

Chapter 9

Set Point and Motive: The Formation and Resolution of Goals

What counts as a dominant problem at any given time in a person's experience? Within the same objective environmental context, two individuals may each initiate behavior directed at the solution of fundamentally different problems. If we are to give a reasonably comprehensive physical explanation of the behavior of an individual in a particular situation, we must not only provide an account of how significant elements of the situation can be perceived, learned, and related to prior experience, we must also explain why formerly neutral aspects are treated as significant, whereas previously significant aspects may be ignored. We must explain the biological bases for the particular complex of cognitive behavior—how it can be selectively initiated, organized, and terminated. In short, we must give a credible account of the processes of motivation.

The commonsense notion of a goal has traditionally served as a core concept for understanding the integrity of intentional behavior. In a domain of arbitrary behavioral affordances, the selection of a goal can serve to organize a whole sequence of neuronal commands (a plan of action) that effectively lead from a given starting situation to the achievement of that goal. Within the framework of the approach taken in this book, this naturally provokes the following questions:

- How do goals develop?
- How are goals represented and stored in the neuronal structures of the brain?
- How is a particular goal selected from the repertoire of stored goals?
- How does an activated goal guide behavior?
- How is the achievement of a goal recognized?
- What happens next?

Set Points and Homeostasis

A commonly presented example of a set point in a physical system is the temperature setting on a home thermostat. If room temperature falls beyond a predetermined margin of tolerance below the set point (temperature setting), the heating system is automatically switched on to bring the temperature back near the set point. When the temperature has risen to within the set point margin, the heating system is automatically switched off. Homeostatic systems in living organisms are mechanisms of this kind (figure 9.1). They initiate processes that tend to maintain internal stability by correcting deviations from a physiological set point.

It is useful to classify homeostatic systems into two kinds, designated here as HS-I and HS-II. HS-I includes, among other mechanisms for maintaining optimal internal states, the vital autonomic mechanisms that ensure the viability of the organism. Type HS-I systems are distinguished from type HS-II by virtue of the fact that the loop of events serving to hold a physiological state near an optimal set point in HS-I does not require that a neuronal token (physical symbol [Newell 1980]) of an external referent be activated.

A clear example of HS-I is the mechanism that causes cardiac output to decrease when there is an increase in arterial pressure and to increase when there is a decrease in pressure (see Cotman and McGaugh 1980), thus reducing possibly dangerous extremes of blood pressure. But consider the hypothesized mechanism for aligning the centroid of an object's visual-afferent representation on the normal foveal axis. While such a system would not ordinarily be called homeostatic, in my view, the automatic balancing of quadrantal excitation on a retinoid can legitimately be regarded as a homeostatic process analogous to that which tends to maintain the stability of arterial pressure. In the case of arterial homeostasis, the set point is normal blood pressure; in the case of centroid homeostasis, the set point is normal quadrantal disparity.

The second type (HS-II), which I will call cognitive homeostasis, contains all of the processes that require the discharge of one or more referential tokens to complete a homeostatic loop that can correct a deviation from an internal set point. Processes of this kind may be entirely covert or may require overt instrumental behavior to close the loop that corrects a deviation from a set point.

Behavior associated with thirst provides a clear example of HS-II. Receptors within the brain that are sensitive to cellular dehydration (osmoreceptors and, possibly, salinity receptors [Cotman and McGaugh 1980]) signal a water deficit and initiate a chain of events

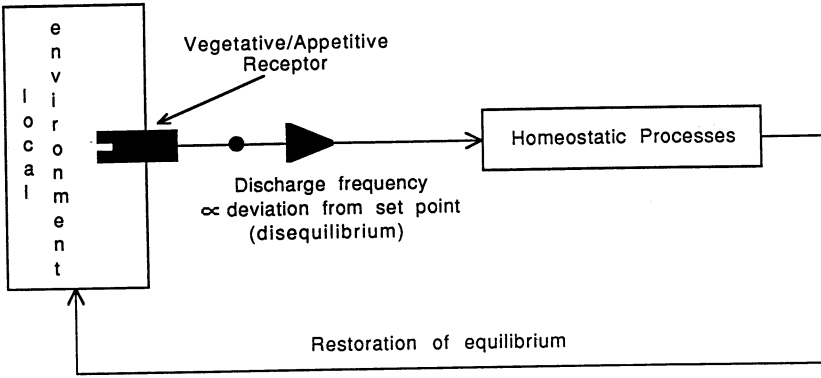


Figure 9.1
A homeostatic system mediated by neuronal discharge proportional to a sensed deviation from a physiological set point.

that correct the deficit. Unlike the homeostatic mechanism for regulating blood pressure, appropriate referential tokens must be discharged and exteroceptive stimulus patterns must be recognized in order to obtain the water needed to bring cellular hydration back to the set point. Thus, if I feel thirsty while sitting in my living room, the neuronal tokens of *kitchen sink*, *faucet*, and *water glass* must be evoked if I am to take advantage of available utilities. Moreover, I must recognize the exteroceptive patterns of these objects in order to guide the instrumental behavior that quenches my thirst.

