

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 shall see later in the chapter.

LGN Input to Layer IVC

Now we want to focus on the details of how the different types of LGN neurons innervate the cells in cortical 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.

Segregation of magnocellular and parvocellular LGN inputs is straightforward. Magnocellular LGN neurons project to layer IVC α , and parvocellular LGN neurons project to layer IVC β . Imagine the two tiers of layer IVC are pancakes, stacked one (α) on top of the other (β). Because the input from

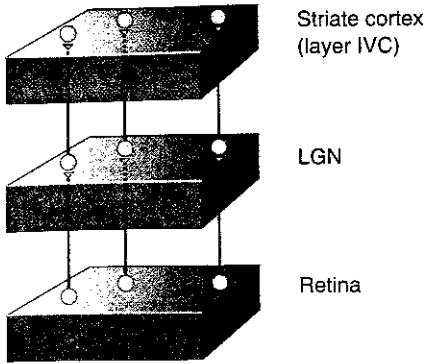


Figure 10.14
Retinotopy is preserved in the LGN projection to layer IVC.

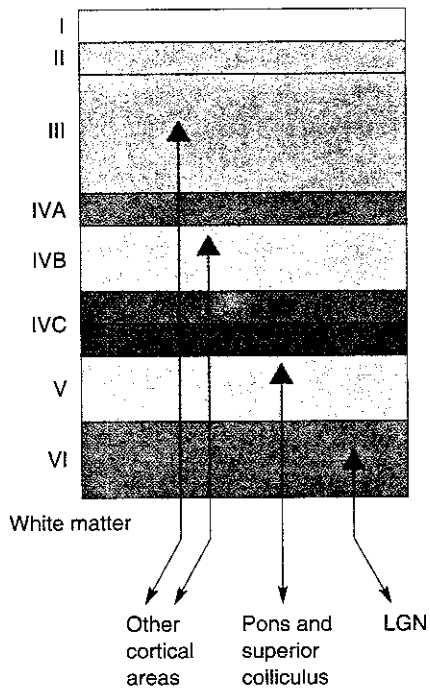


Figure 10.15
The organization of outputs from striate cortex.

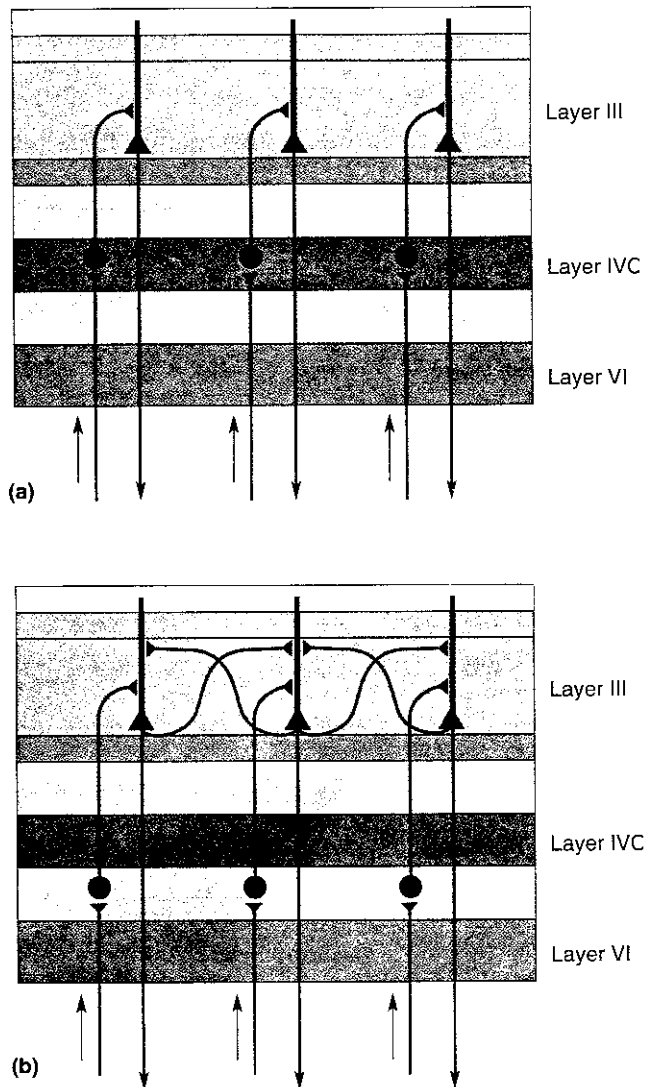


Figure 10.16
Patterns of intracortical connections. (a) Radial connections. (b) Horizontal connections.

the LGN to the cortex is arranged topographically, we see that layer IVC contains two overlapping retinotopic maps, one from the magnocellular LGN ($IVC\alpha$) and the other from the parvocellular LGN ($IVC\beta$).

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 groundbreaking 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 10.17). 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 in 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.

