2. *Neurophysiology and Neural Organization*

A. The Elements of the Network: Neurons

Structure and Function The elongated impulse-carrying cells referred to earlier are called *neurons*. A typical multipolar neuron has the physical structure outlined in figure 7.6: a treelike structure of branching *dendrites* for input, and a single *axon* for output. (The axon is folded for diagrammatic reasons.) This structure reflects what appears to be the neuron's principal function: the processing of inputs from other cells. The axons of many other neurons make contact either with the dendrites of a given neuron, or with the cell body itself. These connections are called *synapses*, and they allow events in one cell to influence the activity of another (figure 7.7).

The influence is achieved in the following ways. When a depolarization pulse—called an *action potential* or *spike*—runs down the axon to its presynaptic ending(s), its arrival causes the terminal bulb to release a chemical called a *neurotransmitter* across the tiny synaptic cleft. Depending on the nature of the bulb's characteristic neurotransmitter, and on the nature of the chemical receptors that receive it on the opposite side of the cleft, the synapse is called either an *inhibitory* or an *excitatory* synapse.

In an inhibitory synapse, the synaptic transmission causes a slight *hyper*polarization or raising of the affected neuron's electric potential. This makes it less likely that the affected neuron will undergo a sudden depolarization and fire off its own spike along its own axon.

In an excitatory synapse, the synaptic transmission causes a slight *de*polarization of the affected neuron, inching its electric potential downward toward the critical minimum point where it suddenly col-

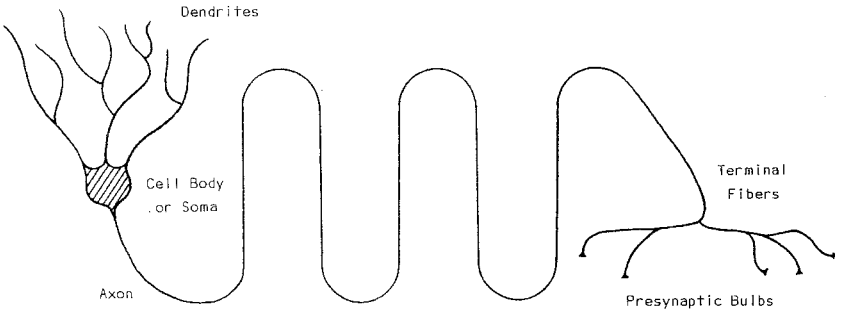

Figure 7.6

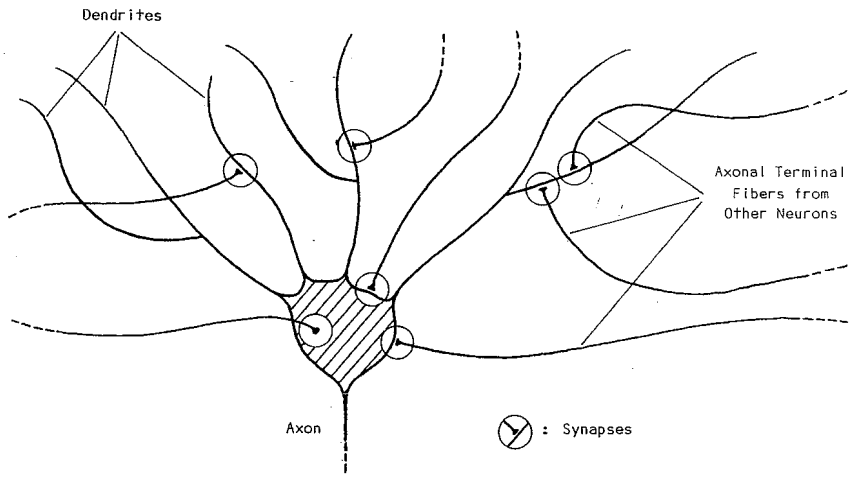

Figure 7.7

lapses entirely, initiating its own axonal output spike. An excitatory synaptic event therefore makes it *more* likely that the affected neuron will fire.

Putting the two factors together, each neuron is the site of a competition between 'fire' and 'don't fire' inputs. Which side wins is determined by two things. First, the relative distribution of excitatory and inhibitory synapses matters greatly—their relative numbers, and perhaps their proximity to the main cell body. If one kind predominates, as often it does, then the deck is stacked, for that neuron, in favor of one response over the other. (In the very short term, these connections are a relatively stable feature of each neuron. But new connections do grow and old ones are lost, sometimes on a time scale of mere minutes or less; hence the functional properties of a neuron are themselves somewhat plastic.)

