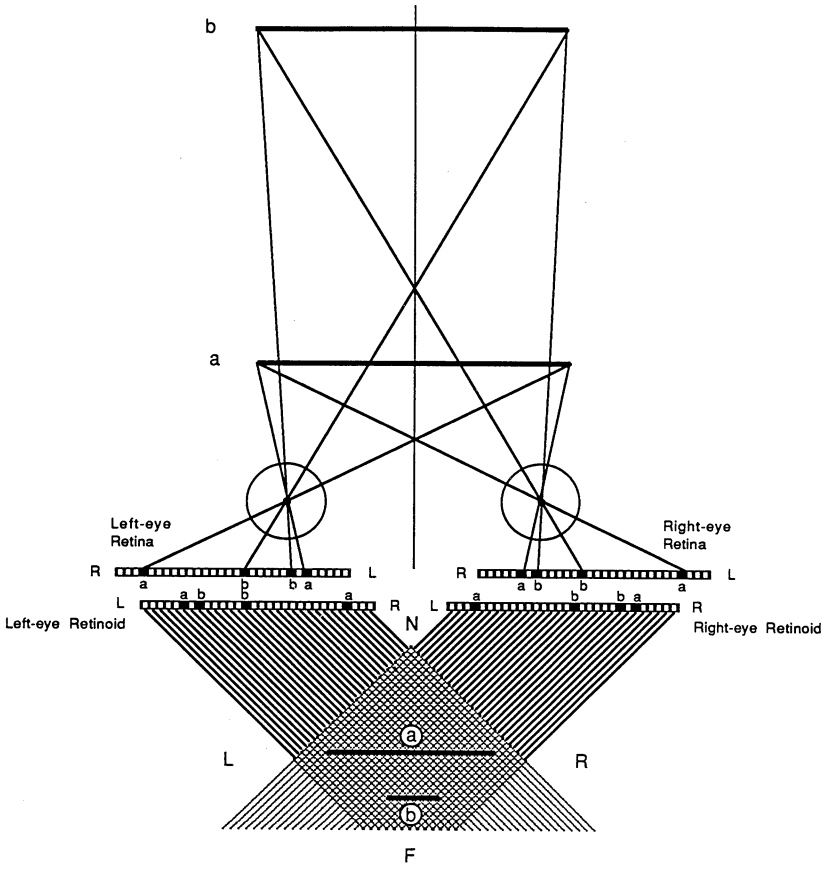

Figure 5.7
Representation in 3-D retinoid of same object at different distances (a and b) from observer.

