Patient P.T.'s case was presented in Portland, Oregon, at the Neurology Grand Rounds, a weekly event at which staff neurologists, internists, and residents gather to review the most puzzling and unusual cases being treated on the ward. The cause of P.T.'s existing neurological problems was not a mystery; he had suffered a left-hemisphere stroke 6 years previously. The physician was confident in his diagnosis that P.T. had suffered a second stroke, especially because multiple strokes are not uncommon. P.T. was referred to the hospital, and the current stroke was confirmed by computed tomography (CT) to be in the right hemisphere.

What was unusual about P.T. was the collection of symptoms that he continued to experience 4 months after the second stroke. The dizziness had ended by the second day after the incident, and the left-sided weakness had mostly subsided during the first month. The only remaining sign of these initial symptoms was that P.T. continued to drag his left leg slightly, although he was not aware of it. Yet P.T. experienced some difficulties as he tried to resume the daily routines required on his small family farm, his home of 66 years.

P.T. had particular difficulty recognizing places and objects. While working on a stretch of fence, for example, he might look out over the hills and suddenly realize that he did not know the landscape. It was hard for him to pick out individual dairy cows—a matter of concern lest he attempt to milk a bull! And, most troubling of all, P.T. no longer recognized the people around him, including his wife. The woman whom he had known for so many years and with whom he had spent all his days was completely unrecognizable to him. He had no trouble seeing her and could accurately describe her actions, but in simply looking at her, he failed to identify her as his wife. He knew that her parts—body, legs, arms, and head—formed a person, but P.T. failed to see these parts as belonging to a specific individual. This deficit was not limited to P.T.'s wife; he had the same problem with other members of his family and friends from his small town.

A striking feature of P.T.'s impairment was that his inability to recognize objects and people was limited to the visual modality. Interestingly, when his wife spoke, he immediately recognized her voice. Indeed, he claimed that, on hearing her voice, the visual percept of her would “fall into place.” The shape in front of him would suddenly morph into his wife. In a similar fashion, he could recognize specific objects by touching, smelling, or tasting them.

Perception is a remarkable human ability, normally involving the integration of each of the five senses: vision, audition, touch, smell, and taste. P.T. lost only a relatively small aspect of his visual perception—an issue we return to later in the chapter—and he was able to compensate for this loss by utilizing his other senses. Similarly, a person who is blind is able to compensate to some degree for this deficit, becoming exquisitely sensitive to sound and touch while navigating about the world.
In normal perception, the interplay of all of the senses is critical. Driving a car down a busy highway in an effective and safe manner requires the successful integration of vision, touch, hearing, and perhaps even smell (warning, for example, if the emergency brake has been left on). We cannot enjoy food intensely without smell. Even visual cues enhance our gustatory experience—a perfectly browned hamburger on a tan-colored bun is much more enticing than a hamburger that is artificially colored green.

In this chapter we begin with a description of what is known about the anatomy and function of the individual senses. Then we return to the issue of how information from our different sensory systems is integrated to produce a coherent representation of the world.

**AUDITORY PERCEPTION**

Knock knock. Beep beep. Ring ring. The sense of hearing, or *audition*, plays an important role in our daily lives and is essential for everything from communication to survival. But how does the brain process sound? What happens as sound waves enter the ear? How does our brain interpret those waves?

**Neural Pathways of Audition**

An overview of the auditory pathways is presented in Figure 5.1. The complex structures of the inner ear provide the mechanisms for transforming sounds (variations in sound pressure) into neural signals. Sound waves arriving at the ear make the eardrum vibrate. These vibrations produce tiny waves in the inner ear's fluid that stimulate tiny *hair cells* located along the surface of the *basilar membrane*. Hair cells are primary auditory receptors. Oscillations of the basilar membrane prompt hair cells to generate action potentials. In this way, a mechanical signal, the fluid oscillations, is converted into a neural signal, the output from hair cells. The basilar membrane and hair cells are located within a

![Image](image-url)
The Cochlear Implant

P.M. became deaf after contracting pneumococcal meningitis when she was 13 months old. She lost the ability to transform sound waves into meaningful neuronal signals that her brain could interpret. At the age of 3 years, she received a cochlear implant. Now 18, P.M. has little difficulty conversing with people and even uses the telephone to talk with her friends.