The second determinant of neuronal behavior is the sheer temporal frequency of inputs from synapses of each kind. If 2,000 inhibitory synapses are active only once per second, and 200 excitatory synapses are active a busy 50 times per second, then the excitatory influence will predominate and the neuron will fire. After repolarization, it will fire again, and again, with a significant frequency of its own.

It is well to keep in mind the relevant numbers here. A typical neuron soma will be almost buried under a layer of several hundred synapsing end bulbs, and its dendritic tree may enjoy synaptic connections with several thousands more. As well, neurons pump themselves back up to resting potential again in rather less than 1/100 second; hence they

can sustain spiking frequencies of up to 100 hertz (= 100 spikes per second), or more. Evidently, a single neuron is an information processor of considerable capacity.

Inevitably, neurons are likened to the logic gates in the CPU of a digital computer. But the differences are as intriguing as the similarities. A single logic gate receives input from no more than two distinct sources; a neuron receives input from well in excess of a thousand. A logic gate emits outputs at a metronomic frequency, 10^6 hertz, for example; a neuron varies freely between 0 and 10^2 hertz. Logic-gate output is and must be temporally coordinated with that of all other gates; neuronal outputs are not thus coordinated. The function of a logic gate is the transformation of binary information (sets of ONs and OFFs) into further binary information; the function of a neuron, if we can even speak in the singular here, seems more plausibly to be the transformation of sets of spiking *frequencies* into further spiking *frequencies*. And last, the functional properties of a logic gate are fixed; those of a neuron are decidedly plastic, since the growth of new synaptic connections and the pruning or degeneration of old ones can change the input/output function of the cell. The dendritic branches can grow tiny spines in a matter of minutes, to effect new synaptic connections, and these changes are themselves induced, in part, by prior neuronal activity.

If neurons are information-processing devices, as almost certainly they are, their basic mode of operation is therefore very different from that displayed in the logic gates of a CPU. This is not to say that systems of the latter, suitably programmed, could not simulate the activities of the former. Presumably they could. But we need to know rather more about the plastic functional properties of neurons, and very much more about their myriad interconnections, before we can successfully simulate their collective activity.

Types of Neurons

An initial classification finds three kinds of neurons: *motor* neurons, *sensory* neurons, and a large variety of *interneurons* (that is, all the rest). Primary motor neurons are found almost exclusively in the spinal cord, and are defined as those neurons whose axons synapse directly onto a muscle cell. The axons of motor neurons are some of the longest in the nervous system, extending from deep within the spinal cord, out the *ventral roots* (see figure 7.1) between the spinal vertebrae, and on out the limbs to the most distant peripheral muscles. Motor neurons secure graded muscle contraction by two means: the spiking frequency of individual motor neurons, and the progressive recruitment of initially quiescent neurons that innervate the same muscle.

Sensory neurons come in greater variety, and are conventionally

defined as those whose input stimulus is some dimension of the world outside the nervous system. For example, the rod and cone receptor cells of the retina are very tiny, with no axon to speak of, and no dendrites at all. They synapse immediately onto more typical neurons in a layer right next to them. Their job is solely to transform received light into synaptic events. The somatosensory cells, by contrast, are as long as the motor neurons. Their axons project from the skin and muscles into the spinal cord by way of the *dorsal roots* (see figure 7.1), and they find their first synapses deep in the spinal cord. Their job is to convey tactile, pain, and temperature information, and information about muscle extensions and contractions—the ever changing positions of the body and its limbs. Other sensory cells have their own idiosyncracies, dictated by the nature of the physical stimulus to which they respond.

The central interneurons also come in a great variety of shapes and sizes, though they all seem variations on the same theme: dendritic input and axonic output. Most, called multipolar cells, have many dendritic branches emerging directly from the cell body. Others, called bipolar cells, have only one dendritic thread emerging, which branches at a point some distance from the cell. Some, such as the Purkinje cells of the cerebellum, have extraordinarily extensive and bushy dendritic trees. Others enjoy only sparse dendritic extensions. The axons of many neurons project across the entire brain, synapsing at distant points. Others make merely local connections among extended concentrations of neurons whose axons project elsewhere.

