Chapter **5**

Biological Motives: Integration by the Nervous System

The axon, so far as we know, does not think.
It only ax.

—GEORGE BISHOP

In previous chapters, we have introduced some of the complexities of motivational systems. They are responsive to multiple inputs, and expressed by multiple outputs, at each level of a hierarchical control system. Internal states affect them. External stimuli affect them. Internal states affect how external stimuli are reacted to, and conversely. And beyond that, an individual's learning history—especially the course of instruction a culture provides for its members—modulates the effects of inputs, the expression of outputs, and the relations between the one and the other.

Even the simplest biological motives must put a formidable number of pieces together. All these influences must be taken into account and translated into action. In all but the simplest animals, that job of integration and translation is done by the *nervous system.*

THE NERVOUS SYSTEM

The cells of the nervous system are the message lines of the body. They permit rapid communication between one part of the body and another,

so that the body as a whole can take effective action. They permit the left hand to know what the right hand is doing—both literally and figuratively.

Nerve Cells and Synapses

Nerve cells, or **neurons**, come in a wide variety of sizes and shapes. Figure 5-1 shows a selection. These are the nerve cells involved in a simple *spinal reflex,* as described below. The thick arrows show the direction in which messages, or nerve impulses, travel from the sensory neuron across the synapse to the motor neuron and from there to the muscle.

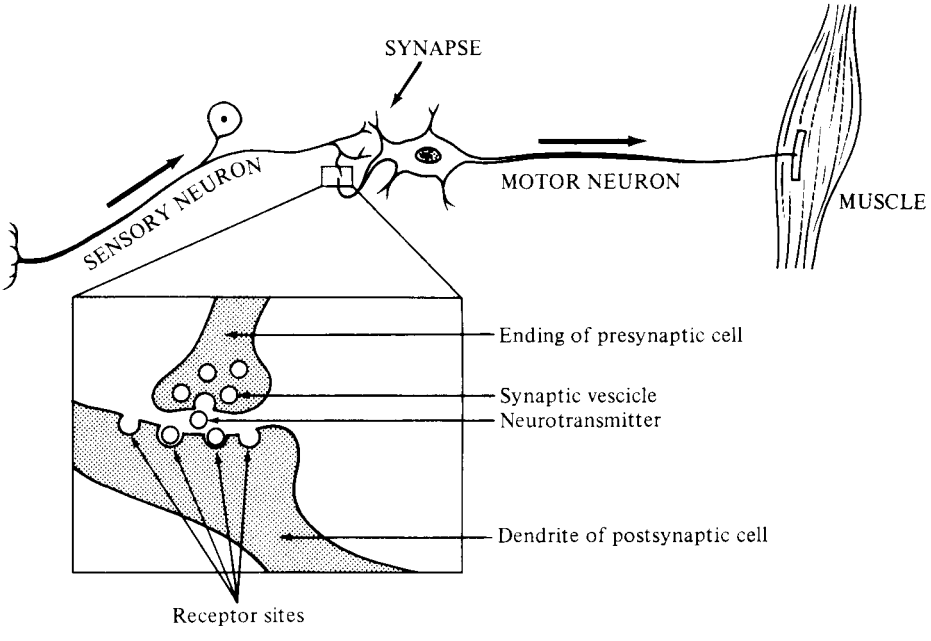


Figure 5-1.
Two neurons and the synapse through which the sensory neuron affects the motor neuron.

Nerve cells communicate with each other across a structure known as a **synapse**, which comes from a Greek word meaning "sitting together and holding hands." The sequence begins with the fingers or *endings* of the presynaptic cell—that is, the cell before the synapse in the path the nerve impulses take. In this case it is the sensory neuron. At the synapse, other fingers grasp the postsynaptic cell—that is, the cell after the synapse. Here it is the motor neuron carrying messages out to the muscle.

As nerve impulses reach the presynaptic endings, they cause opening of the **vescicles** in which packets of chemicals are stored. These chemicals are the **neurotransmitters**, and they carry the messages from one cell to

the next. Once released from their vesicles, they diffuse over to the postsynaptic membrane, where they affect the activity of the postsynaptic cell.

Affect it how? There are two things these neurotransmitters may do. At some synapses, they *excite* the postsynaptic cell. They cause it to generate nerve impulses of its own, or make it more likely to do so. That is how a message is relayed from nerve cell to nerve cell. It is also how nerve cells can add their effects. If two cells converge on a postsynaptic cell, then input from the two of them together may produce more activity in that postsynaptic cell than either alone would do. This is known as summation, or **potentiation.**

At other synapses, the neurotransmitter *inhibits* the postsynaptic cell. It prevents it from generating nerve impulses, or makes it generate fewer. That is how one cell may shush another cell, silencing what it might otherwise say.

We notice that the list of a nerve cell's capabilities is not very long. A nerve cell's message can excite or inhibit the next cell in the chain. That's all it can do—and each synapse does either one or the other. And yet, these simple elements with their simple functions must somehow be able to generate all the subtlety and complexity of behavior. It is a dramatic study in how much can be done with how little.

PRINCIPLES OF NERVOUS SYSTEM FUNCTION

We have seen that what nerve cells do is excite, or inhibit, other cells. These provide the arithmetic of the nervous system—addition and subtraction, respectively. Further complexity and versatility arise from the geometry of the system. One input can trigger a *diverging* system of connections so as to have multiple effects. Or, different inputs can *converge* on a single output so that any or all of these inputs can affect it.

Let us see some examples of how these principles operate in producing behavior.

Inhibition and Excitation: Feeding in the Fly

The analysis of feeding behavior in the fly, strange as it seems, has set the standard for investigation of feeding mechanisms in other species. This is partly because of the elegance of the experimental analysis by Vincent Dethier and his colleagues,[1] but partly too because it shows just how engagingly simple a biological control system can be.

The next time you have a fly as a dinner guest, watch it carefully before chasing it away. Spill a few drops of something sugary on the table where the fly can find it. As it walks along, it probably will encounter your gift with one or the other of its forefeet. Then the feet on that side will stop moving, because movement is inhibited, while the legs on the other side

[1]See Dethier, 1969, 1976.

foot from a pinprick. This occurs because the nerve impulses, traveling from the feet up the sensory nerves, cause excitation of motor nerve cells that in turn drive the proboscis-extension muscles (Figure 5-2A).

This reflex occurs if the fly is hungry. But when is the fly hungry? To make a long story short,[2] the fly is hungry when this feeding reflex is not *inhibited.* If the fly has recently eaten its fill, then the food inside its digestive tract stimulates internal receptors that trigger nerve impulses in another set of nerve cells. These impulses *cancel the excitation provided by the taste input* (Figure 5-2B). Then, even if taste input is there, the fly stubbornly refuses to extend its proboscis and eat. Such a fly is satiated. Of course, once food has left the crucial place in the digestive tract, the inhibition is removed and the fly will be ready to feed again.

Here we have a system that can cause the fly to orient toward food and eat it, when—and only when—the fly has not recently fed. The cycle of hunger and satiety, the process of food identification, the initiation and maintenance of the feeding response itself—all this could be provided for with only that much machinery.* The body can do a great deal with very little, and the fly, a creature of reflex, gets around in the world very well indeed.

Divergence: Reflex Patterns

We now have some grasp of the basic functions of nerve cells—excitation and inhibition, the arithmetic of the nervous system. Now let us see how important the spatial layout of their interconnections is—the geometry of the nervous system.

When we discussed Descartes' theory of the reflex, we used as example a creature that flexes its leg in response to pain. Think again about such a creature. Suppose it has a four-point stance; it is a dog or a cat, let us say. We pinch its toe, and its leg flexes.

Obviously, if such a creature flexed its leg and did nothing else, it would topple over like a chair with a leg missing. Instead, the painful stimulus causes not only flexion but adjustment of the three other legs as well. The opposite foreleg tenses to take up the weight. So does the hind leg on the same side. The diagonally opposite hind leg flexes, to shift the center of gravity away from the limb that is no longer giving support. The result is a **reflex figure**, involving movements of *all four* legs even though only one foot was stimulated.

Each of these adjustments involves a pattern of contraction of one muscle, and relaxation of the opposing one. Each such pattern in turn reflects a pattern of excitation and inhibition of motor neurons, triggered through pathways in the spinal cord by the original painful stimulus.

[2]Dethier, 1976.
*Though again there is in fact a great deal more; see Dethier, 1976.

Thus reflexes need not be isolated muscle twitches. They can be patterns of movement involving the whole body. These patterns are made possible by the *divergence* of nerve cells and their interconnections. A chain of events that begins with one foot, affects the actions of many.

Convergence and Multiple Control

We have seen that one input can have many outputs in the nervous system. Conversely, one output can be responsive to any or all of many inputs.

Think again about the response to pain in the foot. If we step on a tack, we are likely to flex that leg, and adjust the tension on the other leg so that it bears the added weight (a reflex figure).