The cochlear implant is one of the great breakthroughs in biomedical engineering, having partially restored hearing to thousands of individuals. A cochlear implant is not like a traditional hearing aid that amplifies sound. The implant is actually designed to perform the job of the cochlea: to transform sound waves into neuronal impulses that the brain will interpret as sound.

The figure shows the components of the cochlear implant. The sound signal enters the device through a microphone worn over the ear and is then analyzed by a microcomputer called the speech processor. The speech processor converts the information into radio waves that are sent to a receiver. The signal is then relayed by a small wire to the cochlea, where the electrodes artificially stimulate the auditory nerve. The brain, of course, doesn’t know that the cochlea has been artificially stimulated. It processes the information from the auditory nerve just as it would information relayed from a healthy cochlea.

P.M. has this to say about her cochlear implant: “It changed my life dramatically, in such a beautiful way, that I went from being profoundly deaf to being an auditory learner, a social being, and an independent young woman” (personal communication, April 14, 2005).

The cochlear implant mimics natural hearing by activating the auditory nerve artificially via electrical stimulation of the cochlea.

Even at this early stage of the auditory system, information about the sound source can be discerned. Hair cells have receptive fields that code for sound frequency.
Human auditory sensitivity ranges from a low of about 20 Hz to a high of 20,000 Hz, but it is best tuned for 1000 to 4000 Hz, a range that carries much of the information critical for human communication, such as speech or the cries of a hungry infant. Hair cells at the thick end, or base, of the cochlea are activated by high-frequency sounds; cells at the opposite end, or apex, are activated by low-frequency sounds. These receptive fields overlap extensively. Further, natural sounds like music or speech are made up of complex frequencies; thus, sounds activate a broad range of hair cells.

The auditory system contains important subcortical relays. Output from the cochlea is projected to two midbrain structures: the cochlear nucleus and the inferior colliculus. From there, information is sent to the medial geniculate nucleus (MGN) in the thalamus. As with all other senses, except for olfaction, the thalamus serves as a relay station, taking signals from the periphery and passing the information to the primary sensory cortex. For audition, the thalamic outputs project to the primary auditory cortex (A1) in the superior part of the temporal lobe.

Neurons throughout the auditory pathway continue to have frequency tuning. As Figure 5.2 shows, the tuning curves for auditory cells can be quite broad. The fact that individual cells do not give precise frequency information but provide coarse coding indicates that our perception must depend on the integrated activity of many neurons. Such integration is probably facilitated by tonotopic maps found in most auditory areas; such maps show an orderly correspondence between the location of the neurons and their specific frequency tuning. For example, cells in one region of an auditory area will respond to low-frequency stimuli; cells in another region will respond to middle or high frequencies. The tonotopic organization can be seen in humans with the resolution provided by MRI (Figure 5.3). As described in Chapter 4, topographic maps are a defining feature of many cortical areas for the different senses.

Electrophysiological studies of the cat revealed a second general principle of auditory processing. The tuning specificity of auditory receptive fields becomes more refined as the stimulus proceeds through the system. A neuron in the cochlear nucleus that responds maximally to a pure tone of 5000 Hz may also respond to tones ranging from 2000 to 10,000 Hz. A comparable neuron in the auditory cortex is likely to respond over a much narrower range.

**Computational Goals in Audition**

Frequency data are essential for deciphering a sound. Sound-producing objects have unique resonant properties that provide a characteristic signature. The same note played on a clarinet and a trumpet will sound differently even though they share the same base frequency. The resonant properties of each instrument will produce great differences in the note's harmonic structure. In a similar way, we produce our range of speech sounds by varying the resonant properties of the vocal tract. Movements of our lips, tongue, and jaw change the frequency content of the acoustic stream produced during speech. Frequency variation is essential for a listener to identify words or music.

