CHAPTER

10

The Central Visual System

▼ INTRODUCTION

Although our visual system provides us with a unified picture of the world around us, this picture has multiple facets. Objects we see have shape and color. They have position in space, and sometimes they move. For us to see each of these properties, neurons somewhere in the visual system must be sensitive to them. Moreover, because we have two eyes, we actually have two visual images in our head, and somehow they must be merged.

In Chapter 9, we saw that in many ways the eye acts like a camera. But starting with the retina, the rest of the visual system is far more elaborate, far more interesting, and capable of doing far more than any camera. For example, we saw that the retina does not simply pass along information about the patterns of light and dark that fall on it. Rather, the retina *extracts* information about different facets of the visual image. There are more than 100 million photoreceptors in the retina but only 1 million axons leaving the eye carrying information to the rest of the brain. What we perceive about the world around us, therefore, depends on what information is extracted by the retina and how this information is analyzed and interpreted by the rest of the central nervous system (CNS). An example is color. There is no such thing as color in the physical world; there is simply a spectrum of visible wavelengths of light that are reflected by objects around us. Based on the information extracted by the three types of cone photoreceptors, however, our brain somehow synthesizes a rainbow of colors and fills our world with it.

In this chapter, we explore how the information extracted by the retina is analyzed by the central visual system. The pathway serving conscious visual perception includes the *lateral geniculate nucleus* (*LGN*) of the thalamus and the primary visual cortex, also called *area 17*, *V1*, or *striate cortex*. We will see that the information funneled through this geniculocortical pathway is processed in parallel by neurons specialized for the analysis of different stimulus attributes. The striate cortex then feeds this information to more than two dozen different extrastriate cortical areas in the temporal and parietal lobes, and many of these appear to be specialized for different types of analysis.

Much of what we know about the central visual system was first worked out in the domestic cat and then extended to the rhesus monkey, *Macaca mulatta*. The macaque monkey, as it is also called, relies heavily on vision for survival in its habitat, as do we humans. In fact, tests of the performance of this primate's visual system show that in virtually all respects, it rivals that of humans. Thus, although most of this chapter concerns the organization of the macaque visual system, most neuroscientists agree that it approximates very closely the situation in our own brain.

Although visual neuroscience cannot yet explain many aspects of visual perception (some interesting examples are shown in Figure 10.1), significant progress has been made in answering a more basic question: How do neurons represent the different facets of the visual world? By examining those stimuli that make different neurons in the visual cortex respond, and how these response properties arise, we begin to see how the brain portrays the visual world around us.

▼ THE RETINOFUGAL PROJECTION

The neural pathway that leaves the eye, beginning with the optic nerve, is often referred to as the **retinofugal projection**. The suffix *-fugal* is from the Latin word meaning "to flee" and is commonly used in neuroanatomy to describe a pathway that is directed away from a structure. Thus, a centrifugal projection goes away from the center, a corticofugal projection goes

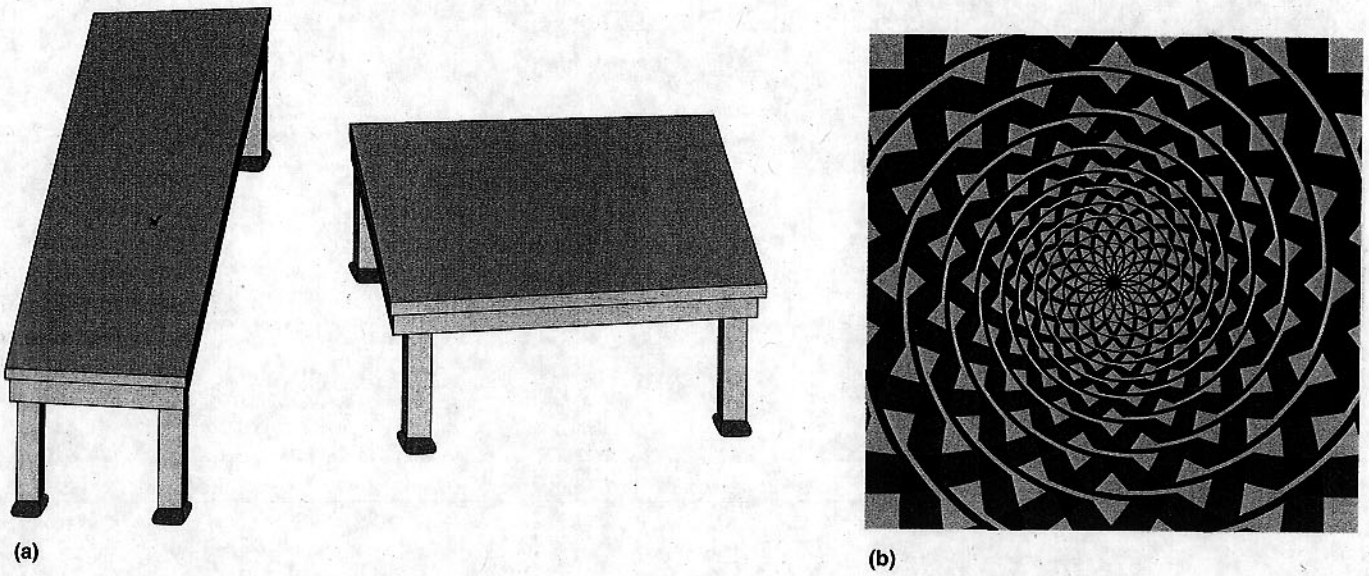


FIGURE 10.1
Perceptual illusions. (a) The two tabletops are of identical dimensions and are imaged on similarly sized patches of retina, but the perceived sizes are quite different. **(b)** This is an illusory spiral. Try tracing it with your finger.

away from the cortex, and the retinofugal projection goes away from the retina.

We begin our tour of the central visual system by looking at how the retinofugal projection courses from each eye to the brain stem on each side, and how the task of analyzing the visual world initially is divided among, and organized within, certain structures of the brain stem. Then we focus on the major arm of the retinofugal projection that mediates conscious visual perception.

The Optic Nerve, Optic Chiasm, and Optic Tract

The ganglion cell axons "fleeing" the retina pass through three structures before they form synapses in the brain stem. The components of this retinofugal projection are, in order, the optic nerve, the optic chiasm, and the optic tract (Figure 10.2). The **optic nerves** exit the left and right eyes

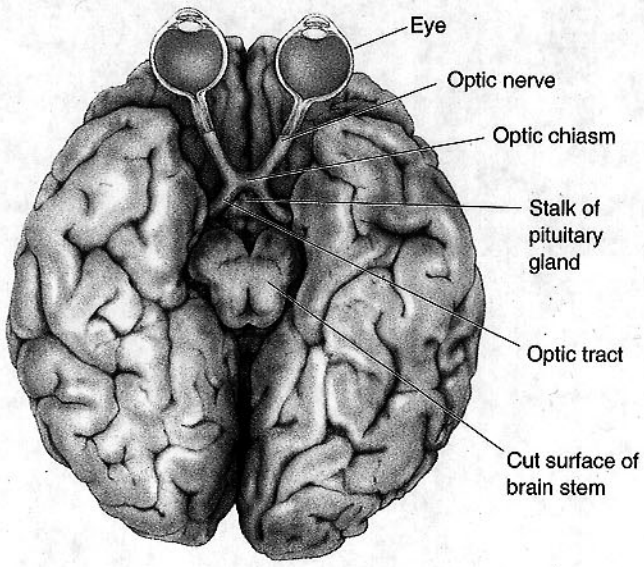


FIGURE 10.2
The retinofugal projection. This view of the base of the brain shows the optic nerves, optic chiasm, and optic tracts.

at the optic disks, travel through the fatty tissue behind the eyes in their bony orbits, then pass through holes in the floor of the skull. The optic nerves from both eyes combine to form the **optic chiasm** (named for the X shape of the Greek letter chi), which lies at the base of the brain, just anterior to where the pituitary gland dangles down. At the optic chiasm, the axons originating in the nasal retinas cross from one side to the other. The crossing of a fiber bundle from one side of the brain to the other is called a **decussation**. Because only the axons originating in the nasal retinas cross, we say that a partial decussation of the retinofugal projection occurs at the optic chiasm. Following the partial decussation at the optic chiasm, the axons of the retinofugal projections form the **optic tracts**, which run just under the pia along the lateral surfaces of the diencephalon.

Right and Left Visual Hemifields

To understand the significance of the partial decussation of the retinofugal projection at the optic chiasm, let's review the concept of the visual field introduced in Chapter 9. The full visual field is the entire region of space (measured in degrees of visual angle) that can be seen with both eyes looking straight ahead. Fix your gaze on a point straight ahead. Now imagine a vertical line passing through the fixation point, dividing the visual field into left and right halves. By definition, objects appearing to the left of the midline are in the left **visual hemifield,** and objects appearing to the right of the midline are in the right visual hemifield (Figure 10.3).

By looking straight ahead with both eyes open and then alternately closing one eye and then the other, you will see that the central portion of both visual hemifields is viewed by *both* retinas. This region of space is therefore called the **binocular visual field**. Notice that objects in the binocular region of the left visual hemifield will be imaged on the nasal retina of the left eye and on the temporal retina of the right eye. Because the fibers from the nasal portion of the left retina cross to the right side at the optic chiasm, all the information about the left visual hemifield is directed to the right side of the brain. Remember this rule of thumb: Optic nerve fibers cross in

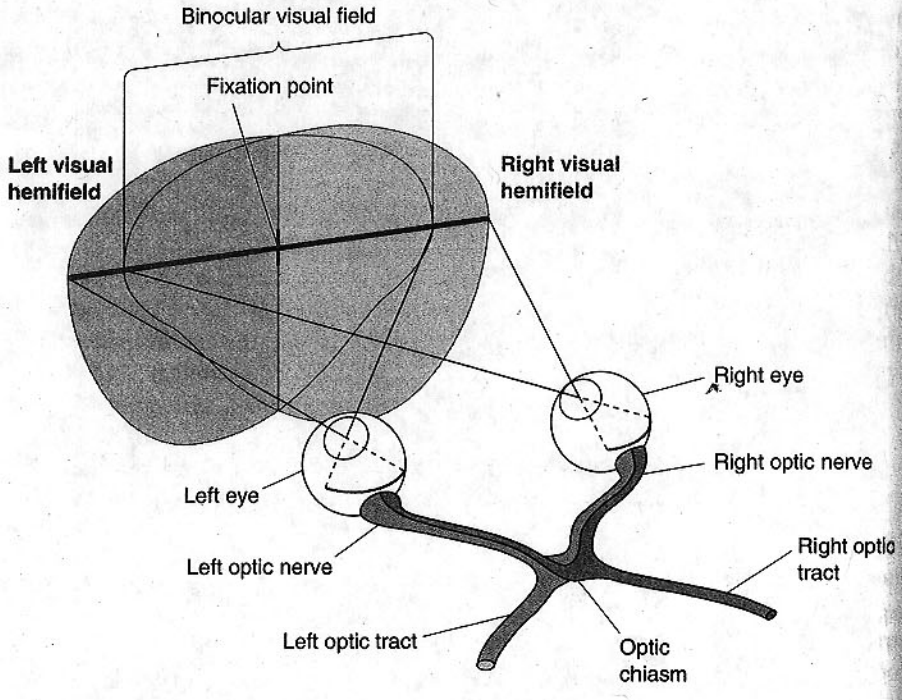


FIGURE 10.3
Right and left visual hemifields.
Ganglion cells in both retinas that are responsive to visual stimuli in the right visual hemifield project axons into the left optic tract. Similarly, ganglion cells "viewing" the left visual hemifield project into the right optic tract.

the optic chiasm so that *the left visual hemifield is "viewed" by the right hemisphere and the right visual hemifield is "viewed" by the left hemisphere.*

Targets of the Optic Tract

A small number of optic tract axons peel off to form synaptic connections with cells in the hypothalamus, and another 10% or so continue past the thalamus to innervate the midbrain. But most of them innervate the **lateral geniculate nucleus** (**LGN**) of the dorsal thalamus. The neurons in the LGN give rise to axons that project to the primary visual cortex. This projection from LGN to cortex is called the **optic radiation**. Lesions anywhere in the retinofugal projection from eye to LGN to visual cortex cause blindness in humans. Therefore, we know that it is this pathway that mediates conscious visual perception (Figure 10.4).

From our knowledge of how the visual world is represented in the retinofugal projection, we can predict the types of perceptual deficits that would result from its destruction at different levels, as might occur from a traumatic injury to the head, a tumor, or an interruption of the blood supply. As shown in Figure 10.5, while a transection of the left optic *nerve* would render a person blind in the left eye only, a transection of the left optic *tract* would lead to blindness in the right visual field as viewed through either eye. A midline transection of the optic chiasm would affect only the fibers that cross the midline. Because these fibers originate in the nasal portions of both retinas, blindness would result in the regions of the visual field viewed by the nasal retinas, that is, the peripheral visual fields on both sides (Box 10.1). Because unique deficits result from lesions at different sites, neurologists and neuro-ophthalmologists can locate sites of damage by assessing visual field deficits.

Nonthalamic Targets of the Optic Tract. As we have said, some retinal ganglion cells send axons to innervate structures other than the LGN. Direct projections to part of the hypothalamus play an important role in synchronizing a variety of biological rhythms, including sleep and wakefulness, with the daily dark-light cycle (see Chapter 19). Direct projections to part

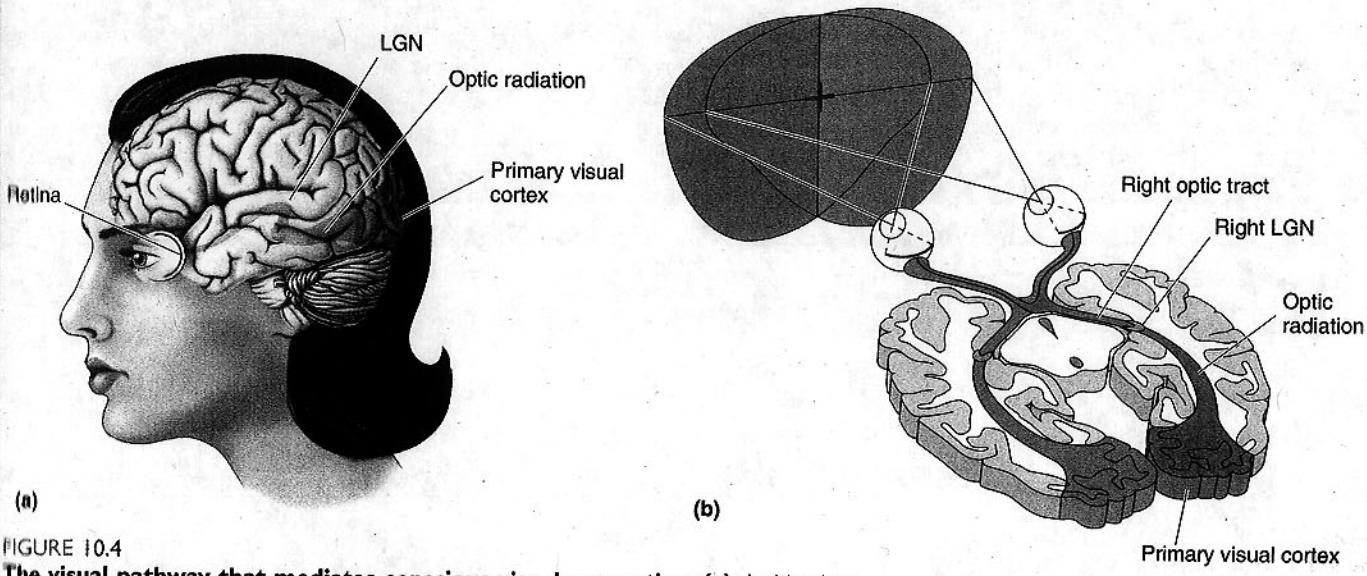


FIGURE 10.4
The visual pathway that mediates conscious visual perception. (a) A side view of the brain with the retinogeniculocortical pathway shown inside (blue). **(b)** A horizontal section through the brain exposing the same pathway.

