

Chapter 15

Other Experimental and Clinical Evidence

This chapter presents additional findings from neurophysiological, psychological, and clinical studies that support the biological plausibility and explanatory competence of the model's principal component mechanisms.

Synaptic Plasticity

In a technically elegant study, the factors responsible for the associative properties of long-term potentiation (LTP) were investigated directly in a hippocampal-slice preparation (Gustaffson et al. 1987). The EPSPs of individual cells resulting from single-volley, low-frequency stimulation of their afferent axons were recorded by an intracellular electrode. The preparation also allowed depolarizing current pulses to be injected through the recording microelectrode in the postsynaptic cell. It was demonstrated that:

1. EPSPs became potentiated when they were paired with depolarizing pulses of sufficient magnitude.
2. The potentiation (LTP) generally reached a peak after 20 to 30 single presynaptic volleys were paired with depolarizing pulses in the postsynaptic cell.
3. LTP was specific to the paired inputs (the coactive synaptic junction).
4. For LTP to occur, the EPSP had to be induced together with the postsynaptic depolarizing pulse or precede it by less than ~100 milliseconds. There was no potentiation when the EPSP immediately succeeded the postsynaptic pulse.
5. LTP was blocked by the application of the NMDA-receptor antagonist 2-amino-5-phosphonovalerate (APV).
6. If an EPSP occurs together with a large postsynaptic depolarization, not much additional LTP can be induced by subsequent coactivation at the modified synapse.

These relatively recent findings extend and clarify earlier notions about LTP, and they provide support for a number of assumptions I made in my proposed model of neuronal plasticity. First, synaptic enhancement is spatially restricted on the postsynaptic dendrite. Second, a long-term increase in synaptic weight (learning) occurs only if there is coactivation of the pre- and postsynaptic cells or if postsynaptic activation follows the EPSP by less than ~ 100 milliseconds. Finally, the efficacy of an adaptive synapse can approach its maximum value (saturation) during a single learning exposure if the activation (depolarization) of the postsynaptic cell is high enough. In the synaptic matrix model, the excitatory bias of arousal is a necessary condition for learning and serves to ensure a high level of postsynaptic activation of filter cells (f) and mosaic cells (M) when stimulus-evoked EPSPs occur. Thus, my theoretical assumptions about the selective locus of synaptic modification, the necessity for the interaction of ATF and DTF within the postsynaptic dendrite, one-trial learning during high arousal, and synaptic saturation seem to be confirmed by microelectrode measurements of LTP processes in single hippocampal neurons.

In an effort to determine whether the effective process underlying LTP is presynaptic or postsynaptic, Kauer, Malenka, and Nicoll (1988) studied synaptic transmission in the CA1 region of the hippocampus. They found that presynaptically released glutamate activates both NMDA and non-NMDA postsynaptic receptors on pyramidal cells, which produces an EPSP with two distinct components. When LTP is induced, there is a selective increase in the non-NMDA component of the EPSP. Thus, it appears that while activation of NMDA receptors is necessary to initiate LTP, the persistence of LTP is caused specifically by a long-lasting increase in the sensitivity of non-NMDA receptors in the postsynaptic neuron (dendrite) to glutamate that is released at spike-activated presynaptic axonal terminals. This finding provides additional physiological support for the proposed learning mechanism essential for the adaptive competence of the synaptic matrix.

A number of the cognitive processes that I have described have required adaptive synaptic coupling between different more or less modular neuronal mechanisms. This is true, for example, in the feed-forward and feed-backward adaptive chaining of class cell tokens (figure 3.6), the linking of motivation to the detection matrix for pattern recognition (figure 9.6), and the cross-coupling between the semantic network and the matrix for episodic learning (figure 13.1). A recent series of studies (Laroche, Jay, and Thierry 1989) provides direct evidence of this kind of adaptive synaptic coupling between

anatomically distinct systems in the neocortex of the rat. Single unit recordings were obtained from 120 neurons in the prelimbic region of the prefrontal cortex. Of these, 42 percent exhibited excitatory responses to single-pulse stimulation of the hippocampal CA1-subicular region, indicating an excitatory pathway between hippocampal cells and these prefrontal neurons. Stimulation of the hippocampus with a high-frequency pulse train (tetanic stimulation) resulted in a significant and persistent potentiation of evoked response (LTP) and a reduction of onset latency in the recorded cells of the prefrontal cortex.

The Synaptic Matrix-Retinoid System

Physiological Evidence

A central architectural feature of the synaptic matrix is the abundance of dynamic cellular junctions created by the intersection of bundles of axons (presynaptic lines) with a large population of orthogonally oriented dendrites (postsynaptic lines). Such an anatomical arrangement requires relatively long cellular processes to accommodate multiple inputs and outputs. The neuronal architectures of the hippocampus and the cerebellum provide clear examples of long dendrites that are transversed and synaptically innervated by long axonal projections. Dendrites range from 500 to 1000 microns in length on hippocampal pyramidal cells and are ~400 microns long on cerebellar Purkinje cells. Apical dendrites of pyramidal cells in the sensory regions of the neocortex range in length from 200 to 1600 microns (Shepherd 1979).

In a recent study of the striate cortex of the macaque monkey, Blasdel, Lund, and Fitzpatrick (1985) established by darkfield microphotography that axonal projections intrinsic to this visual region can extend laterally (parallel to the cortical surface) at least 5000 microns in length. Pyramidal cells in the cortex are tightly packed and oriented with their dendritic shafts orthogonal to the cortical surface. Because of this kind of crossed orientation of axons and dendrites, a single 5000-micron axon can contact the dendrites of many thousands of cells in its lateral passage. Thus, it appears that a basic structural requirement of the synaptic matrix is met in the neuronal architecture of the neocortex.

In a systematic survey employing both simple and complex visual stimuli, the responses of many individual neurons in the inferior temporal (IT) cortex of the macaque were recorded and analyzed (Desimone et al. 1984). A subpopulation of cells was discovered in

this brain area that responded selectively to faces. The magnitude of response in these cells was dependent on the configuration of specific facial features. The selectivity of response was reported to be maintained over a wide range of stimulus sizes, angular orientations, and location in the receptive field. These findings demonstrate that there are individual neurons in the visual system selectively tuned to the holistic patterns of complex stimuli and that serve as detectors of objects characterized by such patterns. Recall that the synaptic matrix model assumes the visual detection mechanism can be thought of as a comb filter within which individual filter cells are selectively tuned to the complex stimulus patterns of significant objects. In addition, pattern recognition in the synaptic matrix occurs after the centroid of an object's representation on a retinoid has been shifted to the normal foveal axis, differential response in tuned filter cells appears to be rather robust over pattern rotation, and the model includes explicated neuronal mechanisms for normalizing pattern size before input to the detection matrix. Thus, the selective responsivity of a filter cell can be effectively maintained despite changes in the position, size, or rotation of an object in the visual field. These properties of the putative synaptic matrix-retinoid system are consistent with the response characteristics of individual neurons in the inferior temporal cortex reported by Desimone and his co-workers.