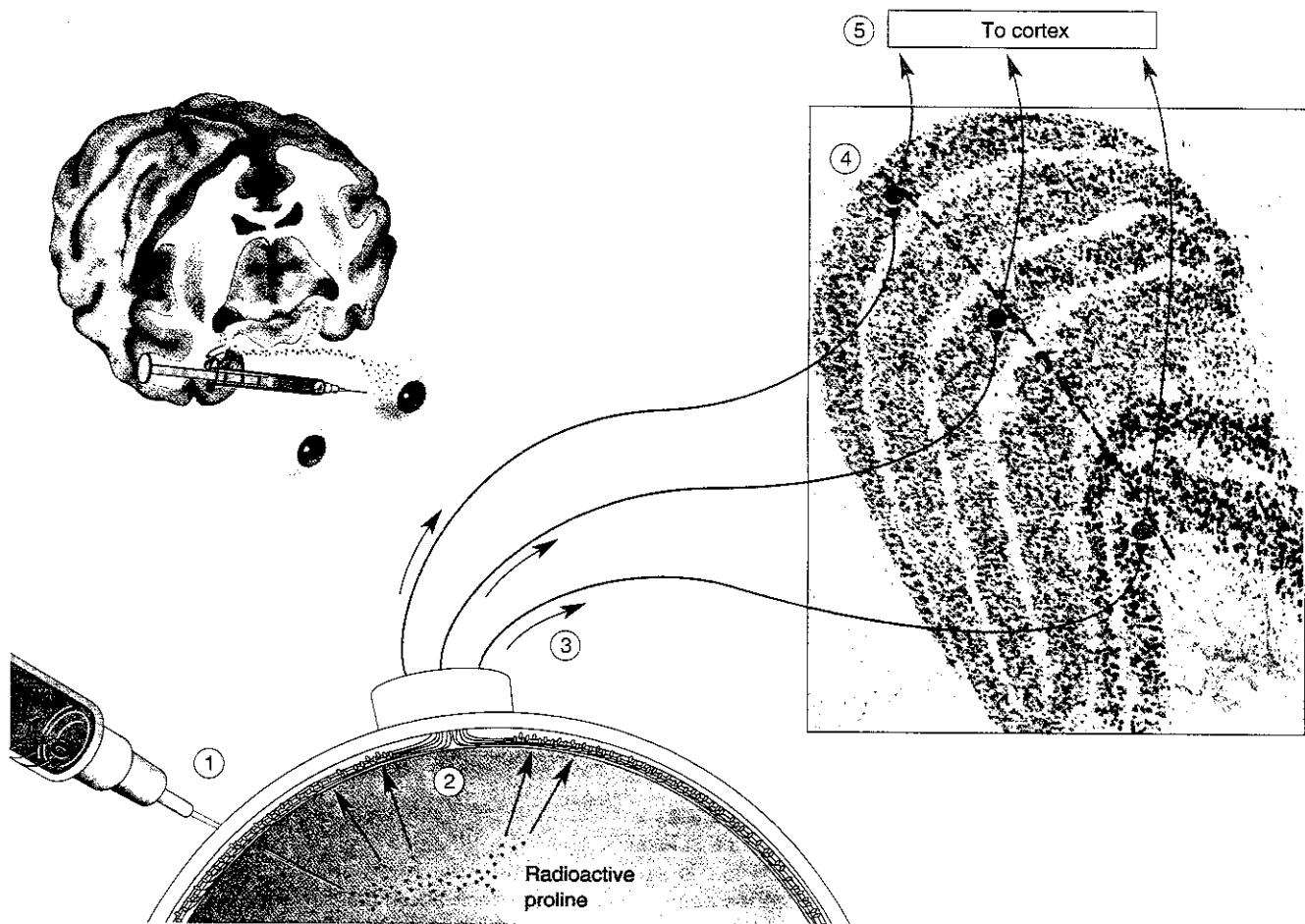


Figure 10.17

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

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 10.18a). 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 10.18b).

Putting the anatomy together, you can see that any chunk of layer IVC measuring 0.5 mm (the thickness of layer IVC) by 1 mm \times 1 mm would contain a full complement of segregated inputs from both magnocellular and parvocellular LGN layers from both left and right eyes.

Layer IVC Innervation of Other Cortical Layers

Layer IVC neurons project axons radially up to layers IVB and III where, for the first time, information from the left eye and right eye begins to mix. Even so, there continues to be considerable anatomical segregation of the magnocellular and parvocellular processing streams. Layer IVC α , which

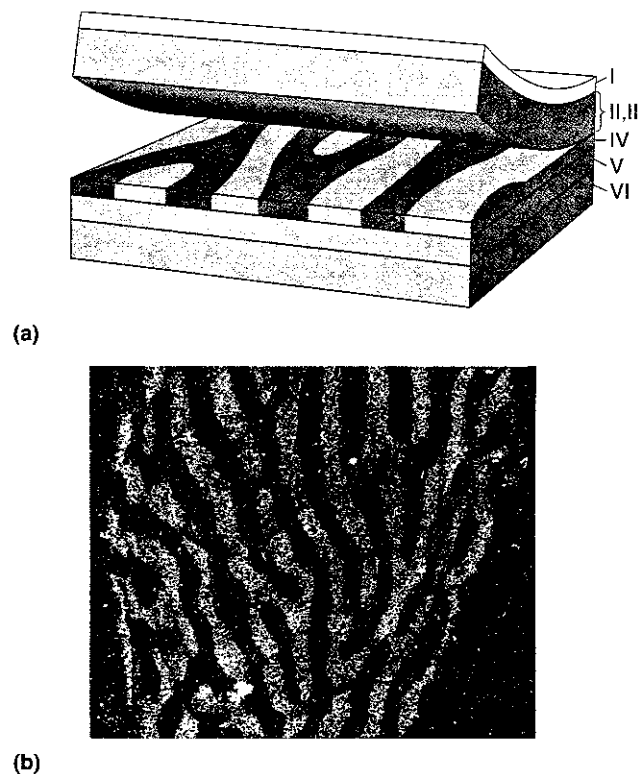


Figure 10.18

Ocular dominance columns in striate cortex. (a) The organization of ocular dominance columns in layer IV of the striate cortex of a macaque monkey. The distribution of LGN axons serving one eye is darkly shaded. In cross section, these eye-specific zones appear as patches, each about 0.5 mm wide, in layer IV. When the superficial layers are peeled back, allowing a view of the ocular dominance columns in layer IV from above, these zones take on the appearance of 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.)

receives magnocellular LGN input, projects mainly to cells in layer IVB. Layer IVC β , which receives parvocellular LGN input, projects mainly to layer III.

Blobs. In addition to the parvocellular input relayed from layer IVC β , very recent research indicates that a subset of cells in layer III receives *direct* input from the LGN. These layer III cells are found in what are known as “blobs.” The blob story began in 1978 when Margaret Wong-Riley, at the University of California, San Francisco, discovered a nonhomogeneity in the anatomy of cortical layer III. She used a staining procedure designed to reveal the presence of **cytochrome oxidase**, a mitochondrial enzyme used for cell metabolism, and found that it 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 (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 in layers II and III, after some debate, eventually were given the name **blobs**. The blobs are in rows, each blob centered on an ocular dominance stripe in layer IV. Between the blobs are “interblob” regions in layers II and III.

It was soon shown that both blobs and interblobs receive parvocellular LGN input via a relay in layer IVC β . In 1994, Johns Hopkins University neuroscientists Stewart Hendry and Takashi Yoshioka discovered that the blob cells in macaque striate cortex also receive direct LGN innervation—from the mysterious koniocellular layers.

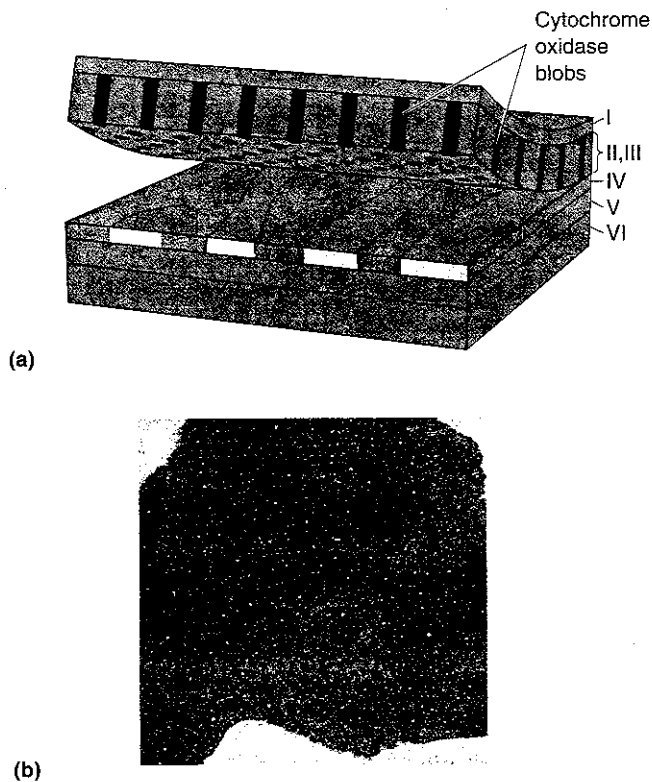


Figure 10.19

Cytochrome oxidase blobs. Distribution of the enzyme cytochrome oxidase in the cells of striate cortex. **(a)** The organization of cytochrome oxidase blobs in layer III of striate cortex in a macaque monkey. **(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.)