That withdrawal reflex can occur without any involvement of the brain. It can be elicited in an animal without a brain, through the interconnections between nerve cells at the spinal cord. But we can make the same flexion movement *voluntarily,* and that does involve the brain. The motor nerve cells that drive the flexor muscles are the same. But this time, it is nerve impulses coming down from the brain that excite those motor nerve cells. In short, we can flex a leg because of a painful stimulus—reflex flexion—*or* because we decide to do so—voluntary flexion. The two possibilities depend on two different communication lines that *converge* on the motor cells.

A Look Backward—and Forward

We have looked over the basic operations that nerve cells perform—excitation and inhibition. We have seen some implications of the spatial arrangements of nerve cells and their interconnections—divergence or multiple outputs, and convergence or multiple inputs.

As we move on to the brain, the thing to remember is that the *same principles continue to apply.* The concepts we have seen in action, in controlling movements, also operate in *whole organized systems* within the brain. Whole systems can be excited or inhibited; whole systems can respond to multiple inputs, with multiple outputs. The levels of complexity are vastly different; but the principles are the same.

Indeed, in looking back over this section, we recognize some familiar ideas. The nervous system provides communication lines by which multiple inputs can affect a single output. And we remember that motivational states, like hunger and thirst, also are affected by multiple inputs. The nervous system provides for divergence, or multiple outputs from a single input. Well, a motivational state can be expressed by different specific actions—it has multiple outputs! And we treated *satiety* as a mechanism that *inhibits* a hunger or a thirst system.

Let us look at how the brain performs these operations at the level of organized action systems.

THE CENTRAL NERVOUS SYSTEM

The **central nervous system** consists of the brain and the spinal cord (Figure 5-3). These structures are densely packed collections of nerve cells and cells of other kinds. We do not show these cells individually, for there are about 100 billion of them in the human nervous system.

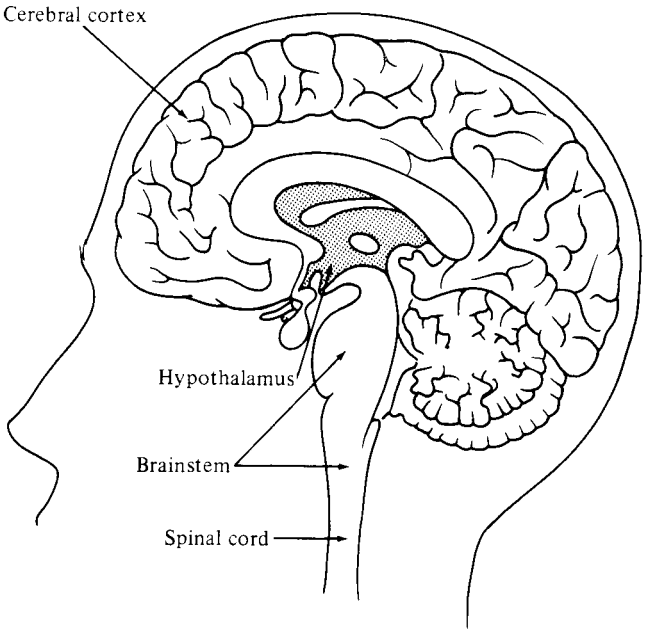


Figure 5-3.
The human central nervous system, seen from the midline out.

In Figure 5-3, the human brain is shown in side view, from the middle out. In other words, imagine that someone has sliced the brain down the middle, from front to back, and that we are looking at the cut. The shaded area shows a cavity in the middle, one of the **ventricles** of the brain. These cavities are filled with **cerebro-spinal fluid**, a kind of filtered blood plasma that can provide the brain with information about the composition of body fluids. The lower wall of that cavity, on each side, consists of clusters of nerve cells known collectively as the **hypothalamus**.

The entire system is a tube-shaped affair, closed at the top, with its fluid-filled central cavity. The figure shows the conventional divisions of the nervous system, which break it up into a series of *levels,* bottom to top. In the human brain especially, the structure appears to get more and more complex as we move upward. It does; and with that increase in anatomical complexity comes increasingly complex control over behavior.

LEVELS OF INTEGRATION IN THE NERVOUS SYSTEM

Now let's look at the *hierarchical organization* of control over movement. We will see how the brain directs action in the environment, and integrates individual movements into *motivational states.*

Bard's Experiments: Hierarchical Organization

A classic series of experiments by the physiologist Philip Bard and his colleagues[3] has shown the *hierarchical organization* of the mammalian nervous system. The principle is: *Higher parts of the brain coordinate and direct the simpler components of action,* which themselves are controlled by levels farther down in the nervous system.

Figure 5-4 presents a highly schematized layout of the mammalian nervous system. It shows that the motor nerve cells, which drive skeletal muscles in mammals, emerge from the spinal cord.* Above the spinal cord we find the brain with its subdivisions. Its influence on movement

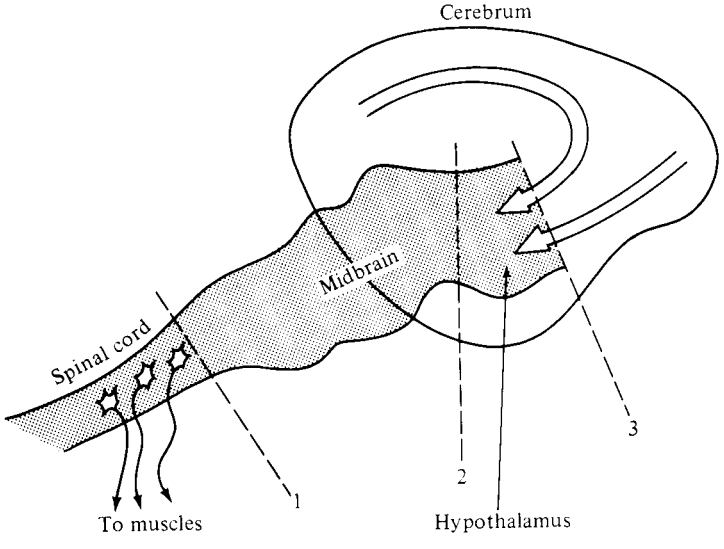


Figure 5-4.
Schematic diagram of the cat central nervous system, showing levels of transection in Bard's experiments. The motor cells that drive the muscles emerge from the spinal cord. The cerebral hemispheres ("cerebrum") fold back over the brainstem on each side; their descending influences funnel down through the brainstem to affect the motor cells, as indicated by the open arrows. (From Gallistel, 1980.)

[3]Bard and Rioch, 1937.
*There are exceptions to this that we will ignore.

is exerted by way of those motor cells that run out from the cord. In other words, the motor cells control the muscles directly, whereas the brain controls the muscles indirectly by controlling the motor cells.

It follows that if we *transect* the nervous system—simply cut it across—we will leave all structures *above the cut* out of communication with the motor cells, and hence unable to influence movement. Therefore, what we see in the behavior of the operated animal is the influence of structures *below the level of the transection.*

The Spinal Cord: Reflex Movements

Suppose then that the system is cut across at Level 1 in the figure. Now the brain is for practical purposes removed as an influence on behavior. The motor cells can only be affected by information coming from the sensory cells that enter the cord below the cut, and synapse with motor neurons there. Movements produced by that part of the system are called **spinal reflexes**, because they involve only the spinal cord, not the brain. The reflex withdrawal from a painful stimulus is such a reflex.

After transection at Level 1, then, the animal should be capable only of simple spinal-reflex responses to stimulation. That is exactly what Bard saw, using cats as experimental subjects. Once they had recovered from the operation, cats with Level 1 transactions were capable of local reflex movements. Prick the paw, and the limb would flex. Stretch the extensor muscle in one leg, and the leg would extend; this is the *extensor reflex.* That reflex is an important component of *standing,* and the Level 1 cat can make the movements involved in standing in each of its limbs individually. But it cannot put the individual movements together. It cannot stand up.

Some of the reflexes that remain, by the way, are specifically associated with higher-order systems. When its genitals are stimulated, a female cat in heat will respond by deflecting its tail to one side. That reflex is part of its mating pattern. A Level 1 cat retains this reflex. And, an interesting point to which we will return, it makes that response to genital stimulation whether it is in heat or not.

The Higher Brainstem: Movement Patterns

If one makes no cut at Level 1, but severs the nervous system at Level 2 instead, the picture is different. One now has a cat that can stand, and even walk. What remains lacking, however, is any adjustment of the movements to external or internal circumstances. The cat continues to deflect its tail upon genital stimulation, and it now may make the treading movements and arching of the back characteristic of the female in heat. But it does these things whether or not its hormonal state is one of heat. It does not approach males. It does not approach food. It can walk, but it walks without apparent purpose. It does not walk to or away from anything.