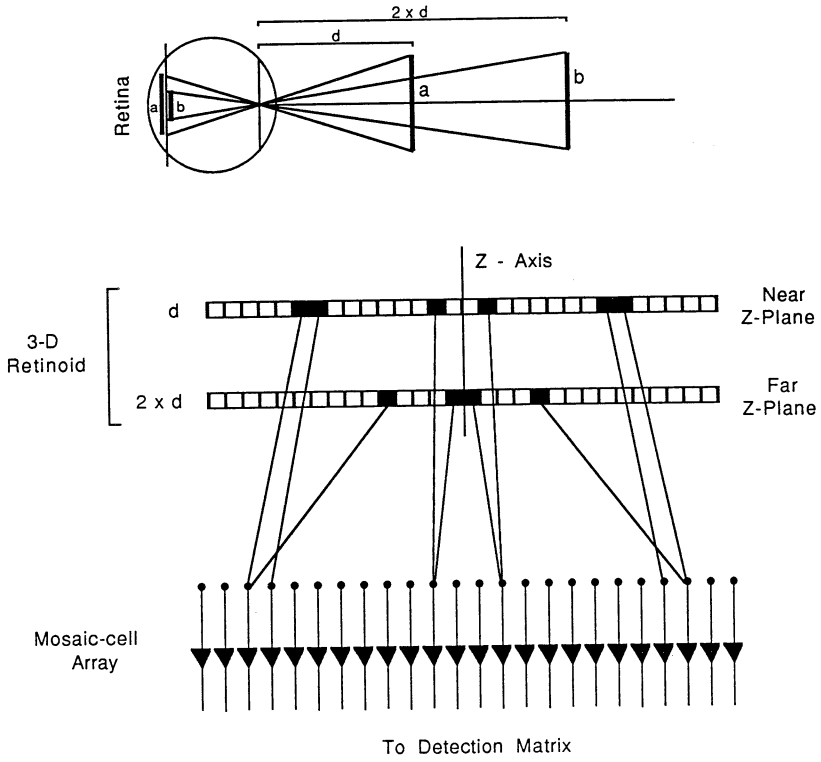

Figure 5.8
Top: Retinal projections of same object at distance d (a) and 2 × d (b). *Bottom:* Normalization of object-size representations on near Z-plane (d) and far Z-plane (2 × d) by compensatory projection of autaptic-cell axons to mosaic cell array.

number of cells activated will always decrease in proportion to object distance, with a corresponding decrease in visual resolution.) This is another instance of the visual system's multiple representation of the properties of objects in the visual world; the relative retinal size of an object is represented in the 3-D retinoid at the same time that its relative inherent size is represented on the mosaic cell array.

This size normalization structure serves a singularly useful purpose for pattern learning and recognition. Since the pattern of excitation on the mosaic cell array will be roughly equivalent for any given object over a wide range of arbitrary distances (although degraded by lower resolution as distance increases), a single filter cell that is tuned to a particular object at a given distance will be an effective detector of that object at other distances. Thus an object need not be learned separately for each distance.

Given the proposed neuronal structure of projection from the 3-D retinoid to the mosaic cells of the synaptic matrix, it is revealing to consider what would happen if the retinal projection of an object to the 3-D retinoid were to remain constant in size as the retinoid representation of the object moved to more distant Z-planes. It would be expected that because of the expanding projection from more distant Z-planes to the mosaic array, the object should be perceived as increasing in size as its distance increased. An anomalous perception of this kind would lend support to the neuronal architecture of the 3-D retinoid and its projections to the mosaic array that I have hypothesized.

To verify that this size illusion does indeed occur, cut out a 1-inch square of black construction paper, and paste it on a sheet of unglossed white posterboard. At normal reading distance, fixate the center of the black square for approximately 1 minute under bright uniform illumination. This will induce a bright after-image of the square on your retinas. If you now shift your gaze to a different region of the posterboard, you will see a square image that is brighter than the surrounding surface. Move the posterboard farther away, and the square will appear to grow larger. Move the posterboard closer than the original fixation distance, and the square will appear to become smaller in size. You can change the perceived shape of the square by tilting the surface of the posterboard after the retinal after-image has formed. If you tilt the fixation surface (the posterboard) away from you on a horizontal axis, the square after-image appears to assume a roughly rectangular shape elongated on the vertical axis. If you tilt it on a vertical axis, you will see a rectangle with horizontal elongation. If the after-image is sharp enough, careful observation reveals that the shape transformations are not quite rectangular; an edge that appears on the near surface of the tilted posterboard is somewhat shorter than its opposite edge on the far surface.

These visual illusions are predictable from the properties of the retinoid system and its projections to the mosaic cell array. It is important to note that the retinal after-image remains constant in size and shape during all the manipulations of fixation. What, then, accounts for the striking changes in the perception of a constant stimulus? The answer lies in the fact that as the fixation surface (the posterboard) is viewed at different distances, the concurrent retinal after-image is necessarily shifted at the same time to the Z-plane that corresponds to the distance of the fixation surface. Thus, with a retinal image of constant size (the after-image), the 3-D retinoid to mosaic array architecture produces an inverse of the size-constancy effect. When the posterboard is slanted, successive slices of the square

after-image are shifted to successively distant Z-planes and magnified anisotropically in the direction of slant away from you. Thus, the true retinal image of a square is perceived as a rectangle.