These densely populated layers of heavily interconnected neuronal cell bodies are called *cortex*. The outer surface of each cerebral hemisphere is one large thin sheet of cortex, heavily folded upon itself like crumpled paper to maximize the total area achieved within the small volume of the skull. The brain's interneural connections are at their heaviest in this folded layer. The surface of the cerebellum is also cortex, and specialized cortical 'nuclei' are distributed throughout the brain stem. These show as gray areas in brain cross sections. The remaining white areas contain axonal projections from one cortical area to another. Which brings us to the matter of the brain's organization.

B. The Organization of the Network

Seeking organization in a network as complex as the human brain is a difficult business. Much structure has emerged, but as much or more remains shrouded in mystery. One can explore the large-scale structure of neuronal interconnections by using special stains that are taken up by a neuron and transported down its axon to the terminal synapses.

If we wish to know where the axons of a stained area project to, successive cross sections of the brain will reveal both the path those stained axons take through its relatively colorless volume, and the region of their ultimate terminus. This technique, applied to postmortem brains, has revealed the major interconnections between the various cortical areas of the brain, the 'superhighways' that involve many thousands of axons strung together. Knowing their locations does not always reveal their functions, however, and the smaller neuronal highways and byways constitute a horizon of ever shrinking detail that defies attempts at complete summary.

With microscopes, thin sections, and a variety of further staining techniques, the microarchitecture of the brain begins to emerge. Cerebral cortex reveals six distinct layers, distinguished by the density of the neuron populations within them, and by the type of neurons they contain. Interneuronal communication is extensive, both within layers and across them. The details are complex and obscure, and the point of this particular arrangement remains mysterious, but we cling to what order we discover, and try to use it to find more. As it happens, this six-layered cytoarchitecture is not entirely uniform through the cerebral cortex: the thickness or density of certain layers is diminished or exaggerated in certain areas of the cortical surface. Tracing areas of identical architecture, and noting their boundaries, has led us to identify about fifty distinct cortical areas, known as *Brodmann's areas* after their discoverer.

Are these areas of any further significance? Many of them are, both in their functional properties and in their more distant connections. A few salient cases will now be outlined.

Sensory Projections within the Brain

As mentioned earlier, the primary somatosensory neurons enter the spinal cord via the dorsal roots, and find their first synaptic connections with neurons in the cord. Those neurons conduct the information up the spinal cord all the way to the thalamus in the forebrain, where they synapse onto neurons in an area called the ventral thalamic nucleus. These neurons project in turn into the cerebral hemispheres, and into a cortical area neatly defined by three connecting Brodmann's areas. This overall area is now known as the *somatosensory cortex*. Damage to various parts of it produces a permanent loss of tactile and proprioceptive awareness of various parts of the body. Moreover, subtle electrical stimulation of neurons in this area produces in the subject vivid tactile sensations 'located' in specific parts of the body. (Brain surgery to correct threats to this area has provided the occasional opportunity for such probing, and since subjects

can be wholly conscious during brain surgery, they can report the effects of such stimulations.)

In fact, the somatosensory cortex constitutes what is called a *topographic map* of the body, since the spatial arrangement of anatomically specific neurons is a projection of the anatomical areas themselves. Each hemisphere represents the opposite one-half of the body. The cross section of one hemisphere in figure 7.8 illustrates this point. The distorted creature represents the areas of the cortex devoted to the body part next to it, and the variations in size represent the relative numbers of cortical cells devoted to that part. This diagrammatic creature is called "the somatosensory homunculus".

The organization and function of the visual system also makes contact with the architecture of the cerebral cortex. Right next to the primary rods and cones of the retina is an interconnected layer of small neurons that performs some initial processing before synapsing onto the long ganglion cells. These cluster together in a thick bundle and exit the back of the retina as the optic nerve. The optic nerve projects to a cortical nucleus (= a local concentration of interconnected cell bodies) at the rear of the thalamus called the *lateral geniculate body*. The cells here also constitute a topographic map of the retina, though it is metrically distorted in that the fovea, the physical and functional center of the retina, is very heavily represented.