THE HYPOTHALAMUS: DIRECTED MOVEMENTS AND INTERNAL CONTROL

If the transection is still higher (Level 3), the result is different again. In this case, the cat does combine fragmentary acts into organized behavioral sequences. Thus, when its genitals are stimulated, such a cat displays the full-blown mating pattern, with loud growling, lowering the head and raising the rump, vigorous treading, and tail deflection. And—another distinct difference—the cat displays that pattern when its hormonal state is one of heat, and only then. Therefore, the pattern is controlled by hormonal state in a Level 3 cat, as in an intact one.

Strikingly similar findings were obtained with feeding behavior. A Level 2 cat could chew and swallow food placed in its mouth, but it never sought food, and it would chew and swallow the food whether it was starved or fully fed. A Level 3 cat would sniff, lick, and explore the floor with its mouth if food was placed in front of it—but only if it was hungry. And the longer it had been without food, the more vigorously it would explore. Clearly, with Level 3 transection, fragmentary movements were organized into effective locomotion and search, and the whole pattern was under the influence of nutritional status.

Also in Level 3 cats, emotional expression changed from isolated fragments to organized patterns. A pinprick on the paw would elicit reflex flexion in a Level 1 cat. A Level 2 cat might look angry or afraid, but would not withdraw or attack effectively. But a Level 3 cat would show the whole integrated pattern of rage, with crouching, hissing, erection of the hair, and striking movements of the forepaws with claws extended.

A Look Backward: Levels of Control

In short, when the hypothalamus and adjacent structures are in communication with the muscles, two things are present that otherwise are absent. First, isolated movements are coordinated into organized behavior patterns and sequences—some writers call them *motivated* behavior sequences. It is tempting to see this as an example of *divergence* in the systems running down from the hypothalamus. Without it, fragments of behavior can be elicited *one by one.* But with it, descending systems diverge to excite *all the fragments at once* (Figure 5-5).

Second, along with the integration of movements into movement patterns, we see control of these patterns by *internal state.* With the hypothalamus in the system, the sexual patterns are responsive to hormones; the food-seeking and feeding patterns are responsive to food deprivation. Without it, they are not.

We should note the definite parallels between levels of neural organization as discovered by Bard's work, the levels of organization of instinctive behavior suggested by Tinbergen (pp. 103–104), and the concept of hierarchical *motivational states* that we developed earlier. Lower-level movements—movements of individual legs in mammals or of fins in fishes—require only lower levels of nervous integration. Movement patterns—

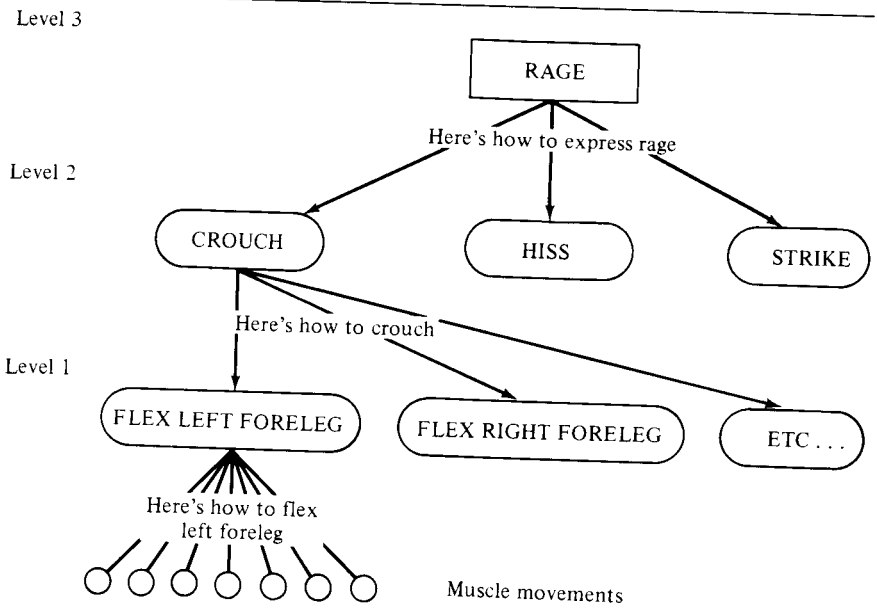


Figure 5-5.
Hierarchical organization of control of movement by the nervous system. Each level combines elements or subsystems organized at lower levels.

action systems at intermediate levels, such as standing or walking—require that the midbrain be intact. Patterned sequences of behavior as in aggression or mating, adjusted to external *and* internal circumstances, are seen only if still higher levels of the brain remain in the controlling system.

Conversely, as we take more and more brain out of the system from the top down, the behavior falls apart. But what falls apart is the *organization* of components into patterns; the components themselves may remain. The organization must therefore be imposed by those higher levels.

DRIVES AND THE HYPOTHALAMUS

In looking over Bard's findings, we notice that the presence or absence of the *hypothalamus* makes a great difference in the organization of many forms of behavior. Since that time, much research in motivation has been concerned with that small bit of brain. It is a nexus within which multiple influences converge.

The next step was made possible by a technical advance. By the late 1930s, scientists had developed techniques that permitted manipulation of small clusters of cells deep in the brain. Small areas of damage, or *lesions,* could be made within the brain and their effects on behavior observed. Or one could place electrodes deep in the brain, fix them in place there, and later stimulate clusters of cells electrically in the con-

scious, awake animal. Finally, one could apply chemical stimuli—drugs, hormones, or neurotransmitters—to local sites within the brain.

The results of such studies led to an influential theory about how drives are mediated by the brain. Let us look first at the theory, and then at some of the findings.

Stellar's Dual-Center Theory

In 1954, Eliot Stellar presented a synthesis of the findings up to that time, and a theory of the role of the hypothalamus in motivation.[4] The basic idea (Figure 5-6) is that motivated behavior reflects the output of **excitatory centers** located in the hypothalamus. These centers, or clusters of cells, are affected by sensory stimuli, whose messages are transmitted to the hypothalamus from elsewhere in the brain; and by stimuli arising from within the body, and carried by the blood and cerebro-spinal fluid. The output of these excitatory cells is directed to other parts of the brain to produce motivated behavior. The consequences of that behavior influence the internal and external stimuli that began the process—the familiar *feedback loop.*

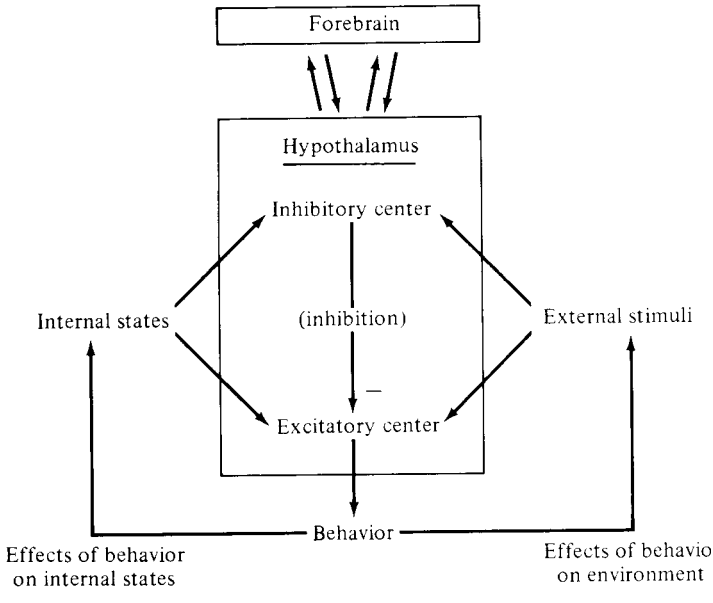


Figure 5-6.
Stellar's theory of brain mechanisms in motivation. Behavior is produced by the activity of *excitatory centers* in the hypothalamus. These centers are held in check by *inhibitory centers.* Both centers are affected in turn by conditions inside and outside the body, and by still higher systems in the forebrain.

[4]Stellar, 1954.

The activity of these excitatory centers is modulated by **inhibitory centers** that suppress them. These in turn receive multiple inputs from inside and outside the body. Thus the behavior that occurs is jointly influenced by inputs to the excitatory center that excites that behavior, and inputs to the inhibitory center that holds it in check. This already has a familiar ring, for we saw earlier that arousal of hunger and thirst (excitation) and their respective satieties (inhibition) both have multiple inputs (Chapter 3).

Notice one thing more before we go on. These excitatory centers are seen as exciting, or calling into play, whole organized motivational hierarchies *as units.* And when the inhibitory centers come into play, they exert their inhibition on entire hierarchies, again as units. This is the idea we looked at earlier: The principles of nerve-cell interaction, such as excitation and inhibition, also apply to *whole organized systems* of behavior.

Since Stellar wrote this paper, the picture has had to be modified in many ways, and many more details are known. Still, it remains a useful way of organizing our thoughts about motivation and the brain. Let us see how the data for specific motives fit into this framework.