Auditory perception does not merely identify the content of an acoustic stimulus. A second important function of audition is to localize sounds in space. Consider the bat, which hunts by echolocation. High-pitched sounds are emitted by the bat and bounce back, as echoes from the environment. From these echoes, the bat's brain creates an auditory image of the environment and the objects within it—preferably a tasty moth. But knowing that a moth (“what”) is present will not lead to a successful hunt. The bat also has to determine the moth’s precise location (“where”). The cognitive neuroscience of audition has focused primarily on this “where” problem. In solving the “where” problem, the...
auditory system relies on integrating information from the two ears.

**Multiple Cues for Sound Localization**

In developing animal models to study auditory perception, we select animals with well-developed hearing. A favorite species for this work has been the barn owl, a nocturnal creature. Barn owls have excellent *scotopia* (night vision), which guides them to their prey. But barn owls also must utilize an exquisitely tuned sense of hearing to locate food because visual information can be unreliable at night. The low levels of illumination provided by the moon fluctuate over the lunar cycle; this and stellar sources can be obscured by dense cloud cover. Sound, such as the patter of a mouse scurrying across a field, offers a more reliable stimulus. Indeed, barn owls have little trouble finding prey in a completely dark laboratory.

Barn owls rely on two cues to localize sounds: the difference in when a sound reaches each of the two ears, the *interaural time*, and the difference in the sound’s intensity at the two ears. Both cues result from the fact that the sound reaching two ears is not identical. Unless the sound source is directly parallel to the head’s orientation, the sound will reach one ear before the other. Moreover, because the intensity of a sound wave becomes attenuated over time, the magnitude of the signal at the two ears will not be identical. The time and intensity differences are minuscule. For example, if the stimulus is located at a 45° angle to the line of sight, the interaural time difference will be approximately 1/10,000 of a second. The intensity differences resulting from sound attenuation are even smaller—indistinguishable from variations due to noise. However, these small differences are amplified by a unique asymmetry of owl anatomy: The left ear is higher than eye level and points downward, and the right ear is lower than eye level and points upward. Because of this asymmetry, sounds coming from below are louder in the left ear than the right. Humans do not have this asymmetry, but the complex structure of the human outer ear, or *pinna*, may amplify the intensity difference between a sound at the two ears.

Interaural time and intensity differences provide independent cues for sound localization. To show this, stimuli are presented over headphones and the owl is trained to turn its head in the sound’s perceived direction. The headphones allow the experimenter to manipulate each cue separately. When amplitude is held constant, asynchronies in presentation times prompt the owl to shift its head in the horizontal plane. Variations in amplitude produce vertical head movements. Combining the two cues by fusing the inputs from the two ears provides the owl with a complete representation of three-dimensional space. If one ear is plugged, the owl’s response indicates that a sound has been detected, but it cannot localize the source.

These two cues are processed by independent neural pathways. The auditory nerve synapses on the cochlear nucleus. Each cochlear nucleus is composed of two parts: the magnocellular nucleus and the angular nucleus (Figure 5.4). The auditory nerve fibers innervating the cochlear nucleus split, sending one axonal branch to the magnocellular nucleus and a second branch to the angular nucleus. This parallelism is maintained in the ascending projections to the midbrain lemniscal nuclei. The magnocellular and angular nuclei...
Figure 5.4 Parallel pathways in the auditory system of the barn owl. The cochlear nucleus is composed of two parts: the magnocellular nucleus and the angular nucleus. The magnocellular pathway is specialized to compute interaural time differences—information essential for locating the lateral position of a stimulus. The angular pathway is specialized to compute intensity differences—information essential for locating the distance to a stimulus.

Mark Konishi of the California Institute of Technology has provided a well-specified neural model of how the owl brain codes interaural time and intensity differences. For detecting time differences, Konishi assumed that the first site of convergence from the two ears is in the anterior lemniscal neurons. These neurons operate as coincidence detectors. To be activated, an anterior lemniscal neuron must simultaneously receive input from each ear. In computer science terms, these neurons act as AND operators. An input from either ear alone or in succession is not sufficient; the neurons will fire only if an input is received at the same time from both ears. Simultaneous activation from a sound source is restricted to only some of these neurons because the magnocellular axons from each half of the brain converge from opposite directions on the coincidence detectors.