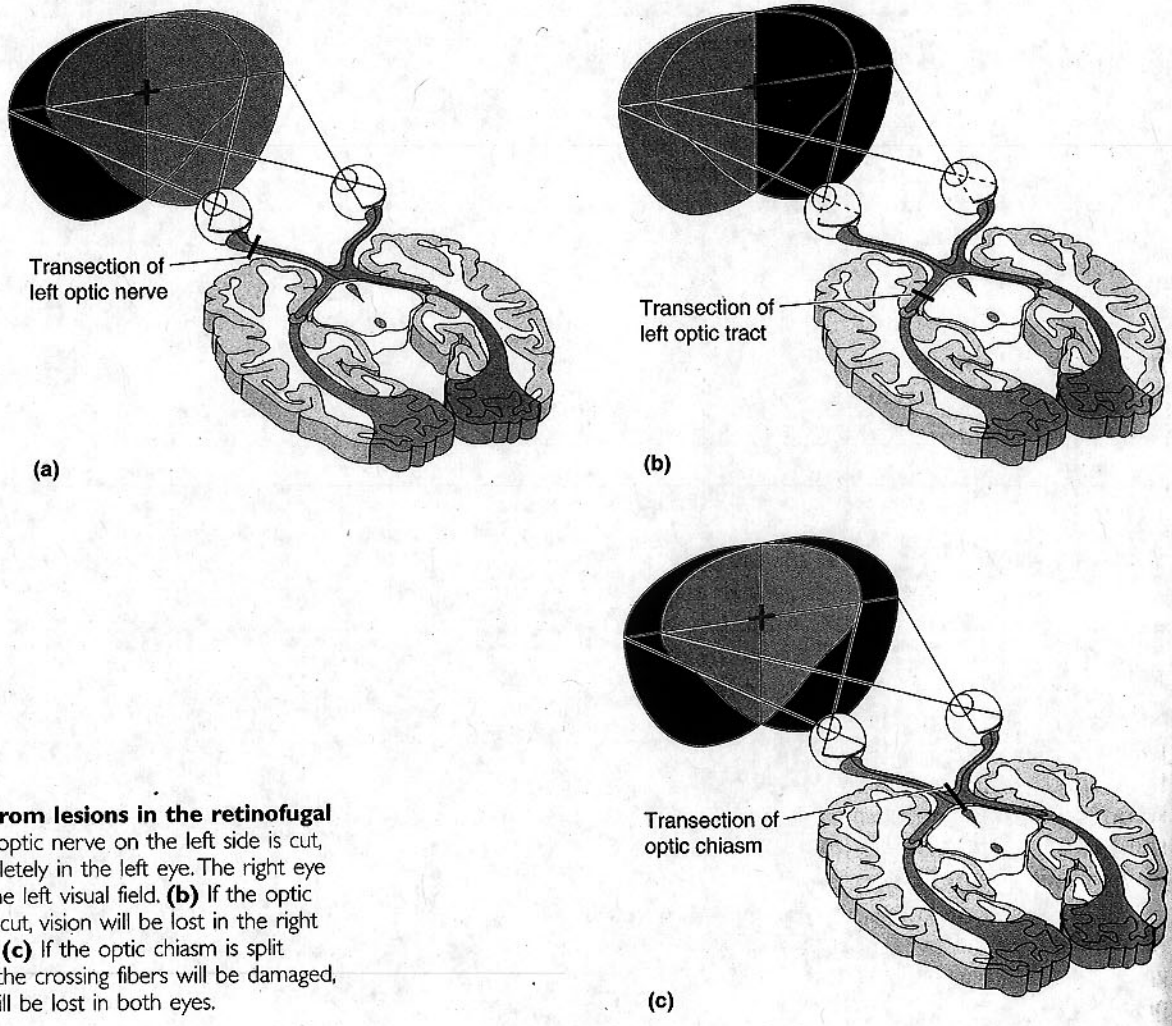


FIGURE 10.5
Visual field deficits from lesions in the retinofugal projection. (a) If the optic nerve on the left side is cut, vision will be lost completely in the left eye. The right eye still sees a portion of the left visual field. **(b)** If the optic tract on the left side is cut, vision will be lost in the right visual field of each eye. **(c)** If the optic chiasm is split down the middle, only the crossing fibers will be damaged, and peripheral vision will be lost in both eyes.

of the midbrain, called the *pretectum*, control the size of the pupil and certain types of eye movement. And about 10% of the ganglion cells in the retina project to a part of the midbrain tectum called the **superior colliculus** (Latin for "little hill") (Figure 10.6).

While 10% may not sound like much of a projection, bear in mind that in primates, this is about 150,000 neurons, which is equivalent to the *total*

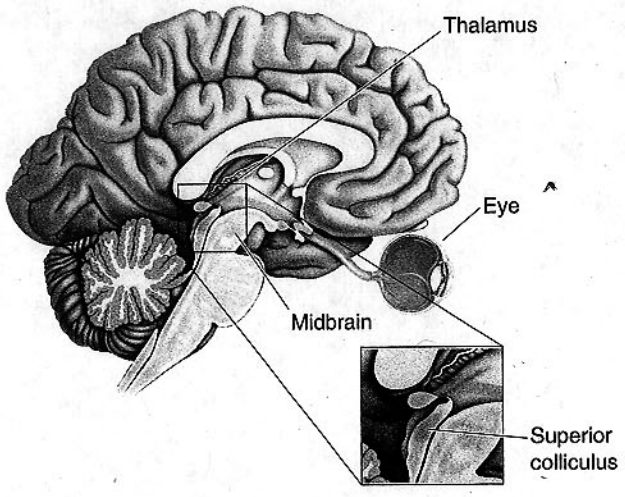


FIGURE 10.6
The superior colliculus. Located in the tectum of the midbrain, the superior colliculus is involved in generating saccadic eye movements, the quick jumps in eye position used to scan across a page while reading.

Box 10.1  OF SPECIAL INTEREST

David and Goliath

Many of you are familiar with the famous story of David and Goliath, which appears in the Hebrew scriptures (Old Testament). The armies of the Philistines and the Israelites were gathered for battle when Goliath, a Philistine, came forth and challenged the Israelites to settle the dispute by sending out their best man to face him in a fight to the death. Goliath, it seems, was a man of great proportions, measuring more than "six cubits" in height. If you consider that a cubit is the distance from the elbow to the tip of the middle finger, about 20 inches, this guy was more than 10 feet tall! Goliath was armed to the teeth with body armor, a javelin, and a sword. To face this giant, the Israelites sent David, a young and diminutive shepherd, armed only with a sling and five smooth stones. Here's how the action is described in the Revised Standard Version of the *Bible* (1 Samuel 17: 48):

> When the Philistine arose and came and drew near to meet David, David ran quickly toward the battle line to meet the Philistine. And David put his hand in his bag and took out a stone, and slung it, and struck the Philistine on his forehead; the stone sank into his forehead, and he fell on his face to the ground.

Now why, you might ask, are we giving a theology lesson in a neuroscience textbook? The answer is that our understanding of the visual pathway offers an explanation, in addition to divine intervention, for why Goliath was at a disadvantage in this battle. Body size is regulated by the secretion of growth hormone from the anterior lobe of the pituitary gland. In some cases, the anterior lobe becomes hypertrophied (swollen) and produces excessive amounts of the hormone, resulting in body growth to unusually large proportions. Such individuals are called pituitary giants and can measure well over 8 feet tall.

Pituitary hypertrophy also disrupts normal vision. Recall that the optic nerve fibers from the nasal retinas cross in the optic chiasm, which butts up against the stalk of the pituitary. Any enlargement of the pituitary compresses these crossing fibers and results in a loss of peripheral vision called *bitemporal hemianopia*, or *tunnel vision*. (See if you can figure out why this is true from what you know about the visual pathway.) We can speculate that David was able to draw close and smite Goliath, because when David raced to the battle line, the pituitary giant had completely lost sight of him.

number of retinal ganglion cells in a cat! In fact, the tectum of the midbrain is the major target of the retinofugal projection in all nonmammalian vertebrates (fish, amphibians, birds, and reptiles). In these vertebrate groups, the superior colliculus is called the **optic tectum**. This is why the projection from the retina to the superior colliculus is often called the **retinotectal projection**, even in mammals.

In the superior colliculus, a patch of neurons activated by a point of light, via indirect connections with motor neurons in the brain stem, commands eye and head movements to bring the image of this point in space onto the fovea. This branch of the retinofugal projection is thereby involved in orienting the eyes in response to new stimuli in the visual periphery. We will return to the superior colliculus when we discuss motor systems in Chapter 14.

▼ THE LATERAL GENICULATE NUCLEUS

The right and left lateral geniculate nuclei, located in the dorsal thalamus, are the major targets of the two optic tracts. Viewed in cross section, each LGN appears to be arranged in six distinct layers of cells (Figure 10.7). By convention, the layers are numbered 1 through 6, starting with the most ventral layer, layer 1. In three dimensions, the layers of the LGN are arranged like a stack of six pancakes, one on top of the other. The pancakes do not lie flat, however; they are bent around the optic tract like a knee joint. This shape explains the name geniculate, from the Latin *geniculatus*, meaning "like a little knee."

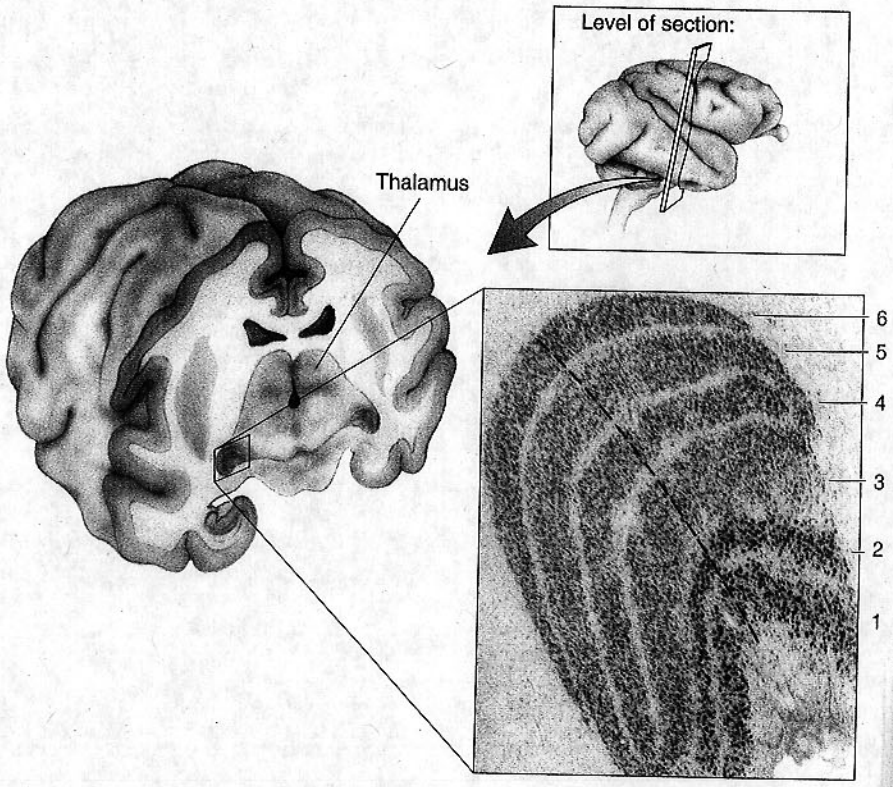


FIGURE 10.7
The LGN of the macaque monkey. The tissue has been stained to show cell bodies, which appear as purple dots. Notice particularly the six layers and the larger size of the cells in the two ventral layers (layers 1 and 2). (Source: Adapted from Hubel, 1988, p. 65.)

The LGN is the gateway to the visual cortex and, therefore, to conscious visual perception. Let's explore the structure and function of this thalamic nucleus.

The Segregation of Input by Eye and by Ganglion Cell Type

LGN neurons receive synaptic input from the retinal ganglion cells, and most geniculate neurons project an axon to primary visual cortex via the optic radiation. The segregation of LGN neurons into layers suggests that different types of retinal information are being kept separate at this synaptic relay, and indeed this is the case: Axons arising from M-type, P-type, and nonM-nonP ganglion cells in the two retinas synapse on cells in different LGN layers.

Recall from our rule of thumb that the *right* LGN receives information about the *left* visual field. The left visual field is viewed by both the nasal left retina and the temporal right retina. At the LGN, input from the two eyes is kept separate. In the right LGN, the right eye (ipsilateral) axons synapse on LGN cells in layers 2, 3, and 5. The left eye (contralateral) axons synapse on cells in layers 1, 4, and 6 (Figure 10.8).

A closer look at the LGN in Figure 10.7 reveals that the two ventral layers, 1 and 2, contain larger neurons, and the four more dorsal layers, 3 through 6, contain smaller cells. The ventral layers are therefore called **magnocellular LGN layers**, and the dorsal layers are called **parvocellular LGN layers**. Recall from Chapter 9 that ganglion cells in the retina may also be classified into magnocellular and parvocellular groups. As it turns out, P-type ganglion cells in the retina project exclusively to the parvocellular LGN, and M-type ganglion cells in the retina project entirely to the magnocellular LGN.

In addition to the neurons in the six principal layers of the LGN, numerous tiny neurons also lie just ventral to each layer. Cells in these **konio-**

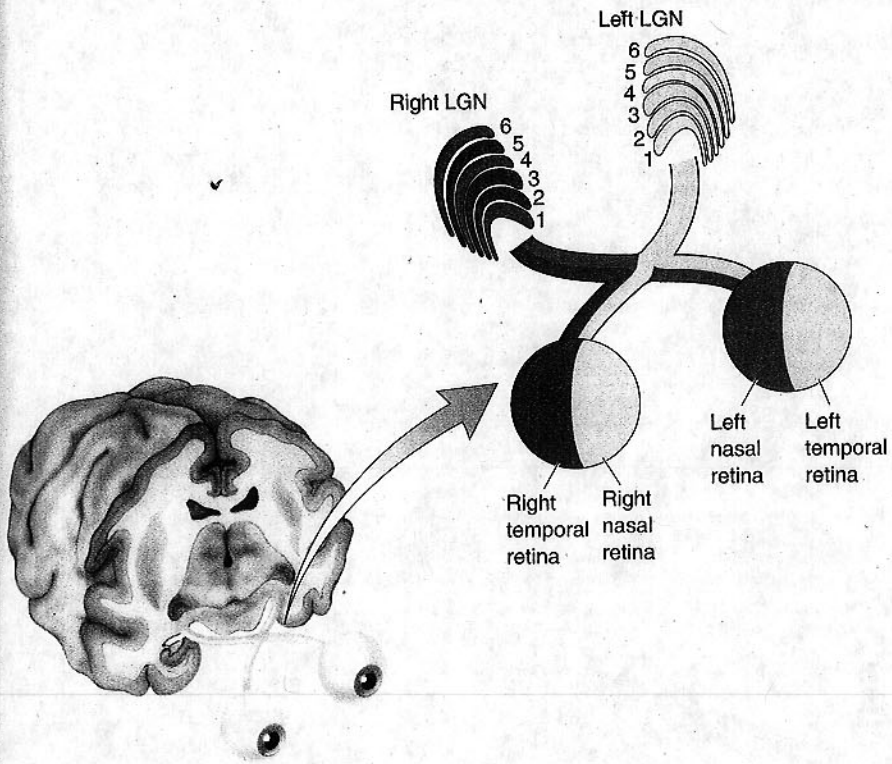


FIGURE 10.8
Retinal inputs to the LGN layers.

cellular layers (*konio* is from the Greek for "dust") receive input from the nonM-nonP types of retinal ganglion cells and also project to visual cortex. Note that the koniocellular layers are not uniquely numbered, because historically, the six thick layers were numbered before cells in the koniocellular layers were discovered. In Chapter 9, we saw that in the retina, M-type, P-type, and nonM-nonP ganglion cells respond differently to light and color. In the LGN, the different information derived from the three categories of retinal ganglion cells from the two eyes remains segregated.