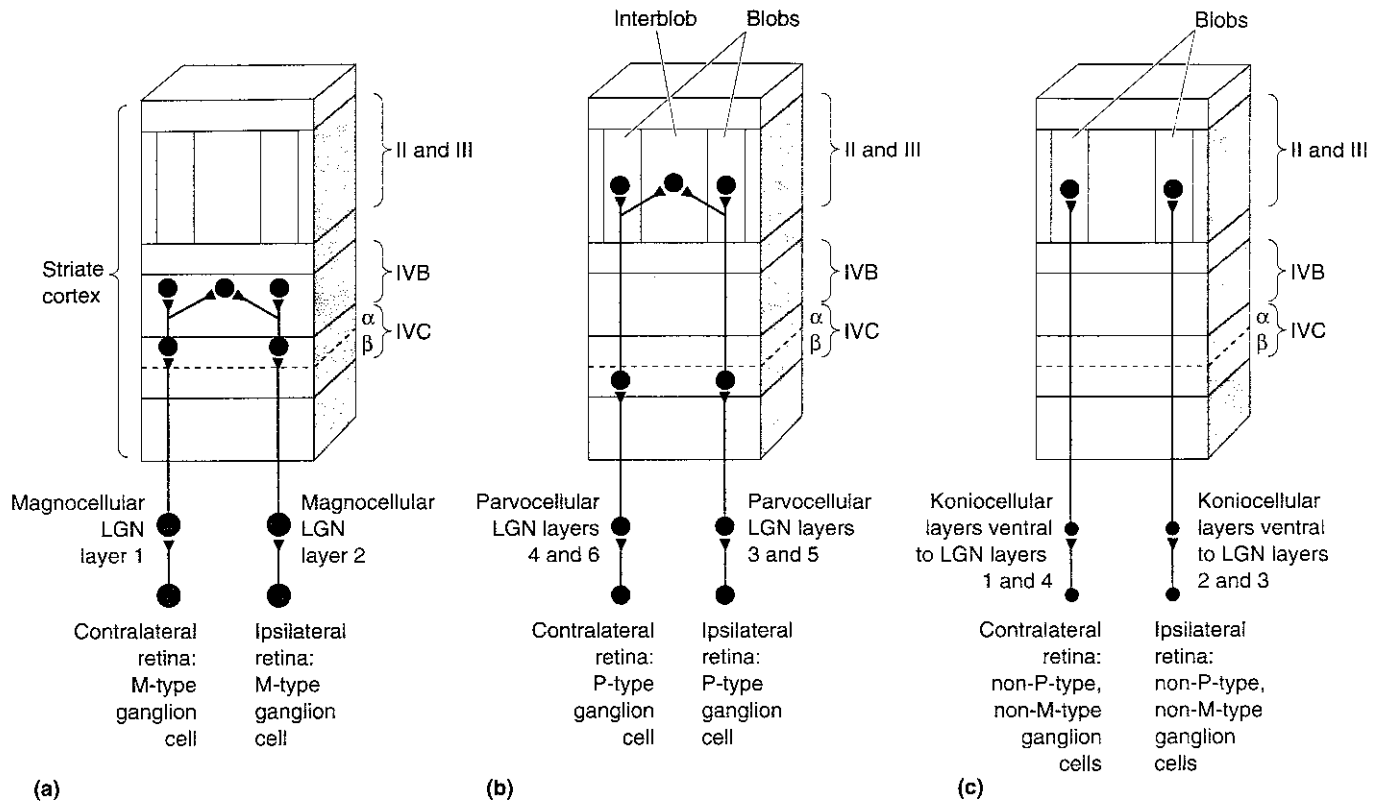


Figure 10.20
Three parallel pathways from the retina to striate cortex. (a) magnocellular pathway, (b) parvocellular pathway, (c) koniocellular pathway.

Parallel Pathways

Figure 10.20 summarizes the connections of striate cortex and its LGN input in terms of anatomically distinct, parallel pathways that arise in the retina. The magnocellular pathway begins with the M-type ganglion cells of the retina. These cells project axons to the magnocellular layers of the LGN. The magnocellular layers of the LGN project to layer IVC α of striate cortex. Layer IVC α neurons project to layer IVB (Figure 10.20a).

The parvocellular pathway originates with the P-type ganglion cells of the retina, which project to the parvocellular layers of the LGN. The parvocellular LGN sends axons to layer IVC β of striate cortex. Layer IVC β neurons project to layer III blob and interblob regions (Figure 10.20b).

The koniocellular pathway arises from the subset of ganglion cells that are neither M-type nor P-type. These poorly characterized cells project to the koniocellular layers of the LGN. The koniocellular LGN projects directly to the layer III blobs (Figure 10.20c). Notice that the blobs are a site of convergence of parvocellular and koniocellular inputs.

Notice also in Figure 10.20 that the information from the two eyes remains segregated all the way through layer IVC. Inputs from the two eyes begin to mix in layer IVB and in the interblob region of layer III. However, note that the blob cells receive input mainly from one eye or the other. Keep these anatomically defined parallel pathways in mind as we look at how neurons in striate cortex respond to visual stimuli in the next section.

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. 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 will be concerned only with 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 (Box 10.2). Their contributions to our understanding of the cerebral cortex were recognized with the Nobel Prize in 1981.

Instead of presenting the physiological results in the order in which they were discovered, we will discuss the physiology of cortical neurons in light of the anatomy described above. The latest research suggests that, functionally, there are three relatively independent channels of visual information processing. The channel that begins with the magnocellular retinal ganglion cells and leads to layer IVB of striate cortex we will call the **magnocellular channel**, or simply the **M channel**. The channel that begins with the parvocellular retinal ganglion cells and leads to the interblob regions of layer III we will call the **parvocellular-interblob channel**, or the **P-IB channel**. The channel that converges on the blobs of layer III, and passes through the parvocellular and koniocellular layers of the LGN, we will call simply the **blob channel**. We'll see that each of these channels appears to process a different facet of vision.

The M Channel

Recall that cells in the magnocellular LGN are activated by only one eye (i.e., they are monocular), have center-surround receptive fields, respond transiently to visual stimulation, and are insensitive to the wavelength of light. At the level of the first synaptic relay in layer IVC α , we see an interesting elaboration of receptive field structure. Instead of being circular, the receptive fields of layer IVC α neurons are elongated along a particular axis, with an ON-center or OFF-center region flanked on one or both sides by an antagonistic surround (Figure 10.21). One gets the impression that these cells receive a converging input from three or more LGN cells with receptive fields that are aligned along one axis (Figure 10.21c). Hubel and Wiesel called neurons of this type **simple cells**. Simple cells respond best to a thin slit of light or dark that is aligned with the long axis of the receptive field, and they respond poorly, if at all, to a stimulus that is perpendicular to this axis. Thus, we say that layer IVC α cells possess **orientation selectivity**.