Thirst

We saw earlier that there are two stimuli for thirst: dehydration inside the cells of the body, and dehydration outside them. It turns out that the two have different brain mechanisms, as well as different arousing conditions.

Excitation of Intracellular Thirst

Gilman's original experiments dehydrated all the cells in the body by injection of strong salt solutions. In an elegant series of experiments, Elliott Blass and Alan Epstein[5] showed that there are certain cells that signal their own dehydration, and thus serve as **receptors**—receiving stations—for the cell-dehydration signal.

The receptors are in the **lateral preoptic area**, which lies just forward of the hypothalamus. Blass and Epstein first showed that localized lesions in this area could produce a rat that no longer drank in response to injections of salt solutions. The rat did drink in response to extracellular-fluid reduction—the other stimulus for thirst—showing that it was capable of drinking. It was only the intracellular stimulus for thirst that it no longer reacted to.

Then Blass and Epstein implanted cannulas in the brain, through which they could inject minute amounts of solution directly into the preoptic area. These injections were so small that they affected only the cells immediately surrounding the cannula tip. A fully hydrated rat,

[5]Blass and Epstein, 1971; see also Peck and Novin, 1971.

Inhibition of Thirst

We have two excitatory systems, then, one for each thirst. What about inhibitory systems? One has been found.

Damage to the **septal area** of the brain, another structure just forward of the hypothalamus, produces a rat that drinks *too much* water.[9] The inhibition here is exerted specifically on the systems that respond to angiotensin, for the overdrinking is seen only when angiotensin injections, or manipulations that activate the renin-angiotensin system, are used to stimulate drinking. If cell dehydration is used to evoke drinking, the amounts taken in by rats with septal damage are quite normal. We have, then, not an *inhibitory center* for thirst as such, but something more specific—an inhibitory system, involving the septal area, that clamps down specifically on drinking stimulated by angiotensin.

Sexual Behavior

We saw in Chapter 4 how, in some species, adult sexual behavior is dependent on hormones. Castrated male rats gradually lose sexual interest, and spayed female rats do so abruptly and totally. Presumably the presence of these hormones in the blood is somehow conveyed to the brain, which in turn organizes and mobilizes the mating behavior itself.

In fact, the functioning of certain bits of brain tissue is quite critical for these hormone effects—and sure enough, the critical locations are in or near the hypothalamus. Mating in female guinea pigs, after it has been abolished by removal of the ovaries, can be restored by injection of estrogen—but only if the hypothalamus is intact. Lesions in the anterior part of the hypothalamus block mating even if estrogen is injected. In male rats, lesions in the preoptic area, forward of the hypothalamus, abolish mating behavior, and testosterone injections do not restore it.

Conversely, we can administer hormones to the brain through cannulas, to stimulate only the cells around the cannula tip. If these cells respond to hormones by promoting mating, then hormonal stimulation at just those sites should be enough to restore mating in spayed or castrated animals. And that is exactly what happens, in females and in males.[10] The whole story is very similar to the thirst one, summarized above. The animal's body may have no sex hormones in it; but if *these* brain cells see hormones, they say to the rest of the brain, "Sex hormones here!" And the brain knows just what to do about that.

It may well be that naturally occurring hormones act on these same brain sites to promote mating behavior. If so, then we have *excitatory* systems in the brain that couple mating behavior to the animal's hormonal state, just as other systems make drinking behavior sensitive to the state of hydration.

[9]Blass, Nussbaum, and Hanson, 1974.
[10]Harris and Michael, 1964; Davidson, 1966.

brain to excite cells artificially. When such stimuli were applied to the lateral hypothalamus, fully satiated rats could be made to eat vigorously, and even to get fat if stimulated often enough.[14]

It all seemed to make a simple and coherent picture. The stimuli for hunger, whatever they are, may act directly or indirectly on cells in the lateral hypothalamus. Perhaps these cells are the generals, who send down a command that tells the body-moving field officers in the midbrain, "Eat!" or "Eat until further notice!" Then the meal ends when the satiety signals activate ventromedial cells. These report, "Mission accomplished!" to the feeding cells; the command to eat ceases, and the animal can go on to other business. It seemed so straightforward that, for a while, researchers spoke of a lateral hypothalamic "feeding center" and a ventromedial "satiety center."

Unfortunately the situation is not so simple as all that; and while the basic idea may hold—an interplay of excitatory and inhibitory systems—few people talk about feeding centers and satiety centers any more. But before we look at the complications, let's explore the general theory further.

Brain and Environment: Modulation of Stimulus Effects

Stellar's dual-center theory, like Bard's work on hierarchical organization, sees the hypothalamus as combining fragmentary response systems, organized at lower levels, and calling them into play as units. How does the system do that? How does it produce movement patterns?

The most likely answer is the one we are familiar with. Systems higher in the brain may prime, or *potentiate,* systems that are in turn responsive to *external* stimuli. Let us see some examples.

Attack Reflexes and Summation

Electrical stimulation of the lateral hypothalamus can induce cats to attack rats or other small animals. It does not just produce the attack movements; if stimulated in an empty cage, the cat will show circling movements and general arousal, but no attack. Present a rat, however, and the cat will attack it at once. The stimulation primes attack, but there must be an attackable stimulus.

J. P. Flynn and his colleagues have investigated the separate movements involved in the attack sequence.[15] For example, when the hypothalamus is stimulated, a touch on the lips will cause head turning and biting. Stimulation of the hypothalamus alone does not produce this; touch alone does not produce it; but the two together do. There must be *potentiation* somewhere in the system.

More convincing still, there is a trade-off between intensity of hypo-

[14]Hoebel and Thompson, 1969.
[15]MacDonnell and Flynn, 1966; Flynn et al., 1970.

thalamic stimulation and adequacy of the touch stimulation; if one is weak, increasing the other will compensate for the weakness. If the current is strong, then a touch anywhere on the cheek or snout, around the mouth, will provoke that response. If the stimulating current is weak, then it takes a touch right on the lip to provoke the biting response. A touch elsewhere will not do. Clearly, the effect of the stimulation is to widen the range of *external* stimuli that are effective in eliciting biting.

Sexual Behavior: The Lordosis Reflex and Disinhibition

A more complex example is the *lordosis reflex*—part of the female mating pattern in rats, cats, and many other mammals. It consists of elevation of the rump, with deflection of the tail to one side.

Now this reflex is elicited by touch on the female's flanks, normally provided by the male. But there is more. The reflex will not occur without the touch; but in the intact cat or rat, even *with* the touch, it will not occur unless the hypothalamus is stimulated by estrogen. Donald Pfaff[16] and his co-workers have traced the pathways through which cells in the hypothalamus, stimulated by estrogen, send messages that are relayed down into the spinal cord, to affect the lordosis-reflex mechanisms that are located there.

Thus far, the picture looks much like Flynn's: External stimuli summing with descending influences from the hypothalamus, to evoke the response. There is a complication, however. We recall that in Bard's cats with transection at Level 1 or 2, the mating reflexes could be elicited by touch, whether or not estrogen was present. In Level 3 cats with the hypothalamus connected to the system, the reflexes could be elicited *only* if estrogen was present. At other times, the same touch would elicit violent rage instead.

The simplest explanation is to suppose that in the intact cat, descending messages from the hypothalamus are *inhibitory.* Their role is not to prime the lordosis reflex when estrogen is present, but to *suppress* that reflex when estrogen is *not* present. Then we can think of estrogen as producing *disinhibition*—literally, it inhibits the inhibitors, directly or indirectly—thus allowing the reflex to occur when its external stimulus, touch, occurs. If this is so, we see again the complex interplay of *excitation* and *inhibition* in behavioral organization.

The Lateral Hypothalamic Syndrome

We noted earlier that damage to the lateral hypothalamus produces failure to eat or drink in rats. But if such a rat is kept alive by force-feeding and hydration, one will see a gradual recovery of ingestive behavior. Philip Teitelbaum and Alan Epstein[17] traced the course of this recovery.

Immediately after the operation, the rat simply refuses to eat or drink

[16]Pfaff, 1982.
[17]Teitelbaum and Epstein, 1962.

anything at all. After a while, however, it will begin to nibble at wet, highly palatable foods: chocolate chip cookies, eggnog, and the like. It does not eat enough to maintain its weight at this stage, and still has to be force-fed. But later still, the rat will begin to eat larger quantities of food; it will now accept ordinary laboratory food, and it regulates its body weight. However, it still needs to be kept hydrated artificially; it will not drink water.

Eventually, most rats do come to accept water, though they seem to use it more as a means of swallowing dry food than as a response to dehydration.[18] But the rats eat and drink, no longer require special maintenance, and appear grossly normal.