The anatomical organization of the LGN supports the idea that the retina gives rise to streams of information that are processed in parallel. This organization is summarized in Figure 10.9.

Receptive Fields

By inserting a microelectrode into the LGN, it is possible to study the action potential discharges of geniculate neurons in response to visual stimuli, just as was done in the retina. The surprising conclusion of such studies is that the visual receptive fields of LGN neurons are almost identical to those of the ganglion cells that feed them. For example, magnocellular LGN neurons have relatively large center-surround receptive fields, respond to stimulation of their receptive field centers with a transient burst of action potentials, and are insensitive to differences in wavelength. All in all, they are just like M-type ganglion cells. Likewise, parvocellular LGN cells, like P-type retinal ganglion cells, have relatively small center-surround receptive fields and respond to stimulation of their receptive field centers with a sustained increase in the frequency of action potentials; many of them exhibit color opponency. Receptive fields of cells in the koniocellular layers are center-surround and have either light/dark or color opponency. Within all layers of the LGN, the neurons are activated by only one eye (i.e., they are monocular) and ON-center and OFF-center cells are intermixed.

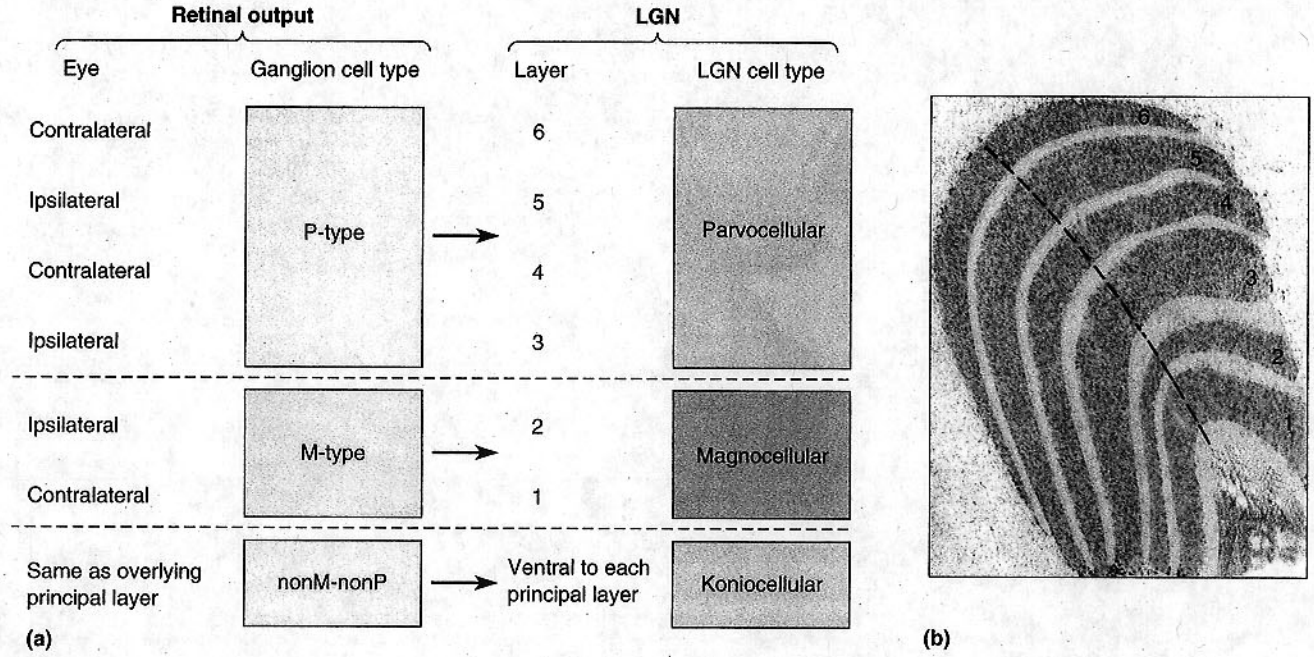


FIGURE 10.9
The organization of the LGN.
(a) Ganglion cell inputs to the different LGN layers. **(b)** A thin koniocellular layer (shown in pink) is ventral to each of the six principal layers.

Nonretinal Inputs to the LGN

What makes the similarity of LGN and ganglion cell receptive fields so surprising is that the retina is not the main source of synaptic input to the LGN. The major input, constituting about 80% of the excitatory synapses, comes from primary visual cortex. Thus, one might reasonably expect that this corticofugal feedback pathway would significantly alter the qualities of the visual responses recorded in the LGN. So far, however, a role for this massive input has not been clearly identified.

The LGN also receives synaptic inputs from neurons in the brain stem whose activity is related to alertness and attentiveness (see Chapters 15 and 19). Have you ever "seen" a flash of light when you are startled in a dark room? This perceived flash might be a result of the direct activation of LGN neurons by this pathway. Usually, however, this input does not directly evoke action potentials in LGN neurons. But it can powerfully modulate the magnitude of LGN responses to visual stimuli. (Recall modulation from Chapters 5 and 6.) Thus, the LGN is more than a simple relay from retina to cortex; it is the first site in the ascending visual pathway where what we see is influenced by how we feel.

▼ ANATOMY OF THE STRIATE CORTEX

The LGN has a single major synaptic target: primary visual cortex. Recall from Chapter 7 that the cortex may be divided into a number of distinct areas based on their connections and cytoarchitecture. **Primary visual cortex** is Brodmann's **area 17** and is located in the occipital lobe of the primate brain. Much of area 17 lies on the medial surface of the hemisphere, surrounding the calcarine fissure (Figure 10.10). Other terms used interchangeably to describe the primary visual cortex are **V1** and **striate cortex**. (The term *striate* refers to the fact that area V1 has an unusually dense stripe of myelinated axons running parallel to the surface that appears white in unstained sections.)

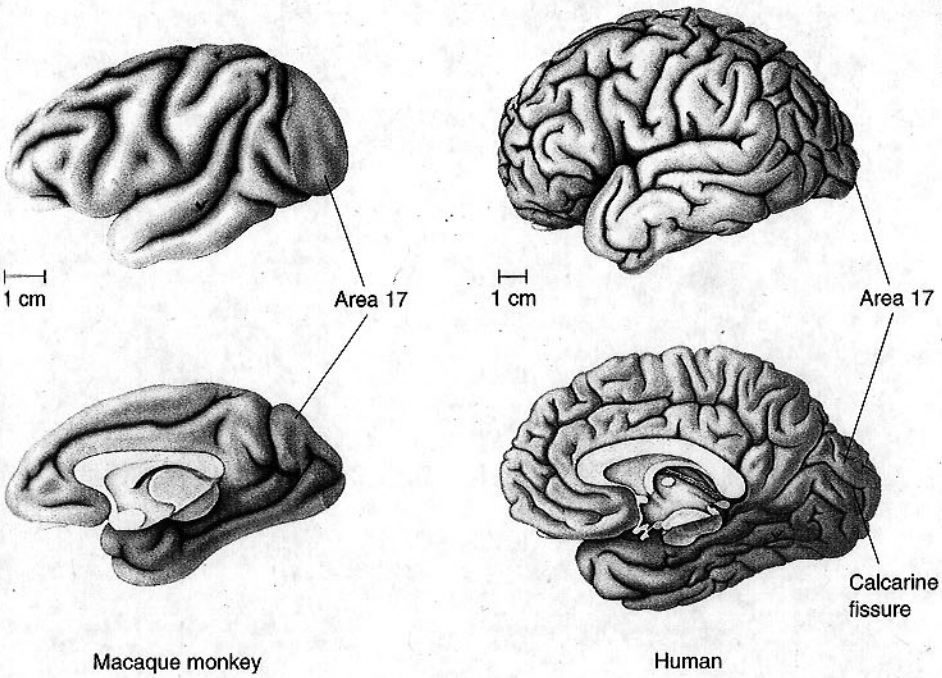


FIGURE 10.10
Primary visual cortex. Top views are lateral; bottom views are medial.

We have seen that the axons of different types of retinal ganglion cells synapse on anatomically segregated neurons in the LGN. In this section, we look at the anatomy of the striate cortex and trace the connections different LGN cells make with cortical neurons. In a later section, we explore how this information is analyzed by cortical neurons. As we did in the LGN, in striate cortex we'll see a close correlation between structure and function.

Retinotopy

The projection starting in the retina and extending to LGN and V1 illustrates a general organizational feature of the central visual system called retinotopy. **Retinotopy** is an organization whereby neighboring cells in the retina feed information to neighboring places in their target structures—in this case, the LGN and striate cortex. In this way, the two-dimensional surface of the retina is *mapped* onto the two-dimensional surface of the subsequent structures (Figure 10.11a).

There are three important points to remember about retinotopy. First, the mapping of the visual field onto a retinotopically organized structure is often distorted, because visual space is not sampled uniformly by the cells in the retina. Recall from Chapter 9 that there are many more ganglion cells with receptive fields in or near the fovea than in the periphery. Thus, the representation of the visual field is distorted in striate cortex: The central few degrees of the visual field are overrepresented, or *magnified*, in the retinotopic map (Figure 10.11b).

The second point to remember is that a discrete point of light can activate many cells in the retina, and often many more cells in the target structure, due to the overlap of receptive fields. The image of a point of light on the retina actually activates a large population of cortical neurons; every neuron that contains that point in its receptive field is potentially activated. Thus, when the retina is stimulated by a point of light, the activity in striate cortex is a broad distribution with a peak at the corresponding retinotopic location.

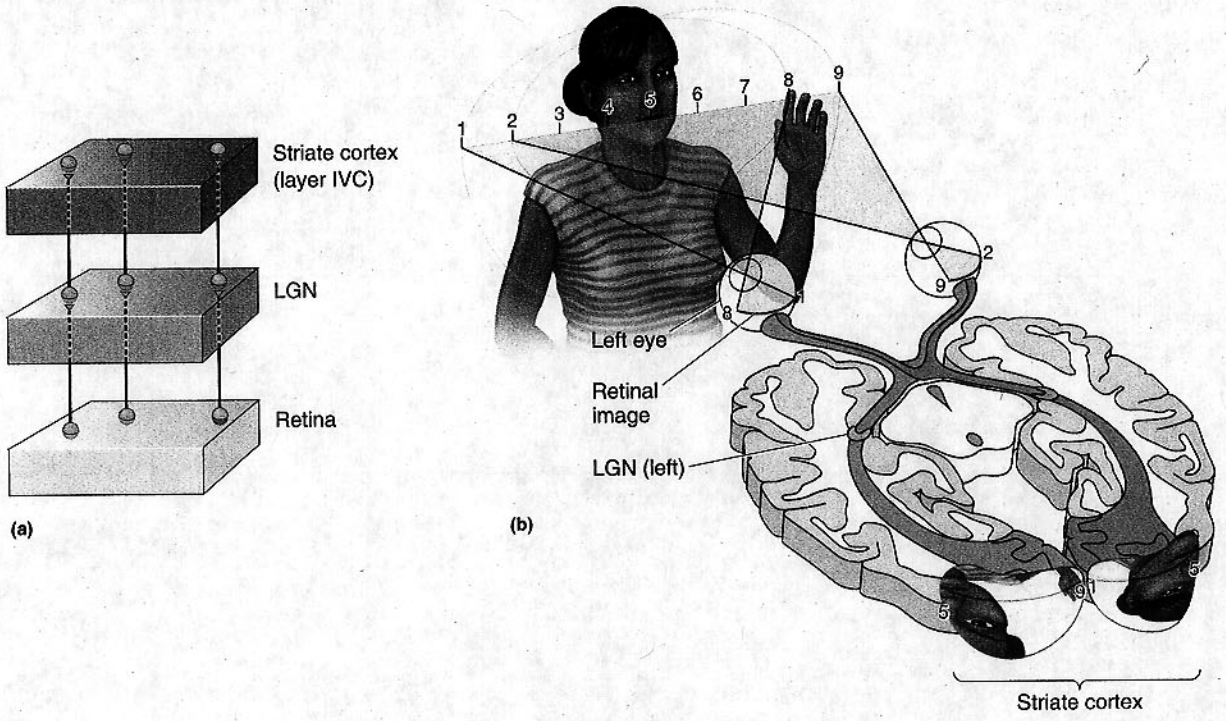


FIGURE 10.11
The retinotopic map in striate cortex. (a) Neighboring locations on the retina project to neighboring locations in the LGN. This retinotopic representation is preserved in the LGN projection to V1. **(b)** The lower portion of V1 represents information about the top half of visual space, and the upper portion of V1 represents the bottom half of visual space. Notice also that the map is distorted, with more tissue devoted to analysis of the central visual field. Similar maps are found in the superior colliculus, LGN, and other visual cortical areas.

Finally, don't be misled by the word "map." There are no pictures in the primary visual cortex for a little person in our brain to look at. While it's true that the arrangement of connections establishes a mapping between the retina and V1, perception is based on the brain's interpretation of distributed patterns of activity, not literal snapshots of the world. (We discuss visual perception later in this chapter.)

Lamination of the Striate Cortex

The neocortex in general, and striate cortex in particular, have neuronal cell bodies arranged into about a half-dozen layers. These layers can be seen clearly in a Nissl stain of the cortex, which, as described Chapter 2, leaves a deposit of dye (usually blue or violet) in the soma of each neuron. Starting at the white matter (containing the cortical input and output fibers), the cell layers are named by Roman numerals VI, V, IV, III, and II. Layer I, just under the pia mater, is largely devoid of neurons and consists almost entirely of axons and dendrites of cells in other layers (Figure 10.12). The full thickness of the striate cortex from white matter to pia is about 2 mm, the height of the lowercase letter m.

As Figure 10.12 shows, describing the lamination of striate cortex as a six-layer scheme is somewhat misleading. There are actually at least nine distinct layers of neurons. To maintain Brodmann's convention that neocortex has six layers, however, neuroanatomists combine three sublayers into layer IV, labeled IVA, IVB, and IVC. Layer IVC is further divided into two tiers called IVCα and IVCβ. The anatomical segregation of neurons into

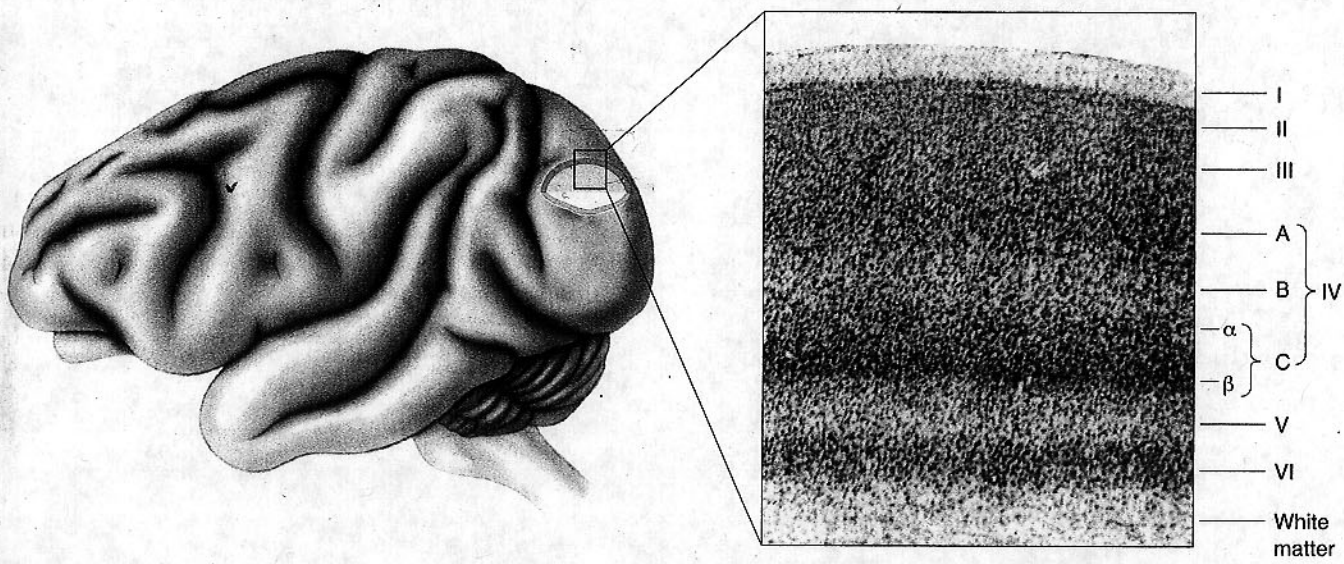


FIGURE 10.12
The cytoarchitecture of the striate cortex. The tissue has been Nissl stained to show cell bodies, which appear as dots. (Source: Adapted from Hubel, 1988, p. 97.)

layers suggests that there is a division of labor in the cortex, similar to what we saw in the LGN. We can learn a lot about how the cortex handles visual information by examining the structure and connections of its different layers.