The neuronal events that constitute a proper response to self-query provide an important but more subtle example of what I mean by cognitive homeostasis. The virtual set point in such cases is a co-occurrence (balancing) of a subject (*W* cell) and a proper predicate (Ω). If a subject token is discharged, the system is in disequilibrium until a familiar predicate token is fired in addition to the subject identity token (figure 9.2). Thus, for example, if one has a neuronal representation (in a semantic network) of both $\langle \text{John's-address} \rangle$ and $\langle \text{is-43-Maple-Street} \rangle$ and has learned the correct association, the query "What is John's address?" will evoke the appropriate predicate token with a latency that signals a familiar response. (The predicate response will include at least two activated class cells—one for the identity relationship $\langle \text{John's-address} \rangle \langle \text{is-John's-address} \rangle$ and the other for the informative predicate $\langle \text{is-43-Maple-Street} \rangle$.) The departure from set point that was initiated by the query is corrected by the Ω response. If John's address has not been learned, no second predicate will fire within the latency for a familiar response, and the system

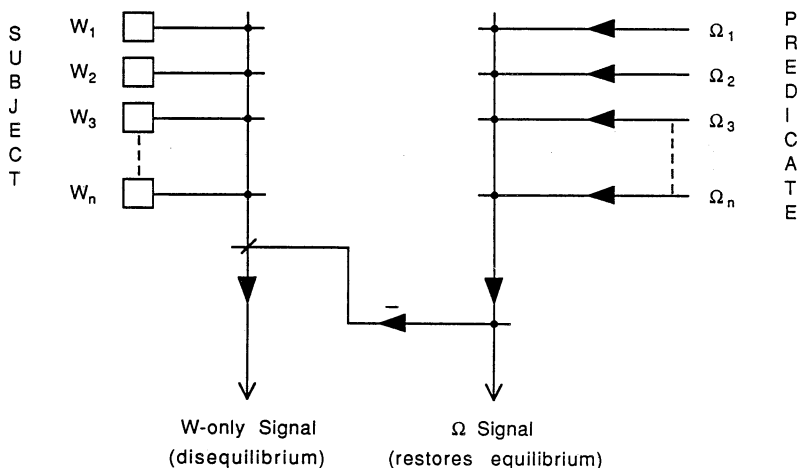


Figure 9.2
Mechanism for restoring equilibrium in self-query.

remains in a state of disequilibrium. In this case, one might resort to overt instrumental behavior to provide the missing predicate and close the homeostatic loop, perhaps by consulting a telephone directory.

Pleasure and Displeasure

The terms *pleasure* and *displeasure* refer to personal and essentially private experiences. There is common agreement about the kinds of overt behavior that indicate the inner experience of pleasure or displeasure and a consensus that these experiences are intimately related to individual motivation. Evidence drawn from experiments in which brain cells are subject to direct electrical stimulation suggests that pleasure and displeasure are modulated by homeostatic events.

During neurosurgical procedures, conscious human patients report electrical stimulation in a variety of deep brain regions (hypothalamus and limbic structures) as pleasurable (Heath 1964; Mark, Ervin, and Sweet 1972). Controlled laboratory experiments provide more detailed information. A rat with an implanted electrode will press a lever thousands of times an hour for many hours simply to receive brief trains of electrical stimulation to a region in its lateral hypothalamus (Olds 1958, 1962). Because of this behavior, together with the observation that the animal will repeatedly cross an apparently painful electrified grid in order to reach the lever, those brain regions that

are the targets of self-produced stimulation have been called both pleasure centers and reward centers. It is significant that the rate of lever pressing for self-stimulation is positively correlated with the strength of hunger, thirst, or sexual drive at the time of stimulation (Rolls 1975). For example, animals that are sated with food press at lower rates than when they are hungry and will even press to turn off hypothalamic stimulation that they eagerly worked for when they were deprived. It appears that when the animal is satiated, brain stimulation that was formerly pleasurable can become aversive.