Cells in the lateral geniculate then project to several of Brodmann's

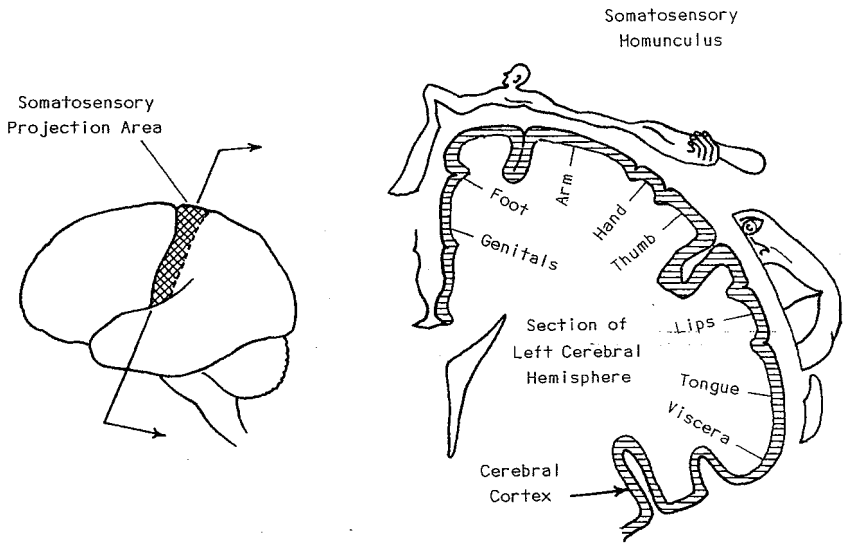

Figure 7.8

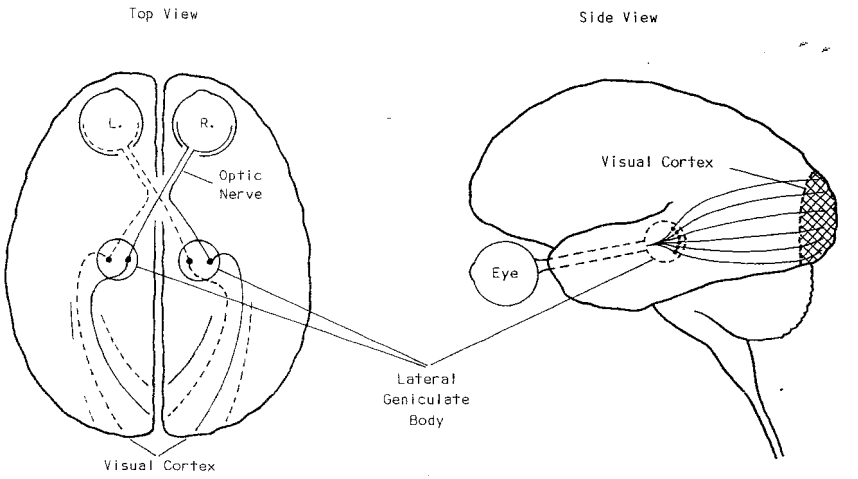

Figure 7.9

areas on the rearmost surface of the cerebral hemispheres: to the striate cortex and thence to the peristriate cortex (figure 7.9). These collected areas are called the *visual cortex*, and still constitute a topographic projection of the retina, each hemisphere representing one-half of the retinal surface. But rather more is going on in the visual cortex, and in its precortical processing, than occurs in the somatosensory system, and the visual cortex represents rather more than just areas of retinal stimulation. Subpopulations of visual neurons turn out to be specialized, in their responses, to highly specific features of the visual information. A cell early in the hierarchy is sensitive only to brightness *differences* within its receptive field (= the retinal area to which it is sensitive). But a higher cell to which these early cells project may be sensitive only to lines or edges of a particular *orientation* within its receptive field. Cells higher still are sensitive only to lines or edges *moving* in a particular direction. And so on. The impression of a cumulative information-processing system is impossible to escape.

Further microstructures promise to explicate the features of binocular vision—in particular, the sophisticated *stereopsis* or three-dimensional vision possessed by humans. Stereopsis requires the systematic comparison of the images from each eye. Close examination reveals the existence of interleaved *ocular dominance columns* in the visual cortex. A column is a narrow cluster of cells organized vertically through the six layers of the cortex, and each has a small receptive field in the retina. Such columns are eye-specific, and their interleaving means that corresponding left and right receptive fields are represented by phys-

ically adjacent columns in the cortex. Comparison of information can thus take place, and further cells have been discovered that are indeed sensitive to binocular disparities between those fields. Such cells are responding to information about the relative distances of the objects in one's visual environment. These discoveries open promising lines of research, and the visual cortex currently commands a great deal of interest.