Looking over these findings, we can see the effects of lateral hypothalamic damage as a sudden shrinking, followed by a gradual widening again, of the *range of effective stimuli* for ingestion. At first, even the most potent elicitors are ineffective. Later, powerful stimuli—special treats—will elicit ingestion in these rats. Later still, less powerful ones—laboratory chow and water—will do so. This is exactly what we would expect of a system that *potentiates responses to stimuli* for eating and drinking. If descending messages from the hypothalamus prime the feeding and drinking responses, then the less priming there is, the more powerful the external stimulus has to be. If lateral hypothalamic damage removes that priming, then only very potent stimuli—very attractive foods—will trigger ingestive behavior. If the gradual recovery of function gradually reinstates the priming. then we would expect that less and less powerful external stimuli will be needed to get ingestion going.* That is exactly what we see.

Hypothalamic Stimulation and Feeding Sequences

If we look at the effects of electrical stimulation in the hypothalamus, we see again the effect of brain activity on responsiveness to external stimuli. And this time, we will see how that mechanism provides for the organization of behavioral *sequences* over time.

When the lateral hypothalamus is stimulated, the rat walks over to where food is, seizes a bit of food with its forepaws, bites off a mouthful, and chews and swallows it. The behavior changes over time. But the brain stimulation does not—it is the same throughout the sequence. If the stimulation is constant, why does the behavior change?

A possible though speculative explanation is as follows.[19] We can think of all these responses—walking, seizing, biting, chewing—as components of the rat's food-getting hierarchy. Perhaps, therefore, *all* these responses are primed by hypothalamic stimulation. But in addition, each one requires a certain stimulus situation before it can occur (Figure 5-7).

[18]Kissileff, 1969; Kissileff and Epstein, 1969.

*The mechanisms by which recovery occurs are not well understood; for discussion see Kolb and Whishaw, 1985.

[19]See Gallistel, 1980.

Here are two examples, both of which have led us to think again about "feeding" and "satiety" centers. They have shown us that the idea is much too simple.

The Ventromedial Syndrome: Direct or Indirect Effects?

Damage to the ventromedial hypothalamus, we have seen, can produce a rat that eats too much. Our first thought was that feeding has been released from inhibition, and so we spoke of a "satiety center." But other, quite different mechanisms operate too—or instead.

Back in Chapter 3, we looked at some findings that just might revolutionize our views about human obesity. Some people, we realized, may not get fat because they overeat. They may overeat because they are getting fat!

Well, exactly the same suggestion has been made about the overeating rat. Within hours after damage to the ventromedial area, there is an outpouring of insulin from the pancreas. This and other internal changes drive fuel out of the blood, making it unavailable to the brain, and the formation of fat from fuel is enhanced.[20] Perhaps such a rat overeats, not because controls over feeding are damaged, but because the brain really *is* starved for fuel, and responds as it should to that state of affairs.

As it turns out, the insulin effect is probably only part of the picture, for rats overeat after such lesions even if insulin level is artificially held constant.[21] But they overeat less; and besides, there are other metabolic effects of the brain damage that also could act back on the feeding system. Any way we look at it, the important point remains: Brain damage, even to a specific area, can have multiple effects, indirect as well as direct; and it can be a challenging experimental task to separate one effect from another.

The Lateral Syndrome: Akinesia and Sensory Neglect

The effects of lateral hypothalamic lesions pose the problem even more dramatically. Such damage produces failure to eat and failure to drink, but it does a great deal more than that. It turns out that lateral hypothalamic damage causes depressed responsiveness to a wide variety of stimuli, not just to food and water.

A rat with lateral hypothalamic damage does not eat and it does not drink; but it also does not attack mice as normal rats do, and it does not orient to sights, touches, and smells as normals do. Such rats may be reluctant to move at all, as if movements were no longer under the control of environmental events and, therefore, seldom occurred. These more global deficits are called **akinesia** (literally, absence of movement) and **sensory neglect**. These symptoms too, like failure to eat and drink, gradually recover over the days or weeks following the operation.[22]

[20]Han and Frohman, 1970; Powley, 1977; Friedman and Stricker, 1976.
[21]Inoue and Bray, 1980.
[22]Marshall, Turner, and Teitelbaum, 1971.

This line of thinking takes us far away from the original notion of a specific disorder in feeding. It is a whole new way of looking at the behavior. A rat with lateral hypothalamic damage may fail to eat, not because hunger has been removed, but because sensory input no longer produces reactions—to *any* object in the environment.[24]

Centers or Throughways?

Thus far we have focused on problems in interpreting the *behavior* we observe. If a rat doesn't eat, is it absence of hunger or absence of arousability? We face similar problems in relating the behavioral changes to the brain systems we assume to underlie them. If we are not sure *what* is happening, we're also not sure *where* it is happening.

If we look at the anatomy of the lateral hypothalamus and the geometry of its connections, the problem becomes clear. There are indeed synapses in the lateral hypothalamus, where information exchange takes place; but there are also nerve fibers running up and down *through* it, connecting brainstem systems with systems in the forebrain (see Figure 5-10, p. 178). It is quite possible that some of the effects of hypothalamic damage are the result of *disconnecting* lower and higher systems from each other. If so, the systems promoting response to food and other commodities may not be *in* the hypothalamus at all; rather, they may involve communications among far-removed systems, where the communication lines run *through* the hypothalamus.

There is evidence that this is so.[25] An important bundle of nerve fibers runs through this area from the midbrain up into the forebrain, where they release the neurotransmitter *dopamine.* Poisoning these cells, so that they no longer deliver their messages to the forebrain, can also produce failure to eat and drink. But, like lateral hypothalamic damage, it does more than that. It also produces akinesia and sensory neglect.

The challenge to experimenters is to tease apart these multiple effects in multiple systems. What part of the system does what? Returning to feeding deficits: Do they result from damage to the synapses *in* the hypothalamus, or from damage to the fibers running *through* it? Probably both. In one study,[26] a cell poison was injected into the lateral hypothalamus; this damaged the synapses *in* the area, but without destroying fibers running *through* the area. This produced failure to eat and drink, suggesting that synapses specifically concerned with feeding and drinking do exist in that area. Perhaps hypothalamic lesions interfere with locally organized feeding and drinking systems, *and* with connections among more remote areas having to do with general arousability and responsiveness.

[24]Stricker, 1983.
[25]Stricker and Zigmond, 1976.
[26]Grossman et al., 1978; see also Dunnett, Björkland, and Stenevi, 1983.

[redacted]

inhibition of another. A human who sees a tiger, with no cage around it, has more important things to do than eat—however hungry she may be.

Stellar's model helps us to remember the ever-present role of stop mechanisms—inhibitory mechanisms—in motivated behavior.

THE PHYSIOLOGY OF REWARD

In the last section we saw how systems involving the hypothalamus can prime the response to stimuli such as food or touch, once those stimuli are encountered. But animals also *seek* appropriate stimuli, making arbitrary learned responses to obtain them; this, we recall, is how we identify *motivational states* as opposed to reflexes and action patterns. To a hungry rat, food is a *reward,* and the rat will learn what is required to produce it. If we knew how the brain handles learning and reward, we would know a great deal about how the brain produces motivated behavior.

We are a long way from understanding these matters, but we do have a starting point.

Brain Stimulation Reward

In the early 1950s, one of the most exciting discoveries in modern psychobiology was made. James Olds and Peter Milner[27] found that electrical stimulation of certain areas of the rat brain, through implanted electrodes, had reinforcing properties. A rat could be given access to a lever, and by pressing the lever could cause delivery of a brief series of electrical pulses to the brain. If the electrode was in the right place, the rat might press the lever over and over again, for hours at a time.

Reward and Aversion Systems

By varying the placement of electrodes in the brain, and seeing which placements would support this *self-stimulation,* Olds and other investigators were able to map out a *reward system* in the brain. Important sites include our old friend the lateral hypothalamus and the systems of nerve fibers that course into and through it, upward and downward. Additional rewarding sites were found in various structures of the brainstem and forebrain.

Over the same period, Olds and others identified electrode placements where stimulation appeared to be aversive. If pressing a lever delivered stimulation to these area, rats might actively avoid the lever, or they might press a lever to turn stimulation off, rather than to turn it on. The *aversion system* so identified runs closer to the midline than does the reward system, through the hypothalamus and down into the floor of the midbrain. It includes the ventromedial area—the old "satiety center." Stimulation there is highly aversive to rats.

In humans, too, stimulation of the brain in conscious patients can

[27]Olds and Milner, 1954.

evoke feelings of pleasure. These tend to be described vaguely as warm, glowing, good feelings.[28] (Science-fiction writers to the contrary, no wallops of unendurably delicious ecstasy have been reported to date.)