Clock and Sequential Priming Circuit

For many kinds of cognitive operations, the temporal relationships among events plays a crucial role; a substantial part of human learning and memory is episodic in character. Sequences of experience are related and made meaningful by their temporal contiguity within particular time frames (Tulving 1972). The common perception of causal relationships in everyday affairs seems to depend, to a large extent, on the frequency with which distinct events occur in close temporal sequence. In the case of planning a course of action, the order in which the elements of the plan are put into effect is an essential factor. To meet the demands for a timing mechanism in such circumstances, the circuit described here (figure 5.9) can control the timing, registration, and location of episodic learning in a synaptic matrix, as well as the relative temporal locus and sequence of recalled episodic experience (Trehub 1983). It can also serve as a controller for temporal indexing in neuronal programs for behavioral routines.

The mechanism illustrated in figure 5.9 depends on the short-term memory properties of autaptic neurons. The whole circuit consists of two rings of autaptic cells with each pair of principal cells within a ring linked by excitatory and inhibitory interneurons.

Consider the inner (clock) ring. For each pair of autaptic cells, we can establish a clockwise direction around the ring. Each autaptic cell innervates an excitatory interneuron, which innervates its clockwise neighbor. Conversely, each autaptic cell also innervates an inhibitory interneuron, which connects to its counterclockwise autaptic neighbor. If any particular principal cell becomes active (discharges), it will transfer its excitation (via the excitatory interneuron) to its clockwise neighbor, which will inhibit (turn off) its counterclockwise donor. In this way, the clock mechanism will be able to maintain a constant circulation of unitary autaptic cell activity in a single direction (clockwise) around the neuronal ring. With fixed integration slopes for EPSP and uniform discharge thresholds for the neurons in the circuit, the rate at which autaptic cell activity circulates over the ring will depend on the level of diffuse excitation (arousal) within the clock module. The higher the level of excitatory bias is, the faster will autaptic cell activity circulate (the clock will run faster), and vice versa. This mechanism provides a neuronal means for temporal ref-

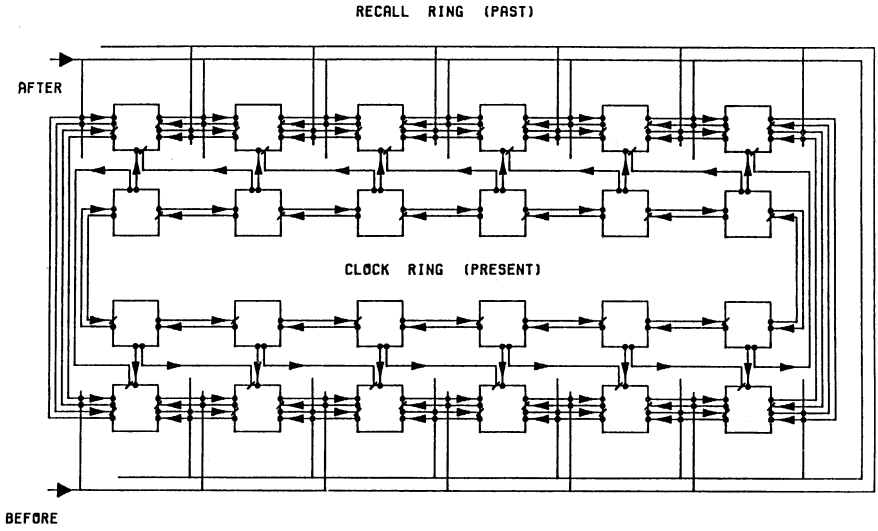

Figure 5.9
Schematic of neuronal clock and temporal priming circuit. Large squares represent autaptic cells that send priming excitatory pulses to synaptic matrix. Small, filled triangles represent control neurons for clock rate and temporal direction (before, after). Inner ring of autaptic cells and interneurons composes clock circuit. Outer ring of autaptic cells and interneurons governs episodic recall. Source: Trehub 1983. Copyright Lawrence Erlbaum Associates, Inc. Reproduced by permission.

erencing and control. If, for example, a particular active autaptic cell should gate the current environmental input in episodic learning, the density of episodically learned experience would be directly proportional to the neuronal clock rate.