Motor Projections Outward Just in front of the somatosensory cortex, on the other side of a largish cleft, is another of Brodmann's areas now known as the *motor cortex*. This is also a clear topographic map, this time of the body's muscle systems. Artificial stimulation of motor cortical neurons produces movement in the corresponding muscles. A 'motor homunculus' is displayed in figure 7.10.

This is only the beginning of the functional story, of course, since motor control is a matter of well-orchestrated *sequences* of muscle contractions—sequences, moreover, that cohere with the body's perceived environment. Accordingly, the motor cortex has axonal projections, not just to the cord and thence to the body's muscles, but to the cerebellum and the basal ganglia, and it receives reciprocal projections from both, primarily through the thalamus, which we already know to be a source of sensory information. The motor cortex is therefore a highly integrated part of general brain activity, and though some of

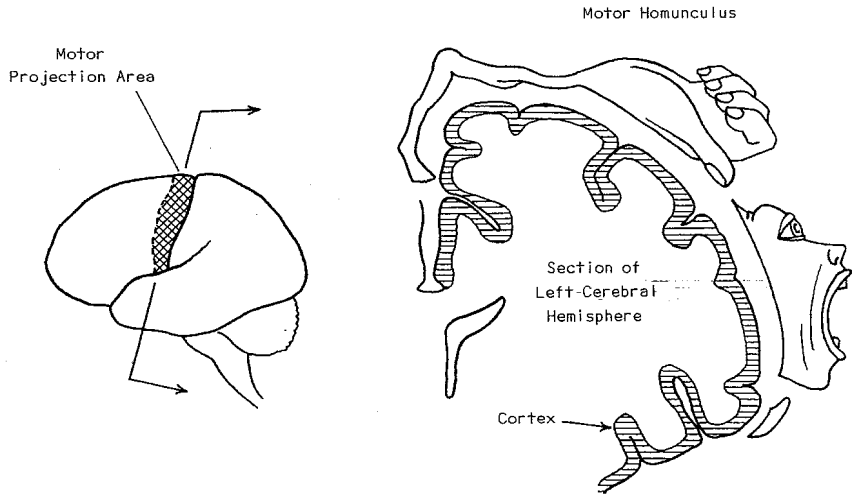

Figure 7.10

its output goes more or less directly to the cord—to provide independent control of fine finger movements, for example—much of it goes through intricate processing in the cerebellum and lower brain stem before entering the spinal cord.

We must think of the brain's output here as a sort of high-level 'fine tuning' of motor capacities more basic still, since the neural organization of the spinal cord itself is sufficient to produce locomotion in most vertebrates. A familiar example is the headless chicken whose body runs around aimlessly for several seconds after slaughter. Even small mammals whose brains have been substantially removed will display locomotor activity upon suitable stimulation of the cord. We have here a reflection of just how very *old* the capacity for vertebrate locomotion is; it was first perfected when primitive vertebrates enjoyed little more than a spinal cord. The progressive additions that survived did so because they added some useful fine tuning of, or intelligent guidance to, that initial capacity. The motor cortex is merely one of the later and higher centers in an extensive hierarchy of motor controls. These extend from the simple reflex arcs—such as will withdraw a hand from a hot stove—up to the highest centers, which formulate abstract, long-term plans of action.

Internal Organization The brain monitors the extra-nervous world, through the primary sensory neurons; but in the process it also monitors many aspects of its own operations. And the brain exerts control over the extra-nervous world; but it also exerts control over many aspects of its own operations. The internal projections among parts of the brain are rich and extensive, and they are critical to its functioning. A good example is the existence of 'descending control' mechanisms. In our earlier discussion of the visual system I did not mention that the visual cortex also sends projections *back* to the lateral geniculate body in the thalamus, where the optic nerve terminates. What this means is that, depending on what the visual cortex is getting from the lateral geniculate, it can exert an influence on the latter to *change* what is being sent, perhaps to highlight certain features of the input, or to suppress others. We have here the elements of some plasticity in the brain's processing activities, the capacity for directing attention and focusing resources. Descending control pathways are especially prominent in the visual system and in the auditory system, which must process speech, but they are common throughout the brain.

Between the sensory areas of the cortex here discussed, and other sensory areas similarly identified, there remains a great deal of highly active brain. The large so-called "association areas", between the various

types of sensory cortex, are not well understood, and neither are the large frontal areas of the cerebral hemispheres, though it is plain from cases of brain damage that these latter are impicated in emotion, drive, and the capacity for planned action.