Layer IVC α cells project up to layer IVB, where the cells also have orientation-selective simple receptive fields. One important difference between the cells in the two layers is that while cells in layer IVC α respond only to stimulation of one eye or the other (just as the anatomy of ocular dominance columns predicts), many cells in layer IVB respond to stimulation of both eyes. Such cells are said to have **binocular receptive fields**. The construction of binocular receptive fields is essential in binocular animals, such as humans. Without binocular neurons, we would be unable to use the inputs from both eyes to form a single image of the world around us.

Besides binocularity, another important physiological property of layer IVB cells is **direction selectivity**. Figure 10.22 shows how a direction-selective cell might respond 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 in the M channel. For this reason, the M channel is thought to be specialized for the *analysis of object motion*.

Box 10.2

PATH OF DISCOVERY

Exploring the Visual Cortex

by David Hubel

Torsten Wiesel and I began what was to be a 25-year collaboration at the Johns Hopkins Hospital in the fall of 1958. For almost all of our first year we worked in the dark and dingy basement of the Wilmer Ophthalmological Institute, in the laboratory of Stephen Kuffler. It was a wonderful place despite its tawdriness; soon after we got there Ed Fushpan and David Potter arrived from Katz' lab in London, and in those days the field was smaller, more intimate, and we had daily discussions ranging from synapses to higher CNS. It was at the Wilmer that Steve Kuffler had done his work on the receptive field organization of retinal ganglion cells in the cat retina, surely one of the most beautiful pieces of work in sensory physiology of this century. Torsten and I had already been working in vision for several years, and we set out to extend Kuffler's receptive field studies into the brain. For several reasons we thought it was likely that the most interesting things would be found in the cortex, so for the time being we skipped the intermediate stage, the LGN, and went right to the primary visual cortex.

At that time recording from single CNS cells, particularly from the cortex, was in its infancy. The most interesting work by far had been done by Vernon Mountcastle (also at Hopkins) in the somatosensory cortex of cats and monkeys, where he had discovered, in 1956, the columnar organization of cortex. The curious thing was that there seemed to be no obvious differences between the cells of the somatosensory cortex and those at lower levels, either thalamus or primary sensory nerves. Nevertheless, just looking at the high complexity and high degree of order in the visual cortex was enough to give us hope that there we might find something more elaborate than the center-surround receptive fields of retinal ganglion cells.

We certainly had no preconceived ideas of what these differences might be. It may be worth remarking here that many sciences are not structured the way much of physics is, with hypothesis, leading to measurements, and then laws, often with much quantification. This dissimilarity holds especially for much of biology. Much of science is akin to geographical exploration in the days of Columbus,

where one sails off not knowing what to expect. This has no bearing on whether a particular area of science is good or bad, or revolutionary or pedestrian. One need only ask what hypotheses Darwin had in mind when he set forth on the *Beagle*; one need only search the *Origin of Species* for equations, to be struck by how great the differences are among different sciences. Our work began with no hypotheses, unless the vague expectation of finding something interesting constitutes a hypothesis; as for quantification, we did not even plot graphs of anything until the 1970s.

Within a few weeks of starting, we began to get results. At first the going was rough. Cells failed to respond to our large patches of light or to the small spots Kuffler had used so effectively. The break came during our third or fourth experiment. We had been recording large, stable impulses from one cell for 3 or 4 hours with no hint of any response to our stimuli. The cell was #3004 in our series, which we had decided to begin at 3000 to bolster our confidence and also to impress Mountcastle, whose series of cells generally ran into the thousands. At that time we were generating our light spots with a most unwieldy concoction designed for retinal work, for which it was ideal—in fact, it was the same stimulator that Kuffler had used so effectively in the early 1950s. To stimulate with a spot of light, one used a metal plate about the shape of a microscope slide into which a small hole had been drilled. The metal plate was inserted into a slot, and the spot was projected onto the retina with an elaborate system of lamps and mirrors. Dark spots on a light background were produced by using a glass microscope slide instead of a brass plate, onto which a tiny opaque disc had been glued. For cell #3004 neither type of stimulus seemed to work, despite hours of searching around in the retina. Suddenly, in mid-afternoon, we started to get hints of a response from the cell just as we inserted the glass slide into the slot. It seemed that what the cell wanted in order to respond was the faint shadow of the edge of the glass as it crossed a particular part of the animal's retina. After working with that one cell for 9 hours, we finally concluded that the shadow only worked over a restricted range of orientations: when the orientation was right, the cell would respond with a roar of impulses.



David Hubel

continued on next page

Box 10.2 PATH OF DISCOVERY

continued from page 260

By now, in 1995, we have seen tens of thousands of such cells, in the visual cortex of cats and monkeys, but it took several years to convince ourselves that the great majority of V1 cells respond to line segments in specific orientations, that all orientations are represented about equally in the population, and to work out such things as the degrees of complexity, binocular properties, responses to movement, and the geometrical distributions of cells by layers and columns. Later we went on to study the effects of visual deprivation, and to compare the responses in adult and newborn animals. On one visit, Vernon remarked, "What a wonderful system! It will be five years before you get this all worked out." We thought he was exaggerating, but now, 36 years later,

the mine is still producing, in dozens of laboratories worldwide.

This might seem to be an example of the importance of luck in science. Of course luck always helps. We were surely lucky to get into such a field early—it fit our personalities not to have to begin by months of reading in the library. We were lucky, if you like, that we had the sense not to spend months or years designing elaborate calibrated stimulators, before having any idea what stimuli the cells might be interested in. We were lucky in being a bit sloppy long before any rigor or fine measurements or statistics were called for. And we were certainly lucky in being stubborn and persistent in our willingness to fight that cell for 9 hours. In retrospect, it was time well spent.

