As we saw in Chapter 3, the brain performs two major functions: it controls the movements of the muscles, producing useful behaviors, and it regulates the body's internal environment. To perform both these tasks, the brain must be informed about what is happening both in the external environment and within the body. Such information is received by the sensory systems. This chapter and the next are devoted to a discussion of the ways in which sensory organs detect changes in the environment and the ways in which the brain interprets neural signals from these organs.

We receive information about the environment from sensory receptors—specialized neurons that detect a variety of physical events. (Do not confuse sensory receptors with receptors for neurotransmitters, neuromodulators, and hormones. Sensory receptors are specialized neurons, and the other types of receptors are specialized proteins that bind with certain molecules.) Stimuli impinge on the receptors and, through various processes, alter their membrane potentials. This process is known as sensory transduction because sensory events are transduced ("transferred") into changes in the cells' membrane potential. These electrical changes are called receptor potentials. Most receptors lack axons; a portion of their somatic membrane forms synapses with the dendrites of other neurons. Receptor potentials affect the release of neurotransmitters and hence modify the pattern of firing in neurons with which these cells form synapses. Ultimately, the information reaches the brain.

People often say that we have five senses: sight, hearing, smell, taste, and touch. Actually, we have more than five, but even experts disagree about how the lines between the various categories should be drawn. Certainly, we should add the vestibular senses; as well as providing us with auditory information, the inner ear supplies information about head orientation and movement. The sense of touch (or, more accurately, somatosensation) detects changes in pressure, warmth, cold, vibration, limb position, and events that damage tissue (that is, produce pain). Everyone agrees that we can detect these stimuli; the issue is whether they are detected by separate senses.

This chapter considers vision, the sensory modality that receives the most attention from psychologists, anatomists, and physiologists. One reason for this attention derives from the fascinating complexity of the sensory organs of vision and the relatively large proportion of the brain that is devoted to the analysis of visual information. Another reason, I am sure, is that vision is so important to us as individuals. A natural fascination with such a rich source of information about the world leads to curiosity about how this sensory modality works. Chapter 7 deals with the other sensory modalities: audition, the vestibular senses, the somatosenses, gustation, and olfaction.

The Stimulus

As we all know, our eyes detect the presence of light. For humans light is a narrow band of the spectrum of electromagnetic radiation. Electromagnetic radiation with a wavelength of between 380 and 760 nm (a nanometer, nm, sensory receptor A specialized neuron that detects a particular category of physical events.
sensory transduction The process by which sensory stimuli are transduced into slow, graded receptor potentials.
receptor potential A slow, graded electrical potential produced by a receptor cell in response to a physical stimulus.
Figure 6.1
The electromagnetic spectrum.

Light, saturation, and brightness. Light travels at a constant speed of approximately 300,000 kilometers (186,000 miles) per second. Thus, if the frequency of oscillation of the wave varies, the distance between the peaks of the waves will similarly vary, but in inverse fashion. Slower oscillations lead to longer wavelengths, and faster ones lead to shorter wavelengths. Wavelength determines the first of the three perceptual dimensions of light: hue. The visible spectrum displays the range of hues that our eyes can detect.

Light can also vary in intensity, which corresponds to the second perceptual dimension of light: brightness. If the intensity of the electromagnetic radiation is increased, the apparent brightness increases, too. The third dimension, saturation, refers to the relative purity of the light that is being perceived. If all the radiation is of one wavelength, the perceived color is pure, or fully saturated. Conversely, if the radiation contains all wavelengths, it produces no sensation of hue—it appears white. Colors with intermediate amounts of saturation consist of different mixtures of wavelengths. Figure 6.2 shows some color samples, all with the same hue but with different levels of brightness and saturation. (See Figure 6.2.)

Anatomy of the Visual System
For an individual to see, an image must be focused on the retina, the inner lining of the eye. This image causes changes in the electrical activity of millions of neurons in the retina, which results in messages being sent through the optic nerves to the rest of the brain. (I said "the rest" because the retina is actually part of the brain; it and the optic nerve are in the central—not peripheral—nervous system.) This section describes the anatomy of the eyes, the photoreceptors in the retina that detect the presence of light, and the connections between the retina and the brain.

The Eyes
The eyes are suspended in the orbit, bony pockets in the front of the skull. They are held in place and moved by six extraocular muscles attached to the tough, white outer coat of the eye called the sclera. (See Figure 6.3.) Normally,

hue One of the perceptual dimensions of color; the dominant wavelength.
brightness One of the perceptual dimensions of color; intensity.
saturation One of the perceptual dimensions of color; purity.
we cannot look behind our eyeballs and see these muscles, because their attachments to the eyes are hidden by the conjunctiva. These mucous membranes line the eyelid and fold back to attach to the eye (thus preventing a contact lens that has slipped off the cornea from “falling behind the eye”). Figure 6.4 illustrates the anatomy of the eye. (See Figure 6.4.)

The eyes make three types of movements: vergence movements, saccadic movements, and pursuit movements. Vergence movements are cooperative movements that keep both eyes fixed on the same target—or, more precisely, that keep the image of the target object on corresponding parts of the two retinas. If you hold up a finger in front of your face, look at it, and then bring your finger closer to your face, your eyes will make vergence movements toward your nose. If you then look at an object on the other side of the room, your eyes will rotate outward, and you will see two separate blurry images of your finger.

When you scan the scene in front of you, your gaze does not roam slowly and steadily across its features. Instead, your eyes make jerky saccadic movements—you shift your gaze abruptly from one point to another. When you read a line in this book, your eyes stop several times, moving very quickly between each stop. You cannot consciously control the speed of movement between stops; during each saccade the eyes move as fast as they can. Only by performing a pursuit movement—say, by looking at your finger while you move it around—can you make your eyes move more slowly.

The outer layer of most of the eye, the sclera, is opaque and does not permit entry of light. However, the cornea, the outer layer at the front of the eye, is transparent and admits light. The amount of light that enters is regulated by the size of the pupil, which is an opening in the iris, the pigmented ring of muscles situated behind the cornea. The lens, situated immediately behind the iris, consists of a series of transparent, onion-like layers. Its shape can be altered by contraction of the ciliary muscles. These changes in shape permit the eye to focus images of near or distant objects on the retina—a process called accommodation.

After passing through the lens, light traverses the main part of the eye, which is filled with vitreous humor ("glassy liquid"), a clear, gelatinous substance. After passing through the vitreous humor, light falls on the retina, the interior lining of the back of the eye. In the retina are located the receptor cells, the rods and cones (named for their shapes), collectively known as photoreceptors.

The human retina contains approximately 120 million rods and 6 million cones. Although they are greatly outnumbered by rods, cones provide us with most of the information about our environment. In particular, they are responsible for our daytime vision. They provide us with information about small features in the environment and thus are the source of vision of the highest sharpness, or acuity (from acus, “needle”). The fovea, or central region

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**vergence movement** The cooperative movement of the eyes, which ensures that the image of an object falls on identical portions of both retinas.

**saccadic movement** (suh kad ik) The rapid, jerky movement of the eyes used in scanning a visual scene.

**pursuit movement** The movement that the eyes make to maintain an image of a moving object on the fovea.

**accommodation** Changes in the thickness of the lens of the eye, accomplished by the ciliary muscles, that focus images of near or distant objects on the retina.

**retina** The neural tissue and photoreceptive cells located on the inner surface of the posterior portion of the eye.

**rod** One of the receptor cells of the retina; sensitive to light of low intensity.

**cone** One of the receptor cells of the retina, maximally sensitive to one of three different wavelengths of light and hence encodes color vision.

**photoreceptor** One of the receptor cells of the retina; transduces photic energy into electrical potentials.

**fovea** (foe vea) The region of the retina that mediates the most acute vision of birds and higher mammals. Color-sensitive cones constitute the only type of photoreceptor found in the fovea.
of the retina, which mediates our most acute vision, contains only cones. Cones are also responsible for color vision—our ability to discriminate light of different wavelengths. Although rods do not detect different colors and provide vision of poor acuity, they are more sensitive to light. In a very dimly lighted environment we use our rod vision; therefore, in dim light we are color-blind and lack foveal vision. You may have noticed, while out on a dark night, that looking directly at a dim, distant light (that is, placing the image of the light on the fovea) causes it to disappear. (See Table 6.1.)

Another feature of the retina is the optic disk, where the axons conveying visual information gather together and leave the eye through the optic nerve. The optic disk produces a blind spot because no receptors are located there. We do not normally perceive our blind spots, but their presence can be demonstrated. If you have not found yours, you may want to try the exercise described in Figure 6.5.

Close examination of the retina shows that it consists of several layers of neuron cell bodies, their axons and den-
Figure 6.5
A test for the blind spot. With your left eye closed, look at the + with your right eye and move the page nearer to and farther from you. When the page is about 20 cm from your face, the green circle disappears because its image falls on the blind spot of your right eye.

drines, and the photoreceptors. Figure 6.6 illustrates a cross section through the primate retina, which is divided into three main layers: the photoreceptive layer, the bipolar cell layer, and the ganglion cell layer. Note that the photoreceptors are at the back of the retina; light must pass through the overlying layers to get to them. Fortunately, these layers are transparent. (See Figure 6.6.)

The photoreceptors form synapses with bipolar cells, neurons whose two arms connect the shallowest and deepest layers of the retina. In turn, these neurons form synapses with the ganglion cells, neurons whose axons travel through the optic nerves (the second cranial nerves) and carry visual information into the brain. In addition, the retina contains horizontal cells and amacrine cells, both of which transmit information in a direction parallel to the surface of the retina and thus combine messages from adjacent photoreceptors. (See Figure 6.6.)

Photoreceptors

Figure 6.7 shows a drawing of two rods and a cone. Note that each photoreceptor consists of an outer segment connected by a cilium to the inner segment, which contains the nucleus. (See Figure 6.7.) The outer segment contains several hundred lamellae, or thin plates of membrane. (Lamella is the diminutive form of lamina, "thin layer".)
Let's consider the nature of transduction of visual information. The first step in the chain of events that leads to visual perception involves a special chemical called a photopigment. Photopigments are special molecules embedded in the membrane of the lamellae; a single human rod contains approximately 10 million of them. The molecules consist of two parts: an opsin (a protein) and retinal (a lipid). There are several forms of opsin; for example, the photopigment of human rods, rhodopsin, consists of rod opsin plus retinal. (Rhod- refers to the Greek rhodon, “rose,” not to rod. Before it is bleached by the action of light, rhodopsin has a pinkish hue.) Retinal is synthesized from vitamin A, which explains why carrots, rich in this vitamin, are said to be good for your eyesight.

When a molecule of rhodopsin is exposed to light, it breaks into its two constituents: rod opsin and retinal. When that happens, the rod opsin changes from its rosy color to a pale yellow; hence, we say that the light bleaches the photopigment. The splitting of the photopigment causes a change in the membrane potential of the photoreceptor (the receptor potential), which changes the rate at which the photoreceptor releases its neurotransmitter glutamate.

The membrane of photoreceptors is different from that of other neurons—it contains cation channels that are normally open (Baylor, 1996). In the dark these ion channels, which admit Na⁺ and Ca²⁺, are held open by molecules of cyclic GMP; thus, the resting membrane potential is less polarized than that of other neurons. As a consequence, photoreceptors continuously release glutamate when light is not falling on them. When light strikes a molecule of photopigment and causes it to split, the resulting series of chemical events activates a G protein known as transducin. In turn, molecules of transducin activate molecules of the enzyme phosphodiesterase, which destroy cyclic GMP, closing the ion channels. Because cations can no longer enter the cell, the membrane then becomes more polarized, and the release of glutamate decreases. (See Figure 6.8.)

In the vertebrate retina, photoreceptors provide input to both bipolar cells and horizontal cells. Figure 6.9 shows the neural circuitry from a photoreceptor to a ganglion cell. The circuitry is much simplified and omits the horizontal cells and amacrine cells. The first two types of cells in the circuit—photoreceptors and bipolar cells—do not produce action potentials. Instead, their release of neurotransmitter is regulated by the value of their membrane potential; depolarizations increase the release, and hyperpolarizations decrease it. The circles indicate what would be seen on an oscilloscope screen recording changes in the cells’ membrane potentials in response to a spot of light shining on the photoreceptor.