Figure 5.5 explains how this model works. In Figure 5.5a, the sound source is directly in front of the animal. In this situation the coincidence detector in the middle is activated because the stimulus is received at each ear at the same time. In Figure 5.5b, the sound source is to the animal’s left, which gives the magnocellular axon from the left ear a slight head start. Simultaneous activation now occurs in a coincidence detector to the left of center. This simple arrangement provides the owl with a complete representation of the sound source’s horizontal position. Physiological studies have confirmed that neurons of the anterior lemniscal nucleus act as coincidence detectors.

A different coding scheme represents interaural intensities. The posterior region of the lemniscal nucleus is again the first point of convergence. But the neural code for intensity is based on the input’s firing rate. The stronger the input signal, the more strongly the cell fires. Neurons in the posterior region use combined signals from both ears to pinpoint the vertical position of the source.

Localization is incomplete at the level of the lemniscal nucleus; horizontal and vertical positions must be combined. Outputs from each region of the lemniscal nucleus converge on another brainstem nucleus, the external nucleus. Neurons within this nucleus are considered space specific. They are activated only when sound comes from a certain location.

Konishi provided an elegant account of how the barn owl can pinpoint a sound in three-dimensional space. The theory stands as an excellent example of the
power of cognitive neuroscience. We know from an owl’s behavior that it can localize sounds precisely. As we shall see later in this chapter, the neural representation of space is more straightforward in vision. The sensory detectors in the retina form a topographic map, and this representation is maintained throughout the processing system. In audition, though, the sensors do not code spatial information in a direct way. The location of a sound source must be computed by integration of the signals received from two ears. Anatomy and physiology led Konishi to realize that temporal information and intensity information are processed independently. Computational models of coincidence detectors and neural summation then guided the search for the derivation of location information.

In Konishi’s model, the problem of sound localization by the barn owl is solved at the level of the brainstem. To date, this theory has not explained higher stages of processing, such as in the auditory cortex. Perhaps cortical processing is essential for converting location information into action. The owl does not want to simply attack every sound; it must decide if the sound is generated by potential prey. Another way of thinking about this is to reconsider the issues surrounding the computational goals of audition. Konishi’s brainstem system provides the owl with a way to solve “where” problems but has not addressed the “what” question. The owl needs a more detailed analysis of the sound frequencies to determine whether a stimulus results from the movement of a mouse or a deer.

OLFACTORY PERCEPTION

Sight, sound, taste, and touch are the senses of which we have the greatest awareness. Yet the more primitive sense of smell is, in many ways, equally essential for our survival. Although the baleen whale probably does not smell the tons of plankton it ingests, the sense of smell is essential for terrestrial mammals, helping in the recognition of foods that are nutritious and safe. Observing the interactions of dogs romping about the beach provides a quick reminder of the importance of smell for mammals. Olfaction may have evolved as a mechanism for evaluating whether a potential food is edible, but it now serves other important roles as well—for instance, in social functions. Before discussing the functions of olfaction, let’s review the neural pathways of the brain that respond to odors.
Neural Pathways of Olfaction

The olfactory pathway to the brain is unique: The receptors are directly exposed to the outside world, and the olfactory nerve arrives at the primary olfactory cortex without going through the thalamus.

Figure 5.6 details the olfactory pathway. First, odor molecules, called odorants, enter the nasal cavity. Odorants can enter the nasal cavity in various ways. They flow in during normal breathing or when we sniff. They will also flow into the nose passively, because air pressure in the nasal cavity is typically lower than in the outside environment, creating a pressure gradient. Finally, via retro nasal olfaction, odorants from the mouth can travel back up into the nasal cavity (e.g., during consumption of food). Odorants then attach to odor receptors embedded in the mucous membrane of the roof of the nasal cavity, called the olfactory epithelium. There are over 1,000 types of receptors, and most of these respond to only a limited number of odorants, though a single odorant can bind to more than one type of receptor.