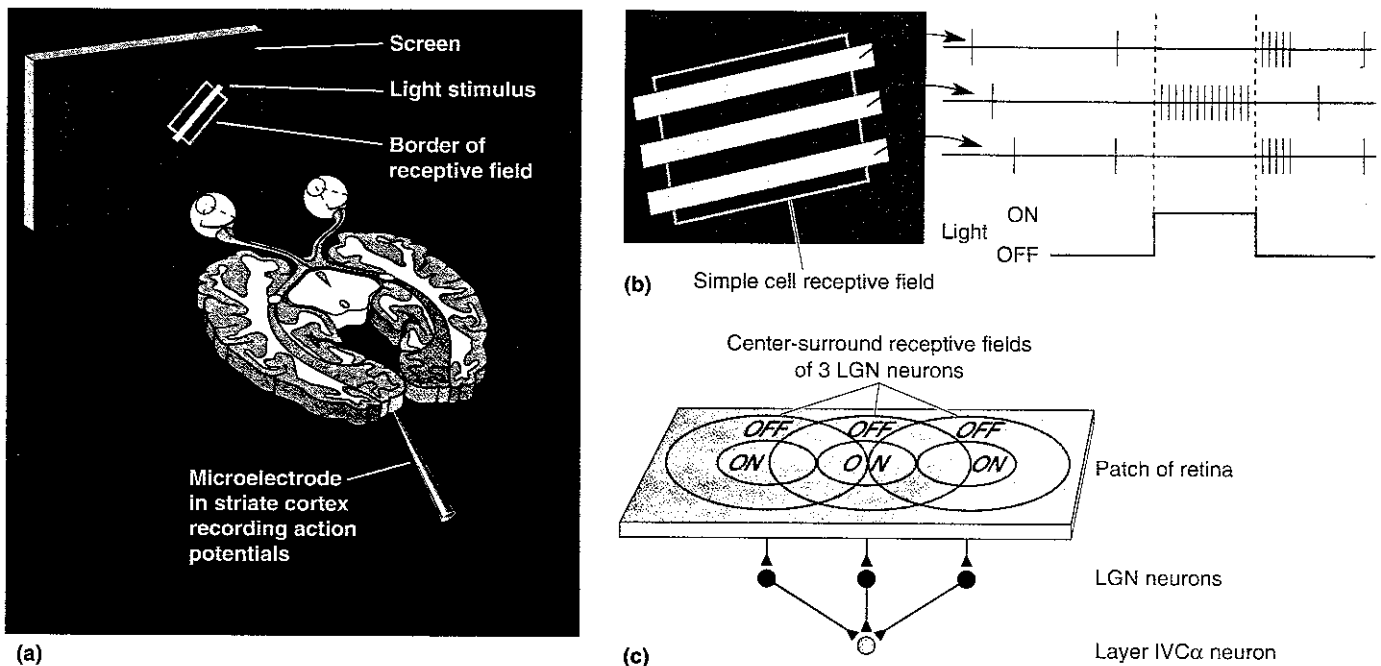


Figure 10.21
Simple cell receptive field. (a) The responses of a neuron in layer IV α are monitored as visual stimuli are presented to its receptive field. The visual stimulus is a light bar. (b) The response of a simple cell to different stimuli. Notice that it responds best to an oriented bar of light, and the response can be ON or OFF depending on where the bar lies in the receptive field. (c) Possible construction of a simple cell receptive field by the convergence of three LGN cell axons with center-surround receptive fields.

The P-IB Channel

Neurons in the parvocellular layers of the LGN have monocular, small, circular receptive fields. About 80% of these cells show red-green or blue-yellow center-surround opponency. In all these respects, the cells of layer IV β act just like the LGN neurons that feed them. Recall that IV β cells relay information up to layers II and III, and that this projection targets both blob and interblob regions.

We will discuss blobs in a moment; for now, let's concentrate on the interblob cells in layer III. Hubel and Wiesel called most of these **complex cells**, because their receptive fields appeared to be more complex than those of simple cells. Unlike simple cells, complex cell receptive fields do not have distinct ON and OFF regions (Figure 10.23). Complex cells are mostly binocular, relatively insensitive to the wavelength of light, and highly selective to stimulus orientation (even more so than simple cells).

The analysis of stimulus orientation appears to be one of the most important functions of striate cortex. Presumably this analysis is required to discriminate and identify objects based on their shape. Because virtually all interblob cells are orientation-selective and have small receptive fields, the P-IB channel is thought to be devoted to the *analysis of object shape*.

Orientation Columns

We have mentioned that many cortical neurons exhibit orientation selectivity. However, because this property may be generated independently by both M channels and P-IB channels, you might wonder whether the orientation selectivity of neurons in different layers is related. From the earliest work of Hubel and Wiesel, the answer to this question is an emphatic yes. As a microelectrode is advanced *radially* from one layer to the next, the preferred orientation remains the same for all the selective neurons encountered. This includes

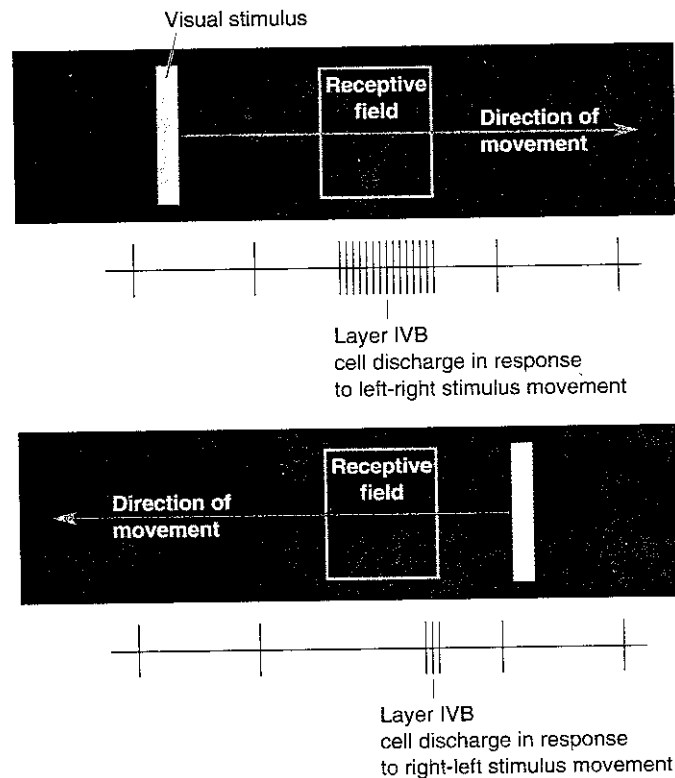


Figure 10.22
Responses of a direction-selective neuron.

all the cells of layers V and VI having both simple and complex receptive fields. Hubel and Wiesel called such a radial column of cells an **orientation column**. Perhaps predictably, as an electrode passes *tangentially* through the cortex in a single layer, the preferred orientation shifts systematically, 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 10.24). Hubel and Wiesel found that a complete 180° shift in preferred orientation required a traverse of about 1 mm within layer III.

Physiology of the Blobs

After Wong-Riley's discovery of the cytochrome oxidase blobs, Hubel, working with Harvard Medical School neurobiologist Margaret

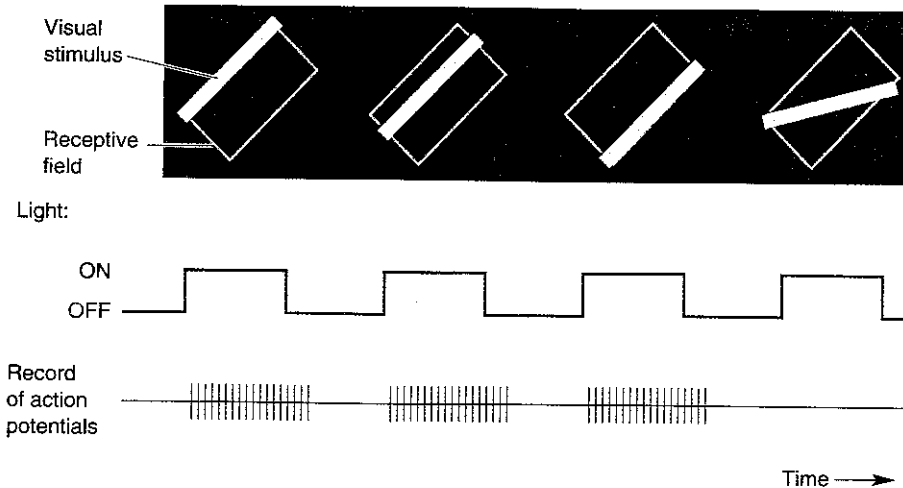


Figure 10.23

Complex cell receptive field. Like the simple cell, the complex cell responds best to an oriented bar of light. However, responses occur at both light ON and light OFF, regardless of position in the receptive field.