The hyperpolarizing effect of light on the membranes of photoreceptors is shown in the left graph. The hyperpolarization reduces the release of neurotransmitter by the photoreceptor. Because the neurotransmitter normally hyperpolarizes the dendrites of the bipolar cell, a reduction in its release causes the membrane of the bipolar cell to depolarize. Thus, light hyperpolarizes the photoreceptor and depolarizes the bipolar cell. (See Figure 6.9.) The depolarization causes the bipolar cell to release more neurotransmitter, which depolarizes the membrane of the ganglion cell, causing it to increase its rate of firing. Thus, light shining on the photoreceptor causes excitation of the ganglion cell.

The circuit shown in Figure 6.9 illustrates a ganglion cell whose firing rate increases in response to light. As we will see, other ganglion cells decrease their firing rate in response to light.

**Photopigment**: A protein dye bonded to retinal, a substance derived from vitamin A, responsible for transduction of visual information.

**Opsin** (op'sin): A class of protein that, together with retinal, constitutes the photopigments.

**Retinal** (ret' i nal'): A chemical synthesized from vitamin A; joins with an opsin to form a photopigment.

**Rhodopsin** (roh dopp' sin): A particular opsin found in rods.

**Transducin**: A G protein that is activated when a photon strikes a photopigment; activates phosphodiesterase molecules, which destroy cyclic GMP and close cation channels in the photoreceptor.
light. These neurons are connected to bipolar cells that form different types of synapses with the photoreceptors. The functions of these two types of circuits are discussed in a later section, “Coding of Visual Information in the Retina.” If you would like to know more about the neural circuitry of the retina, you should consult the book by Rodieck (1998).

Connections Between Eye and Brain

The axons of the retinal ganglion cells bring information to the rest of the brain. They ascend through the optic nerves and reach the dorsal lateral geniculate nucleus of the thalamus. This nucleus receives its name from its resemblance to a bent knee ( genu is Latin for “knee”). It contains six layers of neurons, each of which receives input from only one eye. The neurons in the two inner layers contain cell bodies larger than those in the outer four layers. For this reason, the inner two layers are called the magnocellular layers and the outer four layers are called the parvocellular layers (parvo- refers to the small size of the cells). A third set of neurons in the koniocellular sublayers are found ventral to

dorsal lateral geniculate nucleus. A group of cell bodies within the lateral geniculate body of the thalamus; receives inputs from the retina and projects to the primary visual cortex.

magnocellular layer. One of the inner two layers of neurons in the dorsal lateral geniculate nucleus; transmits information necessary for the perception of form, movement, depth, and small differences in brightness to the primary visual cortex.

parvocellular layer. One of the four outer layers of neurons in the dorsal lateral geniculate nucleus; transmits information necessary for perception of color and fine details to the primary visual cortex.

koniocellular sublayer. One of the sub-layers of neurons in the dorsal lateral geniculate nucleus found ventral to each of the magnocellular and parvocellular layers; transmits information from short-wavelength (“blue”) cones to the primary visual cortex.
each of the magnocellular and parvocellular layers. (Konis is the Greek word for “dust.”) As we will see later, these three sets of layers belong to different systems, which are responsible for the analysis of different types of visual information. They receive input from different types of retinal ganglion cells. (See Figure 6.10.)

The neurons in the dorsal lateral geniculate nucleus send their axons through a pathway known as the optic radiations to the primary visual cortex—the region surrounding the calcarine fissure (calcarine means “spur-shaped”), a hori-

![Figure 6.10](image)

A photomicrograph of a section through the right lateral geniculate nucleus of a rhesus monkey (cresyl violet stain). Layers 1, 4, and 6 receive input from the contralateral (left) eye, and layers 2, 3, and 5 receive input from the ipsilateral (right) eye. Layers 1 and 2 are the magnocellular layers; layers 3-6 are the parvocellular layers. The koniocellular sublayers are found ventral to each of the parvocellular and magnocellular other layers. The receptive fields of all six principal layers are in almost perfect registration; cells located along the line of the unlabeled arrow have receptive fields centered on the same point.


![Figure 6.11](image)

A photomicrograph of a cross section through the striate cortex of a rhesus macaque monkey. The ends of the striate cortex are shown by arrows.


The calcarine fissure is part of the primary visual cortex. (See Figure 6.11.) The optic radiations carry visual information posteriorly from the lateral geniculate nucleus to the primary sensory visual cortex (area 17). The diagram illustrates the horizontal and vertical path the axons take. (See Figure 6.12.) The axons from the nasal half of the retina and the temporal half of the retina cross to the opposite hemisphere and are processed in the visual cortex. (See Figure 6.12.)

Besides the primary retino-geniculo-cortical pathway, several other pathways are taken by fibers from the retina.
When light strikes a molecule of photopigment in a photoreceptor, the retinal molecule detaches from the opsin molecule. This detachment activates a G protein called transducin, which activates the enzyme phosphodiesterase, which in turn destroys the molecules of cyclic GMP that are holding cation channels open. The reduction in the influx of Na\(^+\) and Ca\(^{2+}\) produces the receptor potential—hyperpolarization of the photoreceptor membrane. As a result, the rate of firing of the ganglion cell changes, signaling the detection of light.

Visual information from the retina reaches the striate cortex surrounding the calcarine fissure after being relayed through the magnocellular, parvocellular, and koniocellular layers of the dorsal lateral geniculate nuclei. Several other regions of the brain, including the hypothalamus and the tectum, also receive visual information. These regions help to regulate activity during the day–night cycle, coordinate eye and head movements, control attention to visual stimuli, and regulate the size of the pupils.

### Coding of Visual Information in the Retina

This section describes the way in which cells of the retina encode information they receive from the photoreceptors.

### Coding of Light and Dark

One of the most important methods for studying the physiology of the visual system is the use of microelectrodes to record the electrical activity of single neurons. As we saw in the previous section, some ganglion cells become excited when light falls on the photoreceptors with which they communicate. The receptive field of a neuron in the visual system is the part of the visual field that neuron "sees"—that is, the part in which light must fall for the neuron to be stimulated. Obviously, the location of the receptive field of

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**calcaneal fissure** (kal ka rine) A horizontal fissure on the inner surface of the posterior cerebral cortex; the location of the primary visual cortex.

**striate cortex** (stry at) The primary visual cortex.

**optic chiasm** A cross-shaped connection between the optic nerves, located below the base of the brain, just anterior to the pituitary gland.

**receptive field** That portion of the visual field in which the presentation of visual stimuli will produce an alteration in the firing rate of a particular neuron.
a particular neuron depends on the location of the photoreceptors that provide it with visual information. If a neuron receives information from photoreceptors located in the fovea, its receptive field will be at the fixation point—the point at which the eye is looking. If the neuron receives information from photoreceptors located in the periphery of the retina, its receptive field will be located off to one side.

At the periphery of the retina, many individual receptors converge on a single ganglion cell, bringing information from a relatively large area of the retina—and hence a relatively large area of the visual field. However, foveal vision is more direct, with approximately equal numbers of ganglion cells and cones. These receptor-to-axon relationships explain the fact that our foveal (central) vision is very acute but our peripheral vision is much less precise. (See Figure 6.13.)

Over sixty years ago, Hartline (1938) discovered that the frog retina contained three types of ganglion cells. ON cells responded with an excitatory burst when the retina was illuminated, OFF cells responded when the light was turned off, and ON/OFF cells responded briefly when the light went on and again when it went off. Kuffler (1952, 1953), recording from ganglion cells in the retina of the cat, discovered that their receptive field consisted of a roughly circular center, surrounded by a ring. Stimulation of the center or surrounding fields had contrary effects: ON cells were excited by light falling in the central field (center) and were inhibited by light falling in the surrounding field (surround), whereas OFF cells responded in the opposite manner. ON/OFF ganglion cells were briefly excited when light was turned on or off. In primates these ON/OFF cells project primarily to the superior colliculus (Schiller and Maunsell, 1977); thus, they do not appear to play a direct role in form perception. (See Figure 6.14.)

Figure 6.14 also illustrates a rebound effect that occurs when the light is turned off again. Neurons whose firing is inhibited while the light is on will show a brief burst of excitation when it is turned off. In contrast, neurons whose firing is increased will show a brief period of inhibition when the light is turned off. (See Figure 6.14.)

The two major categories of ganglion cells (ON and OFF) and the organization of their receptive fields into contrasting center and surround provide useful information to the rest of the visual system. Let us consider these two types of ganglion cells first. As Schiller (1992) notes, ganglion cells normally fire at a relatively low rate. Then, when the level of illumination in the center of their receptive field increases or decreases (for example, when an object moves or the eye makes a saccade), they signal the change. In particular, ON cells signal increases and OFF cells signal decreases—but both signal them by an increased rate of firing. Such a system is particularly efficient. Theoretically, a single type of ganglion cell could fire at an intermediate rate and signal changes in the level of illumination by increases or decreases in rate of firing. However, in this case the average rate of firing of the one million axons in each optic nerve would have to be much higher.

Several studies have shown that ON cells and OFF cells do, indeed, signal different kinds of information. Schiller, Sandell, and Maunsell (1986) injected monkeys with APB (2-amino-4-phosphonobutyrate), a drug that selectively blocks synaptic transmission in ON bipolar cells. They found that the animals had difficulty detecting spots that were made brighter than the background but had no difficulty detecting spots that were slightly darker than the background. In addition, Dolan and Schiller (1989) found that an injection of APB completely blocked vision in very dim light, which is normally mediated by rods. Thus, rod bipolar cells must all be of the ON type. (If you think about it, that arrangement makes sense; in very dim light we are more likely to see brighter objects against a dark background than dark objects against a light background.)

The second characteristic of the receptive fields of ganglion cells—their center-surround organization—enhances our ability to detect the outlines of objects even when the contrast between the object and the background is low. Figure 6.15 illustrates this phenomenon. This figure shows six gray squares arranged in order of brightness. The right side of each square looks lighter than the left side, which makes the borders between the squares stand...
out. But these exaggerated borders do not exist in the illustration; they are added by our visual system because of the center-surround organization of the receptive fields of the retinal ganglion cells. (See Figure 6.15.)

Figure 6.16 explains how this phenomenon works. We see the centers and surrounds of the receptive fields of several ganglion cells. (In reality these receptive fields would be overlapping, but the simplified arrangement is easier to understand. This example also includes only ON cells—again, for the sake of simplicity.) The image of the transition between lighter and darker regions falls across some of these receptive fields. The cells whose centers are located in the brighter region but whose surrounds are located at least partially in the darker region will have the highest rate of firing. (See Figure 6.16.)

Enhancement of contrast. Although each gray square is of uniform darkness, the right edge of each square looks somewhat lighter and the left edge looks somewhat darker. This effect appears to be caused by the opponent centers/surround arrangement of the receptive fields of the retinal ganglion cells.

All of the surrounds of the ON cells whose receptive fields fall within the lighter gray are evenly illuminated; this illumination partially inhibits the firing of these cells.

A portion of the inhibitory surrounds of the ON cells near the border receive less illumination; thus, these cells have the highest rate of firing.

A schematic explanation of the phenomenon shown in Figure 6.15. Only ON cells are shown; OFF cells are responsible for the darker appearance of the left side of the darker square.
Coding of Color

So far, we have been examining the monochromatic properties of ganglion cells—that is, their responses to light and dark. But, of course, objects in our environment selectively absorb some wavelengths of light and reflect others, which, to our eyes, gives them different colors. Although monochromatic (black-and-white) vision is perfectly adequate for most purposes, color vision gave our primate ancestors the ability to distinguish ripe fruit from unripe fruit and made it more difficult for other animals to hide themselves by means of camouflage (Mollon, 1989). The retinas of humans, Old World monkeys, and apes contain three different types of cones, which provides us with the most elaborate form of color vision (Jacobs, 1996).

Color Mixing

Various theories of color vision have been proposed for many years—long before it was possible to disprove or validate them by physiological means. In 1802 Thomas Young, a British physicist and physician, proposed that the eye detects different colors because it contained three types of receptors, each sensitive to a single hue. His theory was referred to as the trichromatic (three-color) theory. It was suggested by the fact that for a human observer any color can be reproduced by mixing various quantities of three colors judiciously selected from different points along the spectrum.

I must emphasize that color mixing is different from pigment mixing. If we combine yellow and blue pigments (as when we mix paints), the resulting mixture is green. Color mixing refers to the addition of two or more light sources. If we shine a beam of red light and a beam of bluish green light together on a white screen, we will see yellow light. If we mix yellow and blue light, we get white light. When white appears on a color television screen or computer monitor, it actually consists of tiny dots of red, blue, and green light. (See Figure 6.17.)