These findings highlight what might seem to be a trivial fact but one that allows us to make sense of a variety of motivated behaviors aimed at the achievement of social goals rather than the gratification of vegetative or basic appetitive needs: the experience of pleasure (activation of its cellular substrate) occurs only when there is a deviation from a homeostatic set point in the brain and dissipates when homeostasis is achieved. A deviation from a set point is not a sufficient condition for pleasure, but it appears to be a necessary condition. What is also required under normal circumstances is some action or event that tends to restore equilibrium to the perturbed system. This implies that for humans to experience pleasure when vegetative and appetitive needs are met, other kinds of homeostatic disequilibria must have been induced in the brain. In addition, these disequilibria must be related to ecological domains in which an individual perceives the possibility of achieving a selected goal, thus reestablishing equilibrium.

The Central Hedonic System

As the human brain matures and interacts with its interoceptive and exteroceptive environment, it constructs new homeostatic systems of the HS-II type that serve an elaborate personalized hedonic economy. The development of new cognitive-homeostatic set points provides the opportunity to tap sources of hedonic currency beyond the basic appetitive ones. We can think of these motivational set points as the neuronal representations of particular goals. A homeostatic imbalance associated with the set point of a goal evokes behaviors that can correct the imbalance (reach the goal), and in the process a pleasure mechanism within the brain is stimulated (figure 9.3). It is parsimonious to assume that this is the same kind of mechanism that is at the core of the cellular systems that receive direct electrical input in the self-stimulation experiments cited.

The biological structures that constitute the vegetative and basic appetitive homeostatic systems are genetically determined. Thus, we

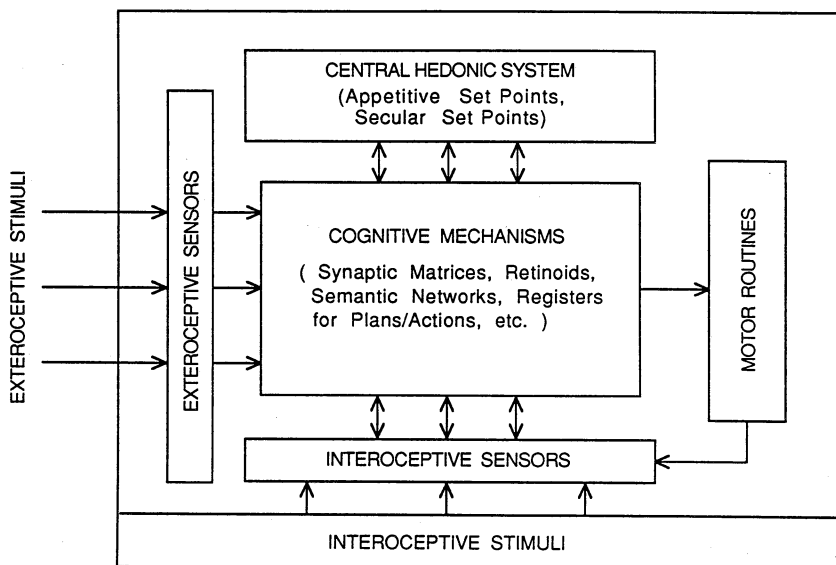


Figure 9.3

Block-flow diagram of the cognitive system of the brain in relation to sensory input, motor routines, and the central hedonic system.

are normally innately endowed with a preset point for benign blood pressure, sensors for detecting potentially destructive changes in vascular pressure, and the physiology for correcting such deviations. Similarly, we are endowed with receptors for glucose and fatty acids that provide hunger signals indicating that concentrations of these substances are below tolerance with respect to our physiological set point (Oomura 1976)—that is, our body cells require nourishment, and we must eat to restore the proper levels of nutrients before the hunger signals will turn off. Homeostatic processes related to thirst and sexual drive are similarly governed by genetically determined factors. But what of the person whose goal is to be a professional basketball player or an entomologist? The set points that represent these goals must somehow be physically instantiated in the brain. Clearly, however, such goals cannot be genetically determined.