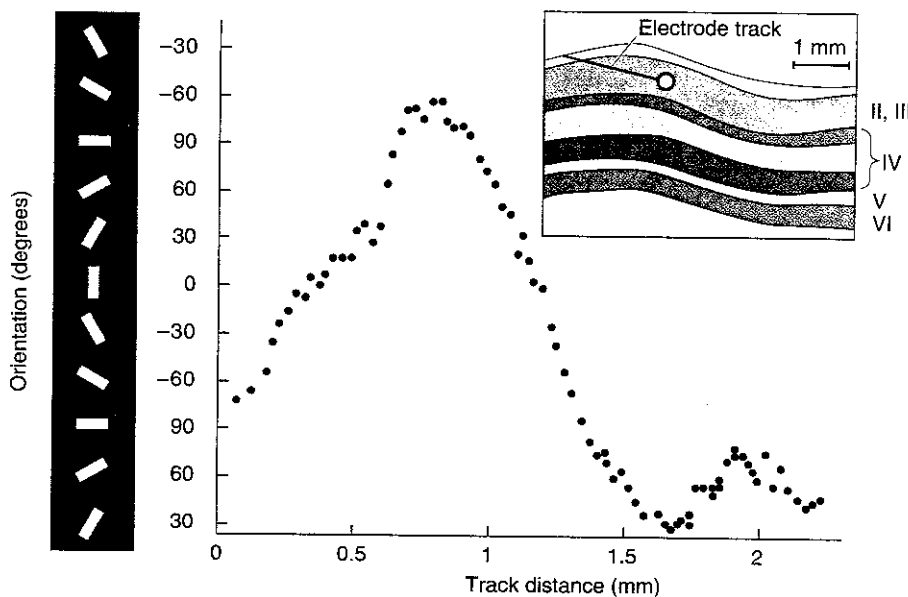


Figure 10.24

Systematic variation among 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.)

Livingstone, recorded from neurons in the blobs and found that they are very different from the interblob cells in layer III. The blob cells are wavelength-sensitive and monocular, and they lack orientation selectivity. The visual responses of blob cells resemble those of their major sources of input: the neurons in layer IV β and the koniocellular layers of the LGN.

The receptive fields of most blob neurons are circular. Some have the color-opponent center-surround organization observed in P-type ganglion cells and in the parvocellular 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 (called double-opponent cells). For present purposes, the most important thing to remember about blobs is that they contain the only color-sensitive neurons outside of layer IVC. Thus, the blob channels appear to be specialized for the *analysis of object color*. Without them, we would be color-blind.

Putting the Pieces Together

We have seen that the anatomy and physiology of the central visual pathways, from retina to striate cortex, are consistent with the idea that there are several, relatively independent, parallel processing channels. Each may be responsible for the analysis of a different facet of the visual scene: the M channel for the analysis of object motion, the P-IB channel for the analysis of object shape, and the blob channel for the analysis of object color.

Each point in the visual world is analyzed by a circumscribed patch of cells in the cortex. Hubel and Wiesel showed that the image of a point in space falls within the receptive fields of neurons across a traverse of about 2 mm of layer III. For a complete analysis, this 2 \times 2 mm patch of active neurons must include representatives from each of the processing channels from right and left eyes.

Fortunately, a 2 \times 2 mm chunk of cortex would contain two complete sets of ocular dominance columns in layer IV, 16 blobs in layer III, and, in the cells between blobs, a complete sampling (twice over) of all 180° of possible orientations. Thus, Hubel and Wiesel have argued that a 2 \times 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 of these modules, and one is shown in Figure 10.25. We can think of a visual scene being simultaneously processed by these modules, each “looking” at a portion of the scene.

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 modules. *How* is the simultaneous activity of widely separated cortical neurons compared, and *where* does this comparison take place?

Let's first explore some general ideas about how the information distributed across striate cortex might be combined. Then we'll examine a couple of cortical areas in the parietal and temporal lobes that depend on striate cortex for their major inputs. We will see that the processing channels that begin in the retina, and are elaborated in striate cortex, are maintained by corticocortical connections. Entirely different regions of association cor-

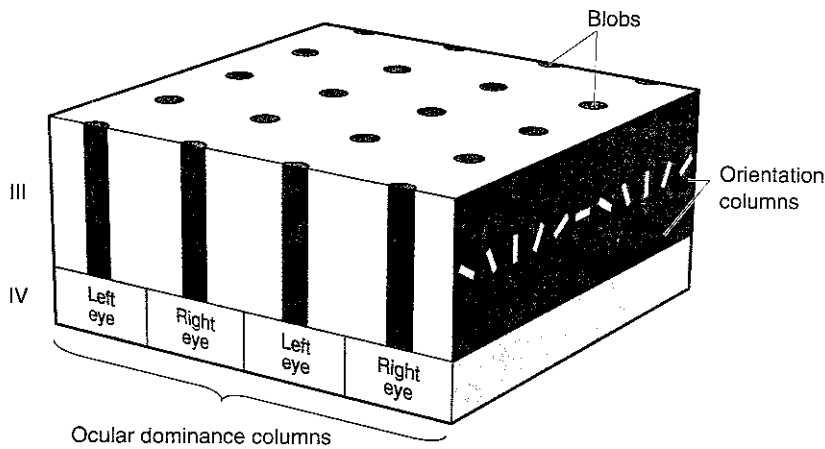


Figure 10.25
A cortical module.

tex appear to be dedicated to the analysis of visual motion and color, for example. We'll see that although neuroscience cannot yet answer the questions, posed above, of how and where the streams of information combine to form seamless perceptions of the world, our best hope lies beyond V1.

Communication Among Modules

Consider the pattern of activity that might arise in striate cortex when you view a simple geometric shape like a stop sign (Figure 10.26). Because the sign takes up a substantial portion of the visual field, neural activity in the cortex is distributed among many cortical modules. The readout of each module provides information about orientation and color at a fraction of the contrast border along the edge of the sign. Somehow, the activity of these modules must be compared, and a decision made, that this activity represents a single object—a stop sign—and not a jumble of line segments. Such a decision might be made by cells in higher cortical areas that are postsynaptic to the active striate cortical cells. In this case, we might expect to find “stop sign cells,” neurons that only respond to an octagonal red shape. Elsewhere we might find “grandmother cells,” neurons that respond only to one’s grandmother, and so on.