Another fact of color perception suggested to a German physiologist, Ewald Hering (1905/1965), that hue might be represented in the visual system as opponent colors. People interested in color perception have long regarded yellow, blue, red, and green as primary colors—colors that seem unique and do not appear to be blends of other colors. (Black and white are primary, too, but we perceive them as colorless.) All other colors can be described as mixtures of these primary colors. The trichromatic system cannot explain why yellow is included in this group—why it is perceived as a pure color. In addition, some colors appear to blend, whereas others do not. For example, one cannot speak of a bluish green or a yellowish green, and orange appears to have both red and yellow qualities. Purple resembles both red and blue. But try to imagine a reddish green or a bluish yellow. It is impossible; these colors seem to be opposite to each other. Again, these facts are not explained by the trichromatic theory. As we shall see in the following section, the visual system uses both trichromatic
and opponent-color systems to encode information related to color.

Photoreceptors: Trichromatic Coding

Physiological investigations of retinal photoreceptors in higher primates have found that Young was right: Three different types of photoreceptors (three different types of cones) are responsible for color vision. Investigators have studied the absorption characteristics of individual photoreceptors, determining the amount of light of different wavelengths that is absorbed by the photopigments. These characteristics are controlled by the particular opsin a photoreceptor contains; different opsins absorb particular wavelengths more readily. Figure 6.18 shows the absorption characteristics of the four types of photoreceptors in the human retina: rods and the three types of cones. (See Figure 6.18.)

The peak sensitivities of the three types of cones are approximately 420 nm (blue-violet), 530 nm (green), and 560 nm (yellow-green). The peak sensitivity of the short-wavelength cone is actually 430 nm in the intact eye, because the lens absorbs some short-wavelength light. For convenience the short-, medium-, and long-wavelength cones are traditionally called “blue,” “green,” and “red” cones, respectively. The retina contains approximately equal numbers of “red” and “green” cones but a much smaller number of “blue” cones (approximately 8 percent of the total).

Genetic defects in color vision appear to result from abnormalities in one or more of the three types of cones (Boynton, 1979; Nathans et al., 1986; Wissinger and Sharpe, 1998). The first two kinds of defective color vision described here involve genes on the X chromosome; thus, because males have only one X chromosome, they are much more likely to have this disorder. (Females are likely to have a normal gene on one of their X chromosomes, which compensates for the defective one.) People with protanopia (“first-color defect”) confuse red and green. They see the world in shades of yellow and blue; both red and green look yellowish to them. Their visual acuity is normal, which suggests that their retinas do not lack “red” or “green” cones. This fact, and their sensitivity to lights of different wavelengths, suggests that their “red” cones are filled with “green” cone opsin. People with deuteranopia (“second-color defect”) also confuse red and green and also have normal visual acuity. Their “green” cones appear to be filled with “red” cone opsin.

Tritanopia (“third-color defect”) is rare, affecting fewer than 1 in 10,000 people. This disorder involves a faulty gene that is not located on an X chromosome; thus, it is equally prevalent in males and females. People with tritanopia have difficulty with hues of short wavelengths and see the world in greens and reds. To them a clear blue sky is a bright green, and yellow looks pink. Their retinas lack “blue” cones. Because the retina contains so few of these cones, their absence does not noticeably affect visual acuity.

Retinal Ganglion Cells: Opponent-Process Coding

At the level of the retinal ganglion cell the three-color code gets translated into an opponent-color system. Daw (1968) and Gouras (1968) found that these neurons respond specifically to pairs of primary colors, with red opposing green and blue opposing yellow. Thus, the retina contains two kinds of color-sensitive ganglion cells: red-green and yellow-blue. Some color-sensitive ganglion cells respond in a center-surround fashion. For example, a cell might be excited by red and inhibited by green in the center of their receptive field while showing the opposite response in the surrounding ring. (See Figure 6.19.) Other ganglion cells

protanopia (pro tan o pe e) an inherited form of defective color vision in which red and green hues are confused; “red” cones are filled with “green” cone opsin.

deuteranopia (dew ter an o pe e) an inherited form of defective color vision in which red and green hues are confused; “green” cones are filled with “red” cone opsin.

tritanopia (try tan o pe e) an inherited form of defective color vision in which hues with short wavelengths are confused; “blue” cones are either lacking or faulty.
The response characteristics of retinal ganglion cells to light of different wavelengths are obviously determined by the particular circuits that connect the three types of cones with the two types of ganglion cells. These circuits involve different types of bipolar cells, amacrine cells, and horizontal cells.

Figure 6.19 helps explain how particular hues are detected by the “red,” “green,” and “blue” cones and translated into excitation or inhibition of the red-green and yellow-blue ganglion cells. The diagram does not show the actual neural circuitry, which includes the retinal neurons that connect the cones with the ganglion cells. The arrows in Figure 6.20 refer merely to the effects of the light falling on the retina. The book by Rodieck (1998) describes the actual neural circuitry in considerable detail.

Detection and coding of pure red, green, or blue light is the easiest to understand. For example, red light excites “red” cones, which causes the excitation of red-green gan-
gion cells. (See Figure 6.20a.) Green light excites "green" cones, which causes the inhibition of red-green cells. (See Figure 6.20b.) But consider the effect of yellow light. Because the wavelength that produces the sensation of yellow is intermediate between red and green, it will stimulate both "red" and "green" cones about equally. Yellow-blue ganglion cells are excited by both "red" and "green" cones, so their rate of firing increases. However, red-green ganglion cells are excited by red and inhibited by green, so their firing rate does not change. The brain detects an increased firing rate from the axons of yellow-blue ganglion cells, which it interprets as yellow. (See Figure 6.20c.) Blue light simply inhibits the activity of yellow-blue ganglion cells. (See Figure 6.20d.)

The opponent-color system employed by the ganglion cells explains why we cannot perceive a reddish green or a bluish yellow: An axon that signals red or green (or yellow or blue) can either increase or decrease its rate of firing; it cannot do both at the same time. A reddish green would have to be signaled by a ganglion cell firing slowly and rapidly at the same time, which is obviously impossible.

**Negative Afterimages**

Figure 6.21 demonstrates an interesting property of the visual system: the formation of a negative afterimage. Stare at the cross in the center of the image on the left for approximately 30 seconds. Then quickly look at the cross in the center of the white rectangle to the right. You will have a fleeting experience of seeing the red and green colors of a radish—colors that are complementary, or opposite, to the ones on the left. (See Figure 6.21.) Complementary items go together to make up a whole. In this context complementary colors are those that make white (or shades of gray) when added together.

The most important cause of negative afterimages is adaptation in the rate of firing of retinal ganglion cells.

When ganglion cells are excited or inhibited for a prolonged period of time, they later show a rebound effect, firing faster or slower than normal. For example, the green of the radish in Figure 6.21 inhibits some red-green ganglion cells. When this region of the retina is then stimulated with the neutral-colored light reflected off the white rectangle, the red-green ganglion cells—no longer inhibited by the green light—fire faster than normal. Thus, we see a red afterimage of the radish.

**Interim Summary**

Recordings of the electrical activity of single neurons in the retina indicate that each ganglion cell receives information from photoreceptors—just one in the fovea and many more in the periphery. The receptive field of most retinal ganglion cells consists of two concentric circles, with the cells becoming excited when light falls in one region and becoming inhibited when it falls in the other. This arrangement enhances the ability of the nervous system to detect contrasts in brightness. ON cells are excited by light in the center, and OFF cells are excited by light in the surround. ON cells detect light objects against dark backgrounds; OFF cells detect dark objects against light backgrounds.

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**negative afterimage** The image seen after a portion of the retina is exposed to an intense visual stimulus; consists of colors complementary to those of the physical stimulus.

**complementary colors** Colors that make white or gray when mixed together.
Color vision occurs as a result of information provided by three types of cones, each of which is sensitive to light of a certain wavelength: long, medium, or short. The absorption characteristics of the cones are determined by the particular opsins that their photopigment contains. Most forms of defective color vision appear to be caused by alterations in cone opsins. The "red" cones of people with protanopia are filled with 'green' cone opsin, and the "green" cones of people with deuteranopia are filled with "red" cone opsin. The retinas of people with tritanopia appear to lack "blue" cones.

Most color-sensitive ganglion cells respond in an opposing center-surround fashion to the pairs of primary colors: red and green, and blue and yellow. The responses of these neurons is determined by the retinal circuitry connecting them with the photoreceptors.

### Analysis of Visual Information: Role of the Striate Cortex

The retinal ganglion cells encode information about the relative amounts of light falling on the center and surround regions of their receptive field and, in many cases, about the wavelength of that light. The striate cortex performs additional processing of this information, which it then transmits to the visual association cortex.

### Anatomy of the Striate Cortex

The striate cortex consists of six principal layers (and several sublayers), arranged in bands parallel to the surface. These layers contain the nuclei of cell bodies and dendritic trees that show up as bands of light or dark in sections of tissue that have been dyed with a cell-body stain. (See Figure 6.22.)

In primates information from the parvocellular and magnocellular layers of the dorsal lateral geniculate nucleus enters the middle layer (layer 4C) of the striate cortex. From there it is relayed upward and downward to be analyzed by circuits of neurons in different layers. Axons bringing information from the koniocellular layers form synapses with neurons in layers 2 and 3.

If we consider the striate cortex of one hemisphere as a whole—if we imagine that we remove it and spread it out on a flat surface—we find that it contains a map of the contralateral half of the visual field. (Remember that each side of the brain sees the opposite side of the visual field.) The map is distorted; approximately 25 percent of the striate cortex is devoted to the analysis of information from the fovea, which represents a small part of the visual field. (The area of the visual field seen by the fovea is approximately the size of a large grape held at arm's length.)

The pioneering studies of David Hubel and Torsten Wiesel at Harvard University during the 1960s began a revolution in the study of the physiology of visual perception (see Hubel and Wiesel, 1977, 1979). Hubel and Wiesel discovered that neurons in the visual cortex did not simply respond to spots of light; they selectively responded to specific features of the visual world. That is, the neural circuitry within the visual cortex combines information from several sources (for example, from axons carrying information received from several different ganglion cells) in such a way as to detect features that are larger than the receptive field of a single ganglion cell. The following subsections describe the visual characteristics that researchers have studied so far: orientation and movement, spatial frequency, texture, retinal disparity, and color.

### Orientation and Movement

Most neurons in the striate cortex are sensitive to orientation. That is, if a line is positioned in the cell's receptive field and rotated around its center, the cell will respond only when the line is in a particular position—a particular orientation. Some neurons respond best to a vertical line, some to a horizontal line, and some to a line oriented somewhere in between. Figure 6.23 shows the responses of a neuron in the striate cortex when lines were presented at various orienta-
tions. As you can see, this neuron responded best when a vertical line was presented in its receptive field. (See Figure 6.23.)

Some orientation-sensitive neurons have receptive fields organized in an opponent fashion. Hubel and Wiesel referred to them as simple cells. For example, a line of a particular orientation (say, a dark 45° line against a white background) might excite a cell if placed in the center of the receptive field but inhibit it if moved away from the center. (See Figure 6.24a.) Another type of neuron, which the researchers referred to as a complex cell, also responded best to a line of a particular orientation but did not show an inhibitory surround; that is, it continued to respond while the line was moved within the receptive field. In fact, many complex cells increased their rate of firing when the line was moved perpendicular to its angle of orientation; thus, they also served as movement detectors. In addition, complex cells responded equally well to white lines against black backgrounds and black lines against white backgrounds. (See Figure 6.24b.) Finally, hypercomplex cells responded to lines of a particular orientation, but had an inhibitory region at the end (or ends) of the lines, which meant that the cells detected the location of ends of lines of a particular orientation. (See Figure 6.24c.)

**Figure 6.23**
Orientation sensitivity. An orientation-sensitive neuron in the striate cortex will become active only when a line of a particular orientation appears within its receptive field. For example, the neuron depicted in this figure responds best to a bar that is vertically oriented.

**Figure 6.24**
Response characteristics of neurons to orientation in the primary visual cortex. (a) Simple cell. (b) Complex cell. (c) Hypercomplex cell.

**Spatial Frequency**
Although the early studies by Hubel and Wiesel suggested that neurons in the primary visual cortex detected lines and edges, subsequent research found that they actually responded best to sine-wave gratings (De Valois, Albrecht, and Thorell, 1978). Figure 6.25 compares a sine-wave grating with a more familiar square-wave grating. A square-wave grating consists of a simple set of rectangular bars that vary in brightness; the brightness along the length of a line

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**simple cell** An orientation-sensitive neuron in the striate cortex whose receptive field is organized in an opponent fashion.