The olfactory receptor is called a bipolar neuron because appendages extend from opposite sides of its cell body. When an odorant binds to a bipolar neuron, a signal is sent to the neurons in the olfactory bulb, called the glomeruli. Tremendous convergence and divergence take place in the olfactory bulb. One bipolar neuron may activate over 8,000 glomeruli, and each glomerulus, in turn, receives input from up to 750 receptors. The axons from the glomeruli then exit the olfactory bulb laterally, forming the olfactory nerve, and travel to the primary olfactory cortex. Most fibers in the olfactory tracts connect to the ipsilateral cortex (the cortex on the same side as the tract), though some do cross to the other side of the brain via the anterior commissure. The primary olfactory cortex is located at the ventral junction of the frontal and temporal cortices. Neurons from this area then connect to the orbitofrontal cortex, which is considered a secondary olfactory processing center.

The Role of Sniffing in Olfactory Perception

Until recently, very little research had been conducted to explore the cognitive neuroscience of human olfaction. This neglect reflects in part our failure to appreciate the importance of olfaction. In addition, some thorny technical challenges must be overcome to apply tools such as fMRI to study the human olfactory system. First is the problem of delivering odors to the subject in a controlled manner (Figure 5.7a). Nonmagnetic systems must be constructed to allow the odorized air to be directed at the subject's nostrils while the subject is in the fMRI magnet. Second, it is hard to determine when an odor is no longer present. The chemicals that carry the odor can linger in the air for a long time. Third, although some odors overwhelm our senses, most are quite subtle, requiring exploration through the act of sniffing to detect and identify. Whereas it is almost impossible to ignore a sound, we can exert considerable control over the intensity of our olfactory experience.

Figure 5.6  The olfactory receptors lie within the nasal cavity, where they interact directly with odorants. The receptors then send information to the glomeruli in the olfactory bulb, the axons of which form the olfactory nerve that relays information to the primary olfactory cortex. The orbitofrontal cortex has also been shown to be a secondary olfactory processing area.
Noam Sobel of the Weizmann Institute in Israel has developed methods to overcome these challenges, conducting neuroimaging studies that have revealed an intimate relationship between smelling and sniffing (Mainland & Sobel, 2006; Sobel et al., 1998). Subjects were scanned while being exposed to either nonodorized, clean air or one of two chemicals: vanillin or decanoic acid. The former smells like vanilla; the latter, like crayons. The odor-absent and odor-present conditions alternated every 40 s. Throughout the scanning session, the instruction, “Sniff and respond, is there an odor?” was presented every 8 s. In this manner, the researchers sought to identify areas in which brain activity was correlated with sniffing and areas in which activity was correlated with smelling (Figure 5.7b).

Surprisingly, smelling failed to produce consistent activation in the primary olfactory cortex. Instead, the presence of the odor produced a consistent increase in the fMRI response in lateral parts of the orbitofrontal cortex, a region typically thought to be a secondary olfactory area. Activity in the primary olfactory cortex was closely linked to the rate of sniffing. Each time the person took a sniff, the fMRI signal increased regardless of whether the odor was present. These results seemed quite puzzling, suggesting that the primary olfactory cortex might be more part of the motor system for olfaction.

Upon further study, however, the lack of activation in the primary olfactory cortex became clear. Neuro-physiological studies of the primary olfactory cortex in the rat had shown that these neurons habituate quickly. Perhaps the primary olfactory cortex lacks a smell-related response because the hemodynamic response measured by fMRI exhibits a similar habituation. To test this idea, Sobel’s group modeled the fMRI signal by assuming a sharp increase followed by an extended drop after the presentation of an odor—an elegant example.

**Figure 5.7** Sniffing and smelling. (a) This special device was constructed to deliver controlled odors during fMRI scanning. (b, top) Regions activated during sniffing. The circled region includes the primary olfactory cortex and a posteromedial region of the orbitofrontal cortex. (b, bottom) Regions more active during sniffing when an odor was present compared to when the odor was absent.
of how single-cell results can be used to interpret imaging data. When analyzed in this manner, the hemodynamic response in the primary olfactory cortex was found to be related not only to sniffing, but also to smell. These results suggest that the primary olfactory cortex role might be essential for detecting a change in the external odor, with the secondary olfactory cortex playing a critical role in identifying the smell itself. Each sniff represents an active sample of the olfactory environment, and the primary olfactory cortex plays a critical role in determining if a new smell is present.