Development of Secular Goals

I use the term *secular* to distinguish a special subclass of set points from those that directly serve vegetative and appetitive homeostasis. A secular set point (goal) is an enduring brain state that represents a future imagined worldly situation. As long as the imagined neuronal

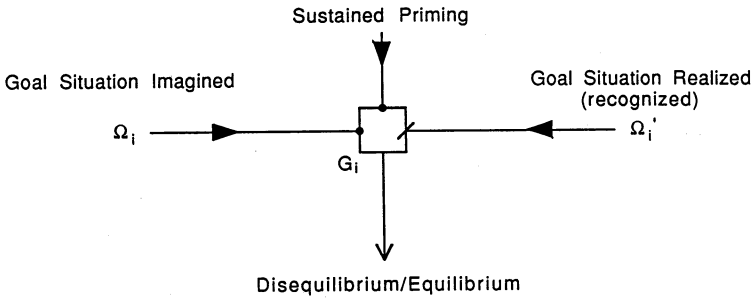


Figure 9.4
Mechanism for activating, sustaining, and terminating a neuronal (autaptic) token of a secular goal (G_i).

representation is not effectively matched by the perception of its corresponding events in the real world, the homeostatic system of which it is a part is in disequilibrium (figure 9.4). This constitutes the neuronal substrate that initiates goal-directed behavior. Those plans and actions that lead to a situation that matches the goal are selectively activated and are said to be motivated by the goal. However, granted that the establishment of a goal can determine a plan to achieve the goal by means of self-query and the intrinsic neuronal logic of a semantic network, what determines the initial establishment of any goal?

The neurophysiological processes and environmental influences that govern the instantiation of a personal secular goal are undoubtedly complex, subtle, and delicately balanced against many competing goal-determining events. I believe, however, that there are some fundamental mechanisms and formative principles that can be reasonably proposed. First, I assume that the instantiation of any secular goal within the microstructure of the brain must be preceded by a heuristic exploration (in imagery) of possible worlds. This kind of fanciful imagery may range over a variety of sensory modalities, and I assume it to be, at least in part, a covert expression of an innate need for exploration and play (Nissen 1951). Under this assumption, one must posit genetically determined homeostatic mechanisms for both exteroceptive and imaginal sensory exploration.

Recall that neuronal tokens can be associatively linked in forward- and backward-going chains. Whatever the signal concomitants of the activation of a pleasure center might be, they too can be represented by a neuronal token and selectively coupled in synaptic matrices to other tokens that are concurrently active. It is assumed that any imagined possible world will be composed of a complex of perceptual

tokens. The greater the number and variety of such component tokens that are linked (directly or indirectly) to pleasure centers in the brain during the course of experience, the more likely will pleasure be evoked when that possible world is fantasied. An activated link to a pleasure center does not necessarily result in a pleasurable experience because it is only in those systems in which there is a current homeostatic imbalance that a pleasure signal can be evoked.

In addition to the pleasure centers, we must take account of brain mechanisms that signal pain, discomfort, and many different aspects of displeasure (Kuffler, Nicholls, and Martin 1984; Mark, Ervin, and Sweet 1972). The hedonic system involves the activity of such centers of aversive experience as well as pleasure, and the signal concomitants of the activation of nociceptive cells can, like those in the pleasure centers, be represented by neuronal tokens. Moreover, particular perceptual tokens in the complex of an imagined possible world may be linked in forward and backward chains to displeasure centers in the brain in the same way that other tokens are linked to pleasure centers.

These considerations suggest a neuronal model that can account for the ontogeny of secular goals. In the discussion that follows, I will use the adjectival terms *adiant* (directed toward) and *abiant* (directed away) to distinguish processes and tokens that are linked to the centers for pleasure and displeasure, respectively.

Figure 9.5 illustrates how the summed activity of adiant and abiant tokens modulates the output of an associated autaptic cell (G_i) that is a token of a secular goal. In order for G_i to be instantiated as an active goal in the cellular structure of the brain, it must be stimulated by the token of an imagined goal situation (Ω_i) and receive a priming margin of excitatory input from the sum of associated adiant events sufficient to overbalance the inhibitory input from the sum of associated abiant events. Under these conditions, G_i will fire. Moreover, G_i will maintain its discharge even when Ω_i is no longer active, provided the margin of input from adiant sources (excitatory) over input from abiant sources (inhibitory) is great enough. The continued discharge of G_i represents the departure from the set point (disequilibrium) of a specific secular goal and signals the need to construct an appropriate plan of action for achieving the goal. The recognition of a realized goal results in the discharge of its class cell token Ω'_i , which sends an inhibitory input to G_i , squelching its autaptic activity and restoring equilibrium.