**complex cell** A neuron in the visual cortex that responds to the presence of a line segment with a particular orientation located within its receptive field, especially when the line moves perpendicularly to its orientation.

**hypercomplex cell** A neuron in the visual cortex that responds to the presence of a line segment with a particular orientation that ends at a particular point within the cell's receptive field.
perpendicular to them would vary in a stepwise (square-wave) fashion. (See Figure 6.25a.) A sine-wave grating looks like a series of fuzzy, unfocused parallel bars. Along any line perpendicular to the long axis of the grating, the brightness varies according to a sine-wave function. (See Figure 6.25b.)

A sine-wave grating is designated by its spatial frequency. We are accustomed to the expression of frequencies (for example, of sound waves or radio waves) in terms of time or distance (such as cycles per second or cycles per meter). But because the image of a stimulus on the retina varies in size according to how close it is to the eye, the visual angle is generally used instead of the physical distance between adjacent cycles. Thus, the spatial frequency of a sine-wave grating is its variation in brightness measured in cycles per degree of visual angle. (See Figure 6.26.)

Most neurons in the striate cortex respond best when a sine-wave grating of a particular spatial frequency is placed in the appropriate part of the visual field. Different neurons detect different spatial frequencies. For orientation-sensitive neurons the grating must be aligned at the appropriate angle of orientation. Albrecht (1978) mapped the shapes of receptive fields of simple cells by observing their response while moving a very thin flickering line of the appropriate orientation through their receptive fields. He found that many of them had multiple inhibitory and excitatory regions surrounding the center. The profile of the excitatory and inhibitory regions of the receptive fields of such neurons looked like a modulated sine wave—precisely what would be needed to detect a few cycles of a sine-wave grating. (See Figure 6.27.) In most cases a neuron's receptive field is large enough to include between 1.5 and 3.5 cycles of the grating (De Valois, Thorell, and Albrecht, 1983).

What is the point of having neural circuits that analyze spatial frequency? A complete answer requires some rather complicated mathematics, so I will give a simplified one here. (If you are interested, you can consult De Valois and De Valois, 1988.) Consider the types of information provided by high and low spatial frequencies. Small objects, details within a large object, and large objects with sharp edges provide a signal rich in high frequencies, whereas large areas of light and dark are represented by low frequencies. An image that is deficient in high-frequency information looks fuzzy and out of focus, like the image seen by a nearsighted person who is not wearing corrective lenses. This image still provides much information about forms and objects in the environment; thus, the most important visual information is that contained in low spatial frequencies. When low-frequency information is removed, the shapes of images are very difficult to perceive. (As we will see, the more primitive magnocellular system provides low-frequency information.)

Many experiments have confirmed that the concept of spatial frequency plays a central role in visual perception,
and mathematical models have shown that the information present in a scene can be represented very efficiently if it is first encoded in terms of spatial frequency. Thus, the brain probably represents the information in a similar way. Here I will describe just one example to help show the validity of the concept. Look at the two pictures in Figure 6.28. You can see that the picture on the right looks much more like the face of Abraham Lincoln, the late U.S. President, than the one on the left does. Yet both pictures contain the same information. The creators of the pictures, Harmon and Julesz (1973), used a computer to construct the figure on the left, which consists of a series of squares, each representing the average brightness of a portion of a picture of Lincoln. The one on the right is simply a transformation of the first one in which high frequencies have been removed. Sharp edges contain high spatial frequencies, so the transformation eliminates them.

**Figure 6.28**
Spatial filtering. Both pictures contain the same amount of low-frequency information, but extraneous high-frequency information has been filtered from the picture on the right. If you look at the pictures from across the room, they look identical.

(From Harmon, L. D., and Julesz, B. Science, 1973, 180, 1191-1197. Copyright 1973 by the American Association for the Advancement of Science.)

If you want to watch the effect of filtering the extraneous high-frequency noise, try the following demonstration. Put the book down and look at the pictures in Figure 6.28 from across the room. The distance "erases" the high frequencies, because they exceed the resolving power of the eye, and the two pictures look identical. Now walk toward the book, focusing on the left figure. As you get closer, the higher frequencies reappear and this picture looks less and less like the face of Lincoln. (See Figure 6.28.)

**Texture**

Several years ago, von der Heydt, Peterhans, and Duerstler (1992) discovered a new class of neurons in the monkey striate cortex. These neurons respond to "periodic patterns." They do not respond when single lines, bars, or edges are placed in their receptive fields, but they do respond vigorously when a grating (square-wave, sine-wave, or thin-line) of a particular spatial frequency and orientation and frequency is presented there. To provide a reliable response, these cells require a minimum of two to seven alternating dark and light bars. They are not spatial-frequency analyzers like the ones I just described. The proof of this fact is difficult to convey in a few words, because it requires an understanding of the underlying mathematics. Those of you who would like to know more should consult the article.

These neurons showed extreme sensitivity to deviations from their optimal frequency and orientation. Figure 6.29 shows three square-wave gratings. The middle one produced the optimal response in a particular neuron in striate cortex. The one on the left, which has a slightly higher spatial frequency, produced only half as much excitation. The one on the right, which is rotated slightly counterclockwise, also produced only half as much excitation. (See Figure 6.29.)

| sine-wave grating | A series of straight parallel bands varying continuously in brightness according to a sine-wave function, along a line perpendicular to their lengths. |
| spatial frequency | The relative width of the bands in a sine-wave grating, measured in cycles per degree of visual angle. |
Von der Heydt and his colleagues estimate that approximately 4 million periodic-pattern-selective cells serve the central four degrees of vision in the monkey striate cortex. They suggest that the function provided by these cells is perception of surfaces. Most surfaces (especially those found in nature) have a rough texture, and many of them contain a repeating pattern. For example, tree trunks, grasslands, boulders, leaves of bushes and trees, pebble-strewn ground—even a close-up view of the fur of another animal—contain periodic patterns that potentially could be detected by these cells. These cells could help to discriminate surfaces that differ only in terms of their texture and could help to determine their orientation. As Figure 6.30 shows, texture gradients provide an important cue for perception of distance. (See Figure 6.30.)

**Retinal Disparity**

We perceive depth by many means, most of which involve cues that can be detected monocularly, by one eye alone. For example, perspective, relative retinal size, loss of detail through the effects of atmospheric haze, and relative apparent movement of retinal images as we move our heads all contribute to depth perception and do not require binocular vision. However, binocular vision provides a vivid perception of depth through the process of stereoscopic vision, or stereopsis. If you have used a stereoscope (such as a View-Master) or have seen a three-dimensional movie, you know what I mean. Stereopsis is particularly important in the visual guidance of fine movements of the hands and fingers, such as we use when we thread a needle.

Most neurons in the striate cortex are binocular—that is, they respond to visual stimulation of either eye. Many of these binocular cells, especially those found in a layer that receives information from the magnocellular system, have response patterns that appear to contribute to the perception of depth (Poggio and Poggio, 1984). In most cases the cells respond most vigorously when each eye sees a stimulus in a slightly different location. That is, the neurons respond to retinal disparity, a stimulus that produces images on slightly different parts of the retina of each eye. This is exactly the information that is needed for stereopsis; each eye sees a three-dimensional scene slightly differently, and the presence of retinal disparity indicates differences in the distance of objects from the observer.

**Color**

In the striate cortex information from color-sensitive ganglion cells is transmitted, through the parvocellular and koniocellular layers of the dorsal lateral geniculate nucleus, to special cells grouped together in cytochrome oxidase (CO) blobs. CO blobs were discovered by Wong-Riley (1978), who found that a stain for cytochrome oxidase, an enzyme present in mitochondria, showed a patchy distribution. Subsequent research with the stain (Horton and Hubel, 1980; Humphrey and Hendrickson, 1980) revealed the presence of a polka-dot pattern of dark columns extending through layers 2 and 3 and (more faintly) layers 5 and 6. The columns are oval in cross section, approximately 150 × 200 μm in diameter and spaced at 0.5-mm intervals ( Fitzpatrick, Itoh, and Diamond, 1983; Livingstone and Hubel, 1987).

Figure 6.31 shows a photomicrograph of a slice through a macaque monkey visual cortex that has been flattened out and stained for the mitochondrial enzyme. You can clearly see the CO blobs within the striate cortex. Because the curvature of the cortex prevents it from being perfectly flattened, some of the tissue is missing in the center of the slice. (See Figure 6.31.)

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**retinal disparity** The fact that points on objects located at different distances from the observer will fall on slightly different locations on the two retinas; provides the basis for stereopsis.

**cytochrome oxidase (CO) blob** The central region of a module of the primary visual cortex, revealed by a stain for cytochrome oxidase; contains wavelength-sensitive neurons; part of the parvocellular system.
tiles in a mosaic mural. Input from the parvocellular, koniocellular, and magnocellular layers of the dorsal lateral geniculate nucleus is received by different sublayers of the striate cortex: The parvocellular input is received by layer 4CB, the magnocellular input is received by layer 4Ca, and the koniocellular input is received by layers 2 and 3.

The modules actually consist of two segments, each surrounding a CO blob. Neurons located within the blobs have a special function: They are sensitive to color and to low spatial frequencies but are relatively insensitive to other visual features. Outside the CO blob, neurons show sensitivity to orientation, movement, spatial frequency, texture, and binocular disparity—but most do not respond to color (Livingstone and Hubel, 1984; Born and Tootell, 1991; Edwards, Purpura, and Kaplan, 1995). Each half of the module receives input from only one eye, but the circuitry within the module combines the information from both eyes, which means that most of the neurons are binocular. Depending on their locations within the module, neurons receive varying percentages of input from each of the eyes.

If we record from neurons anywhere within a single module, we will find that all of their receptive fields overlap. Thus, all the neurons in a module analyze information from the same region of the visual field. Furthermore, if we insert a microelectrode straight down into an interblob region of the striate cortex (that is, in a location in a module outside one of the CO blobs), we will find both simple and complex cells, but all of the orientation-sensitive cells will respond to lines of the same orientation. In addition, they will all share the same ocular dominance—that is, the same percentage of input from each of the eyes. If we move our electrode around the interblob region, we will find that these two characteristics—orientation sensitivity and ocular dominance—vary systematically and are arranged at right angles to each other. (See Figure 6.32.)

The striate cortex is not rectangular in shape, so its architecture is not like a checkerboard, with the borders of the modules following perfectly straight lines. Blasdel (1992a, 1992b) developed an ingenious technique to visualize just how the modules are arranged. He operated on monkeys, removing part of their skull and placing a glass window over the striate cortex. The window was equipped with a fitting that permitted him to inject a voltage-sensitive dye—a dye that changes its color according to the strength of an electrical field that passes through it.

ocular dominance The extent to which a particular neuron receives more input from one eye than from the other.
After the animals recovered from the surgery, Blasdel injected the dye, which spread across the surface of the striate cortex. Then he showed the animals visual stimuli designed to excite neurons that were sensitive to particular features. If a large number of neurons in a particular region were sensitive to that feature, their excitation would change the color of the dye covering that region. Blasdel presented patterns containing lines of different orientations to identify cells that responded to particular orientations, and he presented stimuli monocularly to identify cells that received direct input from either the right or the left eye. During the presentation of the stimuli he used a sensitive video camera to record the pattern of color changes in the dye and analyzed these patterns with a computer.

Figure 6.33 shows some of his results. The colors shown on the figure were produced by the computer and do not represent the actual color of the dye on the surface of the cortex; nor do they represent the sensitivity of cortical neurons to different wavelengths. Instead, the colors represent the orientation sensitivity of the cortical neurons. Neurons in a region colored red are sensitive to horizontal lines; those in a region colored orange are sensitive to a line rotated counterclockwise by 30 degrees; and so on for yellow, green, blue, and violet. You will notice that this sequence of colors corresponds to the sequence of hues in the visual spectrum, as shown in Figure 6.1. Figure 6.33(a) shows how orientation sensitivity (indicated by white lines) is coded by color; as you will see, the red regions contain neurons sensitive to horizontal lines, the green regions contain neurons sensitive to vertical lines, and so on. (See Figure 6.33a.)
Figure 6.33(b) shows the relation between orientation sensitivity and ocular dominance—that is, the degree to which a cell responds to only one eye. The white lines indicate the location of cells that respond exclusively to one eye; thus, cells that respond equally well to stimuli presented to either eye are located midway between these lines. As you will see, the “rainbows” are lined up along the channels defined by the white lines, which means that changes in orientation sensitivity run at right angles to changes in ocular dominance. The CO blobs, which receive information from only one eye and which contain neurons that are not sensitive to orientation, are located at regular intervals along these white lines. (See Figure 6.33b.)