Relation to Natural Motives

We remember that the lateral hypothalamus is an area where electrical stimulation can produce feeding in otherwise satiated rats. It is also an important focus of the reward system. In fact, it is possible to obtain stimulus-bound feeding (with prolonged trains of electrical pulses) and self-stimulation (with briefer trains following a lever-press) through the same electrode in the same rat.[29]

Moreover, the rewarding value of lateral hypothalamic stimulation is affected by some of the same internal factors that affect feeding. If a rat's stomach is full of food, it will not eat spontaneously; it becomes harder to induce it to eat by brain stimulation; and the rate of self-stimulation goes down. Indeed, the previously rewarding stimulation may actually become aversive. A rat that would work to turn the current on when hungry, will work to turn it off when over-fed.[30]

Other experiments have found similar parallels. In the posterior hypothalamus, a bit farther back than the lateral area, electrical stimulation can elicit mating behavior in the male rat, and rats will work to self-stimulate through electrodes implanted there. Sure enough, castration reduces self-stimulation just as it reduces readiness to mate, and treatment with male hormones restores both.[31]

Finally, the effects of deprivation and satiation can be specific to one or another brain site. Filling a rat full of food can depress self-stimulation of the lateral hypothalamus—the old "feeding center"—without affecting self-stimulation through another electrode farther forward in the brain.[32] This tells us at once that the system has multiple components. There is no one reward system; or if there is, it has multiple inputs—convergence again!—that can be affected separately from each other.

Relation to Natural Reinforcers

But how does all this relate to natural motivational states? At first glance, there is a paradox about self-stimulation and stimulus-bound feeding. Stimulation in the lateral hypothalamus can produce feeding, as if it made the rat hungry. But the rat will also work to *produce* stimulation there. Isn't hunger unpleasant? Shouldn't the rat seek to avoid stimulation that mimics hunger?

The paradox disappears if we suppose that the effect of the stimulus is not to produce a drive state, but to set, or mimic, the *reinforcing properties*

[28]Heath, 1964.
[29]Hoebel, 1974.
[30]Hoebel and Thompson, 1969.
[31]Caggiula and Hoebel, 1966.
[32]Hoebel, 1974.

of external stimuli. To a hungry rat, food is a reinforcer. Perhaps the self-stimulation taps into a system that permits, or produces, that reinforcing effect.

An elegantly simple experiment provides support for that idea.[33] Recall that the lateral hypothalamus is a bottleneck through which nerve fibers run up and down in the brain, delivering the neurotransmitter *dopamine* at their terminals. We can block the action of those systems by injecting a drug that blocks dopamine's effects; that way, the cells are undamaged, but the messages they bear do not get through to the rest of the brain.

These experimenters injected such a blocking drug into rats that had been trained to press a lever for food. Then they put the rats in the apparatus and let them work for food as usual. A curious thing happened. Initially, the rats worked for food at their usual high rate. But as the session went on, they responded less and less, as if the lever-press were undergoing extinction through lack of reward.

The simplest interpretation is this. The rats were still hungry, as shown by their initial high rate of response. But the food, when it came, simply wasn't reinforcing any more. The rats discovered as the session went on that the food provided no reinforcement—no pleasure?—and so they quit, just as they would have done if the food itself had stopped coming.*

Reward and External Stimuli: Modulation of Hedonic Response

There are other, quite different lines of evidence that point to the same conclusion: Brain reward and aversion systems modulate *hedonic* responses—responses of pleasure and displeasure, or approach and withdrawal—to external stimuli.

Sensory Neglect and Sensory Rejection

We saw earlier that rats or cats with lateral hypothalamic damage show a decreased responsiveness to stimuli in general. They do not orient to sounds or touches, do not attack mice, and may be reluctant to move at all. This *sensory neglect* can extend to both negative and positive stimuli. Animals with lateral hypothalamic damage may fail to withdraw from a painful stimulus or from an obnoxious odor. They are unresponsive to good *or* bad events.

But there is another common effect of lateral damage, and that is an *exaggerated negative response* to stimuli. Such a rat may gape and turn its head away if even a sweet solution is put in its mouth. It seems to treat *any* stimulation of its mouth as highly aversive. And even after recovery of feeding and drinking, the rat may refuse to drink if the slightest bitter taste is added to its drinking water.[34] In short, after lateral hypothalamic damage we may see not a passive *sensory neglect,* but an active *sensory*

[33]Wise et al., 1978.
*For further discussion see Wise, 1984.
[34]Teitelbaum and Epstein, 1962.

[redacted]

[35]Schaefer and Willshaw, 1978.
[36]Stellar, Brooks, and Mills, 1979.

The effects of these brain systems on feeding might be exerted in part by way of their effects on hedonic reactions to stimuli. Ventromedial hypothalamic tissue—the old "satiety center"—may be part of an inhibitory system that keeps our responses to pleasant stimuli, including foods, in check. We are reminded of the *alliesthesia* idea: Feeding ends when the hedonic response to food shifts from "nice" toward "awful." Conversely, intact lateral hypothalamic tissue—the old "feeding center"—may support the pleasantness of stimulus inputs, including foods, so that in its absence the good is less good and the bad is worse. Remember, though, that these functions are not confined to food-related stimuli; they seem to apply to whatever stimuli the environment offers.[37]

In any case, all this is still too simple. As we have laid out the picture, damage to the ventromedial area should weaken the ain't-it-awful system, and a rat with ventromedial damage ought to respond to stimuli generally as if they were nicer than before. It does not. It shows exaggerated acceptance of nice foods, but it also shows an exaggerated rejection of nasty foods.*[38] And it often shows vicious, rageful reactions to mild provocation—hardly the behavior of a rat for which everything is lovely. Clearly, a full-blown ain't-it-awful reaction can persist after damage to that portion of the brain.

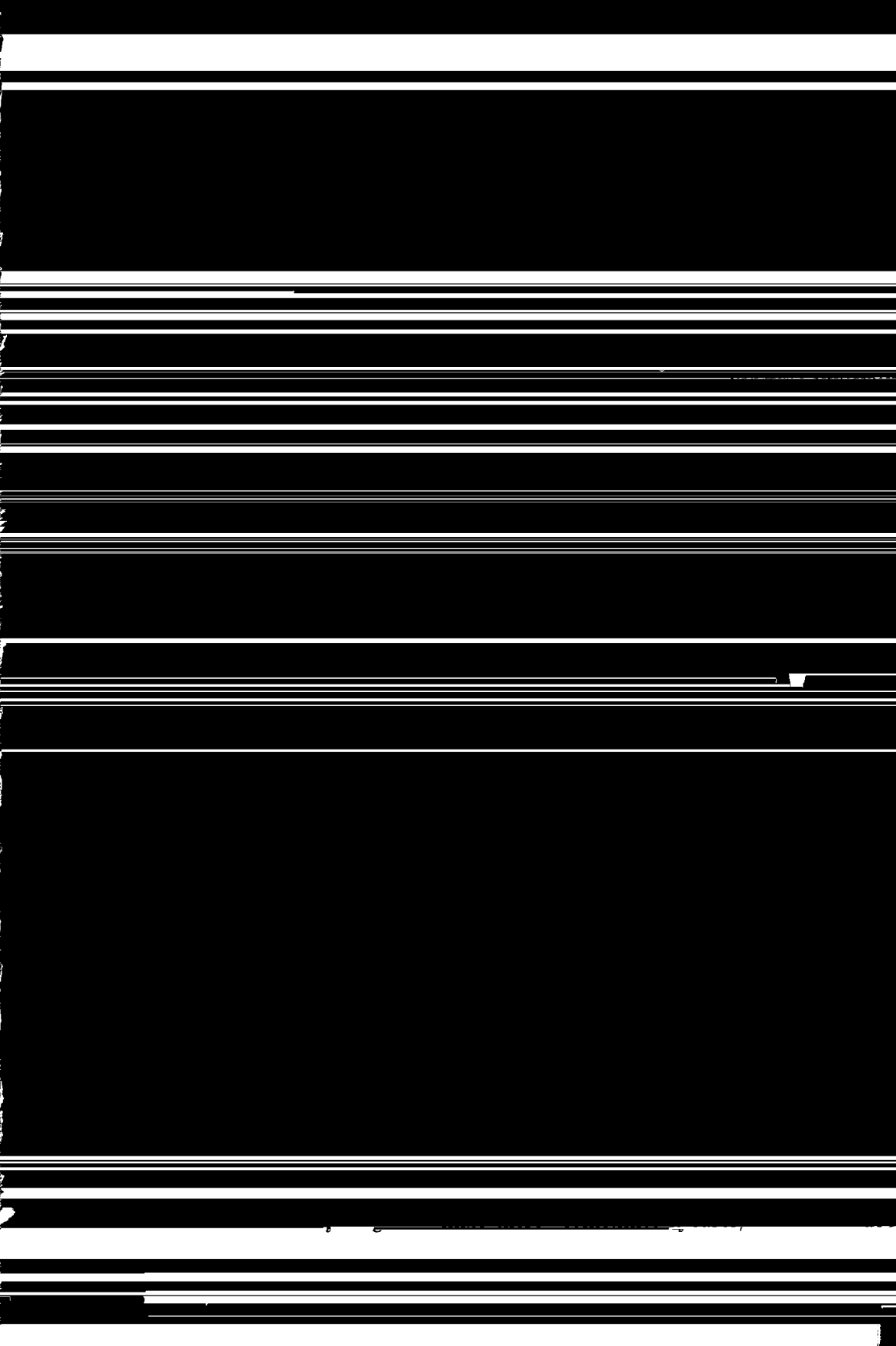
A Look Backward: The Brain and Hedonic Reactions

It appears that among the bundles of fibers running through the hypothalamus, there are at least *three* systems that (1) have hedonic properties themselves, and (2) control the hedonic effects of external stimuli. One system permits *attention* or *arousal* in response to stimuli; it says, "Isn't that interesting!" It may be closely related to other non-specific arousal systems, to be considered in the next chapter.