Damage to the hippocampus or fornix is known to impair the performance of a conditional spatial response task in both monkey and humans. A task of this kind was used together with direct microelectrode recording to investigate the response properties of single neurons in the hippocampus of rhesus monkeys (Miyashita et al. 1989). The animal learned to make a particular spatial (motor) response when one visual stimulus was presented on a video monitor and a different response when a different stimulus was shown. Of 905 single hippocampal cells studied, 14 percent fired differentially to one or the other of the visual-stimulus spatial response associations. Responsive neurons were found throughout the hippocampal structure but were particularly concentrated in the subicular complex and in area CA3. The investigators concluded that single hippocampal neurons respond selectively to combinations of visual stimuli and the specific motor responses with which they must become associated. This finding gives strong support to the proposed model for selective association of an arbitrary sensory input and a discrete motor routine gating output through the adaptive interface of a synaptic matrix.

Motter and Mountcastle (1981) conducted an extensive single-unit study of light-sensitive (LS) neurons in the inferior parietal lobule of

the monkey. They found that the LS cells are sensitive to stimulus movement and the direction of movement over a wide range of velocities. Response was specific to motion that was directed either inward toward the center of the visual field or outward toward the perimeter of the field. In cells with bilateral response fields, motion vectors commonly pointed in opposite directions in the two visual hemifields.

On the basis of their findings, Motter and Mountcastle suggested that LS neurons in the inferior parietal area of the brain "contribute to a continual update of a central neural image of the spatial frame of the immediate behavioral surround and to the perceptual constancy of that space that obtains during body movement." They concluded, "The light-sensitive neurons possess properties suitable for the attraction of gaze and attention toward objects and events in peripheral visual fields." It is clear that the response properties of these parietal neurons meet the dynamic requirements for an environmentally linked input to the shift control mechanism of the retinoid system that will translate images and move the focus of attention (self-locus excursion) within an egocentric frame.

The spatial memory functions of neurons in the dorsolateral prefrontal cortex were investigated in an oculomotor delayed-response task (Funahashi, Bruce, and Goldman-Rakic 1989). Rhesus monkeys were trained to fixate a central target during the presentation of a brief (0.5 second) peripheral visual cue and through a subsequent delay period of 1 to 6 seconds, which was terminated by the extinction of the fixation target, and then to make a saccade to the remembered location of the peripheral cue. Recordings of the activity in single cells within the prefrontal cortex were obtained during the performance of the task. A large proportion of the neurons within and around the principal sulcus and in the frontal eye fields exhibited significant changes in activity during the delay period between the offset of the cue and the visual saccade to its remembered location. Of these cells, most were directionally selective; they exhibited a significant response only following cues in localized regions of the visual field. The investigators concluded that:

1. Prefrontal neurons possess information about the location of visual cues.
2. Information provided by these neurons "appears to be in a labeled line code: different neurons code different cue locations and the same neuron repeatedly codes the same location."
3. These cells exhibit mnemonic activity over a 1- to 6-second interval in the absence of any overt stimuli or movements and

the activity ceases upon the execution of the required behavioral response.

4. "These results strengthen the evidence that the dorsolateral prefrontal cortex participates in the process of working or transient memory and further indicate that this area of the cortex contains a complete 'memory map' of visual space."

The hypothesized neuronal mechanisms of the retinoid system provide just this kind of representation of visual space in short-term memory. Moreover, the retinoid's spatial map is organized in egocentric coordinates—a necessary property for effective visuomotor behavior. In addition to supporting the putative retinoid system, the short-term mnemonic activity of labeled-line cells that has been reported by Funahashi and co-workers lends physiological credence to the other postulated neuronal circuits in the cognitive brain that are organized around short-term memory registers (see, for example, figures 5.9, 8.1, and 8.3).

Excursion of the self-locus over autaptic cells in the retinoid system is assumed to be the biophysical substrate of directed attention over egocentric visual space. Translation of the self-locus to a target region of interest serves (among other functions) to increase the sensitivity of neuronal response to stimuli in the attended region and to direct visual saccades to the target. Andersen and Mountcastle (1983) reported experimental results that link directed visual attention and angle of gaze to the excitability of light-sensitive neurons in the posterior parietal cortex of the macaque monkey. Following up on this work, Andersen, Essick, and Siegel (1985) conducted additional studies of the response properties of neurons in the inferior parietal lobule of rhesus monkey. Their results provide physiological support for the self-locus mechanism of selective attention in the retinoid model of visual representation. With the animal's head restrained and a fixation light straight ahead (coordinates 0,0 on a tangent screen), they first established the retinotopic receptive fields of single cells by briefly flashing a second light at various locations on the tangent screen. After this mapping was completed, the animal fixated at different locations on the screen while the same stimulus that was used for mapping was presented at the center of the receptive field of each cell. It was found that the visual sensitivity of the retinotopic receptive fields changed systematically with the angle of gaze. There was an eye-position-dependent tuning for stimulus location in a head-centered (egocentric) coordinate space. In the retinoid model, the angle of gaze is normally systematically related to the target coordinates of a self-locus excursion, and simple reversal of these coordi-

nates translates an eccentric target to the normal foveal axis. Since retinoid cells around the spatial coordinates of the self-locus automatically receive biasing excitation, the observed selective enhancement of neuronal response as a function of the direction of gaze in a head-centered coordinate space would be expected on the basis of the model.

I have hypothesized that heuristic excursions of the self-locus over retinoid space account for what is commonly called the searchlight of attention. Under certain task demands, the normal coupling of visual fixation to the locus of attention can be interrupted so that the coordinates of fixation and those of the self-locus are dissociated. If the retinoid model of selective attention is correct, it should be possible selectively to facilitate the neuronal response to visual stimuli at targets of attention in arbitrary regions of visual space even while eye position is held constant. A study by Spitzer, Desimone, and Moran (1988) provides direct evidence in support of the retinoid model. Rhesus monkeys were trained on a visual discrimination task with two levels of difficulty. The monkeys maintained fixation on a central target while pairs of visual stimuli (small colored bars) were presented in succession within the receptive fields of single neurons in area V4 of the extrastriate cortex. A sample stimulus appeared for 200 milliseconds, and 400 to 600 milliseconds later a test stimulus was presented at the same location. The animal was required to make one response if the test stimulus was an exact match to the sample and a different response if the test stimulus differed from the sample. In the "easy" discrimination trials, nonmatching test stimuli differed from the samples by either 90 degrees in orientation or 77 nanometers in wavelength. In "difficult" trials, nonmatching test stimuli differed from the samples by 22 degrees in orientation or 19 nanometers in wavelength. An analysis found that the responses of just those neurons mapped to the location of the test stimuli were selectively more vigorous in the difficult task, while at the same time, the monkey's ability to make difficult discriminations improved. The investigators concluded that "increasing the amount of attention directed toward a stimulus can enhance the responsiveness and selectivity of the neurons that process it."

The conditions of this experiment and the conclusions drawn from it support the view that excursions of the self-locus, in the absence of corresponding eye movements, can selectively enhance neuronal responsiveness in the retinoid target region of the self-locus (focal attention). Moreover, if the increased arousal induced by a more difficult task were to increase the excitation of the autaptic cell source of the self-locus (home position at the normal foveal axis), then we

would expect a corresponding spatially selective increase in neuronal response in the difficult task (the empirical finding) when the self-locus makes its excursion to the egocentric coordinates of the test stimulus.