Because the brain is a complex hybrid system of quasi-linear and nonlinear processes and threshold gates, very small differences in initial conditions can result in major differences in the evolution of

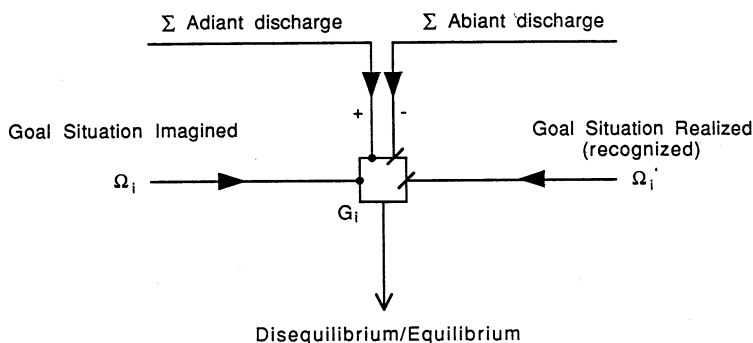


Figure 9.5

Current magnitudes of summed adiant (excitatory) and abiant (inhibitory) inputs to G_i will modulate the moment-to-moment motivational strength (discharge frequency) of the goal represented by G_i . If abiant input approaches the magnitude of adiant input, net excitation will be reduced below the level required to sustain autaptic activity, and G_i will be squelched.

the system. Thus, minor variations in initial neurohumoral and other neurophysiological factors, as well as in subsequent experience, can have profound and unpredictable effects on the selection and expression of personal secular goals. For example, innate physical differences between two individuals can easily result in a different pattern of skills for each, which can affect the development of secular goals. If both individuals initially imagined the same goal situation with the sum of adiant components at the same high level for each, the sum of abiant components might nevertheless be much higher for the individual having the poorer skills related to the goal situation. This is particularly likely to be true if inordinate effort were required for the instrumental behavior needed in the context of the goal or in pursuit of the goal. In this case, the imagined goal might not be instantiated by the person with the poorer skills because net excitatory priming to G_i is insufficient due to an increase in inhibitory abiant input. Or if G_i were instantiated, it might later be squelched before the goal is reached because of an increase in abiant input in the effort to attain the goal.

The role of experience in the setting of secular goals is clear. A goal can be established only on the basis of what can be imagined, and what can be imagined depends on what one has been exposed to and has learned by personal experience. Moreover, the hedonic associations of the neuronal tokens of experience will depend in part on the particular fortuitous coincidences and sequences of the components of experience. In the framework of the model, these associative

adiant and abiant links will determine whether a potential secular goal can be instantiated and sustained.

Motivational Biasing of Attention

The hedonic induction of an energetic set point (a labeled autaptic cell) can determine the selection of a plan of action aimed at getting the goal represented by the set point (the biological substrate of motivated behavior). There is, however, another important aspect of motivation in the realm of both appetitive and secular goals: we pay attention to those things that meet our needs.

During the course of daily experience, out of the enormous number of objects and events that stimulate our senses, we respond selectively with a special sensitivity to relatively few. These are the objects and events that must be discriminated to achieve cognitive homeostasis. According to the model, selective perceptual sensitivity results from the associative priming of filter cells (in synaptic matrices) by the output of those set points that happen to be in a current state of disequilibrium. This represents a mechanism of selective attention that augments the attentional effects of self-locus excursion in retinoid systems.

How can particular motivational states and perceptual sensitivity for particular sensory patterns be adaptively coupled? The neuronal network illustrated in figure 9.6 shows how this can be done. In this example, long-term synaptic changes occur according to the basic learning equation. Axon transfer factor (ATF) has been arbitrarily set at $c = 1$; dendrite transfer factor (DTF) has been set at $k = 12$. The saturation limit in the detection matrix has been arbitrarily set at $Lim = 10$. In the other matrices, the setting is $Lim = 2$. The transfer weight (ϕ) at each learning-modified synaptic junction is displayed to the nearest whole number.