**One Nose, Two Smells**

The importance of sniffing for olfactory perception is underscored by the fact that our ability to smell is continually being modulated by changes in the size of the nasal passages. In fact, the two nostrils appear to switch back and forth—one being larger than the other for a number of hours, and then the reverse. These changes have a profound effect on how smell is processed (Figure 5.8). Why might the nose behave this way?

Maxwell Mozell of the University of Pennsylvania theorizes that the olfactory percept depends not only on how intense the odor is, but also on how efficiently we sample it (Mozell et al., 1991). The presence of two nostrils of slightly different sizes provides the brain with slightly different images of the olfactory environment. Sobel put this theory to test. In each participant, he monitored which nostril was allowing high airflow and which nostril was allowing low airflow, while presenting high-absorption-rate and low-absorption-rate odors to each nostril. As predicted (see Figure 5.8), when sniffed through the high-airflow nostril, the high-absorption-rate odorant was judged to be more intense compared to when the same odorant was presented to the low-airflow nostril. The opposite was true for the low-absorption-rate odorant: here, the odor with a low rate of absorption was judged to be more intense when sniffed through the low-airflow nostril. Some of the participants were monitored when the flow rate of their nostrils reversed. The perception of the odorant presented to the same nostril reversed with the change in airflow.

**Figure 5.8** Although the same odorants enter each nostril, the response across the epithelium will be different for the two nostrils because of variation in flow rates. One nostril always has a greater input airflow than the other with the nostrils switching between the two rates every few hours. This system of having one low-flow and one high-flow nostril has evolved to give the nose optimal accuracy in perceiving odorants that have both high and low rates of absorption.
As we will see in Chapter 11, asymmetrical representations are the rule in human cognition, perhaps providing a more efficient manner of processing complex information. With the ancient sense of olfaction, this asymmetry appears to be introduced at the peripheral level by modulation of the rate of airflow through the nostrils.

**Smell and Memory**

Have you ever noticed that a specific smell can bring you back to a memory from long ago? Perhaps a certain perfume or cologne reminds you of an old friend. Or perhaps, like L.W., the smell of an orange reminds you of Christmas. L.W. is now 82 years old, but the smell of an orange immediately triggers a vivid memory of her childhood Christmases on the family farm in Idaho, when her parents would put an orange in the bottom of her stocking.

Why are smells so closely related to memories? Some scientists believe that the reason is the direct connectivity of the olfactory cortex to the limbic cortex, a region that is intimately involved in memory and emotion (see Chapters 8 and 9). Using functional MRI, Rachel Herz of Brown University found that odors activate the limbic system more robustly than a related visual stimulus does when the stimuli are used to trigger meaningful personal memories (Herz et al., 2004). The link between odor and memory is further supported by studies showing that odor recognition is severely compromised in patients with hippocampal lesions (Levy et al., 2004). Perhaps, then, we should invent a new poem: Roses are red, violets are blue, but it's a flower's odor that captures memories so true.

**GUSTATORY PERCEPTION**

The sense of taste depends greatly on the sense of smell. Indeed, the two senses are often grouped together because they both begin with stimulation by chemicals (odorants or tastants). Because these two senses interpret the environment by discriminating between different chemicals, they are referred to as the chemical senses.

**Neural Pathways of Gustation**

Sensory transduction in the gustatory system begins when a food molecule, or tastant, stimulates a receptor in a taste cell and causes the receptor to depolarize. The taste cells are located in taste buds (Figure 5.9). The mouth contains roughly 10,000 taste buds. Most are found in the tongue, though they are also found, to a lesser degree, in the cheeks and other areas of the mouth. The basic tastes include salty, sour, bitter, sweet, and umami. *Umami* is what you taste when you eat steak or other protein-rich substances.