The outer ring in figure 5.9, designated the recall ring, provides a means for selective priming or gating of neurons for recalling the images of past experience from memory. In this ring, each autaptic cell receives an excitatory input from a paired clock cell at its corresponding sequential position and an inhibitory input from the clock cell in the next clockwise position. In the absence of any other input, this causes the principal cells in the outer ring to fire in synchrony with the inner neuronal clock. In addition, each pair of autaptic cells in the recall ring is linked by a counterbalanced set of excitatory and inhibitory interneurons. If we take any particular autaptic cell in the outer ring as a spatial and temporal reference, a pulse from the command cell marked BEFORE will bias its local interneurons, causing the transfer of its activity to its counterclockwise autaptic neighbor (going back in time in the sense of an earlier autaptic state). A pulse from

the command cell marked AFTER will transfer autaptic cell activity to its clockwise neighbor (going forward in time in the sense of a later autaptic state). The relative direction, rate, and distance of a temporal excursion will be determined by which command cell (BEFORE or AFTER) is discharged, the discharge frequency of the command cell, and the duration of its discharge. (Notice the similarity in the control principles governing operation of this timing module and those for translation of patterns over retinoid surfaces.)

A Network for Episodic Processing

Figure 5.10 illustrates how the clock ring and recall ring are connected to the synaptic matrix so that the system can learn and recall past experiences. The discussion of the basic synaptic matrix in chapter 3 was restricted to a mechanism in which the changes in synaptic transfer weights (ϕ) during the course of learning would be long-lasting; the decay of ϕ over time was not considered as an issue in the initial description of the model. However, synaptic matrices that are involved in episodic processing will have identical filter cells influenced by different afferent patterns on different episodic cycles; consequently, in any given filter cell, the changes in ϕ that occur during learning must decay to approach an initial state before that cell is involved in a new learning cycle. Otherwise the ϕ distributions representing the events learned would be confounded over successive learning cycles.

Each autaptic cell in the clock ring (PRESENT) in figure 5.10 sends an excitatory gating axon to a paired filter cell (f) in the detection matrix. At any given time, only that filter cell that is primed by the output of the neuronal clock can learn the current sensory input. By this scheme, learning (synaptic modification) is transferred over time, sequentially and unidirectionally across the filter cells of the detection matrix, and the sequence of f cell priming is recycled as autaptic cell activity in the clock ring recycles. For any given filter cell, the changes in ϕ due to learning must decay to a base value within an appropriate time period in order to avoid a confounding of learned patterns.

Each autaptic cell in the recall ring (PAST) sends an excitatory axon to its paired class cell (Ω) in the detection matrix. Thus, sequences of Ω discharge can be initiated and synchronously controlled by activity in the recall ring. Because the discharge of any given Ω will evoke its associated (learned) afferent pattern in the imaging matrix, sequences of Ω discharge will recall sequences of learned experiences (images) in their original temporal order, going forward or backward in time