Gnadt and Mays (1989) recorded from single neurons in the posterior parietal cortex and superior temporal sulcus of trained rhesus monkeys during task-driven visual-motor behavior. They manipulated the task demands of ocular vergence, accommodation, and binocular disparity and measured vergence and accommodative responses. They found the following kinds of cells:

1. Disparity-sensitive visual tracking cells, related to binocular disparity during visual tracking in depth.
2. Velocity-sensitive motor cells, which reflected the velocity of either ocular vergence or lens accommodation.
3. Visual fixation cells, which modulated their firing rate during changes in conjugate and disjunctive eye position and their response did not depend on the presence of a visual target.
4. Eye-position-dependent visual cells, which were visually responsive cells in which the response was modulated by the conjugate and disconjugate position of the eyes, even though the stimulus fell on the same locations on the two retinae.

These findings demonstrate the existence of cortical neurons with response properties consistent with the assumed properties of shift control cells in the monocular retinoids and in the 3-D retinoid system. The eye-position-dependent visual cells are of particular interest with respect to the putative 3-D retinoid because they provide direct evidence of visually responsive cells tuned in an egocentric three-dimensional coordinate frame of reference.

In the 3-D retinoid model, the egocentric distance of a target is represented by the selective activity of different distance-ordered populations of autaptic cells (Z-planes). For any given distance, the egocentric direction of a target is represented by the selective activity of autaptic cells within the appropriate Z-plane. A recent experiment utilizing chronic microelectrode implants in single cells of the premotor cortex (Karluk and Ebner 1989) supports the hypothesized neuronal architecture of the 3-D retinoid. Rhesus monkeys were trained to move to targets arranged in concentric circles at three distances from a central start box. Analysis of the single-unit responses in relation to target distance and direction revealed that for a given target direction, different populations of cells are recruited for different target distances, and for a given target distance, roughly the same popu-

lation of cells is activated, while the magnitude of cell discharge within the population varies with the direction of the target.

These findings are consistent with the representation of different distances in the 3-D retinoid by the activation of different populations of cells in their respective Z-planes (Z-plane brightening) and with the representation of different directions by the increased activation of selected cells within a Z-plane population.

Psychological Evidence

All of the work reported in this section studied normal human subjects with no known brain lesions or other significant sensory or motor impairment.

In a follow-up to an earlier study concerning the duration and specificity of memory for events occurring during the first year of life (Myers, Clifton, and Clarkson, 1987) two groups of children who had one learning experience at the age of 6.5 months were tested one or two years later (Perris, Myers, and Clifton 1988). During the original learning occasion, each child had reached in the light and in the dark for a sounding toy that was positioned at five different locations in front of the child: midline, 30 and 60 degrees left, and 30 and 60 degrees right. After an interval of one or two years, the children were tested in the dark only for memory of this experience. It was found that both groups of these children were significantly more likely to reach and grasp the sounding toy than age-matched control groups of children without the single early experience. The investigators concluded that, after a single infant experience, children retain memory for that experience at least as long as two years later. These results are consistent with the rapidity and long-term stability of synaptic modification (learning) in my model of the synaptic matrix.

Object recognition by the visual-cognitive mechanisms that I have proposed can be accomplished solely on the basis of the stimulus contours or edges that are extracted from the brightness distribution of the retinal image by center-surround processes at the retina and other low-level visual preprocessors. Biederman and Ju (1988) performed a series of experiments aimed at uncovering the principle determinants of visual recognition. They presented to their subjects slides of common objects to be named or verified against a target name as quickly as possible. Each object was shown as a full-color photograph (all surface properties displayed) and as a simplified line drawing of the object's major components (only contours displayed). Mean reaction times and error rates were found to be virtually identical for the two types of stimuli. Surface features such as color,

brightness, and texture did not contribute to the speed or accuracy of recognition performance. The investigators concluded that "initial access to a mental representation of an object can be modeled as a matching of an edge-based representation of a few simple components." This conclusion is clearly in agreement with the processes of object recognition in the synaptic matrix.

Shepard and Metzler (1971) conducted a landmark experiment that probed an important aspect of the mental transformation of visual objects. Subjects were shown pairs of perspective line drawings of unfamiliar three-dimensional objects at various orientations. Half of the pairs of objects could be rotated so that their shapes would be congruent with each other (objects the same). The other half of the object pairs differed by a reflection as well as rotation and could not be rotated into congruence. Half of the pairs differed by rotation about a vertical axis; the other half differed by rotation in the picture plane. Angular differences between the paired objects ranged from 0 to 180 degrees. At the presentation of each stimulus pair, subjects were required to signal as quickly as possible whether the objects were the same or different. It was found that the time required to recognize two perspective drawings as depicting an object of the same three-dimensional shape was a linearly increasing function of the angular difference between the two depicted objects. Subjects seemed to perform actual mental rotations of analog representations of the objects in order to test for congruence of shape. Shepard and his co-workers explored phenomena of this kind in many different experiments. The findings consistently suggested an internal process that actively transforms spatial analogs of perceived objects within a two- or three-dimensional spatial frame (Shepard and Cooper 1982). These results and other related studies (Koriat and Norman 1988) lend support to the analog nature of visual representation in the retinoid system and in the mosaic array, the 3-D to 2-D axonal projection from the 3-D retinoid system to the mosaic array, and the local mechanisms for stepwise spatial transformation of excitation patterns in the retinoid system and in the rotation transformer.

Imagination is a powerful and extremely important cognitive faculty that distinguishes the human species (Ferguson 1977, Shepard 1978). The putative neuronal circuitry of the synaptic matrix provides an essential mechanism for the basic processes of imagination. Discharge of a class cell token of a previously learned object or scene can evoke a retinotopically organized pattern of afferent excitation that corresponds to the afferent pattern induced at the original perception of the object. This happens because of a selective reorganization of synaptic transfer weights that automatically occurs in the

imaging matrix during learning. Thus, in my proposed model of the cognitive brain, imagination is a neuronal process having the retinotopic spatial coherence (but not normally the intensity or detail) of ordinary vision (Trehub 1977, 1987).

Over the past two decades, the nature of imagery has been a focus of considerable experimental effort and lively theoretical controversy (Paivio 1971, Pinker and Kosslyn 1983, Pylyshyn 1973, Sheikh 1983). Kosslyn (1980) has undertaken a long-term program of research devoted to the study of imagery. He and his research collaborators have contributed an extensive experimental literature that is relevant to the hypothesized imagery mechanisms in my model of the cognitive brain.

A series of experiments by Kosslyn, Ball, and Reiser (1978) demonstrated that as the distance across a visual image increases, more time is required to scan across the image. This was true even when the amount of visual detail between the starting focus on the image and the final target focus was the same. It was found that the relationship between scanning time and the relative distance between target objects is a linear one. Subjectively larger images required more time to scan than similar but subjectively smaller images. These results are precisely what one would expect according to the neuronal model. An image is first evoked on the array of mosaic cells in the imaging matrix, projected to the retinoid system, and then scanned by translating the retinoid image over the afferent aperture to the detection matrix that is centered on the normal foveal axis (Trehub 1977).