How does spatial frequency fit into this organization? Edwards, Purpura, and Kaplan (1995) found that neurons within the CO blobs responded to low spatial frequencies but were sensitive to small differences in brightness. Outside the blobs, sensitivity to spatial frequency varied with the distance from the center of the nearest blob. Higher frequencies were associated with greater distances. (See Figure 6.34.) However, neurons outside the blobs were less sensitive to contrast; the difference between the bright and dark areas of the sine wave grating had to be greater for these neurons than for neurons within the blobs.

Blindsight

Visual perception depends on the integrity of the connections between the retina and the striate cortex. Thus, damage to the eyes, optic nerves, optic tracts, lateral geniculate nucleus, optic radiations, or primary visual cortex itself results in loss of vision in particular portions of the visual field or in complete blindness if the damage is total. However, an interesting phenomenon is seen in people with cortical blindness—blindness caused by damage to the optic radiations or primary visual cortex.

It has long been recognized that damage to the optic radiations or primary visual cortex on one side of the brain causes blindness in the contralateral visual field. That is, if the right side of the brain is damaged, the patient will be blind to everything located to the left when he or she looks straight ahead. However, Weiskrantz and his colleagues (Weiskrantz et al., 1974; Weiskrantz, 1987) found that if an object is placed in the patient's blind field and the patient is asked to reach for it, he or she will be able to do so rather accurately. The patients are surprised to find their hands repeatedly coming into contact with an object in what appears to them as darkness; they say that they see nothing there. The patient is also sensitive to movement, and, to a certain extent, the orientation of objects in the blind field.

This phenomenon, which Weiskrantz called blindsight, may depend on the connections that the visual association cortex receives from the superior colliculus and from the dorsal lateral geniculate nucleus (Cowey and Stoerig, 1991). The role of these connections in the intact brain is not known. Most of the inputs to the visual association cortex come directly from the striate cortex, and these connections are obviously necessary for normal visual perception.

Besides telling us something about the functions of the various parts of the visual system, the phenomenon of blindsight also shows that visual information can control behavior without producing a conscious sensation. Although the superior colliculi send visual information to parts of the brain that guide hand movements, they do not appear to send them to parts of the brain responsible for conscious awareness. Perhaps that connection is a more recent evolutionary development.
parvocellular, and koniocellular layers of the dorsal lateral geniculate nucleus. The magnocellular system is more primitive, color-blind, and sensitive to movement, depth, and small differences in brightness. The parvocellular system is more recent, color-sensitive (receiving information from "red" and "green" cones), and able to discriminate finer details. The koniocellular system provides additional information about color, received from "blue" cones.

The striate cortex is organized into modules, each surrounding a pair of CO blobs, which are revealed by a stain for cytochrome oxidase, an enzyme found in mitochondria. Each half of a module receives information from one eye; but because information is shared, most of the neurons respond to input to both eyes. The neurons in the CO blobs are sensitive to color and to low-frequency sine-wave gratings, whereas those between the blobs are sensitive to sine-wave gratings of higher spatial frequencies, orientation, retinal disparity, and movement. Some cells are specifically sensitive to orientation and frequency of gratings and probably are involved in detecting the texture of surfaces.

Damage to the visual system up to the striate cortex produces blindness in all or part of the visual field. However, damage limited to the striate cortex or to the optic radiations leading to them produces a syndrome called blindsight. People with blindsight deny seeing anything in the blind part of their visual field but can nevertheless point to objects located there and discriminate their size and orientation. They are also sensitive to movement. But although their behavior can be affected by objects in their blind field, they have no conscious awareness of the presence of these objects. Their ability to respond to visual stimuli apparently depends on connections from the superior colliculus and the lateral geniculate nucleus to the visual association cortex.

Analysis of Visual Information: Role of the Visual Association Cortex

Although the striate cortex is necessary for visual perception, perception of objects and of the totality of the visual scene does not take place there. Each module of the striate cortex sees only what is happening in one tiny part of the visual field. Thus, for us to perceive objects and entire scenes, the information from these individual modules must be combined. That combination takes place in the visual association cortex.

Two Streams of Visual Analysis

Visual information received from the striate cortex is analyzed in the visual association cortex. On the basis of their own research and on a review of the literature, Ungerleider and Mishkin (1982) concluded that the visual association cortex contains two streams of analysis. Subsequent anatomical studies have confirmed this conclusion (Baizer, Ungerleider, and Desimone, 1991). Both streams begin in the striate cortex, but they begin to diverge in the extrastriate cortex. The ventral stream turns downward, ending in the cortex of the inferior temporal lobe. The dorsal stream turns upward, ending in the cortex of the posterior parietal lobe. The ventral stream recognizes what an object is, and the dorsal stream recognizes where the object is located. (See Figure 6.35.)

As we saw, the parvocellular, koniocellular, and magnocellular systems provide different kinds of information. The magnocellular system is found in all mammals, whereas the parvocellular and koniocellular systems are found only
in primates. These systems receive information from different types of ganglion cells, which are connected to different types of bipolar cells and photoreceptors. Only the cells in the parvocellular and koniocellular system receive information about wavelength from cones; thus, these systems analyze information concerning color. Cells in the parvocellular system also show high spatial resolution and low temporal resolution; that is, they are able to detect very fine details, but their response is slow and prolonged. (As far as we know, the koniocellular system does not provide information about fine details). In contrast, neurons in the magnocellular system are color-blind, are not able to detect fine details, and respond very briefly to a visual stimulus. And although they appear to be responsible for vision of lower acuity, these neurons are able to detect smaller contrasts between light and dark. They are especially sensitive to movement. (See Table 6.2.)

At one time, researchers believed that the dorsal stream received its information from the magnocellular system and the ventral stream received its information from the parvocellular system. But more recent research has shown that both systems contribute information to both streams (Maunsell, 1992). The dorsal stream receives mostly magnocellular input, but the ventral stream receives approximately equal input from both systems and from the koniocellular system as well.

Neurons in the striate cortex send axons to the extra-striate cortex, the region of the visual association cortex that surrounds the striate cortex (Zeki and Shipp, 1988). The primate extrastriate cortex (sometimes called the prestriate cortex or circumstriate cortex) consists of several regions, each of which contains one or more independent maps of the visual field. Each region is specialized, containing neurons that respond to a particular feature of visual information, such as orientation, movement, spatial frequency, retinal disparity, or color. So far, investigators have identified twenty-five distinct regions and subregions of the visual cortex of the rhesus monkey. These regions are arranged hierarchically, beginning with the striate cortex (Van Essen, Anderson, and Felleman, 1992). Most of the information passes up the hierarchy; each region receives information from regions located beneath it in the hierarchy, analyzes the information, and passes the results on to "higher" regions for further analysis. Some information is also transmitted in the opposite direction, but axons that descend the hierarchy are much less numerous than those that ascend it. So far, our knowledge of the anatomical details far exceeds our understanding of the functions performed by the subdivisions of the visual cortex.

**Perception of Color**

As we saw earlier, neurons within the CO blobs in the striate cortex respond to colors. Like the ganglion cells in the retina (and the parvocellular and koniocellular neurons in the dorsal lateral geniculate nucleus), these neurons respond in opponent fashion. This information is analyzed by the regions of the visual association cortex that constitute the ventral stream.

**Studies with Laboratory Animals**

In the monkey brain neurons in the CO blobs send information about color to a specific subarea of the extrastriate cortex. Zeki (1980) found that neurons in this subarea (called V4) also respond selectively to colors, but their response characteristics are much more complex. Unlike the neurons we have encountered so far, these neurons respond to a variety of wavelengths, not just those that correspond to red, green, yellow, and blue.

The appearance of the colors of objects remains much the same whether we observe them under artificial light, under an overcast sky, or at noon on a cloudless day. This phenomenon is known as color constancy. Our visual system does not simply respond according to the wavelength of the light reflected by objects in each part of the visual field; instead, it compensates for the source of the

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**Table 6.2**

Properties of the magnocellular, parvocellular, and koniocellular divisions of the visual system

<table>
<thead>
<tr>
<th>Property</th>
<th>Magnocellular division</th>
<th>Parvocellular division</th>
<th>Koniocellular division</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color</td>
<td>No</td>
<td>Yes (from &quot;red&quot; and &quot;green&quot; cones)</td>
<td>Yes (from &quot;blue&quot; cones)</td>
</tr>
<tr>
<td>Sensitivity to contrast</td>
<td>High</td>
<td>Low</td>
<td>?</td>
</tr>
<tr>
<td>Spatial resolution (ability to detect fine details)</td>
<td>Low</td>
<td>High</td>
<td>low</td>
</tr>
<tr>
<td>Temporal resolution</td>
<td>Fast (transient response)</td>
<td>Slow (sustained response)</td>
<td>?</td>
</tr>
</tbody>
</table>
light. This compensation appears to be made by simultaneously comparing the color composition of each point in the visual field with the average color of the entire scene. If the scene contains a particularly high level of long-wavelength light (as it would if an object were illuminated by the light of a setting sun), then some long-wavelength light is "subtracted out" of the perception of each point in the scene.

Schein and Desimone (1990) performed a careful study of the response characteristics of neurons in area V4 of the monkey extrastriate cortex. They found that these neurons responded to specific colors. Some also responded to colored bars of specific orientation; thus, area V4 seems to be involved in the analysis of form as well as color. The color-sensitive neurons had a rather unusual secondary receptive field—a large region surrounding the primary field. When stimuli were presented in the secondary receptive field, the neuron did not respond. However, stimuli presented there could suppress the neuron's response to a stimulus presented in the primary field. For example, if a cell would fire when a red spot was presented in the primary field, it would fire at a slower rate (or not at all) when an additional red stimulus was presented in the surrounding secondary field. In other words, these cells responded to particular wavelengths of light but subtracted out the amount of that wavelength that was present in the background. As Schein and Desimone point out, this subtraction could serve as the basis for color constancy.

Walsh et al. (1993) confirmed this prediction; damage to area V4 does disrupt color constancy. The investigators found that although monkeys could still discriminate between different colors after area V4 had been damaged, their performance was impaired when the color of the overall illumination was changed. But the fact that the monkeys could still perform a color discrimination task under constant illumination means that some region besides area V4 must be involved in color vision.

A study by Heywood, Gaffan, and Cowey (1995) appears to have found that region—a portion of the inferior temporal cortex just anterior to area V4—a region of the monkey brain usually referred to as area TEO. The investigators destroyed area TEO, leaving area V4 intact, and observed severe impairment in color discrimination. The monkeys had no difficulty discriminating shades of gray, so the deficit on this task appeared to be restricted to color perception. (As we will see later, lesions of the inferior temporal cortex also disrupt the ability to perceive and recognize objects.)

When humans from a variety of cultures are asked to provide single names for colors, they universally chose eleven of them: red, orange, yellow, green, blue, purple, pink, brown, white, black, and gray (Boynton and Olson, 1987; Uchikawa and Boynton, 1987). Matsuawa (1983) found that chimpanzees appear to classify colors the same way, which suggests that the classification is based on the characteristics of neural mechanisms responsible for color perception. Indeed, Komatsu (1987) found a good correspondence between the eleven color categories and the responses of color-sensitive neurons in the inferior temporal cortex of the monkey.

Studies with Humans

Lesions of a restricted region of the human extrastriate cortex in the medial occipital lobe can cause loss of color vision without disruption of visual acuity. The patients describe their vision as resembling a black-and-white film. (Damasio et al., 1980; Kennard et al., 1995). The condition is known as achromatopsia ("vision without color"). If the brain damage is unilateral, people will lose color vision in only half of the visual field. In addition, they cannot even imagine colors or remember the colors of objects they saw before their brain damage occurred. As we just saw, Heywood, Gaffan, and Cowey (1995) found a region of the inferior temporal cortex of the monkey brain whose damage disrupted the ability to make color discriminations.