The Cells of Different Layers. Many different neuronal shapes have been identified in striate cortex, but here we focus on two principal types, defined by the appearance of their dendritic trees (Figure 10.13). *Spiny stellate cells* are small neurons with spine-covered dendrites that radiate out from the cell body (recall dendritic spines from Chapter 2). They are seen primarily in the two tiers of layer IVC. Outside layer IVC are many *pyramidal cells*. These neurons are also covered with spines and are characterized by a single thick apical dendrite that branches as it ascends toward the pia mater and by multiple basal dendrites that extend horizontally.

Notice that a pyramidal cell in one layer may have dendrites extending into other layers. It is important to remember that *only pyramidal cells send axons out of striate cortex* to form connections with other parts of the brain. The axons of stellate cells make local connections only within the cortex.

In addition to the spiny neurons, inhibitory neurons, which lack spines, are sprinkled in all cortical layers as well. These neurons form only local connections.

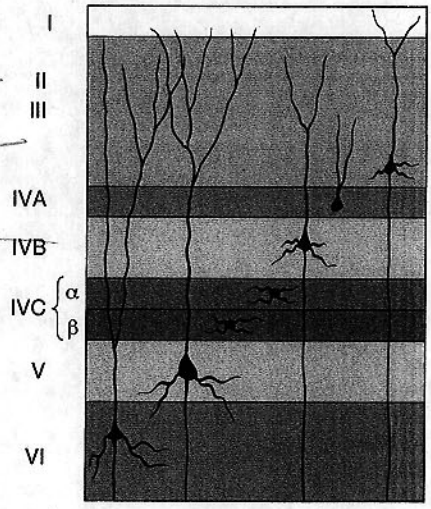


FIGURE 10.13
The dendritic morphology of some cells in striate cortex. Notice particularly that pyramidal cells are found in layers III, IVB, V, and VI and that spiny stellate cells are found in layer IVC.

Inputs and Outputs of the Striate Cortex

The distinct lamination of the striate cortex is reminiscent of the layers we saw in the LGN. In the LGN, every layer receives retinal afferents and sends efferents to the visual cortex. In the visual cortex, the situation is different; only a subset of the layers receives input from the LGN or sends output to a different cortical or subcortical area.

Axons from the LGN terminate in several different cortical layers, with the largest number going to layer IVC. We've seen that the output of the LGN is divided into streams of information, for example, from the magnocellular and parvocellular layers serving the right and left eyes. These streams remain anatomically segregated in layer IVC.

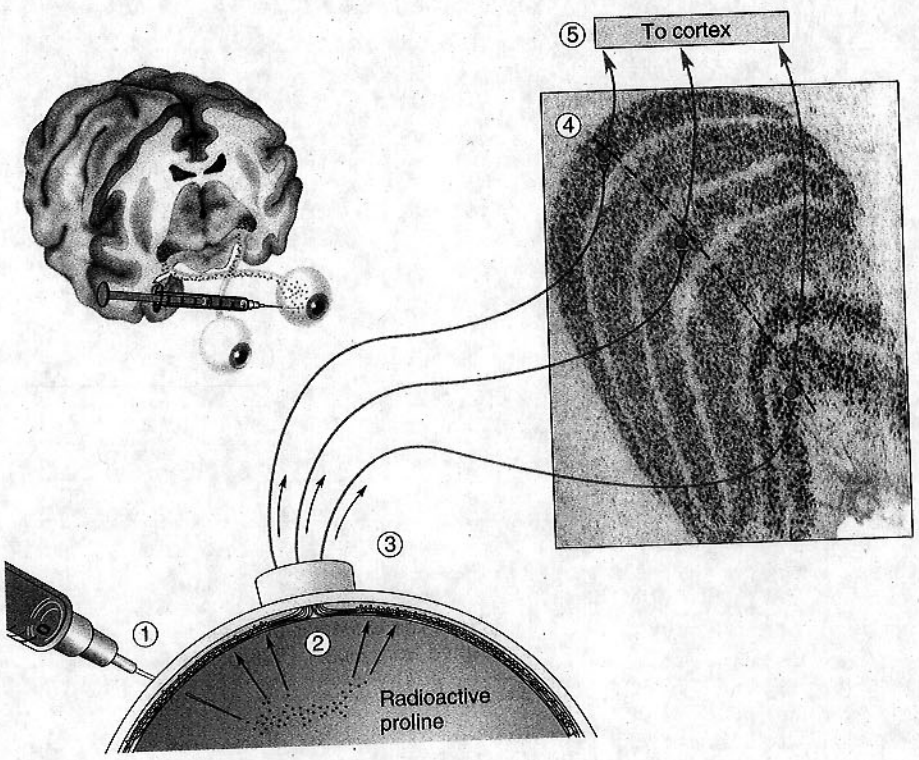
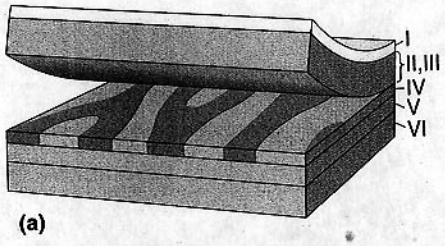


FIGURE 10.14
Transneuronal autoradiography. Radioactive proline is ① injected into one eye, where it is ② taken up by retinal ganglion cells and incorporated into proteins that are ③ transported down the axons to the LGN. Some radioactivity spills out of the retinal terminals and is ④ taken up by LGN neurons that then ⑤ transport it to striate cortex. The location of radioactivity can be determined using autoradiography.



(a)

(b)

FIGURE 10.15
Ocular dominance columns in striate cortex. (a) The organization of ocular dominance columns in layer IV of macaque monkey striate cortex. The distribution of LGN axons serving one eye is shaded blue. In cross section, these eye-specific zones appear as patches, each about 0.5 mm wide, in layer IV. Peeled-back layers reveal that the ocular dominance columns in layer IV look like zebra stripes. **(b)** An autoradiograph of a histological section of layer IV viewed from above. Two weeks prior to the experiment, one eye of this monkey was injected with radioactive proline. In the autoradiograph, the radioactive LGN terminals appear bright on a dark background. (Source: LeVay et al., 1980.)

Magnocellular LGN neurons project to layer IVCα, and parvocellular LGN neurons project to layer IVCβ. Imagine that the two tiers of layer IVC are pancakes, stacked one (α) on top of the other (β). Because the input from the LGN to the cortex is arranged topographically, we see that layer IVC contains two overlapping retinotopic maps, one from the magnocellular LGN (IVCα) and the other from the parvocellular LGN (IVCβ). Koniocellular LGN axons follow a different path, bypassing layer IV to make synapses in layers II and III.

Ocular Dominance Columns. How are the left eye and right eye LGN inputs segregated when they reach layer IVC of striate cortex? The answer was provided by a ground-breaking experiment performed in the early 1970s at Harvard Medical School by neuroscientists David Hubel and Torsten Wiesel. They injected a radioactive amino acid into one eye of a monkey (Figure 10.14). This amino acid was incorporated into proteins by the ganglion cells, and the proteins were transported down the ganglion cell axons into the LGN (recall anterograde transport from Chapter 2). Here, the radioactive proteins spilled out of the ganglion cell axon terminals and were taken up by nearby LGN neurons. But not all LGN cells took up the radioactive material; only those cells that were postsynaptic to the inputs from the injected eye incorporated the labeled protein. These cells then transported the radioactive proteins to their axon terminals in layer IVC of striate cortex. The location of the radioactive axon terminals was visualized by first placing a film of emulsion over thin sections of striate cortex and later developing the emulsion like a photograph, a process called *autoradiography* (introduced in Chapter 6). The resulting collection of silver grains on the film marked the location of the radioactive LGN inputs.

In sections cut perpendicular to the cortical surface, Hubel and Wiesel observed that the distribution of axon terminals relaying information from the injected eye was not continuous in layer IVC, but rather was split up into a series of equally spaced patches, each about 0.5 mm wide (Figure 10.15a). These patches were termed **ocular dominance columns**.

In later experiments, the cortex was sectioned tangentially, parallel to layer IV. This revealed that the left eye and right eye inputs to layer IV are laid out as a series of alternating bands, like the stripes of a zebra (Figure 10.15b).

Innervation of Other Cortical Layers from Layer IVC. Most intracortical connections extend perpendicular to the cortical surface along radial lines that run across the layers, from white matter to layer I. This pattern of *radial connections* maintains the retinotopic organization established in layer IV. Therefore, a cell in layer VI, for example, receives information from the same part of the retina as does a cell above it in layer IV (Figure 10.16a). However, the axons of some layer III pyramidal cells extend collateral branches that make *horizontal connections* within layer III (Figure 10.16b). Radial and horizontal connections play different roles in the analysis of the visual world, as we'll see later in the chapter.

Layer IVC stellate cells project axons radially up mainly to layers IVB and III where, for the first time, information from the left eye and right eye begins to mix (Figure 10.17). Whereas all layer IVC neurons receive only monocular input, most neurons in layers II and III receive binocular input coming from both eyes. Even so, there continues to be considerable anatomical segregation of the magnocellular and parvocellular processing streams. Layer IVCα, which receives magnocellular LGN input, projects mainly to cells in layer IVB. Layer IVCβ, which receives parvocellular LGN input, projects mainly to layer III. In layers III and IVB, an axon may form synapses with the dendrites of pyramidal cells of all layers.

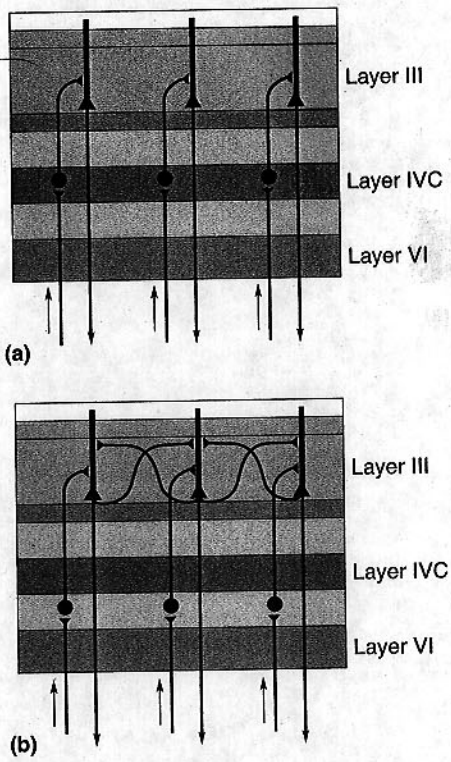


FIGURE 10.16
Patterns of intracortical connections. **(a)** Radial connections. **(b)** Horizontal connections.

Striate Cortex Outputs. As previously mentioned, the pyramidal cells send axons out of striate cortex into the white matter. The pyramidal cells in different layers innervate different structures. Layer II, III, and IVB pyramidal cells send their axons to other cortical areas. Layer V pyramidal cells send axons all the way down to the superior colliculus and pons. Layer VI pyramidal cells give rise to the massive axonal projection back to the LGN (Figure 10.18). Pyramidal cell axons in all layers also branch and form local connections in the cortex.

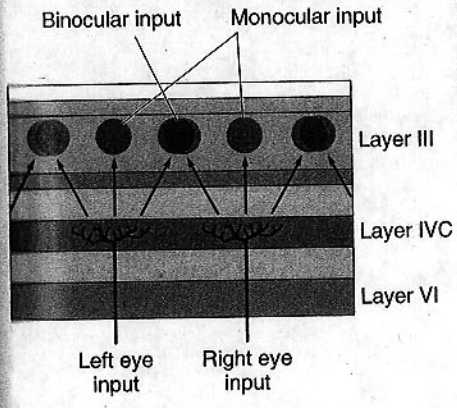


FIGURE 10.17
The mixing of information from the two eyes. Axons project from layer IVC to more superficial layers. Most layer III neurons receive binocular input from both left and right eyes.

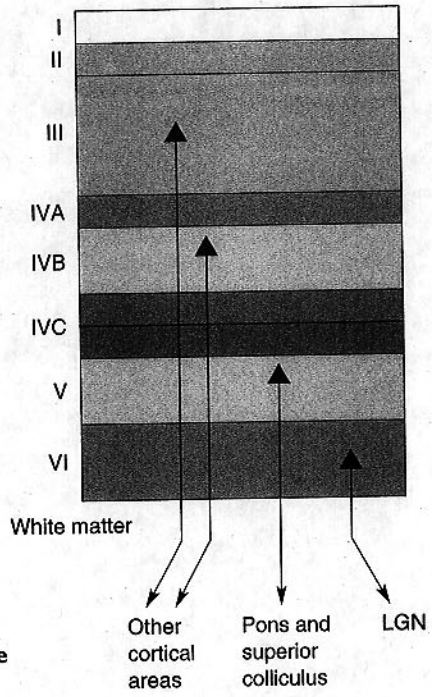
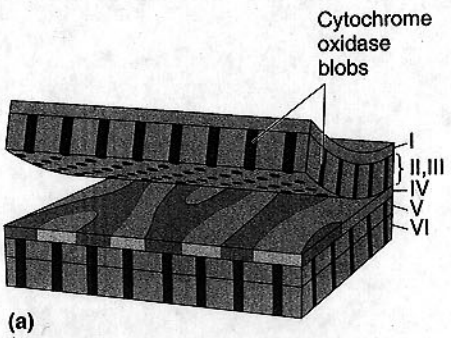


FIGURE 10.18
Patterns of outputs from the striate cortex.



(a)

(b)

FIGURE 10.19
Cytochrome oxidase blobs. (a) The organization of cytochrome oxidase blobs in macaque monkey striate cortex. **(b)** A photograph of a histological section of layer III, stained for cytochrome oxidase and viewed from above. (Source: Courtesy of Dr. S. H. C. Hendry.)

Cytochrome Oxidase Blobs

As we have seen, layers II and III play a key role in visual processing, providing most of the information that leaves V1 for other cortical areas. Anatomical studies suggest that the V1 output comes from two distinct populations of neurons in the superficial layers. When striate cortex is stained to reveal the presence of **cytochrome oxidase**, a mitochondrial enzyme used for cell metabolism, the stain is not uniformly distributed in layers II and III. Rather, the cytochrome oxidase staining in cross sections of striate cortex appears as a colonnade, a series of pillars at regular intervals, running the full thickness of layers II and III and also in layers V and VI (Figure 10.19a). When the cortex is sliced tangentially through layer III, these pillars appear like the spots of a leopard (Figure 10.19b). These pillars of cytochrome oxidase-rich neurons have come to be called **blobs**. The blobs are in rows, each blob centered on an ocular dominance stripe in layer IV. Between the blobs are "interblob" regions. The blobs receive direct LGN input from the koniocellular layers, as well as parvocellular and magnocellular input from layer IVC of striate cortex.