Another scheme for perceptually linking the neurons that respond to a stop sign or one’s grandmother takes advantage of the fact that the cortical modules activated by a single object will be active simultaneously. Thus, the contours of an object could be perceptually bound together if the activity of a single module were in some way sensitive to the activity of other modules that were active at precisely the same time. In fact, there is an anatomical basis for cross-talk between modules: the horizontal connections that connect the cells of layer III. Similar connections even cross from one side of the brain to the other via the corpus callosum to bind together the cells that respond to images falling on the seam between the right and left visual fields. These connections selectively wire up the cells that have common receptive field characteristics; for example, blob cells connect to other blob cells, cells responding only to horizontal lines connect to cells that share that same orientation preference, and so on. In 1989, neuroscientists Charles Gray and Wolf Singer, working together at the Max Planck Institute in Frankfurt, Germany, discovered that visual cortical cells respond with repetitive *bursts* of action potentials to their preferred stimuli, and that the bursts of activity occurring in widely separated modules were often syn-

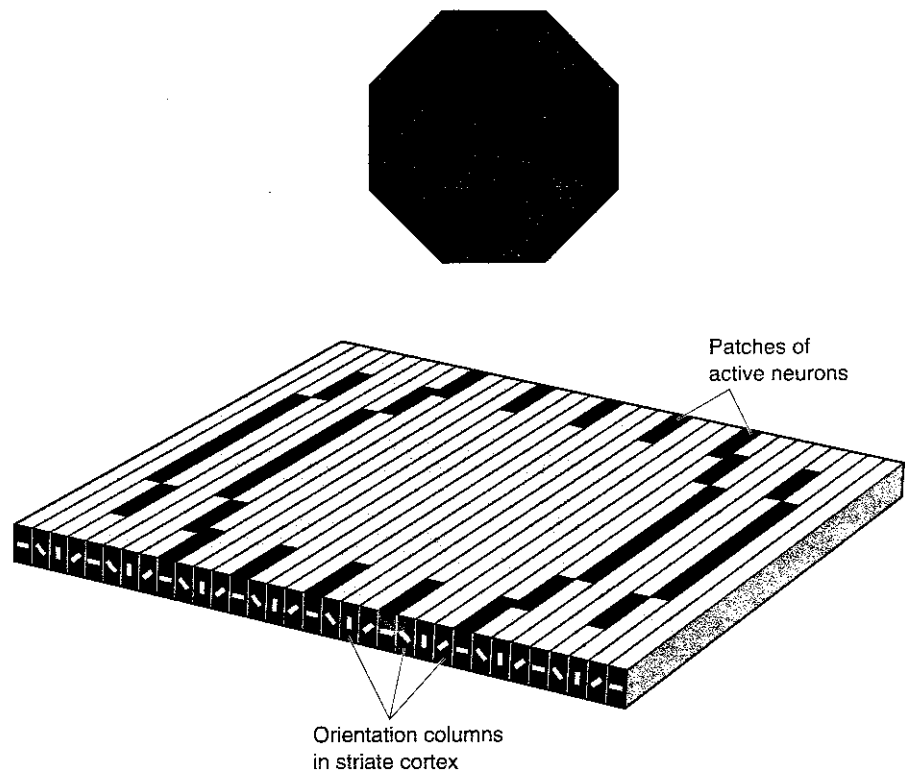


Figure 10.26
Possible distribution of neurons in striate cortex in response to the shape of a stop sign.

chronous when activated by the contours of a single common object. This work suggests that the internal neural representation of a stop sign or one's grandmother might be the synchronous activity of a large population of cells distributed across the cortex.

So is "grandmother" a cell, or a population of cells? The "grandmother cell" theory predicts that we should find neurons in higher cortical areas that are selective to, say, faces. As improbable as this might seem, there are now reports of cells in the temporal lobes of monkeys that respond only to faces! Then again, there do not seem to be neurons tuned to everything we perceive. Most neurons in higher cortical areas appear to represent the things we perceive in a much more abstract manner than the grandmother cell theory suggests. Today, most visual neuroscientists believe that the representation of objects is distributed among a population of many cortical neurons, but there is still little agreement as to the nature of this representation.

Beyond V1

The search for understanding of visual perception takes us beyond the borders of striate cortex. As already mentioned, striate cortex is also called V1, for "visual area one," because it is the first cortical area to receive information from the LGN. Beyond V1, however, lies another two dozen distinct areas of cortex, each of which contains a map of the visual world. Most of the extrastriate areas remain a wild neuroscientific frontier, and their contributions to vision are still being vigorously debated. However, the emerging picture is that there are two large-scale cortical *streams* of visual processing, one stretching from striate cortex toward the parietal lobe and the other projecting toward the temporal lobe (Figure 10.27a).

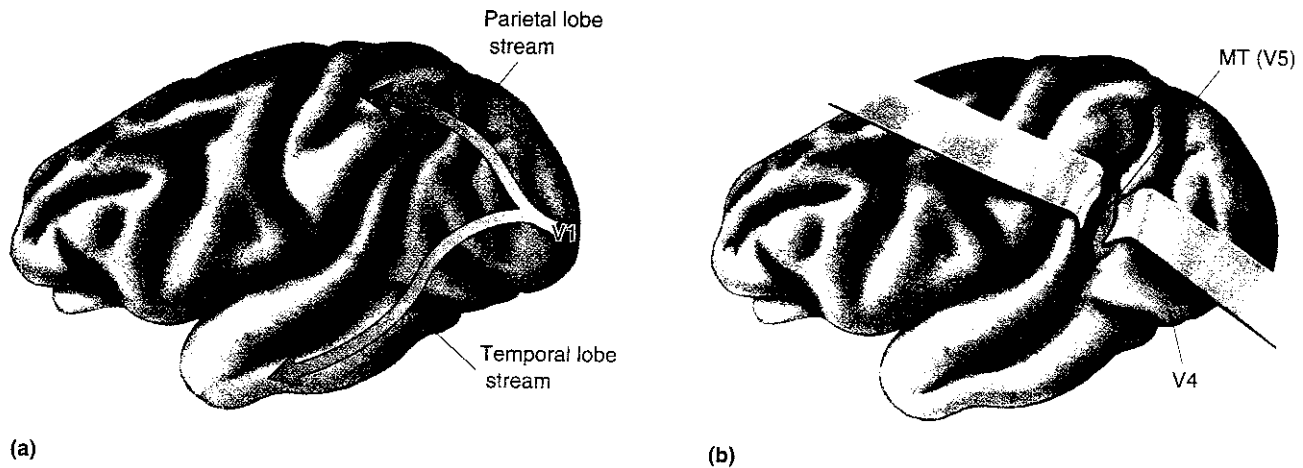


Figure 10.27. Location of (a) parietal and temporal lobe visual processing streams and (b) areas MT and V4 in the macaque brain.

Area MT and the Parietal Lobe Stream. As an example of the areas found in the parietal lobe stream, we consider cortical area V5, or, as it is usually called, area MT. Area MT receives retinotopically organized input from a number of other cortical areas such as V2 and V3, but it also is directly innervated by cells in layer IVB of striate cortex. Recall that layer IVB is part of the magnocellular channel, which is characterized by cells with large receptive fields, transient responses to light, and direction selectivity. Area MT is most notable for the fact that almost all the cells are direction-selective, unlike areas earlier in the parietal stream, or anywhere in the temporal stream. The neurons in MT also respond to types of motion, such as drifting spots of light, that are not good stimuli for other areas. For these reasons and others, MT appears to be specialized for the analysis of motion. Consistent with this idea, lesions in MT seriously disrupt the ability of monkeys to discriminate the direction in which a stimulus moves.