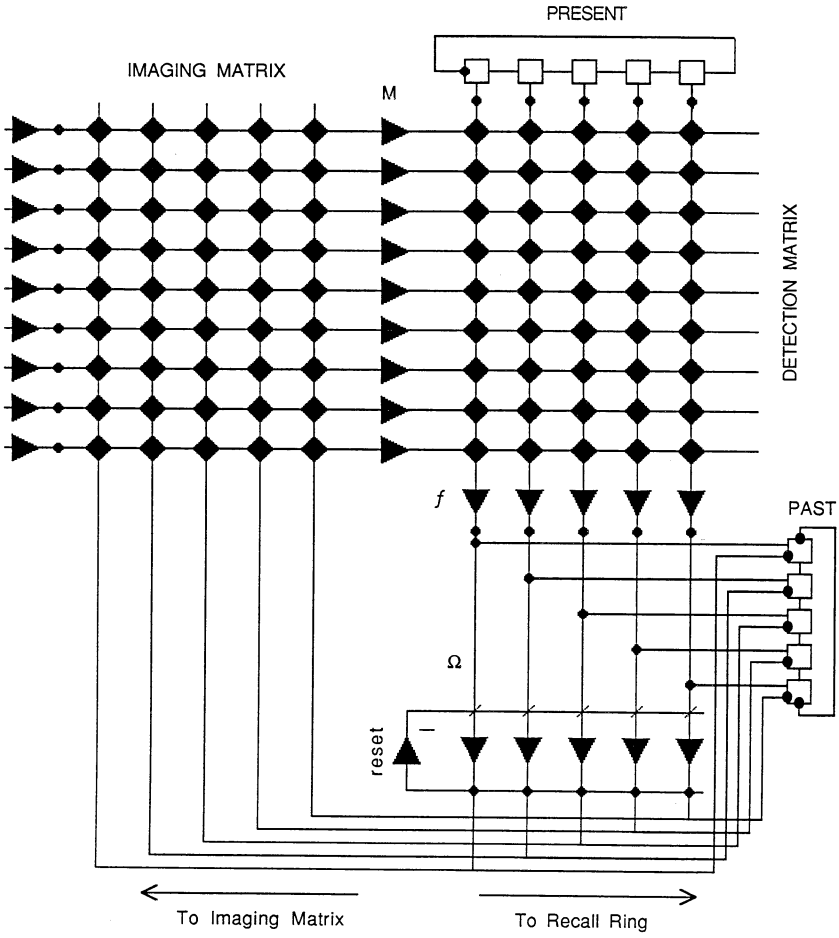

Figure 5.10
Schematic showing synaptic matrix for episodic learning and recall controlled by neuronal mechanism illustrated in figure 5.9. Squares at top represent autaptic cells in clock ring (PRESENT). Squares at bottom right represent autaptic cells governing episodic recall (PAST). Each autaptic cell in the clock ring primes an associated filter cell (f) in the detection matrix. Each autaptic cell in the recall ring can discharge a class cell (Ω), which in turn evokes its related learned afferent pattern in the imaging matrix. The sequence of discharge in the clock ring is unidirectional (forward) in time. The sequence of discharge in the recall ring can vary in direction (forward or backward in "time"). Source: Ibid. Copyright Lawrence Erlbaum Associates, Inc. Reproduced by permission.

from any arbitrary past reference image in accordance with the controlling activity of the recall ring.

If the model were to operate only as described to this point, the initial temporal excursion for the recall of any particular past episode would have to recede sequentially under the control of the autaptic cells in the recall ring starting at the temporal locus that is determined by the currently active principal cell in the neuronal clock (PRESENT). However, a leap back to the beginning of a remembered episode rarely involves a complete sequential playback of intervening images in reverse temporal order from present to past. Rather, a target event is retrieved first, and related episodic recollections are referred to earlier or later times with respect to the temporal locus of the target event. The proposed model can also perform in this way if the clock ring (PRESENT) is momentarily decoupled from the detection matrix while a concurrent increase in diffuse excitation is applied to all filter cells in the synaptic matrix. Under this condition, any input pattern from the mosaic cell array (M), whether exogenously or endogenously evoked, will maximally stimulate that filter cell having the highest sum of synaptic products with the given pattern. Its coupled class cell (Ω) will fire first, and this Ω, through its axon collateral to its paired autaptic cell in the recall ring (figure 5.10), will trigger episodic excursions from this point in the ring. Thus, sequential playback through intervening images from the present to a past target episode is prevented, and recall can begin at a target point in "time" that depends only on the distribution of synaptic transfer weights within the synaptic matrix and the concurrent pattern of evocative excitation arriving from the mosaic cell array.