The relationship between the subjective size of an imaged object and the imagined distance at which it is "viewed" was explored in a number of experiments (Kosslyn 1978). Subjects were instructed to imagine an object far off in the distance and then to imagine moving toward it until all of the object could not be seen in a single image (overflow). It was found that as subjects "approached" objects, their subjective size increased, and the larger the imaged object was, the farther away it seemed at the point of overflow. Thus, subjects could imagine moving closer to a beaver than to an elephant before experiencing subjective overflow. In terms of the visual-cognitive mechanisms that I have modeled, overflow would occur when the size of an image exceeds the bounds of the afferent aperture. The progressive expansion of the subjective size of the image would result from a neuronal linkage of the self-locus with the size transformer. When an object is first imagined far in the distance, one of its exemplars is evoked in the imaging matrix reduced in size, and projected to a distant Z-plane in the 3-D retinoid. Moving toward an object in imagination corresponds to a self-locus excursion to the Z-plane coordinate

of the imagined object. As the self-locus crosses successive Z-planes in its movement toward the "distant" object, it activates the size transformer at each crossing, causing a corresponding succession of incremental expansions in the size of the imaged exemplar. Since the size of the initial image will vary with the intrinsic size of the object imaged, the number of Z-planes crossed by the self-locus in "approaching" the object before the size transformer causes an image overflow will be inversely proportional to the intrinsic size of the imagined object. Thus, the self-locus can get closer to a beaver than to an elephant before overflow is experienced. Put another way, the larger the imaged object is, the farther away it will seem at the point of overflow.

Another series of experiments (Kosslyn et al. 1983) explored the process of amalgamating image units that are stored in memory into a composite form or scene. The principal findings were that:

1. Mental images can be constructed by assembling separate images of individual parts.
2. An increment of time is required to add each additional part to an image.
3. Verbal description can be used to coordinate parts into an imaged scene.
4. Multipart images are assembled by the inspection of parts already in the image.
5. The time required to integrate an image part into a larger image is not a constant.

All of these results can be explained by normal operation of the neuronal mechanisms detailed in earlier chapters. Parts of images (called image units by Kosslyn et al.) are stored in the distribution of synaptic weights (ϕ) over mosaic cells (M) in the imaging matrix. These image parts are activated one at a time by the discharge of their associated class cells (Ω) in the detection matrix and then projected from the mosaic cells to the retinoid system. Each successive part of a complex image is captured on the autaptic cells of a retinoid layer, translated to its proper spatial coordinate, and projected to the scene assembly retinoid where the complete image is constructed (finding 1 above). For multipart images, iteration of the neuronal operation is required. Thus, it is clear that an increment of time will be needed to integrate each additional part within the larger image (finding 2). Given the connective architecture between the synaptic matrix and the networks for lexical assignment and semantic processing, lexical descriptions can evoke selected image components as well as their relative coordinate locations on the scene assembly reti-

noid surface (finding 3). As a complex image is being constructed, it can be inspected (analyzed) by parallel projections from the retinoid system to the synaptic matrix and to the mechanisms for analyzing object relations (finding 4). Inspection of this kind can signal what parts are missing (to be recalled for image completion) and where they should be located with respect to the parts already assembled. One would expect that the more complex the image is to be assembled, the longer will be the inspection time to determine what parts are still missing, what the next image part should be, and where it should be located. Thus, the time required to integrate an image part into a larger image would not be a constant (finding 5).

The results of Cave and Kosslyn's (1989) study of size-scaling processes in visual selection fit nicely with the specific properties of the neuronal scaling mechanisms that I have assumed to exist in the visual system. Subjects were required to evaluate visual stimuli of varying sizes under conditions designed to influence their expectations about the stimuli. The general finding was that when an upcoming stimulus is expected to be a certain size, response time increases with the disparity between the expected size and the actual size. At any given time, the visual system seems set to process information at a particular scale, which can be adjusted to match the size of an object. Beyond this basic result, however, the data indicated that two different kinds of size adjustment processes are involved in size scaling: a slow type of size scaling that is shape specific and can filter out a visual object from a superimposed distractor (see Trehub 1977, 1987) and a faster type that is not shape specific.

Cave and Kosslyn's conclusion that there are two different kinds of size-scaling processes in the visual system conforms to the two kinds of size transformation mechanisms that I have hypothesized. Recall that in my neuronal model of the visual-cognitive system, the size transformer iteratively expands or contracts a ring-ray representation of an object (Trehub 1977, 1987). This is done by transferring a shape-specific pattern of excitation to progressively larger or smaller afferent rings one step at a time. Stepwise size transformations of this kind are necessarily time-intensive processes. However, there is a second size-scaling mechanism with different properties—the neuronal circuitry between Z-planes in the 3-D retinoid system and the mosaic cell array that accounts for size constancy. Any object represented on a given Z-plane will automatically project to the mosaic array at a scale factor associated with that retinoid plane. The nearer the Z-plane is, the smaller is the scale factor, and vice versa. If we assume that visual information is optimally processed at some standard representational size, then small stimuli should be scaled

up to standard and large stimuli should be scaled down to standard. On these grounds, when a small object is expected, a distant Z-plane is primed to capture its excitation pattern so that it will be relatively larger at its projection on the mosaic cell array; when a large object is expected, a near Z-plane is primed so that the projected pattern will be relatively smaller at the mosaic cell array. Because each Z-plane can be independently addressed and primed without requiring the sequential transfer of excitation over neighboring cells on the Z-axis, scale adjustment can be much faster than in the size transformer mechanism.

The influence of parafoveal information on response to foveal information was explored in a series of experiments aimed at gaining a better understanding of cognitive processing during reading (Rayner, McConkie, and Ehrlich 1978; Rayner, McConkie, and Zola 1980). Subjects were presented with a word or letter string in the parafoveal visual field and then, during the saccade to this location, the initial display was replaced by a word that the subject was asked to read. Thus, a parafoveal stimulus was replaced by a word that stimulated the fovea and that the subject had to pronounce as quickly as possible. The visual, lexical, and semantic similarity between the initial parafoveal stimulus and the word to be named was varied. It was found that certain kinds of parafoveal information facilitated the naming of the target word. In particular, facilitation depended on the match between the first two or three letters in the first string and the subsequent word. The matching letters did not have to be the same shape or size to produce facilitation. For example, if the target word was presented in lowercase letters, the preceding parafoveal string would facilitate naming even if it was an uppercase display provided that its initial letters signified the same substring of alphabetical characters that formed the beginning of the target word. The finding that parafoveal lexical information can be integrated with subsequent foveal lexical information is consistent with the operating characteristics of the retinoid-synaptic matrix system. According to the model, the results can be explained as follows:

1. Initial parafoveal letter strings are captured on a retinoid and translated to the normal foveal axis.
2. The translated parafoveal letter patterns are projected to the detection matrix where they selectively excite filter cell-class cell couplets (object tokens) that have previously learned those patterns.
3. Object tokens of letter patterns provide the input to the matrix

for lexical assignment where they selectively excite their associated word tokens.

4. Excitation of a word token provides a facilitating bias (prime) for that word.

5. The initial letter strings of foveal target words are also processed by steps 1–4. If the initial letters of a target word match those of the immediately preceding parafoveal string, its appropriate word token will have been primed by the preceding string, and word naming will be facilitated (the empirical finding).

Similar neuronal processes can explain the integration of pictorial information across eye movements. In a series of experiments (Pollatsek, Rayner, and Collins 1984), line drawings of objects were presented in peripheral vision. During the saccade to the peripheral stimulus, the initially presented picture was replaced by another that the subject was instructed to name as quickly as possible. There was a significant facilitation in response time when the first and second pictures were identical compared to conditions in which the second picture was a different object or to a control in which only the target location was specified on the first stimulus. Changing the size of the picture from the first to the second stimulus had little effect on naming time. When two differently shaped pictures represented the same concept, there was also a significant facilitation effect. The results are consistent with the priming of object tokens in the detection matrix and word tokens in the matrix for lexical assignment on the basis of similarity of stimulus shapes and/or conceptual equivalence.