Each of the basic taste sensations has a different form of chemical signal transduction. In the signal transduction for salty taste, the salt molecule (NaCl) breaks down to Na⁺ and Cl⁻, and the Na⁺ ion is conducted into an ion channel in the cell, thus depolarizing the cell. Other signal transduction pathways, such as sweet carbohydrate tastants, are more complex, involving receptor binding that does not lead directly to depolarization, but rather initiates cascades of chemical “messengers” that eventually lead to cellular depolarization.

![Figure 5.9](image-url) Taste cells form pores on the taste bud. Each cell is sensitive to one of five basic tastes: salty, sweet, sour, bitter, and umami.
to only one type of tastant. The complex range of tastes that we experience, however, must result from the integration of information conveyed from the taste cells and processed in areas like the orbitofrontal cortex.

Gustatory Processing

The basic tastes give the brain information about the types of food that have been consumed. The sensation of umami tells the body that protein-rich food is being ingested; sweet tastes indicate carbohydrate intake and salty tastes give us information that is important for the balance between minerals or electrolytes and water. The tastes of bitter and sour likely developed as warning signals. Many toxic plants taste bitter, and a strong bitter taste can induce vomiting. Other evidence suggesting that bitterness is a warning signal is the fact that we can detect bitter substances 1,000 times better than, say, salty substances. Therefore a significantly smaller amount of bitter tastant will yield a taste response, allowing toxic bitter substances to be avoided quickly. Similarly, but to a lesser extent, sour indicates spoiled food (e.g., “sour milk”) or unripe fruits.

Humans can readily learn to discriminate similar tastes. For instance, Richard Frackowiak and his colleagues at University College London (Castriota-Scanderberg et al., 2005) studied wine connoisseurs (sommeliers), asking how their brain response compared to that of nonexperts when tasting wines that varied in very subtle ways. In primary gustatory areas, the two groups showed a very similar response. However, the sommeliers exhibited increased activation in the insula cortex and parts of the orbitofrontal cortex in the left hemisphere, as well as greater activity bilaterally in dorsolateral prefrontal cortex, a region thought to be important for high-level cognitive processes such as decision making and response selection (see Chapter 13).

The orbitofrontal cortex also appears to play an important role in processing the pleasantness and reward value of eating food. Dana Small and her colleagues (2001) at Northwestern University scanned the brains of people as they ate chocolate. During testing, the subjects rated the pleasantness of the chocolate and their desire to eat more chocolate. Initially, the chocolate was rated as very pleasant and the subjects expressed a desire to eat more. But as the subjects became satiated, their desire for more chocolate dropped. Moreover, although the chocolate was still perceived as pleasant, the intensity of their pleasure ratings decreased.

Comparing the neural activation in the beginning trials with the trials at the end of the study, the researchers were able to determine which areas of the brain participated in processing the reward value of the chocolate (the pleasantness) and the motivation to eat (the desire to have more chocolate). The posteromedial portion of the orbitofrontal cortex was activated when the chocolate was highly rewarding and the motivation to eat was strong. In contrast, the posterolateral portion of the orbitofrontal cortex was activated during the satiated state, when the chocolate was unrewarding and the motivation to eat was low. Thus, the orbitofrontal cortex appears to be a highly specialized taste-processing region containing distinct areas able to process opposite ends of the reward value spectrum associated with eating.
Figure 5.13  Anatomy of the eye (left) and retina (right). Light enters through the cornea and activates the receptor cells of the retina located along the rear surface. There are two types of receptor cells: rods and cones. The output of the receptor cells is processed in the middle layer of the retina and then relayed to the central nervous system via the optic nerve, the axons of the ganglion cells.

rapidly. Thus, cones are most active during daytime vision. Cones are essential for color vision, and we commonly speak of them as being one of three types: red, green, or blue. These names are somewhat misleading, though: Cones do not respond to colors per se; rather, as Figure 5.14 shows, they differ in the sensitivity of their photopigments to different wavelengths of visible light.