Consider the synaptic matrix in the left-hand half of figure 9.6. Five different sensory patterns (objects) have been learned. The presence of each object is signaled by the output of its associated class cell (Ω). The main axon of each class cell token branches to compose two collateral output bundles. One bundle courses back to the imaging matrix; the other carries excitation to a matrix (shown in the right-hand half of figure 9.6) designated O/M Association because it selectively links objects (O) to motives (M). The filter cells in this matrix receive input from three sources: (1) the object-token outputs ($Ob1, Ob2, \dots, Obn$) from the module for learning and detecting exteroceptive patterns, (2) the output from motivational set points in the brain (G_i), and (3) the output from those pleasure centers (Pl) that

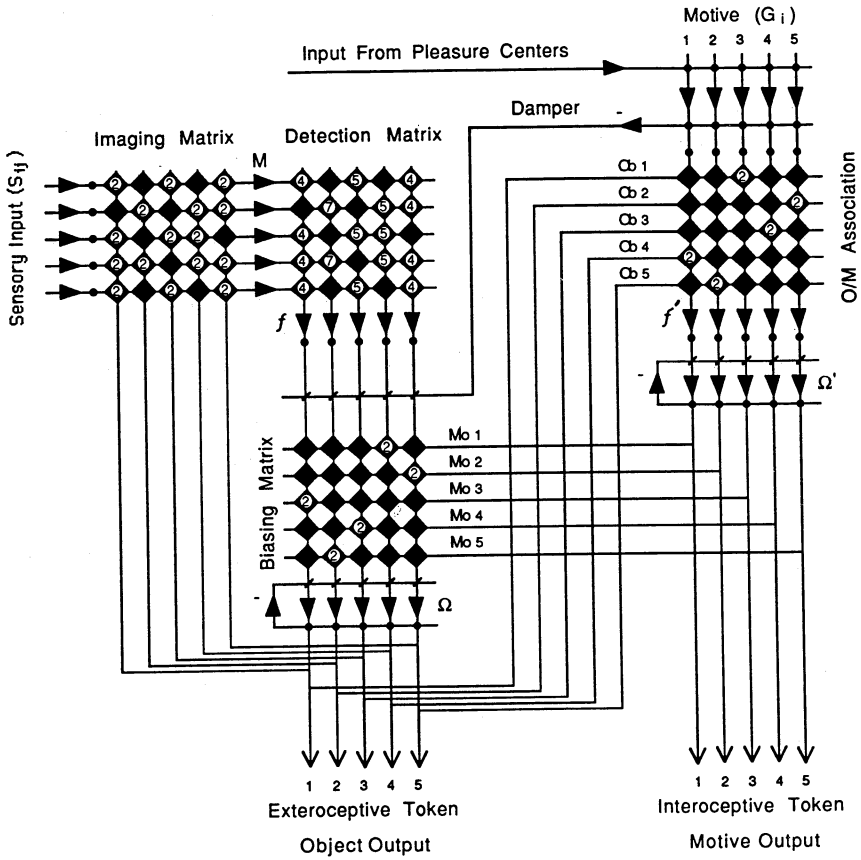


Figure 9.6
Neuronal network that selectively biases perceptual sensitivity in accordance with the strength of a dominant goal.

are activated during the restoration of homeostasis in an energetic motivational system.

Input from motivational set points (G_i) alone is insufficient to fire filter cells (f') in the O/M matrix. Only if there is concurrent biasing input from a pleasure center (Pl) can set point inputs (G_i) discharge their coupled filter cells. Thus Pl is a gating signal distributed to all f' in the matrix. The frequency of spike discharge for any f' is a joint monotonic function of the level of input from its coupled G_i and the level of concurrent input from Pl . As in other detection matrices, if there is simultaneous input from several motivational set points (and if the matrix is gated above threshold by activity from Pl), the class cell (Ω'_i) that is connected to the filter cell (f') receiving input from the most energetic G_i will fire first and inhibit the discharge of competing class cells. Thus, an active class cell in this module is a token of an individual's strongest current motivational/goal-directed state.

Two kinds of output from the O/M matrix feed back to all class cells of the object-detection matrix. One is inhibitory through fixed synapses (see the cell labeled *Damper* in figure 9.6) and serves to raise the threshold (decrease sensitivity) of the class cells (Ω). The other feedback is excitatory through adaptive synapses (Biasing Matrix in figure 9.6) and serves to couple synaptically the token of a currently dominant motive with the tokens of objects that are at the focus of attention during motivational homeostasis.