Morrison and Rayner (1981) asked subjects to read sentences presented on a CRT at different viewing distances while their eye movements were monitored. Sentences at different distances contained different lexical items, but they were matched for word length and grammatical class. It was discovered that there was no significant increase in the number of character spaces traversed by each visual saccade over viewing distances of 36, 53, and 71 centimeters. The average saccade traversed approximately 5.5 characters at each distance. The mean saccade sizes corresponded to visual angles of 3.81, 2.48, and 2.00 degrees at the three distances respectively. Thus, there was a roughly linear inverse relationship between the length of a saccade and the distance of the visual targets. If, during reading, the parsing of five or six characters at a time is optimal for word recognition and comprehension, one would expect saccade size to decrease as the viewing distance of a text increases. This fits in well with the 3-D retinoid-synaptic matrix mechanisms. The more distant lexical

characters would normally be represented by autaptic cell activity on a more distant Z-plane. The particular Z-plane that is activated during reading can provide a neuronal signal that regulates saccade length. Long saccades for near Z-plane activation; short saccades for far Z-plane activation. At the same time, the size-constancy scaling from the 3-D retinoid to the mosaic array of the synaptic matrix will maintain a pattern of input to the detection matrix that will be near optimal size for word recognition despite differences in reading distance.

Additional support for the 3-D retinoid and its accessory mechanisms is provided by a series of experiments undertaken to explore the sort of medium that underlies imagery for 3-D scenes (Pinker 1980, Pinker and Finke 1980). The time that a subject took after viewing a 3-D scene to scan between objects in a mental image of the scene was used to infer the kind of geometric information preserved in the image. On the basis of clear performance differences associated with different kinds of imagery instructions, it was concluded that 3-D information must be preserved in images, 2-D distance information in the original perspective view must also be preserved, and images can be transformed to display 2-D distance information in perspective views never experienced in the stimulus situation.

Pinker (1980) proposed that these results can be explained by a model in which 3-D structure is encoded in long-term memory in a 3-D object-centered coordinate system and mapped onto a 2-D "display" when imaged. Perspective properties specific to a given viewing angle would then be depicted on the 2-D display. The 3-D retinoid-synaptic matrix system and the putative neuronal mechanisms for analyzing and representing object relations conform to this kind of model. The full retinoid system has the capability of representing veridical or imagined objects and scenes and transforming these representations through 3-D space. It has intrinsic axonal projections that map from the 3-D Z-planes to the 2-D mosaic cell array. The mechanisms for analysis and representation of object relations that are specified in my brain model provide 3-D coordinate tokens that can be selectively attached to the visual objects stored in the long-term memory of the synaptic matrix. When objects in the original 3-D scene are later imaged, these X-, Y-, and Z-axis tokens govern the placement coordinates of the object images in 3-D retinoid space. Excursions of the self-locus to the targets at these coordinates and the reverse translation of target objects to the normal foveal axis provide the neuronal means for scanning over the metric distances between objects in 3-D and 2-D retinoid space.

In an investigation of another aspect of mental imagery, subjects were shown an array of dots followed by an arrow in an otherwise

blank field and asked to determine if the arrow pointed to any of the previously seen dots (Finke and Pinker 1983; Pinker, Choate, and Finke 1984). Instructions to form or scan a mental image were never given; nevertheless, subjects almost always reported that they scanned a mental image to make a judgment. Response times were linearly related to the metric distance between the arrow and the nearest dot. After conducting a number of control variants of the basic experimental paradigm, the investigators concluded that "mental scanning along a straight path can be performed on images reconstructed from memory." They also concluded that such mental extrapolations over image patterns did not depend on the perception of a continuous surface or on eye movements.

A neuronal explanation for these findings is provided by the intrinsic properties of the retinoid-synaptic matrix system and the ability of the heuristic self-locus to trace the contours of objects in retinoid space. According to my proposed model, when the array of dots is briefly shown to subjects, it is learned as an arrangement of objects (dots) at a particular egocentric location in the visual field. After the delay period, when the arrow alone is shown, subjects recall the previous dot arrangement as a pattern of excitation in the imaging matrix and project it at its original coordinates in retinoid space. The retinoid surface will now hold a spatially integrated representation of the imaged dots together with the current arrow stimulus within the same coordinate frame. A straight-line trace in the direction of the arrow head along and beyond the shaft of the arrow by an excursion of the self-locus will either hit (response "yes") or miss (response "no") one of the dots in the imaged array. Assuming a uniform rate of self-locus translation, the time taken to give a correct "yes" response should be a linear function of the distance between the arrow head and the imagined dot that it points to (tracing distance), a prediction confirmed by the experimental results. Thus, the proposed brain model can explain in neuronal terms how people can make mental extrapolations in visual patterns constructed from memory.

Two experiments employing somewhat different paradigms (Jolicoeur, Ullman, and MacKay 1986) give additional support to the self-locus model of contour tracing. In the first experiment, several complex, interdigitated curves were displayed, and the subject's task was to determine as quickly as possible whether two Xs in the display lay on the same or different curves. It was found that the mean response time for a "same" response increased monotonically as the distance along the curve between the Xs increased. In the second experiment, the subject's task was to decide as quickly as possible whether a complex curve joining two Xs was unbroken or had a gap.

Again, response time increased as the length of the curve joining the Xs was increased. According to Jolicoeur and his co-investigators, "The results of both experiments suggest that people can trace curves in a visual display internally at high speed (the average rate of tracing was about 40 degrees of visual angle per second)."

An underlying constraint of the putative retinoid-synaptic matrix system is that environmental scenes are normally learned and stored in memory within an egocentric frame of reference. Experiments by Fredrickson and Bartlett (1987) tend to confirm that people learn and recall scenes within an egocentric frame. In one experiment, subjects first verbally encoded and then verbally recalled the lateral location of objects within scenic pictures that were projected in a room with a door on one side and a window on the other. Object location was encoded in two ways: using an egocentric frame (left or right) and an environmental frame (near the window or near the door). Significantly fewer errors were made when recalling locations in egocentric terms (left or right) than when recalling in environmental terms (near the door or near the window), even if the latter proximities had been verbalized at input.

In a second experiment, subjects first viewed half of a set of projected pictures directly, while the remainder of the pictures, together with their near environment in the room (door, window, post), were viewed in a mirror reflection. Subjects were shown (in direct view) a set of pictures of which half had been seen in the first phase (old items) and half were new items. They were required to detect the new items and to classify the old items in terms of their orientation (same or reversed) with respect to their appearance in the original set. Subjects were divided into two groups. One group (the egocentric group) was told that *same* and *reversed* referred to whether what had been to the right (left) of their visual field was still to the right (left). The other group was told that *same* and *reversed* referred to how the picture was presented on the screen without regard to how it looked to them previously. Performance was found to be much better when orientation was egocentrically defined. The investigators concluded that "encoding lateral orientation of complex pictures is cognitively impenetrable in at least one respect: It is strongly constrained, perhaps absolutely constrained, by a viewer-centered reference frame."