The analogous region appears to play a critical role in color perception in humans. A functional MRI study by Hadjikhani et al. (1998) found a color-sensitive region in the inferior temporal cortex in a position corresponding to TEO in the monkey's cortex, which they called area V8. Indeed, lesions that cause achromatopsia damage V8 or other brain regions that provide input to V8. Figure 6.36 shows a "flattened out" map of some of the visual regions of the human and rhesus monkey brain. As you can see, human area V8 appears to correspond to area TEO. (See Figure 6.36.)

Of course, perception of colors is useless in itself. The function of our ability to perceive different colors is to help us perceive different objects in our environment. Thus, to perceive and understand what is in front of us, we must have information about color combined with other forms of information. Some people with brain damage lose the ability to perceive shapes but can still perceive colors (Zeki, 1992). They can identify the colors of objects in their visual field, but they cannot say what these objects are. Presumably, the damaged region of their brain does not include area V8.

**achromatopsia** (ə krə'mə təp'se-ə) inability to discriminate among different hues, caused by damage to the visual association cortex.

**inferior temporal cortex** in primates the highest level of the ventral stream of the visual association cortex; located on the inferior portion of the temporal lobe.
Studies with Laboratory Animals

In primates the recognition of visual patterns and identification of particular objects take place in the inferior temporal cortex, located on the ventral part of the temporal lobe. This region of visual association cortex is located at the end of the ventral stream. It is here that analyses of form and color are put together and perceptions of three-dimensional objects and backgrounds are achieved. The inferior temporal cortex consists of two major regions: areas TE and TEO. Damage to these regions causes severe deficits in visual discrimination (Mishkin, 1966; Gross, 1973; Dean, 1976). (See Figures 6.37 and 6.38.)

The receptive fields of neurons in area TEO are quite variable in size, but generally they are larger than those of neurons in area V4 and smaller than those of neurons in area TE (Boussauod, Desimone, and Ungerleider, 1991). Their primary inputs come from area V4, and their primary outputs go to area TE, suggesting that “the neural coding of visual objects in TEO is based on object features that are more global than those in V4, but not quite as global as those in TE” (Boussauod et al., 1991, p. 574). (As we saw, area TEQ plays a critical role in perception of color.) Lesions of TEO make it almost impossible for monkeys to learn a task that requires them to discriminate between two simple two-dimensional patterns differing in form, size, orientation, color, or brightness (Iwai and Mishkin, 1969; Gross, 1973; Dean, 1982; Ungerleider and Mishkin, 1982; Mishkin,

Analysis of Form

The analysis of form by the visual cortex begins with neurons in the striate cortex that are sensitive to orientation and spatial frequency. These neurons send information to the extrastriate cortex, which consists of several subregions. These subregions analyze the information and send it along the ventral stream toward the temporal neocortex.
Neurons in area TE have the largest receptive fields of all, often encompassing the entire contralateral half of the visual field. In general, these neurons respond best to three-dimensional objects (or photographs of them). They respond poorly to simple stimuli such as spots, lines, or sine-wave gratings. Most of them continue to respond even when these stimuli are moved to a different location, are changed in size, are placed against a different background, or are partially occluded by another object (Rolls and Baylis, 1986; Kovács, Vogels, and Orban, 1995). Thus, they appear to participate in the recognition of objects rather than the analysis of specific features.

Tanaka and his colleagues (reviewed by Tanaka, 1996) investigated the response characteristics of these neurons. First, they located a single neuron with a microelectrode. Then they presented a large number of three-dimensional items, such as toy animals, plants, and "junk" objects, until they found one that produced the best response. Then they used a computerized system to present a series of simplified versions of the picture to find the simplest pattern that would still excite the cell. Figure 6.39 illustrates this procedure. The cell responded when the tiger's head was presented and continued to respond to successively simplified patterns. The cell was activated by a pair of black rectangles superimposed on a white square but not by either of the two components of this stimulus (See Figure 6.39.)

Obviously, the fact that the cell responded to the tiger's face does not mean that it was a "tiger's face analyzer." As Tanaka observed, no single cell could recognize a complex stimulus found in nature. Instead, particular stimuli would be represented by the activity of a large group of cells, each sensitive to slightly different patterns. It is the pattern of activity in circuits of neurons in area TE that represents the perception of particular objects.

Figure 6.40 shows the responses of a cell in area TE that responded to the sight of a water bottle oriented with its spout at ten o'clock. The computer program found that the simplest stimulus that this neuron would respond to was pear-shaped. The neuron would not respond to a circle without the neck; nor would it respond to a pear-shaped object rotated away from ten o'clock. The neuron still responded if the neck was extended or retracted but did not respond at all if the neck was squared off at the end. (See Figure 6.40.)
Like other regions of the visual cortex, the inferior temporal cortex is arranged in columns. Neurons in adjacent regions usually respond to slightly different versions of the same stimuli. For example, several studies (for example, Desimone et al., 1984) have found neurons in the temporal lobe that are specifically excited by the sight of another face—either that of another monkey or that of a human. Some of these neurons respond to full-face views, and others respond to profiles. Most of these face-sensitive cells are located in area TE and in the cortex that lines the anterior bank of the superior temporal sulcus (area STS). (Refer to Figures 6.37 and 6.38.)

A study by Wang, Tanaka, and Tanifuji (1996) used an optical recording technique to study the functional organization of the inferior temporal cortex in monkeys. They placed transparent "windows" over the surface of the cortex that permitted them to monitor the surface of the brain. They did not inject a voltage-sensitive dye, as Blasdel did in his study of the striate cortex. Instead, they used a special computer-driven video camera to record changes in the appearance of the cortex caused by changes in the oxidation level of hemoglobin in the cortical capillaries—changes that correlate with neural activity. Figure 6.41 shows the response to different views of a doll's head as it rotated. As you can see from the movement of the dark spot (arrows) from frame to frame, adjacent clusters of neurons were activated by different views of the head. (See Figure 6.41.)

Clearly, neurons in the primate inferior temporal cortex respond to very complex shapes, including things the animals have already seen, such as water bottles and faces. The complexity and the specific nature of these features suggest that the development of the circuits responsible for detecting them must involve learning. Indeed, that seems to be the case. For example, several studies have found neurons in the inferior temporal cortex that respond specifically to objects that the monkeys have already seen many times but not to unfamiliar objects (Kobatake, Tanaka, and Tamori, 1992; Logothetis, Pauls, and Poggio, 1995). Such studies will be discussed in more detail in Chapter 14.

Studies with Humans

Damage to the human visual association cortex can cause a category of deficits known as visual agnosia. Agnosia ("failure to know") refers to an inability to perceive or identify a stimulus by means of a particular sensory modality, even though its details can be detected by means of that modality and the person retains relatively normal intellectual capacity. Apperceptive visual agnosias are failures in high-level perception, whereas associative visual agnosias are disconnections between these perceptions and verbal systems. The distinction will be described in more detail later in this section.

People with visual agnosia cannot identify common objects by sight, even though they have relatively normal visual acuity (Warrington and James, 1983). In some cases they can read small print but fail to recognize a common object, such as a wristwatch. However, if they are permitted to hold the object (say, the wristwatch), they can immediately recognize it by touch and say what it is. Therefore, they have not lost their memory for the object or forgotten how to say its name.

- Apperceptive Visual Agnosia. People with apperceptive visual agnosia may have normal visual acuity, but they cannot successfully recognize objects visually by their shape. For example, a brain-damaged patient studied by Benson

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visual agnosia (eg no zhe). Deficits in visual perception in the absence of blindness; caused by brain damage.

apperceptive visual agnosia. Failure to perceive objects, even though visual acuity is relatively normal.
and Greenberg (1969) was initially believed to be blind but was subsequently observed to navigate his wheelchair around the halls of the hospital. Testing revealed that his visual fields were full (there were no blind spots other than ones we all have) and that he could pick up threads placed on a sheet of white paper. He could discriminate among stimuli that differed in size, brightness, or hue but could not distinguish those that differed only in shape.

Are Faces Special? A common symptom of apperceptive visual agnosia is prosopagnosia, an inability to recognize particular faces (prosopon means “face”). That is, the patients can recognize that they are looking at a face, but they cannot say whose face it is—even if it belongs to a relative or close friend. They see eyes, ears, a nose, a mouth—but cannot recognize the particular configuration of these features that identifies an individual face. They still remember who these people are and will usually recognize them when they hear their voice. As one patient said, “I have trouble recognizing people from just faces alone. I look at their hair color, listen to their voices... I use clothing, voice, and hair. I try to associate something with a person one way or another... what they wear, how their hair is worn” (Buxbaum, Glosser, and Coslett, 1999, p. 43). Some investigators believe that facial recognition is mediated by special circuits in the brain that are devoted to the specific analysis of facial features. Others believe that the distinction between prosopagnosia and visual agnosia for common objects is quantitative, not qualitative; that is, visual agnosia for common objects is simply a more severe deficit, caused by more extensive damage to the relevant parts of the visual association cortex. They argue that distinguishing one person’s face from another is a more complex task than recognizing a common object, such as a key or a wristwatch.

The most recent evidence suggests that faces are, indeed, recognized by special circuits in the inferior temporal lobe but that these circuits are not genetically programmed as a “face-recognizing device,” but develop through experience. The earliest evidence that the perception of faces is a special process comes from studies that demonstrated the inversion effect. People can quickly learn to recognize pictures of faces they have never seen before, but they have difficulty learning to recognize pictures of faces when they are turned upside-down. In contrast, learning to recognize inverted pictures of houses is not much harder than learning to recognize such pictures when they are in their normal orientation (Yin, 1970).

Yin and others have suggested that we have special circuits devoted to the analysis of subtle differences in the configuration of eyes, eyebrows, nose, cheeks, and chin, and all the other features that distinguish one face from another. When a face is presented upside-down, these special circuits are of no use because the configuration of these features is abnormal. Studies with brain-damaged people and functional imaging studies suggest that these special face-recognizing circuits are found in the fusiform gyrus, located

prosopagnosia (prah sopp ahg noh zha) Failure to recognize particular people by the sight of their faces.

inversion effect The increased difficulty in learning to recognize visual stimuli that are normally seen in a particular orientation when they are inverted—turned upside-down.
in the inferior temporal lobe (see Haxby et al., 1999, for specific references). Furthermore, most studies indicate that the right hemisphere is more important than the left.

Several studies have shown that people with prosopagnosia do not show an inversion effect for faces (Yin, 1970; Farah et al., 1995). Their recognition of faces presented in a normal orientation is very poor, but their performance is no worse than that of normal subjects when they are shown inverted photos. These studies suggest that if damage is limited to the face-recognition region of the cortex, patients must use their general-purpose object-recognition circuits to recognize faces.

Perhaps the strongest evidence for a special face-recognition region comes from a report by Moscovitch, Winocur, and Behrmann (1997), who studied a man with a visual agnosia for objects but not for faces. For example, he recognized the face shown in Figure 6.42 but not the flowers and vegetables that compose it. (See Figure 6.42.) Furthermore, he showed a very strong inversion effect for faces, presumably because his general-purpose object-recognition circuits were damaged.

So there seems to be a special region devoted to recognition of faces. But must we conclude that the development of this region is a result of natural selection? Several kinds of evidence suggest that the answer is no—that the facial-recognition circuits develop as a result of the experience we have seeing people's faces. First, consider the fact that some neural circuits appear to be devoted to the rapid and efficient recognition of written words. Damage to one part of the brain can impair people's ability to read but not affect their ability to recognize objects, and damage to another region can impair object recognition but not reading. (This evidence is reviewed in Chapter 16, which discusses the neural mechanisms of spoken and written language.) The process of natural selection cannot possibly be responsible for the development of these circuits because the invention of written languages occurred very recently—only a few thousand years ago. In addition, until very recently, the vast majority of the world's population was illiterate, so there has not been enough time for the evolution of innate word-recognition circuits to take place. So if experience looking at words can cause the development of circuits that recognize words, perhaps experience looking at faces can cause the development of face-recognition circuits.

Because of the extensive experience we have looking at faces, we are all experts at recognizing them, and we show an inversion effect for faces. Diamond and Carey (1986) found that people who have extensive experience looking at dogs show an inversion effect for pictures of them. The investigators showed pictures of faces and dogs to college students and judges of show dogs. The college students, who had certainly seen many dogs but had not received any special training at distinguishing and rating the special features of show dogs, showed a much greater inversion effect for faces than for dogs. The judges showed a large inversion effect for both types of pictures—faces and show dogs—which suggests that their experience had led to the development of circuits that recognize subtle details in the appearance of show dogs. Another study (Gauthier and Tarr, 1997) found that when people had spent a long time becoming familiar with computer-generated objects they called "greebles," they showed an inversion effect for these stimuli. People without this experience did not—their performance was the same regardless of the greeble's orientation. (See Figure 6.43.)