Imagine the network without the O/M matrix. The distribution of synaptic weights (ϕ) in the biasing matrix would be at a uniform basal level, and learning and recognition of environmental objects would occur without motivational bias. This would also be true if the O/M matrix were connected as shown in figure 9.6 but without prior concurrent discharge of class cells (Ω) evoked by objects (S_{ij}) and class cells (Ω') evoked by the gratification of motives (G_i). Suppose, however, that a number of objects had been learned and that among these was one (say Ob1) critical for the instrumental behavior needed to gratify a motive (say Mo3). It is assumed that when G_3 is energized and dominant, it will evoke instrumental behavior aimed at homeostasis. In the course of behavior that restores equilibrium, objects with utility relevant to the current state of motivation will tend to be at the focus of perception and will evoke the discharge of their corresponding class cell tokens. Thus, we can assume a high likelihood that the token for Ob1 (Ω_1) will fire at some time during the discharge of the token for Mo3 (Ω'_3). This will result in an adaptive change in the biasing matrix at just that synaptic junction between the axon of Ω'_3 and the dendrite of Ω_1 . Thereafter, whenever G_3 evokes the discharge of Ω'_3 , class cell Ω_1 in the sensory detection

matrix will receive an excitatory bias through its enhanced synapse with Ω'_3 . At the same time, all class cells in the sensory detection matrix will receive an inhibitory bias from the damper cell. It is assumed that the inhibitory bias to all class cells in this matrix is less than the excitatory bias to the cell selectively coupled to Ω'_3 .

The general effect, then, is this: when a dominant motive controls behavior that is instrumental in need gratification, there is an automatic and selective facilitation of neuronal response to those objects that have utility in relation to the current motive, and there is an inhibition or dampening of neuronal response to all other objects that are not relevant to the current motive (Moran and Desimone 1985). By neuronal mechanisms like the one proposed in the circuit diagram of figure 9.6, attention can be selectively focused on stimulus patterns (veridical or imagined) important to an individual's governing motives.

The Resolution of Goals

A person will have many different secular goals simultaneously. The output of these energetic set points (G_i) will wax and wane independently according to the momentary relative magnitudes of adiant and abiant input to each. Given this multiplicity of current goals, how can goal-directed behavior be adaptively gated and modulated? What determines which goal will govern during any arbitrary epoch? It would be maladaptive if the various activities motivated by the repertoire of one's goals were unrelated to the perceived or anticipated (imagined) properties of current or future environmental contexts. In situations where there are no affordances that can lead to the achievement of a particular goal (say G_1), persistence of behavior directed toward G_1 would be ineffective and preclude the attainment of other goals.

If the coupling between goals and motor behavior were modulated by the perception of relevant affordances, there would be an increased likelihood that the pursuit of a specific goal will be successful. According to this design, the competition among many different energetic goals to capture and control overt behavior would be resolved on the joint basis of the current relative strength (demand) of each goal and the presence of those affordances needed to continue on a path to a given goal.

From moment to moment, behavior is controlled by plans of action that are initiated and sustained by the most powerful current motive (the G_i with the highest discharge frequency) unless the means for pursuing the goal are initially absent or have become unavailable.