Shulman, Remington, and McClean (1979) performed several experiments to determine whether shifts of visual attention can occur in a continuous analog fashion across the visual field in the absence of eye movements. In their experimental paradigm, four light-emitting diodes (LEDs) were positioned 8 degrees and 18 degrees to the left

and to the right of a central fixation point. Subjects were instructed to press a key as soon as any one of the four LEDs was turned on. An arrow pointing left or right was presented at the fixation point. The arrow served as a cue informing the subject that the far LED on the indicated side had a very high probability of being the target light. Thus, the far light on the indicated side was always the expected target light. Subjects were instructed to pay attention to that light but to maintain their fixation on the center. Eye movements were recorded by electrooculograms, and any trial showing eye movement was discarded. Target lights were illuminated at a randomly determined interval following each onset of the cue (arrow). The difference between the reaction time to the far expected light (18 degrees) and to the intermediate light (8 degrees) was measured and plotted as a function of the delay time between the cue and the onset of a target light. Analysis of the data indicated that an intermediate delay time facilitated response to the intermediate light relative to the far (expected) light, whereas a longer delay time facilitated response to the far light relative to the intermediate light. This result suggests that the focus of visual attention moves through space in a continuous analog fashion.

In a variation of the experimental paradigm employed by Shulman and colleagues, Tsal (1983) confirmed and extended their conclusions. Tsal's data suggest that when attention is directed to a peripheral target, it moves through visual space at a constant velocity of ~ 1 degree per 8 milliseconds. The results of both studies are consistent with the neuronal mechanism of directed attention that I have proposed: heuristic excursions of the self-locus evoke a "spotlight" of local excitatory bias that moves over retinoid space in a continuous analog fashion from the normal foveal axis (the home position of the self-locus) to the peripheral target.

Experiments by Chambers and Reisberg (1985) and by Finke, Pinker, and Farah (1989) provide interesting examples of both a specific limitation and a high degree of flexibility in manifestations of human imagery, which can be seen as natural consequences of the putative retinoid-imaging matrix mechanism. In the study reported by Chambers and Reisberg, subjects were shown ambiguous figures that have been traditionally employed to demonstrate multistability in visual perception. They were then instructed to form mental images of these shapes and to try to see reversals in their images. It was found that reversals of the imaged figures that are normally experienced under the condition of direct perception were never reported by the subjects. Why should this be the case? Recall that perceptual reversal of ambiguous shapes is fully explained by shifts

in the capture of pattern centroids during visual parsing of the overall figure. Normal operation of the hypothesized neuronal mechanisms results in the alignment of different centroids of an extended figure on the normal foveal axis as a consequence of shifts in the locus of visual fixation on the stimulus pattern at different times. In the usual perception of unstable figures, the partial pattern that is automatically parsed (captured within the afferent aperture) in association with a particular centroid will trigger an object token (class cell) appropriate to its particular shape and different pattern parts will evoke different object tokens (figure reversal). However, whenever a single object is imaged, it can be represented only on the mosaic array oriented around one centroid—the one at which it was learned and stored in the synaptic memory of the imaging matrix. Thus, in simple imagery, there are no alternative parsings of the object pattern that can spontaneously evoke alternative object interpretations.

It would be a mistake, however, to conclude from Chambers and Reisberg's study that new patterns cannot be "seen" in the form of an image. When the full capabilities of the transformer circuits, the retinoid system, and aperture control are utilized, then spatial transformations, analytical decomposition of unitary image patterns, constructive and destructive modification of a source pattern, controlled assembly of multiple component images, and retinoid tracings of the self-locus can all contribute to reconstructions of old images or the creation of entirely novel images. The series of experiments by Finke, Pinker, and Farah (1989) clearly demonstrated that initial visual patterns in mental imagery can be transformed and combined with other images in response to verbal instructions so that emergent imaginal objects are induced and recognized. In their most rigorous procedure, subjects were first instructed to imagine a familiar pattern—for example, a square or the capital letter H. Then they were instructed to perform a series of changes of the original imagined figure such as spatial transformations and pattern additions or deletions. After each instructional step, subjects were asked to inspect their images and guess the identity of the final emergent pattern—for example, "Imagine a capital letter 'D'. [Guess 1] Rotate the figure 90 degrees to the left. [Guess 2] Now place a capital letter 'J' at the bottom. [Final identification]."

The complete set of transformations for this experiment is illustrated in figure 15.1. It was found that emergent patterns were never identified at the end of the first step and were identified only 4.2 percent of the time at the end of the second step. But when the sequence of imaginal transformations was correctly performed (48 trials), a significant proportion of correct identifications were made

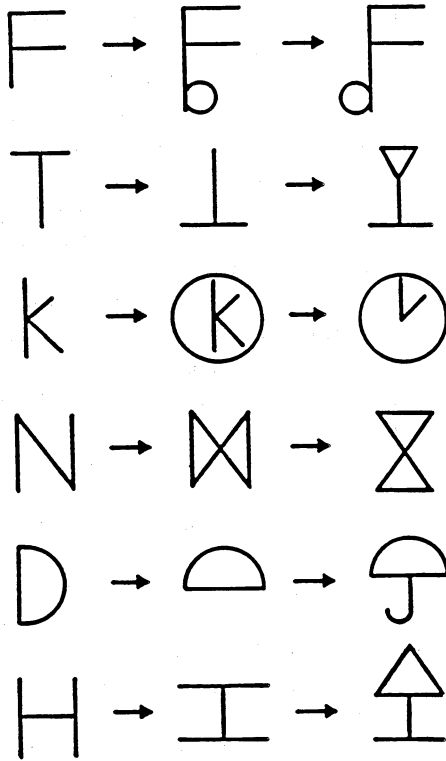


Figure 15.1

Subjects were instructed to begin by imagining the patterns shown at the left of each row and then to imagine transforming the patterns as the illustration depicts. The final patterns in each sequence are the emergent patterns that subjects were to try to recognize. Source: Finke, Pinker, and Farah 1989. Copyright Cognitive Science Society, Inc. Reproduced by permission.

after the third step (44 percent). These results are consistent with the capability for imaginal transformation, reconstruction, and recognition inherent in the putative neuronal mechanisms that I have described.

Clinical Evidence

Clinical investigations of the effects of localized brain pathology on cognitive performance can illuminate both the functional topology of the brain and the issue of functional modularity—the degree to which an identified cognitive process can be dissociated from other related processes. My cognitive brain model is a complex biological system composed of many intercommunicating special-purpose neuronal

mechanisms. A system of this kind is characterized by a high degree of modularity in distinction from systems that might operate on the basis of global distributed processes. When there is localized damage in such a modular brain architecture, one would expect that dissociated functional loss would often occur, and, in fact, there is abundant evidence in the clinical literature that this is the case (Brown 1989).

In a review of specific deficits of visual gnosis, Benson (1989) cites a variety of dissociated failures of function associated with brain damage. For example, patients having apperceptive visual agnosia are unable to name, copy, or recognize visually presented objects but can immediately identify the object if they are given tactile or auditory cues. At the same time, they are able to identify light intensity and the direction and dimension of visual stimuli. This disorder can be explained by discrete damage of neurons in the detection matrix or to its afferent channel. In another class of disorders, patients are able to copy words but unable to read them (alexia).

In terms of the synaptic matrix model, such a condition can arise if filter cells that have been tuned (in prior learning) to the visual pattern of words or their coupled class cells that constitute word tokens are damaged. The same syndrome would also result if the cellular interface between word tokens in the synaptic matrix and mosaic cells in the lexical assignment matrix were disconnected by brain pathology (localized lesion, neoplasm, or vascular deficit). Another specific clinical deficit cited by Brown is in the ability to make simple constructions on the basis of visual stimulus patterns. He reports a relatively pure visual-spatial discrimination loss in patients with focal damage in the right parietal-occipital area of the brain and a language-dependent construction loss with left parietal damage. Pure visual-spatial deficits would be expected to occur with damage to the mechanisms for coordinate location on the representational surfaces of the retinoid module, while a specific loss in language-dependent construction would result from an interruption of normal projections from lexical matrices to the cells that govern object location on the scene-assembly retinoid.