▼ PHYSIOLOGY OF THE STRIATE CORTEX

Beginning in the early 1960s, Hubel and Wiesel were the first to systematically explore the physiology of striate cortex with microelectrodes. They were students of Stephen Kuffler, who was then at Johns Hopkins University and later moved with them to Harvard. They extended Kuffler's innovative methods of receptive field mapping to the central visual pathways. After showing that LGN neurons behave much like retinal ganglion cells, they turned their attention to striate cortex, initially in cats and later in monkeys. (Here we focus on the monkey cortex.) The work that continues today on the physiology of striate cortex is built on the solid foundation provided by Hubel and Wiesel's pioneering studies. Their contributions to our understanding of the cerebral cortex were recognized with the Nobel Prize in 1981.

Receptive Fields

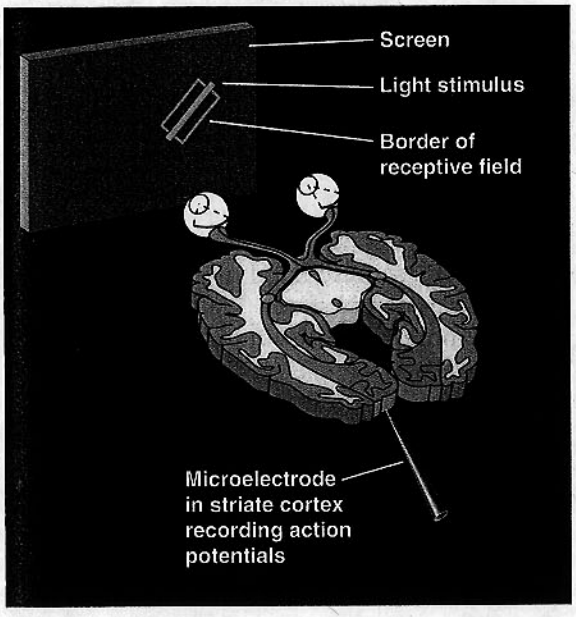
By and large, the receptive fields of neurons in layer IVC are similar to the magnocellular and parvocellular LGN neurons providing their input. This means they are generally small monocular center-surround receptive fields. In layer IVCα the neurons are insensitive to the wavelength of light, whereas in layer IVCβ the neurons exhibit center-surround color opponency. Outside layer IVC, new receptive field characteristics, not observed in the retina or LGN, are found. We will explore these in some depth, because they provide clues about the role V1 plays in visual processing and perception.

Binocularity. Each neuron in layers IVCα and IVCβ receives afferents from a layer of the LGN representing either eye. Monocular neurons from either eye are also clumped together in V1 rather than randomly intermixed. This accounts for ocular dominance columns that can be visualized in layer IVC with autoradiography. As we have already seen, the axons leaving layer IVC diverge and innervate more superficial cortical layers. As a consequence of the divergence, there is a mixing of inputs from the two eyes (see Figure 10.17). Microelectrode recordings confirm this anatomical fact; most neurons in layers superficial to IVC are binocular, responding to light in either eye. We say that the neurons have **binocular receptive**

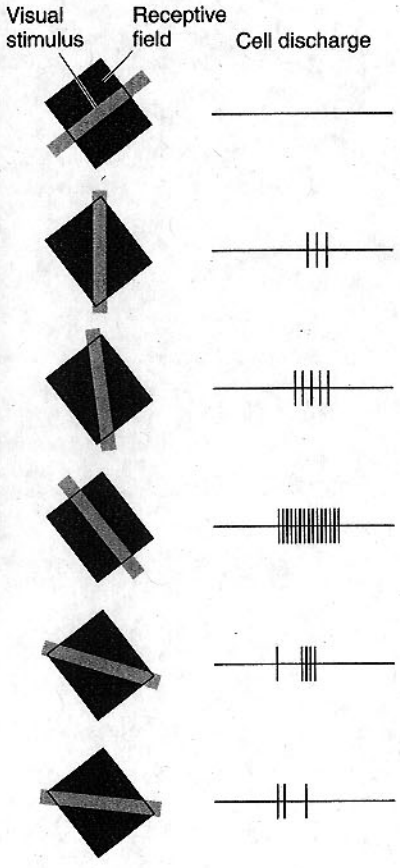
fields, meaning that they actually have two receptive fields, one in the ipsilateral and one in the contralateral eye.

The construction of binocular receptive fields is essential in binocular animals, such as humans. Without binocular neurons, we would probably be unable to use the inputs from both eyes to form a single image of the world around us. Retinotopy is preserved because the two receptive fields of a binocular neuron are precisely placed on the retinas such that they are "looking" at the same point in space. We still speak of ocular dominance columns in superficial cortical layers. However, now instead of the sharp monocular columns of layer IVC, there are patches of neurons that are more strongly driven by one eye than the other (i.e., they are dominated by one eye), even though they are binocular.

Orientation Selectivity. Most of the receptive fields in the retina, LGN, and layer IVC are circular and give their greatest response to a spot of light matched in size to the receptive field center. Outside layer IVC, we encounter cells that no longer follow this pattern. While small spots can elicit a response from many cortical neurons, it is usually possible to produce a much greater response with other stimuli. Rather by accident, Hubel and Wiesel found that many neurons in V1 respond best to an elongated bar of light moving across their receptive fields. But the orientation of the bar is critical. The greatest response is given to a bar with a particular orientation; perpendicular bars generally elicit much weaker responses (Figure 10.20). Neurons having this type of response are said to exhibit **orientation selectivity**. Most of the V1 neurons outside layer IVC (and



(a)



(b)

FIGURE 10.20
Orientation selectivity. (a) The responses of an orientation-selective neuron are monitored as visual stimuli are presented in its receptive field. The visual stimulus is a bar of light. **(b)** Light bars of various orientations (left) elicit very different responses (right). The optimal orientation for this neuron is 45° counterclockwise from vertical.

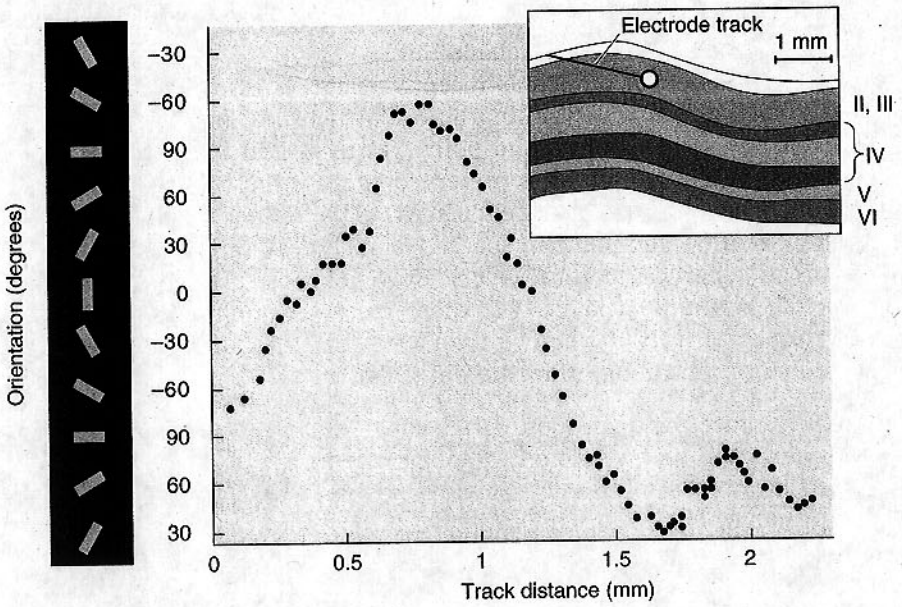


FIGURE 10.21
Systematic variation of orientation preferences across striate cortex. As an electrode is advanced tangentially across layer III of striate cortex, the orientation preference of the neurons encountered is recorded and plotted. Notice that there is a periodic, regular shift in preferred orientation. (Source: Adapted from Hubel and Wiesel, 1968.)

some within) are orientation selective. The optimal orientation for a neuron can be any angle around the clock.

If V1 neurons can have any optimal orientation, you might wonder whether the orientation selectivity of nearby neurons is related. From the earliest work of Hubel and Wiesel, the answer to this question was an emphatic yes. As a microelectrode is advanced radially (perpendicular to the surface) from one layer to the next, the preferred orientation remains the same for all the selective neurons encountered from layer II down through layer VI. Hubel and Wiesel called such a radial column of cells an **orientation column**.

As an electrode passes tangentially (parallel to the surface) through the cortex in a single layer, the preferred orientation progressively shifts. We now know, from the use of a technique called optical imaging, that there is a mosaiclike pattern of optimal orientations in striate cortex (Box 10.2). If an electrode is passed at certain angles through this mosaic, the preferred orientation rotates like the sweep of the minute hand of a clock, from the top of the hour to ten past to twenty past, and so on (Figure 10.21). If the electrode is moved at other angles, more sudden shifts in preferred orientation occur. Hubel and Wiesel found that a complete 180° shift in preferred orientation required a traverse of about 1 mm, on average, within layer III.

The analysis of stimulus orientation appears to be one of the most important functions of striate cortex. Orientation-selective neurons are thought to be specialized for the *analysis of object shape*.

Direction Selectivity. Many V1 receptive fields exhibit **direction selectivity**; they respond when a bar of light at the optimal orientation moves perpendicular to the orientation in one direction but not in the opposite direction. Direction-selective cells in V1 are a subset of the cells that are orientation selective. Figure 10.22 shows how a direction-selective cell responds to a moving stimulus. Notice that the cell responds to an elongated stimulus swept across the receptive field, but only in a particular direction of movement. Sensitivity to the direction of stimulus motion is a hallmark of neurons receiving input from the magnocellular layers of the LGN. Direction-selective neurons are thought to be specialized for the *analysis of object motion*.

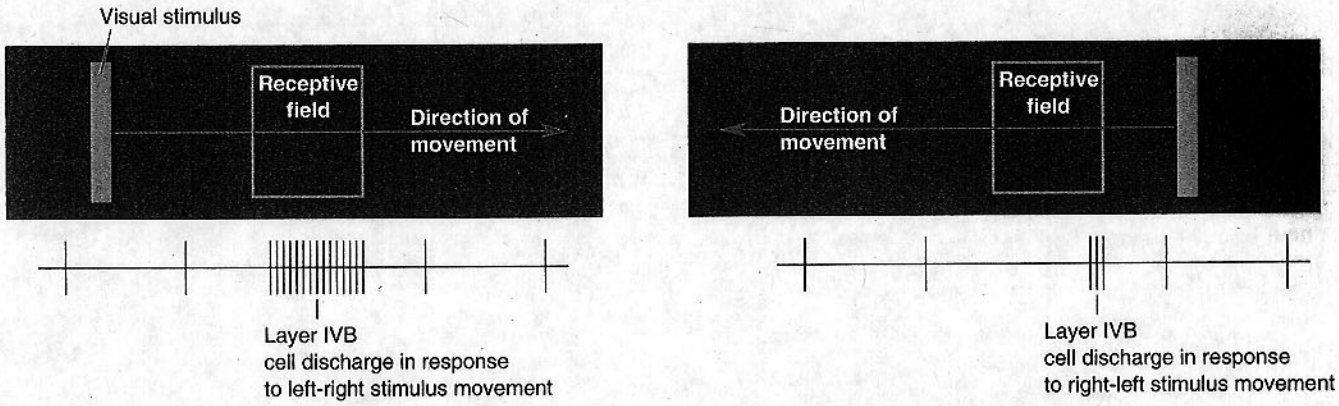


FIGURE 10.22
Direction selectivity. With a bar stimulus at the optimal orientation, the neuron responds strongly when the bar is swept to the right but weakly when it is swept to the left.

Simple and Complex Receptive Fields. Neurons in the LGN have antagonistic center-surround receptive fields, and this organization accounts for the responses of neurons to visual stimuli. For example, a small spot in the center of the receptive field may yield a much stronger response than a larger spot also covering the antagonistic surround. What do we know about the inputs to V1 neurons that might account for binocularity, orientation selectivity, and direction selectivity in their receptive fields? Binocularity is easy; we have seen that binocular neurons receive afferents from both eyes. The mechanisms underlying orientation and direction selectivity have proven more difficult to elucidate.

Many orientation-selective neurons have a receptive field elongated along a particular axis, with an ON-center or OFF-center region flanked on one or both sides by an antagonistic surround (Figure 10.23a). This linear arrangement of ON and OFF areas is analogous to the concentric antagonistic areas seen in retinal and LGN receptive fields. One gets the impression that the cortical neurons receive a converging input from three or more LGN cells with receptive fields that are aligned along one axis (Figure 10.23b). Hubel and Wiesel called neurons of this type **simple cells**. The segregation of ON and OFF regions is a defining property of simple cells, and it is because of this receptive field structure that they are orientation selective.

Other orientation selective neurons in V1 do not have distinct ON and OFF regions and are therefore not considered simple cells. Hubel and Wiesel called most of these **complex cells**, because their receptive fields appeared

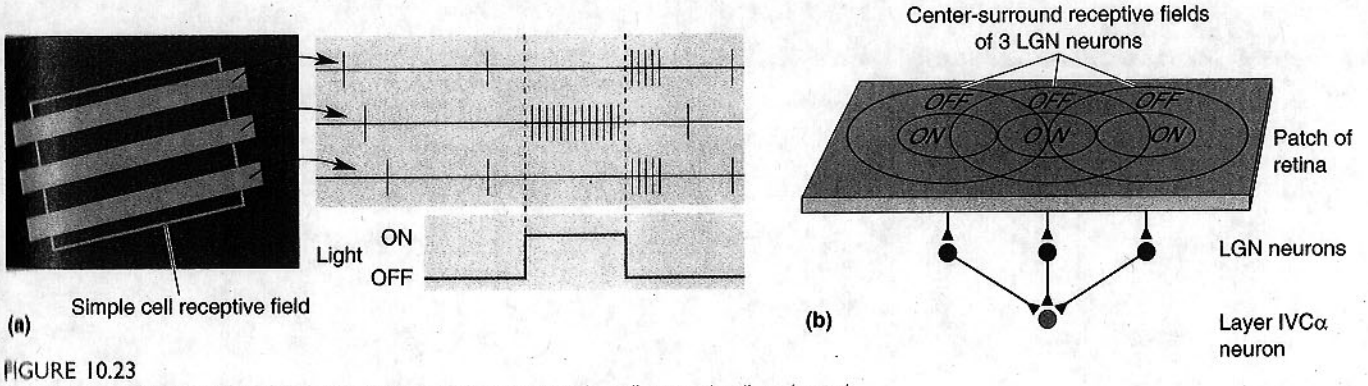


FIGURE 10.23
A simple cell receptive field. (a) The response of a simple cell to optimally oriented bars of light at different locations in the receptive field. Notice that the response can be ON or OFF depending on where the bar lies in the receptive field. **(b)** A possible construction of a simple cell receptive field by the convergence of three LGN cell axons with center-surround receptive fields.

Box 10.2 BRAIN FOOD

Optical Imaging of Neural Activity

Most of what we know about the response properties of neurons in the visual system, and every other system in the brain, has been learned from intracellular and extracellular recordings with microelectrodes. These recordings give precise information about the activity of one or a few cells. However, unless one inserts thousands of electrodes, it is not possible to observe patterns of activity across large populations of neurons.

What if we could simultaneously record signals from thousands of neurons simply by aiming a camera at the brain's surface? Incredibly, one can observe brain activity with this optical recording approach, and the resulting images have yielded new insight about the organization of the cerebral cortex. In one version of optical recording, a voltage-sensitive dye is applied to the surface of the brain. The molecules in the dye bind to cell membranes, and they change their optical properties in proportion to variations in membrane potential. The change is detected with either an array of photodetectors or a video camera. If this technique is used to record from a single neuron, the output of the optical detector is similar to an intracellular recording. In recordings from the cerebral cortex, the activity of individual neurons cannot be resolved, and the optical signal represents a summation of the changes in membrane potential of the neurons and glial cells in an area about 100 μm across.