Another system permits *positive reactions* to stimuli; it says, "Isn't that nice!" Stimulation in that system exaggerates positive responses to stimuli, and is positively reinforcing in its own right. Conversely, chemical blocking of the system blocks the reward properties of food in hungry rats, and damage to the system reduces the niceness of external stimuli in general.

Still a third system promotes *negative reactions;* it says, "Ain't it awful!" Stimulation there exaggerates negative reactions to stimuli, and is punishing in its own right. And damage within the system, in the ventromedial hypothalamus, can release exaggerated positive reactions to *some* stimuli such as palatable foods. However, there can be exaggerated negative reactions as well, so there must be more to the ain't-it-awful system than this simple picture implies.

[37]Compare Kelley and Stinus, 1984; Neill et al., 1982.
*There is dispute about whether this happens because it is brain-damaged or because it is fat; see, for example, Weingarten, 1982; Graff and Stellar, 1962.
[38]Teitelbaum, 1955.



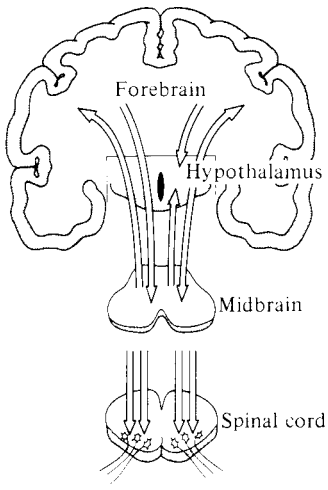


Figure 5-10.
The forebrain includes the cerebral cortex (shaded) and various subcortical structures (not shown). The forebrain, the hypothalamus, and the midbrain are interconnected, so that mechanisms at any level can communicate with all levels. And all levels of the brain are connected, directly or indirectly, with the motor neurons in the spinal cord.

evoke motivated behavior with hypothalamic stimulation. But it does not end there. The excited cells reach down into the brainstem and spinal cord to activate movements and movement patterns, and they reach up into the forebrain to interact with the cells there (Figure 5-10). We are dealing with systems that can be disrupted at many levels. And many of the symptoms produced by hypothalamic damage have their parallels after damage to the forebrain.

Akinesia: Parkinson's Disease

We recall from earlier discussion that the functions ascribed to the lateral hypothalamus may not occur *in* that structure at all, but involve lines of communication between midbrain and forebrain. A particular neurological disorder results from disruption of this system, and turns out to have some points in common with the lateral hypothalamic syndrome.

This is *Parkinson's disease,* a disorder of the motor system. It is characterized by tremor of the muscles when at rest, muscular rigidity, and *akinesia*—our familiar sluggishness and reluctance of voluntary movement. These patients may sit motionless in wheelchairs for hours at a time, requiring complete nursing care; their faces may be masklike and unexpressive, and even swallowing may be so difficult that drooling becomes a problem. The capacity for movement is not absent; rather, what seems to be missing is the background *arousability* of the system, needed to produce voluntary movement of any kind.[39]

[39]Kolb and Whishaw, 1985.

or at most investigate, is enough to trigger violent attack from a cat with its forebrain disconnected.[42]

It appears that in intact cats, forebrain mechanisms hold rage in check until there is something worth raging about. Lacking that inhibitory check, the cat flies off its handle at any little thing—hardly a recipe for effective action in the world.

Finally, notice that it is the whole pattern that is held in check, as a unit. Once again we see how the principles of nerve-cell interaction—inhibition, in this case—apply to the interactions among whole organized systems.

Forced Grasping

With more localized damage to the brain, we may see more specific deficits in inhibitory control. An example is the *forced-grasping syndrome* sometimes seen in humans after damage to the frontal lobes. In such a patient, any prominent visual stimulus may cause involuntary reaching out and grasping at it. "In bed, when he sees a nurse passing by, [the patient's] hand may automatically reach out and clutch at her dress, an act particularly distressing if she happens to be carrying a tray loaded with food or medicine."[43] The patient may be distressed by such forced actions, but unable to control them.

Very often in such cases, the reach-and-grasp pattern is well organized, aimed and guided all too accurately, and indeed quite normal. What is missing is not the organization of the movement pattern itself. It is the inhibitory control over the whole pattern, *as a unit,* which normally restricts it to situations in which it is appropriate. The whole movement pattern is released from inhibition as a unit, and so it occurs when normally it would be held in check.

Such inhibitory control is, in large part, what makes our voluntary behavior truly *voluntary.* When a pattern of movement is released from those inhibitory checks, it becomes involuntary and robot-like—even though the pattern itself is perfectly well organized.

The Temporal-Lobe Syndrome

Yet another kind of release from inhibition was discovered in the 1940s by Heinrich Klüver and Paul Bucy.[44] This occurred after damage, in monkeys, to the temporal lobes and the subcortical structures underlying them.

After the operation, the monkeys expressed simple motivated behaviors —eating, mating, exploring—in a perfectly normal way. What was missing was the *selectivity* in expressing them toward appropriate objects. The monkeys would eat monkey food, but they would also eat meat—which monkeys normally do not eat—or wooden blocks or paper bags or, it

[42]Bard and Mountcastle, 1948.
[43]Teitelbaum, 1967.
[44]Klüver and Buoy, 1939.

seemed, anything that could be put into the mouth. They masturbated frequently and mated, or tried to mate, with animals of different species or even with inanimate objects. They would carefully explore small objects such as nuts and bolts, find them useless, and throw them away in normal monkey fashion—but then, on encountering them again, they would carefully explore them again as if seeing them for the first time.

Since it is *removal* of brain tissue that produced these indiscriminate mating, eating, and exploring reactions, we must assume that systems involving that tissue had been keeping them discriminate before. In such cases, it is tempting to speculate that the failure is of inhibitory control on the *stimulus elicitation* of the action. The operation must have damaged systems that say to intact monkeys, "Eat, but don't eat *that;*" or "Mate, but not with *that;*" or "Explore new things, but not *that;* it isn't a new thing and is not worth exploring."

The syndrome also occurs in humans. One such patient

> seemed unable to recognize a wide variety of common objects. He examined each object placed before him as though seeing it for the first time, explored it repetitively and seemed unaware of its significance. . . . [When imitating another's actions, a common neurological test], he would perseverate, copying all movements made by another for extended periods of time. . . . All objects that he could lift were placed in his mouth and sucked and chewed. He was commonly observed to place his fingers in his mouth and suck them . . . he ingested virtually everything within reach, including the plastic wrapper from bread, cleaning pastes, ink, dog food, and feces.[45]

A Look Backward: The Importance of Inhibition

Inhibitory systems are central to the organization of action. If we have an animal or human that eats, mates with, grasps, or becomes enraged at any old object in the environment, we do not have organized behavior at all. Behaving effectively means focusing on food, and only food, if one is hungry; reaching for objects if appropriate, but not otherwise; and so on. Forebrain mechanisms have a great deal to do with keeping behavior focused on objects and stimuli that are *appropriate* for the expression of motivated behavior.

Control of Movement Sequences

In the normally-functioning brain, motivational states can be expressed by calling upon complex patterns and sequences of movement. The structures of the forebrain have a great deal to do with the control of movement, especially fine, skilled sequences of movement and especially in human beings. After damage to the brain, these ways of *implementing* one's purposes, attaining one's goals, may be unavailable.

[45]Marlowe et al., 1975.

Skilled Sequences

Consider a pianist playing a fast passage. Her finger movements in depressing successive keys may be as rapid as sixteen per second. We might suppose that each movement is triggered individually by the corresponding note in the score; or perhaps each movement provides stimuli that in turn trigger the next one, so that we have a chain of responses, as in stimulus-bound feeding. However, Karl Lashley[46] showed by simple arithmetic that it cannot be like that. We simply are not capable of reacting that quickly to either visual input or kinesthetic input produced by our own movements.

If that is so, then the phrase as a whole must be played by a preprogrammed sequence of operations that imposes the necessary interval between each output and the next, and diverts each output to the appropriate finger. If we cannot play notes that fast one at a time, we must turn over control to an integrated, skilled system that can program the whole *pattern* of movements over time.