There is a hypothesis that makes general sense of these areas, of their function and of their axonal connections with other areas. Consider figure 7.11. The cross-hatched areas are the areas of *primary* sensory cortex: somatosensory, auditory, and visual. The vertically striped areas are *secondary* sensory cortex. Cells in the primary cortex project to cells in the secondary cortex, for all three sensory modalities, and these secondary cells are responsive to more complex and abstract features of the sensory input than are the cells in the primary cortex. Secondary cortex, in turn, projects into the unshaded areas, called *tertiary* or *association* cortex. Cells in the association cortex are responsive to still more abstract features of the original sensory inputs, but here we find a mixture of cells, some responsive to visual input, some to auditory input, some to tactile input, and some to combinations of all three. It would appear that the brain's most abstract and integrated analysis of the sensory environment takes place in the association cortex between the several sensory areas.

From this rear or 'sensory' half of the brain, information can make its way by a variety of underlying pathways in the midbrain to the frontal or 'motor' half of the brain, into what we may call the tertiary

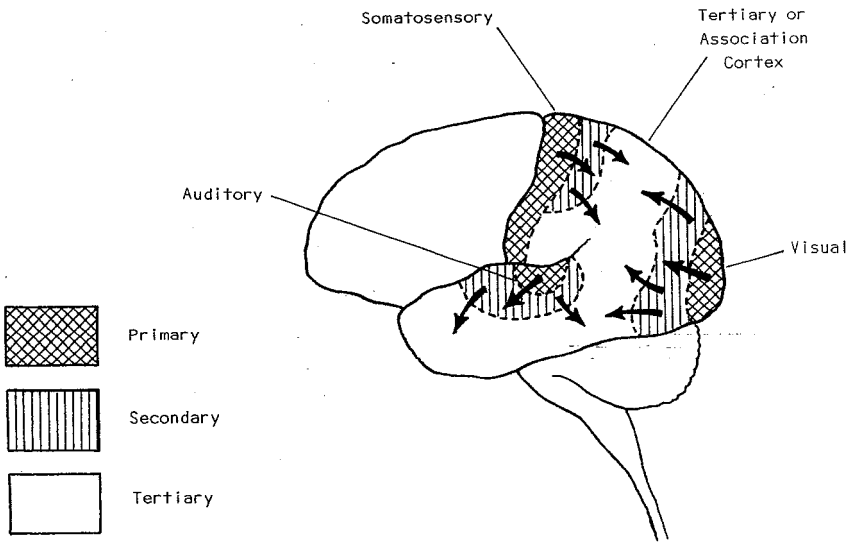

Figure 7.11

motor areas. This is the unshaded frontal area in figure 7.12. This area appears responsible for the formation of our most general plans and intentions. Cells here project into the secondary motor cortex, which appears to be the locus of more specifically conceived plans and sequences of behavior. This area projects finally to the primary motor cortex, which is responsible for highly specific motions of the various parts of the body.

This hypothesis is consistent with the neuroarchitecture of the brain, with its overall capacities as a sensorily guided control of bodily behavior, and with detailed studies of the specific cognitive deficits produced by lesions at various sites in the brain. Damage to the extreme frontal lobe, for example, leaves the victim unable to conceive of, or to distinguish in a caring fashion between, alternative possible futures beyond the most immediate and simple matters.

The preceding sketch of the global organization of the brain represents the classical view, but the reader should be warned that it presents a provisional and oversimplified picture. Recent studies indicate that distinct topographic maps of the retina are scattered throughout the cortical surface, and enjoy distinct projections from the lateral geniculate, or from elsewhere in the thalamus. The hierachical system of topographic maps discussed earlier, which culminates in the 'secondary visual cortex' at the rear of the brain, is thus only one of several parallel systems, each processing different aspects of visual input. The 'classical' system