Topographagnosia and environmental agnosia are two interesting pathologies that would seem to implicate the accessory control mechanisms of the retinoid system in one disorder and the recollection of previously schematized spatial representations in the other. Individuals with topographagnosia are typically able to perform normally in real-life situations (drive long distances, navigate city streets) but are unable to interpret artificial displays of topography. They cannot place their own location on a map, draw a plan of their own house, or identify rooms on a plan drawn for them. These patients typically

show right parietal lobe damage. In chapter 7, I detailed the neuronal mechanisms that work together with the retinoid system to enable analysis and schematic representations of visual space. It would appear that topographagnosia results from damage to these mechanisms.

Patients with environmental agnosia are able to read maps and house plans but are unable to find their way in familiar environments. They can get lost even in their own homes. In this case, brain pathology always involves the medial aspects of the right occipital lobe (Landis et al. 1986). The phenomenon can be explained by an interruption of axon collaterals from the detection matrix for local stimuli to the imaging matrix of another module, which normally evokes the previously associated schematic representation of the spatial-environmental context.

Riddoch and Humphreys (1989) report patients who appeared to have lost all experience of 3-D vision although their 2-D vision remained intact. These individuals perceive the world as utterly flat. In such cases, one would expect that the 3-D retinoid system is severely damaged or disconnected while the monocular retinoids function properly.

A striking as well as informative example of a specific representational deficit in patients with brain lesions involving the right temporal-parietal-occipital junction has been reported by Bisiach and Luzzatti (1978). These patients were asked to imagine they were standing in a very familiar setting, the main square in Milan. They were first to imagine themselves facing the cathedral and to describe what they could see in their mind's eye. They reported a greater number of details to the right than to the left of their imaginary line of sight and often neglected salient aspects on the left side. They were then asked to perform the same imagery task facing in the opposite direction, away from the cathedral. In this case, they were able to report previously neglected details within the right half of the new imaginal perspective but ignored items in the left half that they had reported a few moments before. This example of unilateral neglect indicates that the deficit is not due to a loss of sensory input, a fault in sensory processing, or a discrete memory loss but rather to a failure in the internal representation of egocentric space or in the ability to classify objects represented within a particular region of egocentric space. The cognitive deficit can be explained by damage to the left hemifield of the retinoid system (representational loss), selective damage to that part of the shift control mechanism that translates retinoid images from left to right in order to bring them to the normal foveal axis for detection and classification in the synaptic

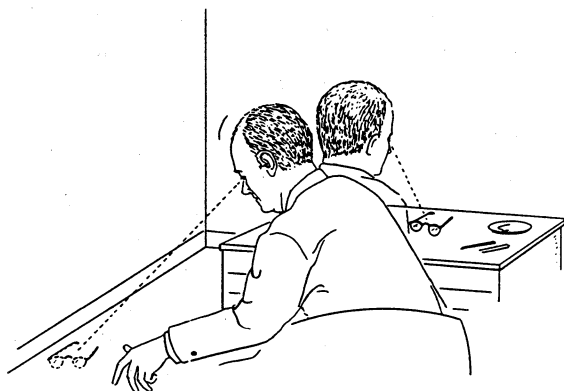


Figure 15.2

Palinopsia. Patient initially viewed his eyeglasses placed horizontally on a desk in front of him. Seconds later he looked away but continued to see an image of the eyeglasses to his left on the floor. Source: Jacobs 1989. Copyright Lawrence Erlbaum Associates, Inc. Reproduced by permission.

matrix (analytical loss), or selective damage to that part of the shift control mechanism that drives the self-locus into the left hemifield of retinoid space (attentional loss).

While perceptual-cognitive deficits constitute the typical sequelae of brain damage, intrusive perceptual anomalies can also occur. Palinopsia is a particularly interesting instance of such an anomaly in the visual domain. It is characterized by the persistence or recurrence of a visual percept after the exciting stimulus object has been removed and is usually associated with lesions in the parieto-occipital region (Bender, Feldman, and Sobin 1968; Jacobs 1989; Michel and Troost 1980). An example of the kind of intrusive visual experience typical in the palinopsic patient is given in the following case report (Jacobs 1989):

On one occasion he initially saw his eyeglasses placed horizontally before him on a desk. Seconds later, after turning away, the eyeglasses were no longer in view, but he continued to see an image of them off to his left on the floor (figure 15.2). The illusory eyeglasses appeared so realistic that he reached out attempting to pick them up from the floor before he realized that they were still there on the desk in front of him. He continued to see an image of the eyeglasses in his left visual field wherever he looked for the next five minutes.

If one were still undecided about the essential visuo-spatial nature of visual imagery (Farah 1988), the evidence of palinopsia leaves little doubt that images share the spatially extended property of normal visual perception. But can palinopsia be explained in neuronal terms—specifically by the properties of the mechanisms in our putative cognitive brain model?

What mainly distinguish the palinopsic image from normal imagery are its vividness (to the point of being mistaken for an actual visual percept) and its involuntary persistence; in addition, the image seems to be located wherever the focus of gaze comes to rest in the immediate environment. Recall that an image is normally evoked in the synaptic matrix when an axon collateral of an activated class cell induces the pattern of discharge in the mosaic cell array that was present at the time the original stimulus object was learned. Suppose that local cell irritation associated with a lesion in the visual system were to result in phasic hyperexcitation of the synaptic matrix. Under this abnormal condition, a perceived object would immediately be learned; it would be stored in the synaptic transfer weights of a filter cell and on the mosaic cells in the imaging matrix through a coupling class cell. Moreover, the lesion-induced hyperexcitation would induce a more vigorous response in the imaging matrix than one would experience in normal imagery, and this could account for the abnormal vividness of the palinopsic image. In addition, the class cell that evokes the palinopsic image would be locked in sustained discharge during the phase of hyperexcitation, and the patient would be unable to exercise voluntary control of the intrusive image. In accordance with the ordinary operation of the visual-cognitive mechanisms, the visual pattern on the mosaic array would consist of the vivid palinopsic image superposed on normal perceptions transmitted from the retina. Thus, the intrusive object would be “seen” wherever the patient looked. The fact that in palinopsia a visual object can be captured as a vivid memory during a single brief exposure provides strong support for the learning principle of one-trial reorganization of synaptic efficacy that I have assumed to be true of the human brain (Trehub 1975a, Lewis 1979).

Other Mechanisms

In chapters 8 and 9, I presented the circuitry of neuronal mechanisms that are competent for composing, storing, and recalling plans, as well as initiating actions in accordance with concurrent motivational states. These mechanisms are variants of the basic synaptic matrix

and adaptively couple sensory tokens with motor-control circuits to construct sequenced plans of action. They are characterized by multimodal inputs representing the convergence of complex multisensory information that defines tokens of environmental affordances, tokens of simple motor commands, and tokens of concurrently activated goals. Outputs serve to select plans of action (latent sensory motor sequences) on the basis of affordances and goals. These neuronal structures, in turn, project to motor regions for the initiation of overt action sequences. Recent investigations by Joaquin Fuster and his co-workers provide direct neurophysiological evidence that tends to support these putative brain mechanisms.