Neurons in area MT have large receptive fields that respond to stimulus movement in a narrow range of directions. As with orientation-selective cells in striate cortex, these direction-selective cells are arranged into a system of columns. A complete analysis requires that each point in visual space be "viewed" by cells in columns that respond to a full 360° range of directions. Presumably, the perception of movement at any point in space then depends on a comparison of the activity across these direction columns. This idea was recently tested directly in a fascinating experiment performed by William Newsome and his colleagues at Stanford University. They used a method pioneered at the National Institutes of Health by Edward Everts, in which electrodes are painlessly lowered into the cortex of awake monkeys (see Box 14.1). In Newsome's experiment, a monkey watched a video display of moving dots. The monkey would report the direction in which he believed the dots were moving by moving his eyes in that direction. (Monkeys, like humans, appear to enjoy video games.) Amazingly, Newsome and his colleagues discovered that by electrically stimulating the cells in a single-direction column of area MT, they could shift the monkey's perception of stimulus movement (Box 10.3). This is perhaps the closest visual neurophysiology has come to visual perception, and it bodes well for our eventual understanding of the neurophysiological basis of vision.

Beyond area MT in the parietal lobe stream are several cortical areas involved in directing eye movements. It is likely that one of the main reasons the brain exerts so much effort on analyzing motion is that it is critical

Box 10.3

PATH OF DISCOVERY

Influencing a Perceptual Judgment

by William T. Newsome

By the summer of 1989, my colleagues and I were fairly sure that MT was an important source of the directional signals that enabled monkeys to discriminate the direction of motion in our visual displays. We had used microelectrode recordings to “listen” to the traffic of electrical signals in MT while monkeys worked on the task, and we knew that MT neurons were exquisitely sensitive to the motion signals in the displays. We also knew that small lesions in MT would selectively disrupt a monkey’s ability to perform the task. But we lacked a “smoking gun”—an experiment that could directly show that changes in the activity of MT neurons *cause* corresponding changes in a monkey’s choices in the direction discrimination task.

From the beginning of our work with the direction discrimination task, I had considered trying just such an experiment. The idea was to use electrical microstimulation to activate a column of directionally selective neurons in MT while a monkey inspected the visual display and judged the direction of motion. In other words, rather than “listen” to the natural electrical signals of MT neurons, we would use our microelectrodes to artificially “boost” the signals related to a particular direction of motion. If MT neurons actually provided the signals used by the monkey in choosing the direction of motion, stimulating a particular column should increase the probability of a choice favoring the direction of motion encoded by that column.

Though intriguing, this experiment stayed on the back burner because the chances of success seemed very slim. Columns encoding different directions of motion are interspersed among each other in MT, and it would thus be necessary to restrict activation to one or a very few columns in order to avoid “smearing” the microstimulation signal across an arbitrary collection of directions. Even if the stimulating current could be placed with sufficient precision, it seemed unlikely that activation of a *single* cortical column could exert a measurable influence on perceptual behavior.

In August of 1989, however, Daniel Salzman (a Stanford University medical student) and I gave the

experiment a try. I warned Daniel not to expect much from this experiment because it was fairly far-fetched. Remarkably, the very first experiment we attempted yielded a significant effect—the monkey’s choices were indeed biased toward the direction encoded by the neurons at the electrode tip! Not believing the result, I insisted that we immediately repeat the experiment with the stimulator turned off to determine whether any undetected bugs in our computer software could cause the effect. To my surprise, the result held up in that experiment and in several more experiments over the next few days.

In the tenth experiment we got our first “whopper,” a term we came to apply to experiments with spectacularly large effects. In this experiment, the monkey chose the direction encoded by the stimulated neurons on 51 of the first 54 trials! It was almost as though the monkey could not help but choose that direction when we “tickled” the cortex at exactly the right place. I distinctly remember that Daniel and I had the same reaction as we watched this amazing result unfold—uncontrollable laughter. The most analogous experience I can imagine is hitting a jackpot at a Las Vegas slot machine; laughter is probably the most appropriate response to such a preposterous turn of events.

One of the most important lessons I learned from this investigation was to be suspicious of my own preconceptions. I would never have trained a monkey simply to do the microstimulation experiment; the investment of time and money seemed unwarranted, given what appeared to be a very small probability of success. Since the monkeys were already trained for the single-unit recordings, however, only a modest amount of additional effort was necessary to try a few microstimulation experiments. How many other important results are we missing even now because our most basic assumptions about the brain are somehow flawed?



William T. Newsome

for our eyes to be able to follow objects and move toward the objects when necessary.

Area V4 and the Temporal Lobe Stream. One of the most-studied areas in the temporal lobe is area V4 (Figure 10.27b). V4 receives input from the blob and interblob regions of striate cortex via a relay in V2. Neurons in V4 have larger receptive fields than cells in striate cortex, and many of the cells are both orientation-selective and color-selective. Neurons in this area also have more elaborate spatial and color interactions than those in striate cortex. Although there is a good deal of ongoing speculation concerning the function of V4, this area appears to be important for both shape and color perception. If this area is damaged, there are perceptual deficits involving both shape and color.

Beyond V4 in the temporal lobe are cortical areas that contain neurons with complicated spatial receptive fields. Although some of these neurons have been found to be particularly responsive to identifiable objects such as faces, the receptive fields of many temporal neurons are more abstract. As we will see in Chapter 19, these areas in temporal cortex appear to be important for both visual perception and visual memory.

In this chapter, we have outlined the organization of the sensory pathway from eye to thalamus to cortex. We saw that the sensory modality we call vision actually may be divided into a number of submodalities—color, contrast, form, movement—and these are processed in parallel by different cells of the visual system. This processing of information in the visual system 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.

CONCLUDING REMARKS

The Retinofugal Projection

retinofugal projection
optic nerve
optic chiasm
partial decussation
optic tract
visual hemifield
binocular visual field
lateral geniculate nucleus (LGN)
optic radiation
superior colliculus
optic tectum

retinotectal projection
retinotopy

The Lateral Geniculate Nucleus

magnocellular LGN layer
parvocellular LGN layer
koniocellular LGN layer

Anatomy of the Striate Cortex

area 17
V1
striate cortex

KEY TERMS

ocular dominance column
cytochrome oxidase
blob

Physiology of the Striate Cortex

magnocellular channel (M channel)
parvocellular-interblob channel
(P-IB channel)
blob channel
simple cell

orientation selectivity
binocular receptive field
direction selectivity
complex cell
orientation column
cortical module

From Single Neurons to Perception
area MT



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 a M-type retinal ganglion cell to a neuron in striate cortical layer IVB?
6. Which pathway, magnocellular or parvocellular, constitutes four-fifths 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?