**Associative Visual Agnosia** A person with apperceptive agnosia who cannot recognize common objects also cannot draw them or copy other people's drawings; therefore, we properly speak of a deficit in perception. However, people with an associative visual agnosia appear to be able to

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**associative visual agnosia** Inability to identify objects that are perceived visually, even though the form of the perceived object can be drawn or matched with similar objects.
perceive normally but cannot name what they have seen. In fact, they seem to be unaware of these perceptions. For example, a patient studied by Ratcliff and Newcombe (1982) could copy a drawing of an anchor (better than I could have done). Therefore, he could perceive the shape of the anchor. However, he could not recognize either the sample or the copy that he had just drawn. When asked on another occasion to draw (not copy) a picture of an anchor, he could not do so. Even though he could copy a real image of an anchor, the word anchor failed to produce a mental image of one. (See Figure 6.44.) When asked (on yet another occasion) to define anchor, he said, "a brake for ships," so we can conclude that he knew what the word meant.

Associative agnosia also extends to prosopagnosia. For example, Sergent and Signoret (1992) reported the case of a patient who could match photos of different views of the same face but could not identify the faces—even when they were pictures of the patient herself. The lesion seems to have affected the ability to identify faces without severely damaging perceptual analysis.

Associative visual agnosia appears to involve difficulty in transferring visual information to verbal mechanisms. That is, the person perceives the object well enough to draw it (or to match it with similar stimuli), but his or her verbal mechanisms do not receive the necessary information to produce the appropriate word. David Margolin and I studied a man who had sustained brain damage from an inflammatory disease that affected his cerebral blood vessels. (The damage was diffuse, so we could not make any conclusions about the anatomy of his disorder.) Suffering from an apparent visual agnosia, he failed to identify most pictures of objects. However, he sometimes made unintentional gestures when he was studying a picture that gave him enough of a clue that he could identify it. For example, on one occasion while puzzling over a picture of a cow, he started making movements with both hands that were unmistakably ones he would make if he were milking a cow. He looked at his hands and said, "Oh, a cow!" (He was a farmer, by the way.)

We might speculate that his perceptual mechanisms, in the visual association cortex, were relatively normal but that connections between these mechanisms and the speech mechanisms of the left hemisphere were disrupted. However, the connections between the perceptual mechanisms and the motor mechanisms of the frontal lobe were spared, permitting him to make appropriate movements when looking at some pictures. In fact, a particularly observant and conscientious speech therapist helped the pa-
tient learn how to read by these means. She taught him the manual alphabet used by deaf people, in which letters are represented by particular hand and finger movements. (This system is commonly called *finger spelling.*) He could then look at individual letters of words he could not read, make the appropriate movements, observe the sequence of letters that he spelled, and decode the word.

Recent studies suggest that associative visual agnosia is best explained as a disruption of connections between the ventral stream of the visual cortex and the brain's verbal mechanisms without damage to the connections between these mechanisms and the dorsal stream. I will say more about these studies in the next subsection.

**Perception of Movement**

We need not only to know what things are, but also where they are and where they are going. Without the ability to perceive the direction and velocity of movement of objects, we would have no way to predict where they will be. We would be unable to catch them (or avoid letting them catch us). This section examines the perception of movement; the final section examines the perception of location.

**Studies with Laboratory Animals**

One of the regions of the extrastriate cortex—area V5, also known as area MT, for *medial temporal*—contains neurons that respond to movement. Damage to this region severely disrupts a monkey's ability to perceive moving stimuli (Siegel and Andersen, 1986). Area V3 receives input directly from the striate cortex and from several regions of the extrastriate cortex. It also receives input from the superior colliculus. (Refer to Figures 6.37 and 6.38.)

Accurately determining the velocity and direction of movement of an object is an important ability. That moving object could be a prey animal trying to run away, a predator trying to catch you, or a projectile you are trying to catch (or keep from hitting you). If we are to accurately track moving objects, the information received by V5 must be up-to-date. In fact, the axons that transmit information from the magnocellular system are thick and heavily myelinated, which increases the rate at which they conduct action potentials. Petersen, Miezin, and Allman (1988) recorded the responses of neurons in areas V4 and V5. As you can see in Figure 6.45, visual information reached the V5 neurons sooner than it reached those in area V4, whose neurons are involved in the analysis of form and color. (See Figure 6.45.)

The input from the superior colliculus contributes in some way to the movement sensitivity of neurons in area V5. Rodman, Gross, and Albright (1989, 1990) found that destruction of the striate cortex or the superior colliculus alone does not eliminate the movement sensitivity of V5 neurons, but destruction of both areas does. The roles played by these two sources of input are not yet known. Clearly, both inputs provide useful information; Seagraves et al. (1987) found that monkeys still could detect movement after lesions of the striate cortex but had difficulty estimating its rate.

Albright, Desimone, and Gross (1984) mapped the characteristics of movement-sensitive neurons in area V5. They found that all V5 neurons responded better to moving stimuli than to stationary ones and that most of them gave the same response regardless of the color or shape of the test stimulus. Most neurons showed directional sensitivity; that is, they responded only to movements in a particular direction. They also found that, like the striate cortex, area V5 is divided into rectangular modules. Traveling along the long axis of a module, they encountered neurons with directional sensitivities that varied systematically, in a clockwise or counterclockwise fashion. The receptive fields of movement-sensitive neurons in area V5 are elongated, with most neurons showing movement sensitivity in a direction at right angles to the long axis. A large antagonistic surround shows sensitivity to movement in the opposite direction (Raguel et al., 1995).

A region adjacent to area V5 (sometimes called V5a but more often referred to as MST, for *medial superior temporal*)
receives information about movement from V5 and performs a further analysis. MST neurons respond to complex patterns of movement, including radial, circular, and spiral motion (see Vaina, 1998, for a review). One important function of this region—in particular, the dorsolateral MST, or MSTD—appears to be analysis of optic flow. As we move around in our environment or as objects in our environment move in relation to us, the sizes, shapes, and locations of environmental features on our retinas change. Imagine the image seen by a video camera as you walk along a street, pointing the lens of the camera straight in front of you. Suppose your path will pass just to the right of a mailbox. The image of the mailbox will slowly get larger. Finally, as you pass it, it will veer to the left and disappear. Points on the sidewalk will move downward, and branches of trees that you pass under will move upward. Analysis of the relative movement of the visual elements of your environment—the optic flow—will tell you where you are heading, how fast you are approaching different items in front of you, and whether you will pass to the left or right (or under or over) these items. If the retinal image of an item does not move, but simply looms up in front of you, you will bump into it. Similarly, we use optic flow to determine whether an object approaching us will hit us or pass us by.)

Rizzolatti and his colleagues (Gallese et al., 1996; Rizzolatti et al., 1996) recorded from the frontal cortex of monkeys and found a group of neurons with particularly interesting response characteristics. These neurons, found in the rostral part of the ventral premotor cortex, responded when the monkeys either saw or performed various grasping, holding, or manipulating movements. For example, one of these neurons might respond when the monkey saw an experimenter pick up a piece of food from a tray. It would not respond to the sight of the experimenter’s hand alone, to the sight of the tray alone, or to the sight of the experimenter picking up the piece of food with a pair of pliers. The same neuron would also respond when the monkey picked up a piece of food from the tray, whether it did so when the lights were on and its movement was guided by vision or when the lights were off so it had to pick up the piece of food in the dark. The investigators called the cells mirror neurons because they responded to a particular visual stimulus or to the movement that produces this stimulus. Presumably, they are involved in a monkey’s ability to recognize and imitate gestures made by other monkeys.

**Studies with Humans**

Bilateral damage to parts of the visual association cortex of the human brain can produce an agnosia for movement. For example, Zihli et al. (1991) reported the case of a woman with bilateral lesions of the lateral occipital cortex and area V5. The woman (Patient L.M.) had an almost total loss of movement perception. She was unable to cross a street without traffic lights, because she could not judge the speed at which cars were moving. Although she could perceive movements, she found moving objects very unpleasant to look at. For example, while talking with another person, she avoided looking at the person’s mouth because she found its movements very disturbing. When the investigators asked her to try to detect movements of a visual target in the laboratory, she said, “I first the target is completely at rest. Then it suddenly jumps upwards and downwards” (p. 2244). She was able to see that the target was constantly changing its position, but she was unaware of any sensation of movement.

As we saw in the previous subsection, area V5 is the one region of the monkey brain that is most important for perception of movement. Several PET and functional MRI studies suggest that the region of the human brain that performs this function is located near the junction of the lateral occipital and temporal lobes. For example, Malach et al. (1995) showed people two types of stimuli: pictures of objects, faces, and textures, and moving random patterns of dots. As Figure 6.46 shows, these stimuli activated different regions of extrastriate cortex. The red region was activated by objects, faces, and textures, and the green region (presumably corresponding to area V5) was activated by movement. (See Figure 6.46.)

Walsh et al. (1998) used a procedure known as transcranial magnetic stimulation (TMS) to temporarily inactivate area V5 in normal human subjects. The TMS procedure applies a strong localized magnetic field into the brain by passing an electrical current through a coil of wire placed on the scalp. The magnetic field induces a weak electrical current in the brain that temporarily disrupts normal neural activity. The investigators found that during the stimulation, people were unable to detect which of several objects displayed on a computer screen was moving. When the current was off, the subjects had no trouble detecting the motion. The current had no effect on the subject’s ability to detect stimuli that varied in their form.

Perception of movement can even help us perceive three-dimensional forms—a phenomenon known as structure from motion. Johansson (1973) demonstrated just how much information we can derive from movement. He dressed actors in black and attached small lights to several points on their bodies, such as their wrists, el-

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**optic flow** The complex motion of points in the visual field caused by relative movement between the observer and environment; provides information about the relative distance of objects from the observer and of the relative direction of movement.
circular optic flow—but could not perceive structure from motion. Thus, perception of motion and perception of structure from motion involve different regions of the visual association cortex.

Perception of structure from motion may not seem like a phenomenon that has any importance outside the laboratory. However, this phenomenon does occur under natural circumstances, and it appears to involve brain mechanisms different from those involved in normal object perception. For example, people with visual agnosia can still perceive actions (such as someone pretending to stir something in a bowl or deal out some playing cards) even though they cannot recognize objects by sight. They may be able to recognize friends by the way they walk, even though they cannot recognize their faces. Exactly what brain regions must be spared for perception of form from motion is not yet known.

So far, this discussion has been confined to movement of objects in the visual field. But if a person moves his or her eyes, head, or whole body, the image on the retina will move even if everything within the person's visual field remains stable. Often, of course, both kinds of movements will occur at the same time. The problem for the visual system is to determine which of these images are produced by movements of objects in the environment and which are produced by the person's own eye, head, and body movements.

To illustrate this problem, think about how the page of this book looks as you read it. If we could make a videotape of one of your retinas, we would see that the image of the page projected there is in constant movement as your eyes make several saccades along a line and then snap back to the beginning of the next line. Yet the page seems perfectly still to you. On the other hand, if you look at a single point on the page (say, a period at the end of a sentence) and then move the page around while following it with your eyes, you perceive the book as moving, even though the image on your retina remains relatively stable. (Try it.) And then think about the images on your retina while you are driving in busy traffic, constantly moving your eyes around to keep track of your own location and that of other cars moving in different directions at different speeds. And you are perceiving not only the simple movement of objects, but optic flow as well, which helps you keep track of the trajectories of the objects relative to each other and to yourself.

McCleod et al. (1996) suggest that the ability to perceive structure from motion does not involve area V5. They reported that Patient L.M. (studied by Zihl et al., 1991) could recognize people depicted solely by moving points of light even though she could not perceive the movements themselves. Vaina and his colleagues (reported by Vaina, 1998) found a patient with a lesion in the medial right occipital lobe who showed just the opposite deficits: Patient R.A. could perceive movement—even complex radial and
eye movements, any movement of a retinal image was perceived as movement of the environment.

As we saw in the previous subsection, mirror neurons in the ventral premotor area of the monkey brain respond when the monkey either sees or performs a particular action. Functional imaging studies suggest that a region containing mirror neurons is also found in the human prefrontal cortex. Rizzolatti and Arbib (1998) suggest that these neurons might be involved in recognizing and imitating other people’s gestures—including those made by deaf people when they communicate by sign language.