Lashley believed that such skilled patterns are organized in the cerebral cortex. He may well have been right. Certainly cortical tissue is necessary for the proper sequencing of other skilled activities. Laboratory rats have been required to emit a series of responses, in the proper sequence, to earn a food reward. Rats without cortex are unable to perform such tasks.[47] They can make the movements, all right, but they are incapable of keeping them in the proper sequence. It is the same with human patients: After frontal lobe damage,

> [t]he patient's handwriting is altered and every stroke of a letter requires a special effort; the typist loses the speed and smoothness of her work, the muscles cannot play a tune smoothly, and the skilled worker is unable to carry out automatically the successive system of operations constituting a habitual motor act.[48]

Special Forebrain Systems

The forebrain organizes more specific skills as well. These skills are like tools in a toolbox, available for special purposes when they are needed. As just one example of these specialized systems, let us consider *speech.*

Now at first glance, a discussion of speech systems in the brain doesn't seem to belong in a discussion of *motivation.* But if we think about it, we realize that speech is very often used as a tool for implementing motivated behavior. If we are hungry, we can ask where a restaurant is, and we can understand and follow the directions when we get them. We can read the menu and order from it; we can ask our partner to pass the potatoes. Thus the hunger system, in human beings, must be able to reach

[46]Lashley, 1951.
[47]See Morgan, 1951; Kolb and Whishaw, 1985.
[48]Luria, 1973, p. 180.

beds over there the same thing . . ."[49]

[49]Gardner, 1974, p. 68.

Here the speech is adequately articulated (though there was a problem with the words "thing," "trying," and "recuperation"), and most of the phrases make sense in isolation. What is missing is the *inhibitory control* that keeps the discourse within bounds, and prevents it from drifting off the track.

A Look Backward: Access to Special Systems

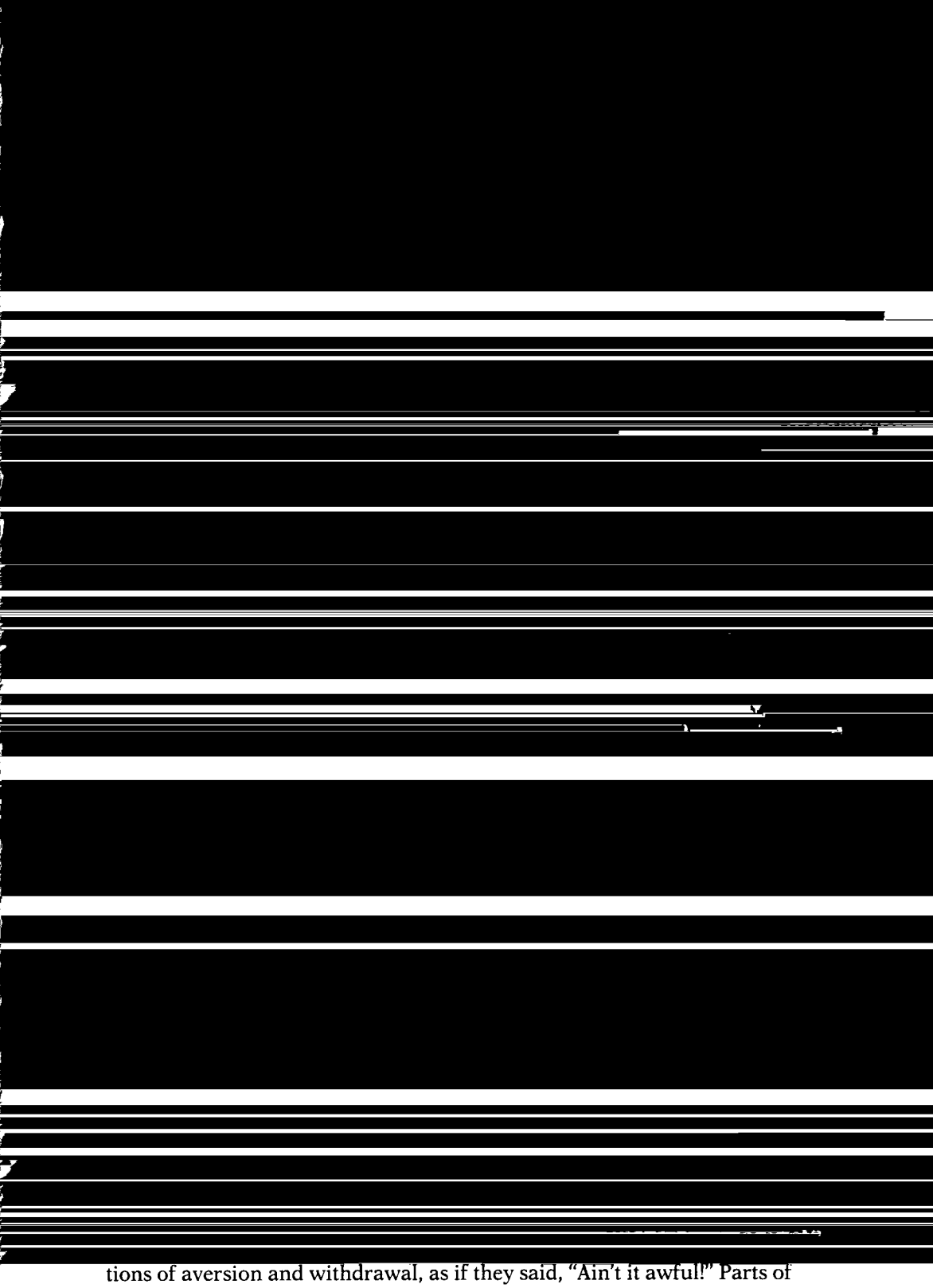
We cannot discuss these special tools any further, for it would take us into the entire field of neuropsychology. But that is the point. To express even a relatively simple motive like hunger, we depend on access to systems that involve the highest levels of the brain—skilled movement sequences like using a fork, movement patterns like reaching for the potatoes, special-tool operations like asking for the salt. Then again, we may *inhibit* any or all of these—until other people at the table have been served, perhaps. Clearly, much more is involved than just a state of the hypothalamus.

Finally, how are these tools brought into use? How does hunger or the sight of food call the reaching pattern or the speech system into play? How do motivational states take hold of patterns of muscle contractions? That is one of the most fundamental mysteries in the psychology of motivation *or* of brain function. We are approaching that mystery from both sides; we know that the motor programs are there, and we know some of the things that lead us to access one or another of them. But how do we do it? We do not know.

SUMMARY

The nervous system is composed of nerve cells, or *neurons.* A nerve cell communicates with another cell by liberating a chemical messenger, a *neurotransmitter,* which can either *excite* or *inhibit* the activity of the receiving cell. Even this much permits a surprising degree of organization in behavior. An example is the blowfly, whose reflex feeding movements are excited by taste stimuli, but inhibited by food in the digestive tract; thus food identification, feeding behavior, and satiety are all provided for. In addition to the arithmetic of excitation (addition) or inhibition (subtraction), the geometry of the nervous system adds possibilities for complexity. Connections in the nervous system can permit a signal to *diverge* so as to have multiple effects. Or different signals can *converge* on a common output, permitting multiple-control systems in which different inputs can have similar effects.

Bard's experiments in animals with transected nervous systems showed a hierarchical organization, in which fragments of behavior at lower levels are organized into units at higher levels. In particular, if the *hypothalamus* was left in the system, movement patterns were organized into sequences of goal-directed action, and brought under the control of



tions of aversion and withdrawal, as if they said, "Ain't it awful!" Parts of

the reward system are affected by the same states of the body that affect motivational systems. In a well-fed rat, lateral hypothalamic stimulation becomes less rewarding. It is possible that stimulation in these systems mimics the rewarding properties of natural reinforcers. Blocking such a system with drugs can block the rewarding properties of food without, apparently, reducing hunger. In nature, these systems may be essential for the rewarding effects of natural commodities such as food or opportunity to mate.

Forebrain systems are closely connected to lower systems in the brain, and damage to the forebrain can mimic effects of damage lower down. *Parkinson's disease,* characterized by akinesia and difficulty of movement, results from deterioration of a midbrain-to-forebrain communication link. Damage to the cerebral cortex can produce *sensory neglect.* In addition, the forebrain has important *inhibitory* functions, so that the effects of damage there can be seen as *release from inhibition.* Animals without cortex may display the hair-trigger *sham rage* reaction. In animals and humans, temporal-lobe damage can produce the *temporal-lobe syndrome,* in which mouthing, mating, and exploring are done in a normal way but are no longer restricted to appropriate objects. Frontal-lobe damage can also produce release phenomena such as *forced grasping.*

Finally, the forebrain plays an important role in the fine control of precise movements, and in the organization of skilled movement sequences. This function includes the organization of specialized systems such as *speech,* so that brain damage can block access to the speech system or release it from inhibition. Inasmuch as humans use speech to seek their goals or meet their needs, motivational systems must be able to call the speech system into play; this applies as well to the other specialized skills that the brain makes available.