In a study of cross-temporal integration of sensory and sensory motor information, the activity of 295 single neurons was recorded in the dorsolateral prefrontal cortex of rhesus monkeys (Quintana, Yajeya, and Fuster 1988). The monkeys were given two visual discrimination tasks: a delayed matching to sample that required the animal to remember a colored cue to be matched 18 seconds later and a delayed conditional position discrimination that required the animal to remember the color of a cue so that a correct spatial response could be made 18 seconds later. On the basis of their analysis of single unit responses, the investigators concluded:

1. During visual delay tasks, neurons in the dorsolateral prefrontal cortex may process both spatial and nonspatial information.
2. Because of their protracted differential discharge during the delay between cue and response, some units seem involved in the transfer of sensory information across time.
3. The findings suggest that prefrontal neurons have a role in the representation of multiple attributes of sensory stimuli, including their associated motor connotations.
4. The findings are consistent with the role of the prefrontal cortex in the cross-temporal mediation of sensory motor contingencies and the temporal organization of behavior.

The observed properties of cells in the prefrontal cortex fit well with the neuronal details of my hypothesized register for plans and actions; for example (figure 8.3), sustained stimulation of register cells by input from autaptic cells in the step ring would account for their protracted discharge during the delay between cue and response (conclusion 2). Multiple sensory attributes would normally be represented by the activity of register cells that compose specification of the *find* mode, and particular outputs from the register are selective for particular motor routines (conclusion 3). Finally, the hypothesized mechanism provides an explicit neuronal basis for mediating sensory

motor contingencies over time and achieving effective temporal organization of behavior (conclusion 4).

A follow-up study was performed to explore the role of behavioral significance on the neuronal representation of stimulus attributes during delay tasks (Yajeya, Quintana, and Fuster 1988). Rhesus monkeys were given two visual discrimination tasks requiring a delayed response after 18 seconds. In both tasks, the correct response depended on the color of a visual cue. Red and green guided the response in one task, yellow and blue in the other. A fifth color (violet) was not relevant to either task and was presented at random in the same location as the significant cues. As in the previous experiment, the activity of 294 single units in the dorsolateral prefrontal cortex was recorded and analyzed. It was found that cellular reactions to the insignificant stimulus were of lesser magnitude than the reactions to the significant cues. Cell response differences as a function of stimulus significance outnumbered and overshadowed differences as a function of cue color or any other task variable. The investigators concluded that neurons in the prefrontal cortex differentiate stimuli by their behavioral significance as well as by other stimulus attributes and that "these results support the notion that the prefrontal cortex integrates motivational inputs into the structure of behavioral action."

Mechanisms detailed in chapter 9 can clearly account for these empirical findings. For example, the neuronal network shown in figure 9.6 selectively enhances cellular response to significant stimuli by providing discrete excitatory bias to just those cells that are relevant to a currently activated motive and by inhibiting the response of other sensory tokens. Figure 9.7 and table 9.1 illustrate how motivational inputs are integrated into the structure of behavioral action by the putative brain mechanisms. Thus, the activity of individual cells in the prefrontal cortex reported by Yajeya and colleagues is consistent with the model.

I have described neuronal mechanisms that can analyze the visually perceived environment, represent pathways within the environment, and formulate plans for navigating the environment, but I have not explicitly dealt with processes whereby the representation of spatial properties of the physical environment might be mediated by nonvisual information. An example of the latter kind of cognitive ability is highlighted in a study by Landau, Gleitman, and Spelke (1981) who demonstrated in a series of experiments that a congenitally blind 2½-year-old child, as well as sighted but blindfolded children and adults, were able to determine the appropriate path between two objects in a room (on a route never before followed by

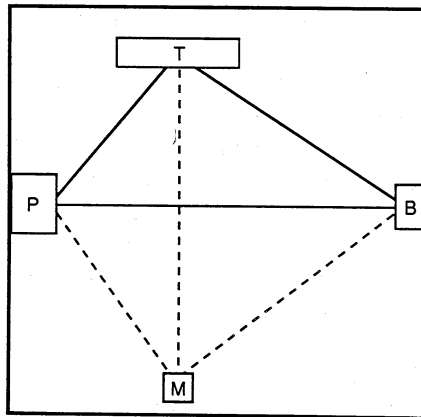


Figure 15.3

Room layout for spatial inference experiment. Dashed lines indicate trained routes. Solid lines indicate test routes. Landmarks: M = mother; P = pillows; T = table; B = basket. After Landau, Gleitman, and Spelke 1981.

the subjects) after traveling under guided locomotion to each of those objects from a third object (figure 15.3). The distances and angular relationships of paths between the initial pairs of target objects were detected and internally represented without visual input, solely on the basis of locomotion. Moreover, potential paths between objects in the environment were apparently “deduced” from the internal motor-generated representation.

This finding can be explained if we assume that the retinoid system may be spared even in congenitally blind individuals and feedback signals from the motor system and/or signals of motion from the vestibular system can control excursions of the self-locus in retinoid space. If these assumptions are true, then the location of objects will be usefully (if not precisely) represented in egocentric retinoid space despite the absence of visual input, and all potential paths among the represented locations can be traced by heuristic excursions of the self-locus. Now we need only assume that these new cognitive-neuronal representations can govern locomotion.

Recall that while the neuronal mechanism of the semantic network is normally accessed by the words of a culture-bound lexicon and its output normally selects words from the contents of such a lexicon, semantic knowledge and the making of logical inferences are intrinsic to the mechanism. It is assumed to be independent of any particular lexicon. A recently reported clinical experiment supports this aspect of the hypothesized semantic network mechanism (Anderson et al.

1989). A left temporo-parietal infarct in a 28-year-old man caused severe aphasia for English. Although the patient had no prior knowledge of American Sign Language (ASL), he was able to acquire ASL at a rate equal to that of an age-matched normal control. This finding was replicated in another patient with left temporo-parietal damage and severe aphasia. The fact that specific semantic knowledge can be readily expressed by a visual-gestural symbolic system after damage to posterior language-related cortices and profound aphasia for English indicates that semantic knowledge is represented in the brain independently of any particular lexicon. This conforms with the assumption of the model.

The logical structure of the semantic network enables two modes of query: (1) defining a subject (evoking a predicate, given a subject) and (2) inferring a subject (evoking a subject, given a predicate). A query of the first kind depends on a direct input to a selected mosaic cell (subject token) in the semantic network. A query of the second kind depends on a direct input to a selected class cell (predicate token) in the network. Because each mode of query is served, in part, by a discrete bundle of neurons, selective brain damage might disrupt the ability to answer one kind of question while sparing the ability to answer the other kind of question. For example, having learned that a bird flies, an individual with focal brain damage that interrupts extramodular excitation of predicate tokens might be able to answer "flies" in response to the question "What does a bird do?" yet be unable to answer "bird" when asked "What flies?" This, at least, is an implication of the brain mechanism for semantic processing that I have hypothesized.

Interestingly, a patient described by Damasio and Tranel (1989) seems to exhibit just this kind of semantic dissociation when tested for his knowledge of cities in states. When the patient was given the names of cities and asked to tell what state each city is in (defining a subject), his performance was accurate. However, when given the names of the states and asked to tell what cities were in the states (inferring a subject), he was almost completely unable to do so. This clinical observation is clearly consistent with the operating principles of the putative semantic network mechanism.