### Perception of Spatial Location

As we just saw, all subareas of the extrastriate cortex send information to the inferior temporal cortex, the region in which object perception appears to take place. In addition, three subareas of the extrastriate cortex—those involved with color, orientation, and movement—send information through area V5 to the parietal cortex. (Refer to Figures 6.37 and 6.38.) The parietal lobe is involved in spatial perception, and it is through these connections that it receives its visual input. Damage to the parietal lobes disrupts performance on a variety of tasks that require perceiving and remembering the locations of objects (Ungerleider and Mishkin, 1982).

Haxby et al. (1994) had human subjects perform two different discrimination tasks: one for form (human faces) and the other for spatial location (human faces and random patterns). In both cases the subjects saw a display showing a face or a pattern, followed by a second display. In the form discrimination task the subjects had to ignore the location of the faces but decide whether the second display contained the same face as the first. In the spatial location task they had to ignore the nature of the forms but decide whether the form shown in the second display was in the same location as the one shown in the first display. While the subjects were performing the discrimination tasks, the investigators used a PET scanner to record their regional cerebral blood flow. They found that performance of both tasks increased the metabolic activity of much of the extrastriate cortex. However, only the form discrimination task activated the ventral stream and only the location discrimination task activated the dorsal stream. (See Figure 6.47.)

A functional imaging study by Mellet et al. (1996) showed that the dorsal stream is involved in the construction of mental images of three-dimensional objects according to verbal instructions. The investigators asked people to imagine an assembly of cube-shaped blocks, put together one by one. For example, the assembly in Figure 6.48 begins with the block shown in blue. The second block goes to the right of the first, the third goes below the second, the fourth goes below the third, and so on. (See Figure 6.48a.) Functional MRI images that were taken while the subjects were constructing the mental images of these objects found increased activity in a bilateral occipitoparietal-frontal network that included the superior extrastriate cortex of the occipital lobe, inferior parietal cortex, and dorsal premotor cortex of the frontal lobe. Activity was also seen in the right inferior temporal cortex. (See Figure 6.48b.) Thus, imagining the construction of a three-dimensional assembly involves the dorsal stream (where spatial perception takes place) and the frontal lobes (where planning of movements takes place). The involvement of the ventral stream of the right hemisphere may reflect the people’s recognition of the imaginary shape they had constructed.

A particularly interesting phenomenon called Balint’s syndrome occurs in people with bilateral damage to the parieto-occipital region—the region bordering the parietal lobe and occipital lobe (Balint, 1909; Damasio, 1985). Balint’s syndrome consists of three major symptoms: optic ataxia, ocular apraxia, and simultanagnosia. All three symptoms are related to spatial perception.

Optic ataxia is a deficit in reaching for objects under visual guidance (ataxia comes from the Greek word for “disorderly”). A person with Balint’s syndrome might be
Analysis of Visual Information: Role of the Association Cortex

![Diagram of cube with directional labels: Up, Down, Left, Right, Front, Back]

Figure 6.48

Construction of a mental image. Subjects imagined the construction of an assembly of cubes as the experimenter indicated the location of each new block. (a) An assembly produced by the following directions: right, down, down, back, back, back, up, up, back, back, right. (b) Neural activity during the imaginary construction task, as measured by functional MRI.


able to perceive and recognize a particular object, but when he or she tries to reach for it, the movement is often misdirected. Ocular apraxia (literally "without visual action") is a deficit of visual scanning. If a person with Balint's syndrome looks around a room filled with objects, he or she will see an occasional item and will be able to perceive it normally. However, the patient will not be able to maintain fixation; his or her eyes will begin to wander, and another object will come into view for a time. The person is unable to make a systematic scan of the contents of the room and will not be able to perceive the location of the objects he or she sees. If an object moves or if a light flashes, the person may report seeing something but will not be able to make an eye movement that directs the gaze toward the target.

Simultanagnosia is the most interesting of the three symptoms (Rizzo and Robin, 1990). As I just mentioned, if the gaze of a person with Balint's syndrome happens to fall on an object, he or she will perceive it. But only one object will be perceived at a time. For example, if an examiner holds either a comb or a pen in front of a patient's eyes, the patient will recognize the object. But if the examiner holds a pen and a comb together (for example, so that they form the legs of an X), the patient will see either the comb or the pen but not both. The existence of simultanagnosia means that perception of separate objects takes place at least somewhat independently, even when the outlines of the objects overlap in the visual field.

Goodale and his colleagues (Goodale and Milner, 1992; Goodale et al., 1994) suggested that the primary function of the dorsal stream of the visual cortex is to guide actions rather than simply to perceive spatial locations. As Ungerleider and Mishkin (1982) originally put it, the ventral and dorsal streams tell us "what" and "where." Goodale and his colleagues suggested that the better terms are "what" and "how." First, they noted that the visual cortex of the parietal lobe is extensively connected to regions of the frontal lobe involved in controlling eye movements, reaching movements of the limbs, and grasping movements of the hands and fingers (Cavada and Goldman-Rakic, 1989; Gentilucci and Rizzolatti, 1990; Broussaud, di Pellegrino, and Wise, 1996). Second, they noted that optic ataxia and ocular apraxia of Balint's syndrome, which are caused by bilateral damage to the dorsal stream, are deficits in visually guided movements. They cited the case of a person with such lesions who had no difficulty recognizing line drawings (that is, the ventral stream was intact) but who had trouble picking up objects (Jakobson et al., 1991). The patient could easily perceive the difference in size of wooden blocks set out before her, but she failed to adjust the distance between her thumb and forefinger to the size of the block she was about to pick up. In contrast, a patient with profound

<table>
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<th>Ocular apraxia (kə prak'si)</th>
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<td>Simultanagnosia (sī mə lā nə ˈgō nə zə)</td>
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visual agnosia could not distinguish between wooden blocks of different sizes but could adjust the distance between her thumb and forefinger when she picked them up. She made this adjustment by means of vision, before she actually touched them (Milner et al., 1991; Goodale et al., 1994).

The suggestion by Goodale and his colleagues seems a reasonable one. Of course, the dorsal stream is involved in perception of the location of object's space—but then, if its primary role is to direct movements, it must be involved in location of these objects, or else how could it direct movements toward them? In addition, it must contain information about the size and shape of objects, or else how could it control the distance between thumb and forefinger?

I mentioned earlier that I would attempt to explain associative visual agnosia as a disruption of the connections between the ventral stream and the brain's verbal mechanisms. As we saw, people with associative agnosia cannot verbally identify visually presented objects or pictures of them, but they can copy them and sometimes they can make hand movements that enable them to guess what the object is. Sirigu, Duhamel, and Ponec (1991) reported the case of a patient with bilateral lesions of the anterior temporal cortex who was able to copy drawings of objects but was unable to name them. However, he was able to say or demonstrate what to do with these objects. For example, he said, “You open on one side, stick something on it, close it, and it stays in. I can tell you how it works, but I don't see its exact use” (p. 2555). And what had the investigators shown him? A safety pin. When they showed him a picture of a jackhammer, he acted as if he were holding one, and made striking movements. What was it for? “Probably to make holes ... in the wall ... when you want to hang a picture” (p. 2566).

It is important to realize that the patient recognized what to do with objects he saw, not what they were used for. He was able to describe or mime behaviors, not functions. Certainly, one would not use a jackhammer to hang a picture on the wall. Consider what he said when shown a pair of pliers: “It is used manually, when you pull apart here [points to handle] it opens up at the other end.” So far, so good. But then he went on to say, “Perhaps to hold several pieces of paper together” (p. 2566). When shown an iron, he said “You hold it in one hand, and move it back and forth horizontally.” He then mimed the action, as if he were pressing some clothes on an ironing board. “Maybe you can spread glue evenly with it” (p. 2566).

Even though the patient could not identify most objects visually, he accurately answered questions about their physical properties, such as “Which one would feel the heaviest?”, “Which one is the softest?”, or “Which one would feel the coldest?” The fact that he could answer these questions (and could mime what to do with them) indicates that the circuits responsible for visual form perception (those in the ventral stream) were relatively intact but that they were no longer connected to the circuits responsible for speech (and for consciousness). His dorsal stream and its connections with speech mechanisms were undamaged, and it was apparently through these connections that he was able to describe how to use the objects. This interpretation is consistent with Goodale and Milner’s conclusion that the dorsal stream is primarily occupied with controlling movements, not simply perceiving the location of objects.

The visual cortex consists of the striate cortex, the extrastriate cortex (also called the prestriate or circumsstriate cortex), and the visual association cortex of the inferior temporal lobe and the posterior parietal lobe. There are at least twenty-five different subregions of the visual cortex, arranged in a hierarchical fashion. The extrastriate cortex receives information from the striate cortex and from the superior colliculus. The color-sensitive cells in the CO blobs in the striate cortex send information to areas V4 and V8 of the extrastriate cortex. Damage to the area V4 abolishes color constancy (accurate perception of color under different lighting conditions), and damage to area V8 causes achromatopsia, a loss of color vision.

The visual cortex is organized into two streams. The ventral stream, which ends with the inferior temporal cortex, is involved with perception of objects. Lesions of this region disrupt visual object perception. Also, single neurons in the inferior temporal cortex respond best to complex stimuli and continue to do so even if the object is moved to a different location, changed in size, placed against a different background, or partially hidden. The dorsal stream, which ends with the posterior parietal cortex, is involved with perception of location, movement, and control of eye and hand movements. Damage to area V5 (also called area MT) disrupts an animal's ability to perceive movement, and damage to the posterior parietal cortex disrupts perception of the spatial location of objects. Area MSTd, a region of extrastriate cortex that receives information from area V5, appears to be specialized for perceiving optic flow, one of the cues we use to perceive the direction in which we are heading. The visual association cortex receives information about eye movements from the motor system and information about movement of retinal images from the visual cortex and determines which movements are caused by eye
movements and which are caused by movements in the environment. At least one patient with extrastriate damage is unable to compensate for eye movements; when his eyes move, he perceives movement in the environment.

PET studies indicate that specific regions of the cortex are involved in perception of form, movement, and color, and these studies are enabling us to discover the correspondences between the anatomy of the human visual system and that of laboratory animals. Studies with humans who have sustained damage to the visual association cortex have discovered two basic forms of visual agnosia. Apperceptive visual agnosia involves difficulty in perceiving the shapes of objects, even though the fine details can often be detected. Prosopagnosia—failure to recognize faces—appears to be caused by damage to a particular region of the right inferior temporal cortex. The development of this region appears to be a result of extensive experience looking at faces; expertise with other complex stimuli causes the development of circuits devoted to the perception of these stimuli as well. The second basic form of visual agnosia, associative visual agnosia, is characterized by relatively good object perception (shown by the fact that the patients can copy drawings of objects) but the inability to recognize what is perceived. This disorder is probably caused by damage to axons that connect the visual association cortex with regions of the brain that are important for verbalization and thinking in words. Some patients with this disorder can describe or mime actions appropriate to the objects they see but cannot recognize.

Damage to the human visual association cortex corresponding to area V5 disrupts perception of movement, and transcranial magnetic stimulation of this area causes a temporary disruption. Sometimes people with visual agnosia caused by damage to the ventral system can still perceive the meanings of actions or recognize friends by the way they walk, which indicates that the dorsal stream of their visual cortex is largely intact. Mirror cells in the ventral premotor cortex become active when a monkey sees a particular limb movement or makes the movement itself. This region (which appears to exist in humans as well) may play a role in learning to imitate the movement of others. Balint's syndrome, which is caused by bilateral damage to the parieto-occipital region (the dorsal stream), includes the symptoms of optic ataxia, ocular apraxia, and simultanagnosia.

suggested readings


suggested websites

Retina Reference
http://retina.anatomy.upenn.edu/~lance/retina/retina.html
The anatomy of the retina is the focus of this site. The site contains some marvelous images and diagrams of the retina and visual system that would be useful in lectures on vision.

Perception: An Introduction to the Gestalt Theoriat by Kurt Koffka (1922)
This site contains a translation of portions of a book by Kurt Koffka (1922) which outlines the general Gestalt view of perception.

Tutorials in Sensation and Perception
http://psych.hanover.edu/krantz/sen_tut.html
This perception site contains tutorials and demonstrations on visual perception including visual aftereffects, motion illusions, and receptive fields.

Blindsight Demonstration
http://serendip.brynmawr.edu/ bk/blindsight.html
This site provides an online demonstration of the phenomenon known as blindsight.