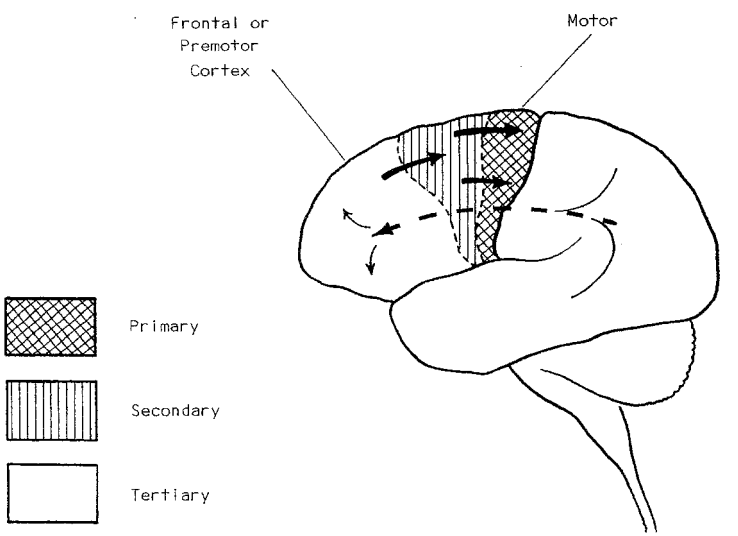

Figure 7.12

for vision may be the dominant one, but it has company, and all of these systems interact. Similar complexities attend the 'somatosensory cortex', which emerges as only one of several parallel systems processing different types of somatosensory information: light touch, deep pressure, limb position, pain, temperature, and so forth. Sorting out the functional differences between these distinct maps and tracing their functional interconnections is a job that has only begun. As that information does emerge, our appreciation of the intricate and occasionally unsuspected achievements of our perceptual system must grow in equal measure.

One further area of intrigue is worthy of mention, not because it is large, but because it is the ultimate target of a hierarchy of projections from very large and varied areas of the cerebral cortex. The smallish *hippocampus* is at the back end of the limbic system, a forebrain structure just underneath the great cerebral hemispheres. If we trace the inputs to the hippocampus back to their origins, against the flow of incoming information, we fairly quickly implicate the entire cerebral cortex. Damage to the hippocampus, it emerges, blocks the transfer of information from short-term into long-term memory. Victims of such damage live in a nightmare world of no memories reaching longer than a few minutes into the past, save those original memories, of those ever more distant events, entrenched before the injury occurred.

It is natural to think of the brain as something which is interposed between the peripheral sensory nerves and the peripheral motor nerves, something controlled by the former and in control of the latter. From an evolutionary perspective, this makes sense, at least in the early stages. But with the brain at the level of articulation and self-modulation found in humans, a certain autonomy has crept into the picture. Our behavior is governed as much by our past learning, and by our plans for the long-term future, as by our current perceptions. And through self-directed learning, the long-term development of the brain's internal organization is to some extent under the control of the brain itself. We do not by this means escape the animal kingdom, but we are become its most creative and unpredictable members.

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3. Neuropsychology

Neuropsychology is the discipline that attempts to understand and explain psychological phenomena in terms of the neurochemical, neurophysiological, and neurofunctional activities of the brain. We have already seen some tentative but intriguing neuropsychological results in the preceding section: how the hierarchical structure of the visual system permits us to discriminate selected features from a scene, how interleaved retinal representations on the cortical surface make stereo vision possible, and how the overall organization of the cortex makes it possible for highly processed sensory information to guide the formation and execution of general plans of action.

Unfortunately, the greater portion of the data traditionally available to neuropsychology derives from cases of brain damage, degeneration, and disequilibrium. What we understand best is the neural basis of *abnormal* psychology. Brain tissue can be physically disrupted by invasive objects; it can be crushed by growing tumors or fluid pressure; it can starve and atrophy from localized loss of blood supply; or it can be selectively destroyed by disease or degeneration. Depending on the specific *location*, within the brain, of the lesion produced by any of these means, very specific losses in the victim's psychological capacities typically result.

Such losses may be minor, as with an inability to identify perceived colors (lesions to the connections between the secondary visual cortex and the secondary auditory cortex of the left hemisphere). Or they may be serious, as with the permanent inability to recognize faces, even those of family members (lesions in the association cortex of the right hemisphere). And they can be devastating, as with the total and permanent loss of speech comprehension (lesions to the secondary auditory cortex of the left hemisphere), or the inability to lay down new memories (bilateral damage to the hippocampus).

Using postmortem examination, and other diagnostic techniques, neurologists and neuropsychologists can find the neural correlates of these and hundreds of other losses in cognitive and behavioral function. By this means we can slowly piece together an overall *functional map* of the brain. We can come to appreciate the functional specializations and the functional organization of the brain in a *normal* human. This information, in conjunction with a detailed understanding of the neuroarchitecture and microactivity of the relevant areas, can lead to a real understanding of how our cognitive capacities are actually produced. Recall our glimpse into feature extraction and stereopsis in the visual system. Once we know where to look for them, we can start to find specific neural structures that account for the specific features of the

cognitive capacity at issue. Overall, there is cause for much optimism here, even though our ignorance still dwarfs our understanding.