A second way to optically study cortical activity is to image intrinsic signals. When neurons are active, numerous changes occur in the neurons themselves and in the surrounding tissue. Examples of such changes are ion movement, neurotransmitter release, and alterations in blood volume and oxygenation. Because these factors are correlated with the level of neural activity and they have (very small) effects on the reflection of light from the brain, they are called intrinsic signals for optical recording. Thus, when intrinsic signals are used to study brain activity, membrane potentials or action potentials are not directly measured. To record intrinsic signals, light is projected onto the brain, and a video camera records the reflected light. With the wavelengths of light usually used for illumination, the intrinsic signal is dominated by changes associated with activity-dependent increases in blood volume or blood oxygen saturation. One disadvantage of this technique is that its reliance on slow vascular changes makes it incapable of the millisecond temporal resolution possible with voltage-sensitive dyes.

Figure A shows the vasculature in a portion of primary visual cortex. Figure B shows ocular dominance columns in the same patch of striate cortex obtained by imaging areas in which blood flow changes occurred during visual stimulation. This figure is actually a subtraction of two images—one made when only the right eye was visually stimulated, minus another when only the left eye was stimulated. Consequently, the dark bands represent cells dominated by the left eye, and the light bands represent cells dominated by the right eye.

Figure C is a color-coded representation of preferred orientation in the same patch of striate cortex. Four different optical images were recorded while bars of light at four different orientations were swept across the visual field. Each location in the figure is colored according to the orientation that produced the greatest response at each location on the brain (blue = horizontal; red = 45°; yellow = vertical; turquoise = 135°). Consistent with earlier results obtained with electrodes (see Figure 10.21), in some regions, the orientation changes progressively along a straight line. However, the optical recording technique reveals that cortical organization based on orientation is much more complex than an idealized pattern of parallel "columns."

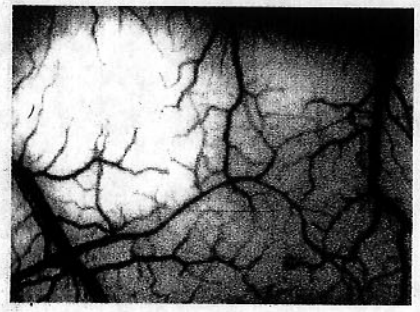


FIGURE A
Vasculature on the surface of primary visual cortex. (Source: Ts'o et al., 1990, Fig. 1A.)

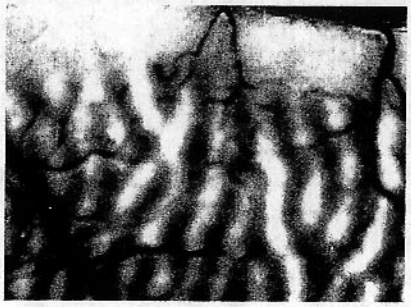


FIGURE B
Ocular dominance columns. (Source: Ts'o et al., 1990, Fig. 1B.)

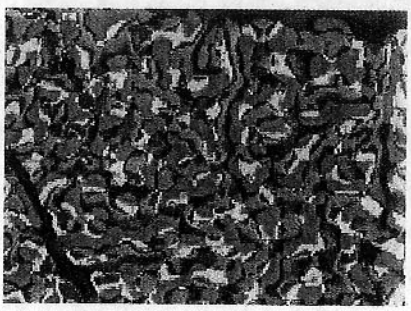


FIGURE C
A map of preferred orientations. (Source: Ts'o et al., 1990, Fig. 1C.)

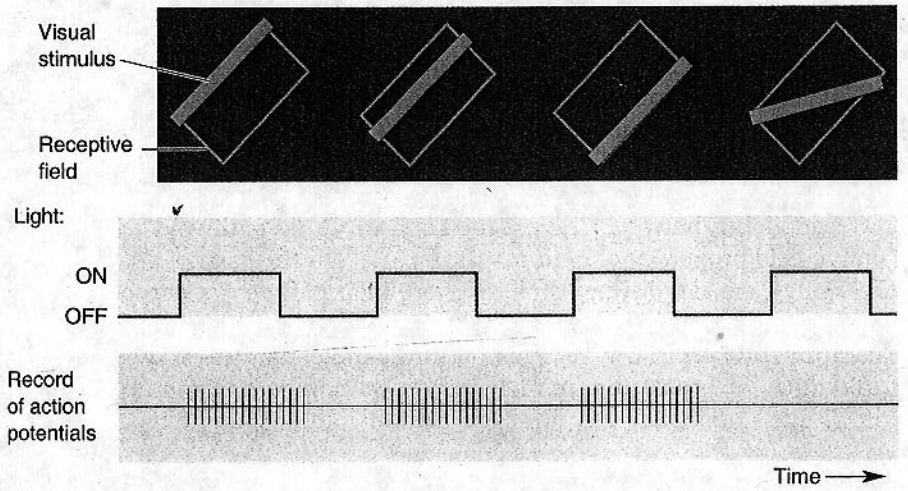


FIGURE 10.24
A complex cell receptive field. Like a simple cell, a complex cell responds best to a bar of light at a particular orientation. However, responses occur to both light ON and light OFF, regardless of position in the receptive field.

to be more complex than those of simple cells. Complex cells give ON and OFF responses to stimuli throughout the receptive field (Figure 10.24). Hubel and Wiesel proposed that complex cells are constructed from the input of several like-oriented simple cells. However, this remains a matter of debate.

Simple and complex cells are typically binocular and sensitive to stimulus orientation. While less is known about the mechanism, many are also direction selective. In general, they are relatively insensitive to the wavelength of light, although color sensitivity is sometimes observed.

Blob Receptive Fields. The old adage says, where there's smoke, there's fire. This idea appropriately describes the connection between structure and function in the brain. We have seen repeatedly in the visual system that when two nearby structures label differently with some anatomical technique, there is good reason to suspect the neurons in the structures are functionally different. For example, we have seen how the distinctive layers of the LGN segregate different types of input. Similarly, the lamination of striate cortex correlates with differences in the receptive fields of the neurons. The presence of the distinct cytochrome oxidase blobs outside layer IV of striate cortex immediately raises the question of whether the neurons in the blobs respond differently from interblob neurons. The answer is clearly yes. The neurons in the interblob areas have some or all of the properties we discussed above: binocularity, orientation selectivity, and direction selectivity. They are both simple cells and complex cells and generally are not wavelength sensitive. Most blob cells, on the other hand, are wavelength sensitive and monocular, and they lack orientation and direction selectivity. The blobs receive input directly from the koniocellular layers of the LGN and magnocellular and parvocellular input via layer IVC. The visual responses of blob cells most resemble those of the koniocellular and parvocellular input.

The receptive fields of most blob neurons are circular. Some have the color-opponent center-surround organization observed in the parvocellular and koniocellular layers of the LGN. Other blob cell receptive fields have red-green or blue-yellow color opponency in the center of their receptive fields, with no surround regions at all. Still other cells have both a color-opponent center and a color-opponent surround; they are called double-opponent cells. For present purposes, the most important thing to remember about blobs is that they contain the great majority of color-sensitive neurons outside layer IVC. Thus, the blob channels appear to be specialized for the *analysis of object color.* Without them, we might be color-blind.

Parallel Pathways and Cortical Modules

The anatomy and physiology of the central visual pathways, from retina to striate cortex, are consistent with the idea that there are several channels that process visual input in parallel. Each one appears to be specialized for the analysis of different facets of the visual scene. Dr. Margaret Livingstone and her colleagues at Harvard University have explored the fascinating correspondence between the organization of visual pathways and the receptive field properties of neurons (Box 10.3). On the basis of anatomy and physiology, we can distinguish a magnocellular pathway, a parvo-interblob pathway, and a blob pathway. These pathways are summarized in Figure 10.25. In addition to this segregation into parallel pathways, there appears to be modular processing in V1 based on retinotopy and the organization into ocular dominance columns, orientation columns, and blobs.

Parallel Pathways. The **magnocellular pathway** begins with M-type ganglion cells of the retina. These cells project axons to the magnocellular layers of the LGN. These layers project to layer IVCα of striate cortex, which in turn projects to layer IVB. The pyramidal cells in layer IVB have binocular receptive fields of the simple and complex types. They are orientation selective, and many are direction selective. They are generally not wavelength sensitive. Because this pathway contains neurons with transient responses, relatively large receptive fields, and the highest percentage of direction-selective neurons, it is thought to be involved in the *analysis of object motion and the guidance of motor actions.*

The **parvo-interblob pathway** originates with P-type ganglion cells of the retina, which project to the parvocellular layers of the LGN. The parvocellular LGN sends axons to layer IVCβ of striate cortex, which project to layer II and III interblob regions. These neurons are not generally direction selective or wavelength sensitive. The binocular receptive fields are orientation selective and simple or complex. Neurons in this pathway have the smallest

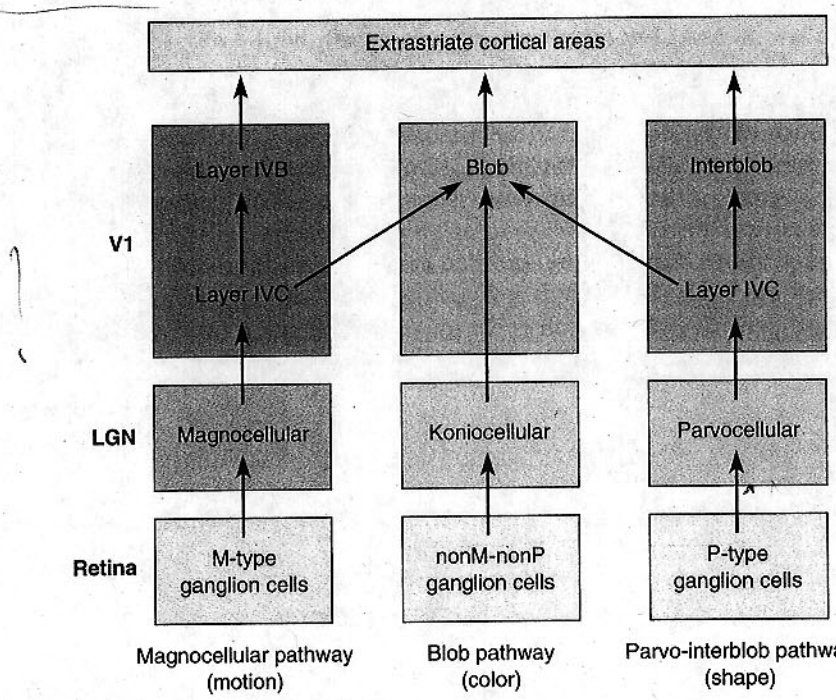


FIGURE 10.25
Three parallel pathways reaching into primary visual cortex. The function indicated below each pathway name is a "best guess" based on unique receptive field properties. Additional interactions between the pathways exist, but they are not shown.

Box 10.3 PATH OF DISCOVERY

Vision and Art

by Margaret Livingstone

In thinking about what factors led to my favorite discoveries, I recall that there always seemed to be a combination of luck and having the right question percolating around in my mind. David Hubel and I worked out the interlacing connectivity between the different subdivisions of V1 and V2 because we had been recording from the V1 blobs and were very curious about their connectivity. The lucky part was that a colleague gave us a very large number of squirrel monkeys—otherwise, we would not have had enough money to do the study.

We started looking at the roles of the magno and parvo systems in perception after seeing Patrick Cavanagh's astonishing demonstration of the slowing down of motion perception at equiluminance (i.e., when the differently colored object and background are equally bright). On seeing the demo, I immediately said, "That's because the magno system is color-blind." David Hubel replied, "That's ridiculous; if that were the case, then stereopsis (depth perception from binocular vision) should be color-blind." So we looked at some stereograms at equiluminance, and sure enough, we couldn't see stereopsis at equiluminance. Each time I thought we had settled the hypothesis that magno functions should be diminished at equiluminance, David would object, saying that some other visual task should be similarly affected. After 2 years of arguing, and doing every experiment he could think up, we finally convinced ourselves that it was the case, and published a very long paper on the parallel processing of form, color, motion, and depth.

We looked at all kinds of visual functions to see which ones were diminished at equiluminance, to see whether they might be carried selectively by the magno system, and one of the things we found that was adversely affected at equiluminance was reading. This got me interested in looking at dyslexia. People with dyslexia often complain that ordinary text seems jittery, just like what non-dyslexics experience when reading equiluminant text. I got lucky in telling this idea to Al Galaburda because he turned out to have an entire collection of dyslexic and control brains, and this collaboration led to our developing a theory (still disputed) about the etiology of dyslexia.

Whenever I would give scientific talks about the parallel processing of form, color, motion, and depth, I would use works of art to illustrate the points about how various visual functions would disappear at equiluminance, because a lot of op art uses just this principle. I found that people in the audience were often more interested in the art than the science, so I started putting more art and less science in my lectures. I also started collecting the best examples I could find of works of art that illustrated various points in my lectures. After a while, I had so many of them that I started writing an article, thinking I would publish it in *Scientific American*, but I had collected so many examples that it turned into a book.

An editor I was working with on the book told me that although it was obvious I knew a lot about art, it was equally obvious that I knew nothing about art history, and he recommended I read an art history book. So I did, and when I got to the Renaissance, the author urged the reader to look carefully at the *Mona Lisa* and observe how lifelike she seemed, and how her expression seemed to change. I noticed that her expression did change, but it changed systematically with my gaze direction. I realized this was because her smile was blurry and therefore more visible to my low-resolution peripheral vision than to my high-acuity central vision.

From my work on dyslexics, I got interested in the possibility that artistic talent might have some biological basis. An astonishing number of talented artists, musicians, actors, and computer programmers contacted me and told me that they were dyslexic. It became clear that some of them were so talented that their success couldn't be simply compensation for being bad at reading, and the idea that something that might be a disability in one realm of life might be an asset in another was forced upon me.

I began thinking that one small component of artistic talent in dyslexics might be poor depth perception, because a painter's job is to flatten the 3-D world onto a flat canvas, and I started looking for evidence of poor depth perception in artists. Mostly I looked at photographs of famous artists, because you can legitimately diagnose strabismus, which would result in stereoblindness, from photographs. During vacation, I noticed that all four of the Rembrandt self-portraits in the Louvre look wall-eyed. I looked at a very large number of Rembrandt self-portraits, but I couldn't see any pattern as to which eye deviated outward, which you would expect if Rembrandt had had one bad eye. One of my students, Bevil Conway, is himself a stereoblind artist, and he pointed out that we should look at etchings and paintings separately, because etchings are mirror image reversed from the plate. Then we saw the pattern!

So for me, Pasteur's maxim that luck favors the prepared mind has repeatedly been true.

orientation-selective receptive fields, suggesting that they are involved in the *analysis of fine object shape*.

The origin of the **blob pathway** is more mixed than that of the magnocellular and parvo-interblob pathways. Unique input to the blob pathway arises from the subset of ganglion cells that are neither M-type cells nor P-type cells. These nonM-nonP cells project to the koniocellular layers of the LGN. The koniocellular LGN projects directly to the cytochrome oxidase blobs in layers II and III. The blobs are a site of convergence of parvocellular, magnocellular, and koniocellular inputs. Typical receptive fields in the blobs are center-surround and color-opponent. They are often monocular and lack orientation selectivity. The uniquely high incidence of wavelength sensitivity in the blobs suggests that the neurons are involved in the *analysis of object color*.

While parallel pathways are a compelling feature of the visual system, it is important to note that they are not "pure." There is some mixing both within V1 and beyond, resulting in the interaction of signals from the magnocellular, parvo-interblob, and blob pathways. At present, we do not know whether this mixing is useless "contamination" that degrades information transmission within pathways or the source of valuable integration of different visual attributes.