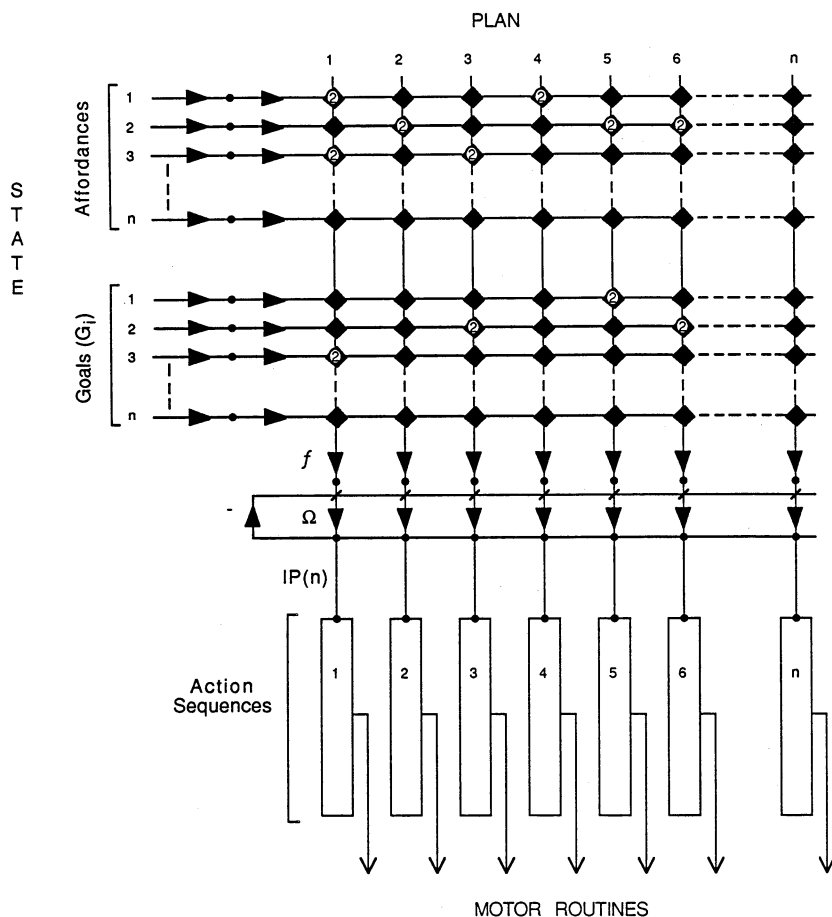


Figure 9.7

Neuronal matrix that selectively activates plans of action in accordance with recognized affordances and the strength profile of current goals. Each numbered box represents a mechanism similar to the one shown in figure 8.3 for sequencing a series of goal-related actions. In this module, activation of filter cells (f) by either goals or affordances alone is insufficient to exceed the threshold of their coupled class cells (Ω). Only joint excitation of a filter cell by both kinds of input can fire its paired class cell.

When this happens, supporting tokens of critical affordances are no longer active, and the current peak of motivational excitation alone is insufficient to dominate class cell output. Control of behavior is then captured by the strongest motive having the supporting bias of available affordances. The pursuit of goals that have been interrupted can be resumed at the proper step in the sequence of situation-action transitions when the appropriate affordances are encountered. Of course, behavior governed by secular goals will also be periodically interrupted by more urgent appetitive needs (hunger, thirst) and harm-avoiding action evoked by abiant signals.

Figure 9.7 shows a schematic of a neuronal matrix that is stimulated in parallel by active tokens of recognized affordances and the outputs of the motivating autaptic set points (G_i) that represent the relative strengths of current goals. Given the particular distribution of synaptic weights shown in this example, it is possible to determine the governing goals and their associated plans that will be induced under a variety of state contingencies.

A sampling of possible goal-affordance conditions and their outcomes is presented in table 9.1. In accordance with figure 9.7, there are three goals and three affordances. For each of the 22 states in this example, the values within the cells for the goals represent the relative discharge frequencies of the set points of the goals (their relative motivational strengths). Cells that represent affordances have a value of 1 if the designated affordance is recognized; otherwise the cell has a value of 0. The bottom two rows of table 9.1 show which goal controls behavior and which plan is evoked under the designated state. With identical patterns of motivation, different goals can con-

Table 9.1
Output of the network shown in figure 9.7

		STATE																					
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
Goal 1		5	5	5	6	6	6	3	3	3	4	4	4	5	5	5	9	9	9	1	1	1	5
Goal 2		3	3	3	7	7	7	5	5	5	2	2	2	8	8	8	3	3	3	3	3	3	0
Goal 3		2	2	2	9	9	9	4	4	4	8	8	8	2	2	2	1	1	1	9	9	9	0
Affordance 1		1	0	0	1	0	0	1	0	0	1	0	0	1	1	0	1	1	0	1	1	0	1
Affordance 2		0	1	0	0	1	0	0	1	0	0	1	0	1	0	1	1	0	1	1	0	1	0
Affordance 3		0	0	1	0	0	1	0	0	1	0	0	1	0	1	1	0	1	1	0	1	0	1
Controlling Goal		3	1	2	3	2	3	3	2	2	3	1	3	2	2	2	1	2	2	3	3	2	*
Plan Evoked		1	5	3	1	6	1	1	6	3	1	5	1	6	3	3/6	5	3	3/6	1	1	6	*

* = none

trol behavior and evoke different plans because of the recognition of different affordances. Moreover, the same controlling goal can evoke different plans depending on which affordances are recognized (states 8 and 9). Under some conditions, a controlling goal can evoke one of a number of appropriate plans with equal probability (state 15). In such a case, the final selection among the suitable plans would depend on chance or on biasing factors outside the mechanisms considered here. Finally, even if there is an energetic goal having no competition from other goals, it will not control behavior, and no plan of action will be evoked without the recognition of a related affordance (state 22).

The examples presented derive from just six state parameters. If similar mechanisms govern secular motivation in the human brain, it is clear that idiosyncratic behavioral contingencies of great depth and complexity will evolve as the number of goals and affordances increases and as adiant and abiant processes ramify through learning in the course of personal development.