The functional sleuthing just described requires caution in two respects. First, the simple correlation of a lesion in area *x* with the loss of some cognitive function *F* does not mean that area *x* has the function *F*. It means only that some part of area *x* is typically involved in some way in the execution of *F*. The key neural structures that sustain *F* may be located elsewhere, or they may not be localized at all, being distributed over large areas of the brain.

Second, we must not expect that the functional losses and functional localizations that we do find will always correspond neatly with cognitive functions represented in our common-sense psychological vocabulary. Sometimes the deficit is difficult to describe, as when it involves a global change in the victim's personality, and sometimes its description is difficult to credit. For example, some lesions produce a complete loss of awareness, both perceptual and practical, of the *left half* of the victim's universe, including the victim's own body (hemineglect). A victim will typically dress only the right side of his body, and even deny ownership of his own left arm. Other lesions leave the victim able to write lucid, readable prose, but *un*able to read and understand what she or anyone else has written, even though her vision is wholly normal (alexia without agraphia). Further lesions leave the victim 'blind', in the sense that his visual field has disappeared and he insists that he cannot see; and yet he can 'guess' where a light is placed in front of him with an accuracy approaching 100 percent (blind-sight). Still other lesions leave the victim genuinely and utterly blind, but the victim perversely insists that she *can* see perfectly, as she stumbles about the room confabulating excuses for her clumsy behavior (blindness denial).

These cases are surprising and confusing, relative to the familiar conceptions of folk psychology. How could one possibly be blind and not know it? See with no visual field? Write freely but not read a word? Or sincerely deny ownership of arms and legs attached to oneself? These cases violate entrenched expectations. But we cannot expect that folk psychology represents anything more than one stage in the historical development of our self-understanding, a stage the neurosciences may help us to transcend.

Beneath the level of structural damage to our neural machinery, there is the level of chemical activity and chemical abnormalities. The reader will recall that transmission across the synaptic junction is a critical element in all neural activity, and that such transmission is chemical in nature. Upon receipt of an impulse or spike, the axonal end bulb releases a chemical called a *neurotransmitter* that swiftly diffuses across the synaptic cleft to interact with chemical receptors on the far side.

This interaction leads to the breakdown of the neurotransmitter chemical, and the breakdown products are eventually taken up again by the end bulb for resynthesis and reuse.

Evidently, anything that frustrates, or exaggerates, these chemical activities will have a profound effect on neural commmunication and on collective neural activity. This is precisely how the many psychoactive drugs work their effects. The various types of neurons make use of distinct neurotransmitters, and different drugs have different effects on their activity, so there is room here for a wide variety of effects, both chemical and psychological. A drug may block the synthesis of a specific neurotransmitter; or bind to its receptor sites, thus blocking its effects; or block the uptake of its breakdown products, thus slowing its resynthesis. On the other hand, a drug may enhance synthesis, increase receptor sites, or accelerate the uptake of breakdown products. Alcohol, for example, is an antagonist to the action of noradrenaline, an important neurotransmitter, whereas the amphetamines enhance its activity, producing the very opposite psychological effect.

Most important, extreme doses of certain of the psychoactive drugs produce symptoms that closely resemble those of the major forms of mental illness—depression, mania, and schizophrenia. This suggests the hypothesis that these illnesses, as they occur naturally, involve the same neurochemical abnormality as is artificially produced by these drugs. Such hypotheses are of much more than purely theoretical interest because if they are true, then the naturally occurring illness may well be correctable or controllable by a drug with an exactly opposite neurochemical effect. And thus it seems to be, though the situation is complex and the details are confusing. *Imipramine* controls depression, *lithium* controls mania, and *chlorpromazine* controls schizophrenia. Imperfectly, it must be said, but the qualified success of these drugs lends strong support to the view that the victims of mental illness are the victims primarily of sheer chemical circumstance, whose origins are more metabolic and biological than they are social or psychological. If so, this fact is important, since better than 2 percent of the human population has a significant encounter with one of these conditions at some point in their lives. If we can discover the nature and origins of the complex chemical imbalances that underlie the major forms of mental illness, we may be able to cure them outright or even prevent their occurrence entirely.

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