Cortical Modules. Each point in the visual world is analyzed by thousands of cortical neurons. The retinotopic organization of the projections from retina to LGN to the primary visual cortex ensures that all the neurons analyzing a point in visual space are within a circumscribed patch of the cortex. Hubel and Wiesel showed that the image of a point in space falls within the receptive fields of neurons within a 2×2 mm region of layer III. For a complete analysis, this 2×2 mm patch of active neurons must include representatives from each of the processing channels from right and left eyes.

Fortunately, a 2×2 mm chunk of cortex would contain two complete sets of ocular dominance columns, 16 blobs, and, in the cells between blobs, a complete sampling (twice over) of all 180° of possible orientations. Thus, Hubel and Wiesel argued that a 2×2 mm chunk of striate cortex is both necessary and sufficient to analyze the image of a point in space, *necessary* because its removal would leave a blind spot for this point in the visual field and *sufficient* because it contains all the neural machinery required to analyze the participation of this point in oriented and/or colored contours viewed through either eye. Such a unit of brain tissue has come to be called a **cortical module**.

Striate cortex is constructed from perhaps a thousand cortical modules, and one is shown in Figure 10.26. We can think of a visual scene being

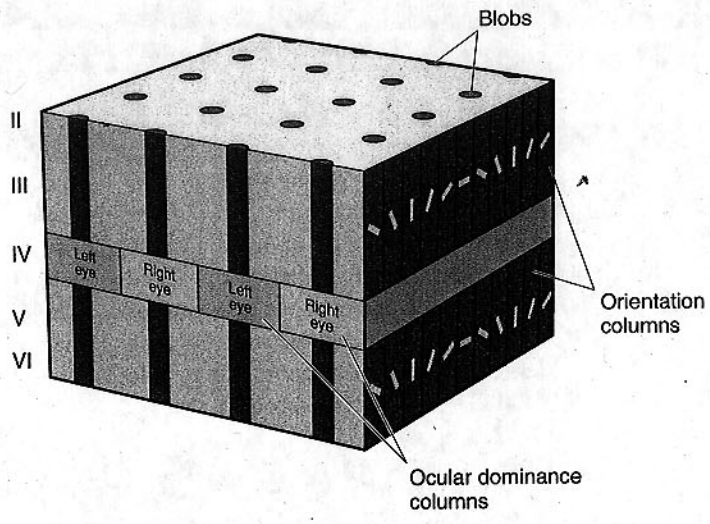


FIGURE 10.26
A cortical module. Each cortical module contains ocular dominance columns, orientation columns, and cytochrome oxidase blobs to fully analyze a portion of the visual field. The idealized cube shown here differs from the actual arrangement, which is not as regular or orderly.

simultaneously processed by these modules, each "looking" at a portion of the scene. Just remember that the modules are an idealization. Optical images of V1 activity reveal that the regions of striate cortex responding to different eyes and orientations are not nearly as regular as the "icecube model" in Figure 10.26 suggests.

▼ BEYOND STRIATE CORTEX

Striate cortex is called V1, for "visual area one," because it is the first cortical area to receive information from the LGN. Beyond V1 lie another two dozen distinct areas of cortex, each of which contains a representation of the visual world. The contributions to vision of these *extrastriate areas* are still being vigorously debated. However, the emerging picture is that there are two large-scale cortical streams of visual processing, one stretching dorsally from striate cortex toward the parietal lobe and the other projecting ventrally toward the temporal lobe (Figure 10.27).

The *dorsal stream* appears to serve the analysis of visual motion and the visual control of action. The *ventral stream* is thought to be involved in the perception of the visual world and the recognition of objects. These processing streams have primarily been studied in the macaque monkey brain, where recordings from single neurons can be made. However, functional magnetic resonance imaging (fMRI) research has begun to identify areas in the human brain that have properties analogous to brain areas in the macaque (Figure 10.28).

The dorsal and ventral streams in extrastriate cortex are related to the magnocellular, parvo-interblob, and blob pathways in V1. As we will see below, the properties of dorsal stream neurons are most similar to magnocellular neurons in V1, and ventral stream neurons have properties combining features of parvo-interblob and blob cells in V1. It appears to be a reasonable approximation to view the dorsal stream as an extension of the V1 magnocellular pathway and the ventral stream as an extension of V1 parvo-interblob and blob pathways. However, each extrastriate stream re-

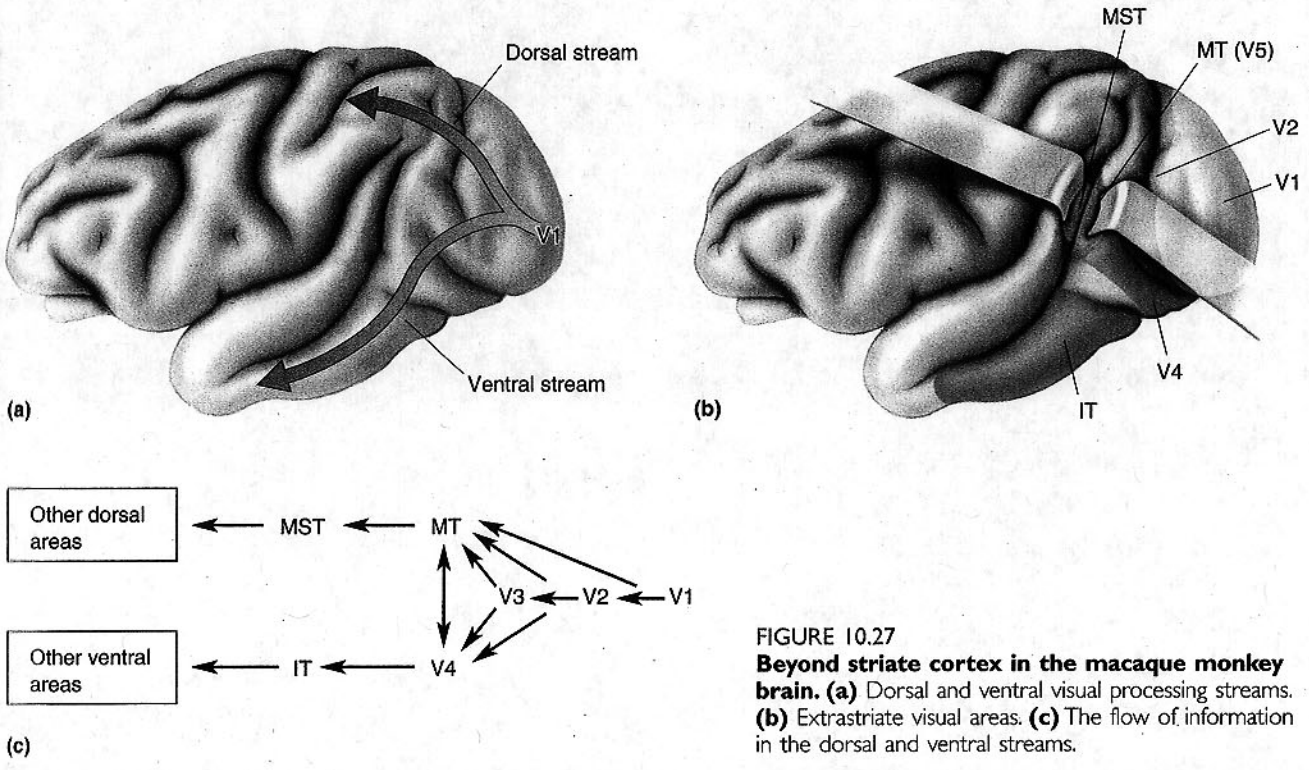


FIGURE 10.27
Beyond striate cortex in the macaque monkey brain. (a) Dorsal and ventral visual processing streams. **(b)** Extrastriate visual areas. **(c)** The flow of information in the dorsal and ventral streams.

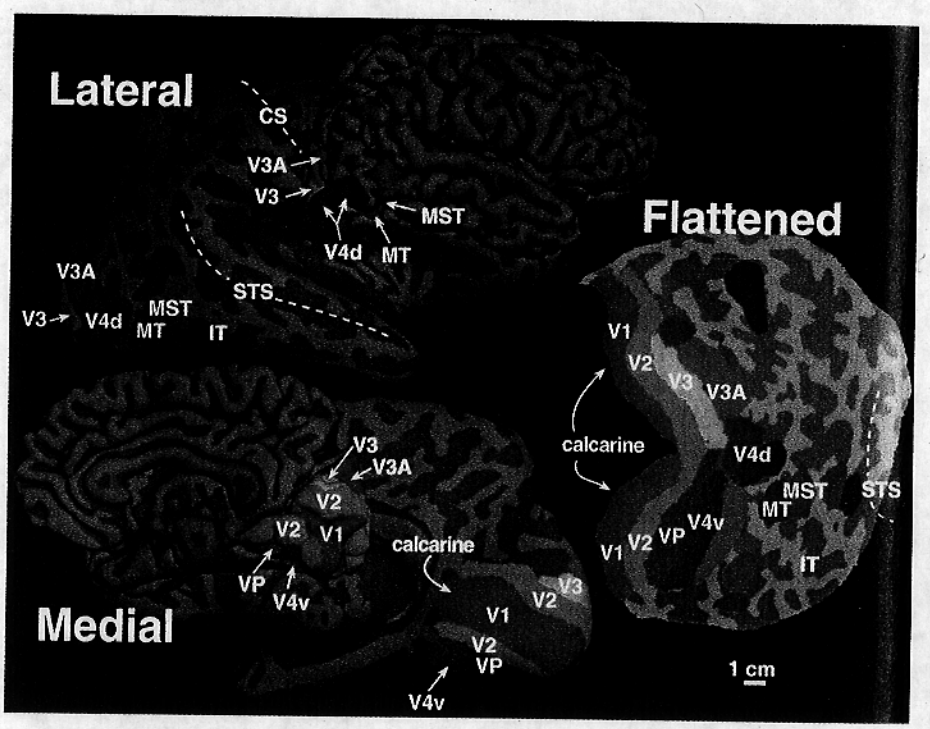


FIGURE 10.28
Visual areas in the human brain, medial and lateral views. For each view, next to the conventional picture of the brain is a "computer-inflated" brain in which the sulci have been flattened to expose hidden cortex. On the right is a flattened map of the cerebral cortex with the visual areas colored. Visual areas are represented by their common abbreviation (V1, V2, V3, V4, MT, MST, IT). VP is the ventral posterior area. Sulci shown are the superior temporal sulcus (STS), calcarine fissure, and central sulcus (CS). (Source: Courtesy of Dr. M. Sereno.)

ceives some amount of input from all the pathways that are segregated in the primary visual cortex. Thus, the extrastriate streams appear to be *dominated by* input from particular V1 pathways rather than exclusive extensions of them.

The Dorsal Stream

The cortical areas composing the dorsal stream are not arranged in a strict serial hierarchy, but there does appear to be a progression of areas in which more complex or specialized visual representations develop. Projections from V1 extend to areas designated V2 and V3, but we will skip farther ahead in the dorsal stream.

Area MT. In an area known as V5 or MT (because of its location in the middle temporal lobe in some monkeys), strong evidence indicates that specialized processing of object motion takes place. **Area MT** receives retinotopically organized input from a number of other cortical areas, such as V2 and V3, and it also is directly innervated by cells in layer IVB of striate cortex. Recall that in layer IVB the cells have relatively large receptive fields, transient responses to light, and direction selectivity. Neurons in area MT have large receptive fields that respond to stimulus movement in a narrow range of directions. Area MT is most notable for the fact that almost all the cells are direction-selective, unlike areas earlier in the dorsal stream, or anywhere in the ventral stream.

The neurons in MT also respond to types of motion, such as drifting spots of light, that are not good stimuli for cells in other areas—it appears that the motion of the objects is more important than their structure. Further specialization for motion processing is evident in the organization of MT. This cortical area is arranged into direction-of-motion columns analogous to the orientation columns in V1. Presumably, the perception of movement at any point in space depends on a comparison of the activity across columns spanning a full 360° range of preferred directions.

William Newsome and his colleagues at Stanford University have shown that weak electrical stimulation in area MT of the macaque monkey appears

to alter the perceived direction in which small dots of light move. For example, if electrical stimulation is applied to cells in a direction column preferring rightward movement, the monkey makes behavioral decisions suggesting that it has perceived motion in that direction. The artificial motion signal from electrical stimulation in MT appears to combine with visual motion input. The fact that the monkey behaviorally reports a perceived direction of motion based on the combination suggests that MT activity plays an important role in motion perception.

Dorsal Areas and Motion Processing. Beyond area MT, in the parietal lobe, are areas with additional types of specialized movement sensitivity. For example, in an area known as *MST*, there are cells selective for linear motion (as in MT), radial motion (either inward or outward from a central point), and circular motion (either clockwise or counterclockwise). We do not know how the visual system makes use of neurons with complex motion-sensitive properties in MST or of the "simpler" direction-selective cells in V1, MT, and other areas. However, three roles have been proposed:

1. *Navigation:* As we move through our environment, objects stream past our eyes, and the direction and speed of objects in our peripheral vision provide valuable information that can be used for navigation.
2. *Directing eye movements:* Our ability to sense and analyze motion must also be used when we follow objects with our eyes and when we quickly move our eyes to objects in our peripheral vision that catch our attention.
3. *Motion perception:* We live in a world filled with motion, and survival sometimes depends on our interpretation of moving objects.

Striking evidence that cortical areas in the vicinity of MT and MST are critical for motion perception in humans comes from extremely rare cases in which brain lesions selectively disrupt the perception of motion. The clearest case was reported in 1983 by Josef Zihl and his colleagues at the Max Planck Institute for Psychiatry in Munich, Germany. Zihl studied a woman who experienced a stroke at the age of 43, bilaterally damaging portions of extrastriate visual cortex known to be particularly responsive to motion (Figure 10.29). Although some ill effects of the stroke were evident,

FIGURE 10.29
Human brain activity in response to visual motion. In this PET image, an area on the lateral surface of the occipital lobe (shown in red and yellow) is particularly active. (Source: Zeki, 1993, Plate 2.)

such as difficulty naming objects, neuropsychological testing showed the patient to be generally normal and to have relatively normal vision, except for one serious deficit: She appeared to be incapable of visually perceiving motion. Before you decide that not seeing motion would be a minor impairment, imagine what it would be like to see the world in snapshots. Zihl's patient complained that when she poured coffee into a cup, it appeared at one moment to be frozen at the bottom of the cup and then suddenly it had filled the cup and covered the table. More ominously, she had trouble crossing the street—one moment she would perceive cars to be in the distance, and the next moment they would be right next to her. Clearly, this seemingly minor loss of motion perception had profound ramifications for the woman's lifestyle. The implication of this case is that motion perception may be based on specialized mechanisms located beyond striate cortex in the dorsal stream.

The Ventral Stream

In parallel with the dorsal stream, a progression of areas from V1, V2, and V3 running ventrally toward the temporal lobes appears specialized for the analysis of visual attributes other than motion.

Area V4. One of the most-studied areas in the ventral stream is **area V4** (see Figures 10.27 and 10.28). V4 receives input from the blob and interblob regions of striate cortex via a relay in V2. Neurons in area V4 have larger receptive fields than cells in striate cortex, and many of the cells are both orientation selective and color selective. Although there is a good deal of ongoing speculation concerning the function of V4, this area appears to be important for both shape perception and color perception. If this area is damaged in monkeys, perceptual deficits involving both shape and color result.

A rare clinical syndrome in humans known as *achromatopsia* is characterized by a partial or complete loss of color vision despite the presence of normal functional cones in the retina. People with this condition describe their world as drab, consisting of only shades of gray. Imagine how unappetizing a gray banana would be! Because achromatopsia is associated with cortical damage in the occipital and temporal lobes, without damage to V1, the LGN, or the retina, the syndrome suggests that there is specialized color processing in the ventral stream. Consistent with the coexistence of color-sensitive and shape-sensitive cells in the ventral stream, achromatopsia is usually accompanied by deficits in form perception. Some researchers have proposed that V4 is a particularly critical area for color and form perception, but whether the lesions associated with achromatopsia correspond to a human V4 area is controversial.

Area IT. Beyond V4 in the ventral stream are cortical areas that contain neurons with complicated spatial receptive fields. A major output of V4 is an area in the inferior temporal lobe known as **area IT**. A wide variety of colors and abstract shapes have been found to be good stimuli for cells in IT. As we will see in Chapter 24, this area appears to be important for both visual perception and visual memory. One of the most intriguing findings concerning IT is that a small percentage of the neurons responds strongly to pictures of faces. These cells may also respond to stimuli other than faces, but faces produce a particularly vigorous response, and some faces are more effective stimuli than others. This finding in monkeys appears consistent with images made using fMRI in humans, which indicate that there is a small area in the human brain that is more responsive to faces than to other

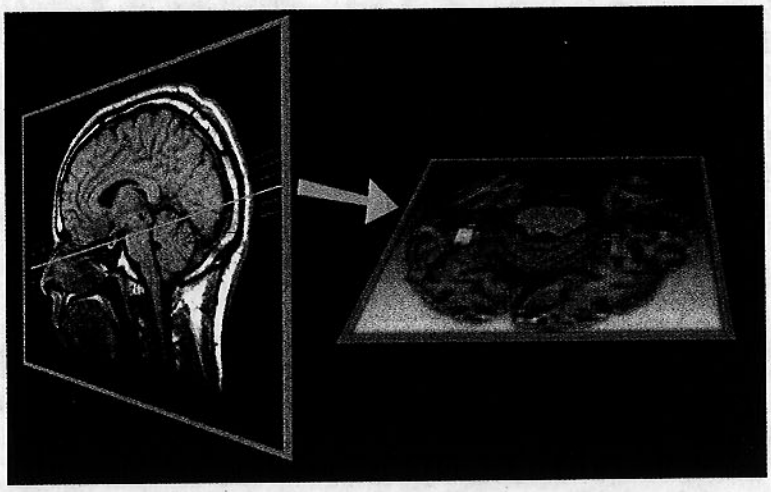


FIGURE 10.30
Human brain activity elicited by pictures of faces. Using fMRI, brain activity was recorded first in response to faces and second in response to nonface stimuli. The colored area in the brain section on the right showed significantly greater responses to faces. (Source: Courtesy of Drs. I. Gauthier, J. C. Gore, and M. Tarr.)

stimuli (Figure 10.30). The finding of face-selective cells has sparked much interest, in part because of a syndrome called *prosopagnosia*—difficulty recognizing faces even though vision is otherwise normal. This rare syndrome usually results from a stroke and is associated with damage to extrastriate visual cortex.

Could it be that our brains contain a group of cells highly specialized for face recognition? The answer is not known. While most scientists agree that faces are particularly good stimuli for a small percentage of cells, this does not mean that these cells are not involved in processing other types of information.

▼ FROM SINGLE NEURONS TO PERCEPTION

Visual *perception*—the task of identifying and assigning meaning to objects in space—obviously requires the concerted action of many cortical neurons. But which neurons in which cortical areas determine what we perceive? How is the simultaneous activity of widely separated cortical neurons integrated, and where does this integration take place? Neuroscience research is only just beginning to tackle these challenging questions. However, sometimes basic observations about receptive fields can give us insight into how we perceive (Box 10.4).

From Photoreceptors to Grandmother Cells

Comparing the receptive field properties of neurons at different points in the visual system might provide insight about the basis of perception. The receptive fields of photoreceptors are simply small patches on the retina, whereas those of retinal ganglion cells have a center-surround structure. The ganglion cells are sensitive to variables such as contrast and the wavelength of light. In striate cortex, we encounter simple and complex receptive fields that have several new properties, including orientation selectivity and binocularity. We have seen that in extrastriate cortical areas, cells are selectively responsive to more complex shapes, object motion, and even faces. It appears that the visual system consists of a hierarchy of areas in which

Box 10.4  OF SPECIAL INTEREST

The Magic of Seeing in 3D

You have probably seen books or posters showing patterns of dots or splotches of color that supposedly contain pictures in 3D, if you contort your eyes just the right way. But how is it possible to see three dimensions on a two-dimensional piece of paper? The answer is based on the fact that our two eyes always see slightly different images of the world because of the distance between them in the head. The closer objects are to the head, the greater the difference in the two images. You can easily demonstrate this to yourself by holding a finger up in front of your eyes and alternately viewing it, at different distances, with the left or right eye closed.

Long before anything was known about binocular neurons in visual cortex, stereograms were a popular form of recreation. Two photographs were taken with lenses separated by a distance roughly the same as that of human eyes. By looking at the left photograph with the left eye and the right photograph with the right eye (by relaxing the eye muscles or with a stereoscope), the brain combines the images and interprets the different views as cues for distance (Figure A).

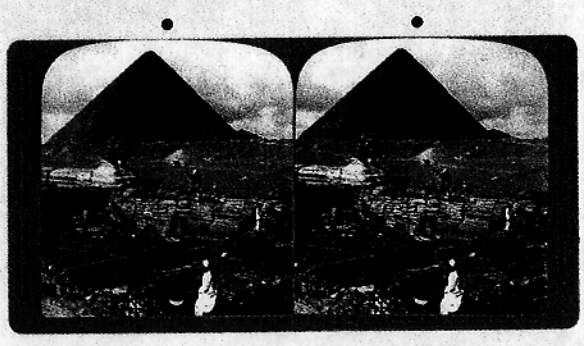


FIGURE A
A nineteenth-century stereogram. (Source: Horibuchi, 1994, p. 38.)

In 1960, Bela Julesz, working at the Bell Telephone Laboratories, invented random-dot stereograms (Figure B). These paired images of random dots are, in principle, the same as the nineteenth-century stereograms. The big difference is that no image can be seen with normal binocular viewing. To see the image in 3D, you must direct your left and right eyes to left and right images. The principle in constructing the stereo images is to create a background of randomly spaced dots, and wherever an area should be closer or farther away in the fused image, the dots shown to one eye are horizontally shifted relative to those in the other eye. Imagine looking at a white index card covered with random black dots while you hold it in front of a large piece of white paper covered with similar dots. By alternately closing your eyes, the dots on the index card will shift horizontally more than those on the more distant piece of paper. The pair of stereo images captures this difference in viewpoint and erases any other indication that there is a square in front, such as the edge of the index card. Random-dot stereograms shocked many scientists, because in 1960, it was

receptive fields become increasingly more complex, moving away from V1. Perhaps our perception of specific objects is based on the excitation of a small number of specialized neurons in some ultimate perceptual area that has not yet been identified. Is our recognition of our grandmother based on the responses of 5 or 10 cells with receptive field properties so highly refined that the cells respond only to one person? The closest approximation to this is the face-selective neurons in area IT. However, even these fascinating cells do not respond to only one face.

While it is by no means settled, there are several arguments against the idea that perception is based on extremely selective receptive fields such as those of "grandmother cells." First, recordings have been made from most parts of the monkey brain, but there is no evidence that a portion of cortex has cells tuned to each of the millions of objects that we all recognize. Second, such great selectivity appears to be counter to the general principle of broad tuning that exists throughout the nervous system. Photoreceptors respond to a range of wavelengths, simple cells respond to many orientations, cells in MT respond to motion in a range of directions, and face cells usually respond to many faces. Moreover, cells that are selective for one property—orientation, color, or whatever—are always sensitive to

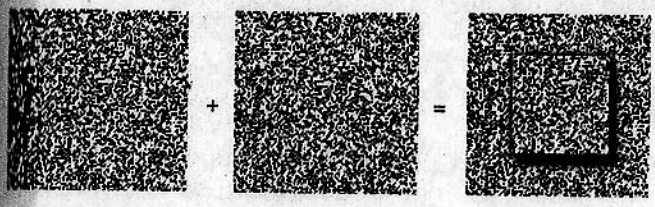


FIGURE B
A random-dot stereogram and the perception that results from binocularly fusing the images. (Source: Julesz, 1971, p. 21.)

commonly thought that depth was perceived only after the images in each eye were separately recognized.

In the 1970s, Christopher Tyler at the Smith-Kettlewell Eye Research Institute created autostereograms. An autostereogram is a single image that, when properly viewed, gives the perception of objects in 3D (Figure C). The colorful, and sometimes frustrating, autostereograms you see in books are based on an old illusion called the wallpaper effect. If you look at wallpaper that contains a repeating pattern, you can cross (or diverge) your eyes and view one piece of the pattern with one eye and the next cycle of the pattern with the other eye. The effect makes the wallpaper appear to be closer (or farther away). In an autostereogram, the wallpaper effect is combined with random-dot stereograms. To see the 3D skull in Figure C, you need to relax your eye muscles so that the left eye looks at the left dot on top and the right eye the right dot. You will know you are getting close when you see three dots at the top of the image. Relax and keep looking, and the picture will become visible.

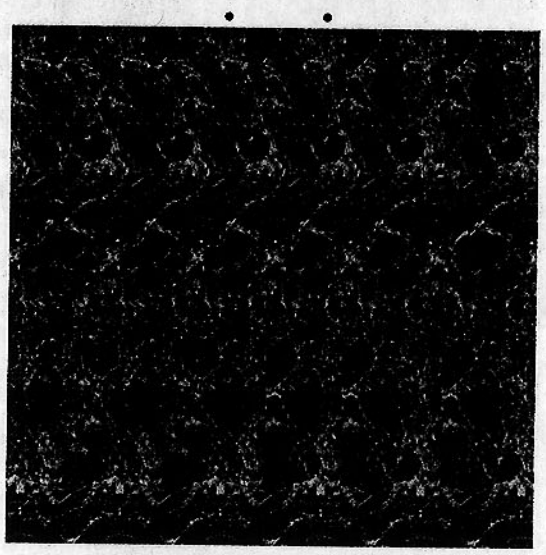


FIGURE C
An autostereogram. (Source: Horibuchi, 1994, p. 54.)

One of the fascinating things about stereograms is that you often must look at them for tens of seconds or even minutes, while your eyes become "properly" misaligned and your visual cortex "figures out" the correspondence between the left and right eye views. We do not know what is going on in the brain during this period, but presumably it involves the activation of binocular neurons in the visual cortex.

other properties. For example, we can focus on the orientation selectivity of V1 neurons and the way in which this might relate to the perception of form, neglecting the fact that the same cells might selectively respond to size, direction of motion, and so on. Finally, it might be too "risky" for the nervous system to rely on extreme selectivity. A blow to the head might kill all five grandmother cells, and in an instant, we would lose our ability to recognize her.

Parallel Processing and Perception

If we do not rely on grandmother cells, how does perception work? One alternative hypothesis is formulated around the observation that parallel processing is used throughout the visual system (and other brain systems). We encountered parallel processing in Chapter 9 when we discussed ON and OFF and M and P ganglion cells. In this chapter, we saw three parallel channels in V1. Extending away from V1 are dorsal and ventral streams of processing, and the different areas in these two streams are biased, or specialized, for various stimulus properties. Perhaps the brain uses a "division of labor" principle for perception. Within a given cortical area, many broadly

tuned cells may serve to represent features of objects. At a bigger scale, a relatively large group of cortical areas may contribute to perception, some dealing more with color or form, others more with motion. In other words, perception may be more like the sound produced by an orchestra of visual areas than the end product of an assembly line.

▼ CONCLUDING REMARKS

In this chapter, we have outlined the organization of the sensory pathway from eye to thalamus to cortex. We saw that vision actually involves the perception of numerous different properties of objects—color, form, movement—and these properties are processed in parallel by different cells of the visual system. This processing of information evidently requires a strict segregation of inputs at the thalamus, some limited convergence of information in striate cortex, and finally a massive divergence of information as it is passed on to higher cortical areas. The distributed nature of the cortical processing of visual information is underscored when you consider that the output of a million ganglion cells can recruit the activity of well over a billion cortical neurons throughout the occipital, parietal, and temporal lobes! Somehow, this widespread cortical activity is combined to form a single, seamless perception of the visual world.

Heed the lessons learned from the visual system. As we shall see in later chapters, the basic principles of organization in this system—parallel processing, topographic mappings of sensory surfaces, synaptic relays in the dorsal thalamus, cortical modules, and multiple cortical representations—are also features of the sensory systems devoted to hearing and touch.

KEY TERMS

The Retinofugal Projection
retinofugal projection (p. 310)
optic nerve (p. 311)
optic chiasm (p. 312)
decussation (p. 312)
optic tract (p. 312)
visual hemifield (p. 312)
binocular visual field (p. 312)
lateral geniculate nucleus (LGN) (p. 313)
optic radiation (p. 313)
superior colliculus (p. 314)
optic tectum (p. 315)
retinotectal projection (p. 315)

The Lateral Geniculate Nucleus
magnocellular LGN layer (p. 316)
parvocellular LGN layer (p. 316)
koniocellular LGN layer (p. 316)

Anatomy of the Striate Cortex
primary visual cortex (p. 318)
area 17 (p. 318)
V1 (p. 318)
striate cortex (p. 318)
retinotopy (p. 319)
ocular dominance column (p. 322)
cytochrome oxidase (p. 324)
blob (p. 324)

Physiology of the Striate Cortex
binocular receptive field (p. 324)
orientation selectivity (p. 325)
orientation column (p. 326)
direction selectivity (p. 26)
simple cell (p. 327)
complex cell (p. 327)
magnocellular pathway (p. 330)
parvo-interblob pathway (p. 330)
blob pathway (p. 332)
cortical module (p. 332)

Beyond Striate Cortex
area MT (p. 334)
area V4 (p. 336)
area IT (p. 336)

REVIEW QUESTIONS

1. Following a bicycle accident, you are disturbed to find that you cannot see anything in the left visual field. Where has the retinofugal pathway been damaged?
2. What is the source of most of the input to the *left* LGN?
3. A worm has eaten part of one lateral geniculate nucleus. You can no longer perceive color in the right visual field of the right eye. What layer(s) of which LGN have been damaged?
4. List the chain of connections that link a cone in the retina to a blob cell in striate cortex.
5. How are receptive fields transformed at each of the synaptic relays that connect an M-type retinal ganglion cell to a neuron in striate cortical layer IVB?
6. Which pathway, magnocellular or parvocellular, provides a greater percentage of the input to striate cortex? What are two analyses of the visual world that are thought to involve mainly this pathway? What about the other pathway?
7. What is meant by parallel processing in the visual system? Give two examples.
8. If a child is born cross-eyed and the condition is not corrected before the age of 10, binocular depth perception will be lost forever. This is explained by a modification in the circuitry of the visual system. From your knowledge of the central visual system, where do you think the circuitry has been modified?
9. In what ways is area MT more specialized for the detection of visual motion than area V1?
10. For many years, it was thought that depth perception involved the recognition of objects in each eye separately, followed by binocular integration. How do the stereograms discussed in Box 10.4 disprove this hypothesis? What areas of the brain are possible sites for binocular integration?

FURTHER READING

Alonso JM. 2002. Neural connections and receptive field properties in the primary visual cortex. *Neuroscientist* 8:443–456.

Callaway EM. 1998. Local circuits in primary visual cortex of the macaque monkey. *Annual Review of Neuroscience* 21:47–74.

Courtney SM, Ungerleider LG. 1997. What fMRI has taught us about human vision. *Current Opinion in Neurobiology* 7:554–561.

Gegenfurtner KR. 2003. Cortical mechanisms of colour vision. *Nature Reviews Neuroscience* 4:563–572.

Goodale MA, Westwood DA. 2004. An evolving view of duplex vision: separate but interacting cortical pathways for perception and action. *Current Opinion in Neurobiology* 14:203–211.

Hendry SHC, Reid RC. 2000. The koniocellular pathway in primate vision. *Annual Review of Neuroscience* 23:127–153.

Paradiso MA. 2002. Perceptual and neuronal correspondence in primary visual cortex. *Current Opinion in Neurobiology* 12:155–161.

Sherman SM, Guillery RW. 2002. The role of the thalamus in the flow of information to the cortex. *Philosophical Transactions of the Royal Society of London B* 357:1695–1708.