Rods and cones are not distributed equally across the retina. Cones are densely packed near the center of the retina, in a region called the fovea. Few cones are in the more peripheral regions of the retina. In contrast, rods are distributed throughout the retina. You can easily demonstrate the differential distribution of rods and cones by having a friend slowly bring a colored marker into your view from one side of your head. Notice that you see the marker and its shape well before you identify its color because of the sparse distribution of cones in the retina’s peripheral regions.

From the Eye to the Central Nervous System

The extensive processing of visual information performed within the retina is characterized by elaborate convergence of information. Indeed, though humans have an estimated 260 million photoreceptors, there are only 2 million ganglion cells, the output neurons from the retina. This compression of information suggests that higher-level visual centers should be efficient processors to recover the details of the visual world. Axons of the ganglion cells form a bundle, the optic
Figure 5.14 Spectral sensitivity functions for rods and the three types of cones. The short-wavelength ("blue") cones are maximally responsive to light with a wavelength of 430 nm. The peak sensitivities of the medium-wavelength ("green") and long-wavelength ("red") cones are shifted to longer wavelengths. White light such as daylight activates all three receptors because it contains all wavelengths.

Figure 5.15 The primary projection pathways of the visual system. The optic fibers from the temporal half of the retina project ipsilaterally, and the nasal fibers cross over at the optic chiasm. In this way, the input from each visual field is projected to the primary visual cortex in the contralateral hemisphere after the fibers synapse in the lateral geniculate nucleus (geniculocortical pathway). A small percentage of visual fibers of the optic nerve terminate in the superior colliculus and pulvinar nucleus.

nerve. By way of this nerve, visual information is transmitted to the central nervous system.

Figure 5.15 diagrams how visual information is conveyed from the eyes to the central nervous system. Before entering the brain, each optic nerve splits into two parts. The temporal (lateral) branch continues to traverse along the same (ipsilateral) side. The nasal (medial) branch crosses over to project to the opposite (contralateral) side; this crossover place is called the optic chiasm. Given the eye's optics, the crossover of nasal fibers ensures that visual information from each side of external space will be projected to contralateral brain structures. Because of the retina's curvature, for example, the temporal half of the right retina is stimulated by objects in the left visual field. In the same fashion the nasal hemiretina of the left eye is stimulated by this same region of external space. Because fibers from each nasal hemiretina cross, all information from the left visual field is projected to the right hemisphere, and information from the right visual field is projected to the left hemisphere (see Figure 5.15).

Once inside the brain, each optic nerve divides into pathways that differ with respect to where they terminate
in the subcortex. Figure 5.15 focuses on the retinogeniculate pathway, the projection from the retina to the lateral geniculate nucleus (LGN) of the thalamus. This pathway contains more than 90% of the axons in the optic nerve and provides input to the cortex via the geniculocortical projections. The remaining 10% of the fibers innervate other subcortical structures, including the pulvinar nucleus of the thalamus and the superior colliculus of the midbrain. However, the fact that these other receiving nuclei are innervated by only 10% of the fibers does not mean that these pathways are unimportant. The human optic nerve is so large that 10% of it constitutes more fibers than are found in the entire auditory pathway. The superior colliculus and pulvinar nucleus play a large role in visual attention, and the retinocollicular pathway is sometimes viewed as a more primitive visual system (see the discussion of blindsight later in this chapter).

The final projection to the visual cortex is via the geniculocortical pathway. This bundle of axons exits the LGN and ascends to the cortex, with almost all of the fibers terminating in the primary visual cortex (V1) of the occipital lobe. Thus, visual information reaching the cortex has been processed by at least four distinct neurons: photoreceptors, bipolar cells, ganglion cells, and LGN cells.

**Cortical Visual Areas**

Figure 5.16 shows a map of the visual areas of the cortex. Each box in the figure stands for a region of cortex that is a distinct region of visual processing. More than 30 distinct cortical visual areas have been identified in the monkey—an increase of about 200% since similar maps were published in 1983. Some say that physiologists "discover" new visual areas faster than rabbits reproduce. Note that Figure 5.16 follows the nomenclature developed by physiologists for functional maps (see Chapter 3). Striate cortex, or V1, is the first projection region of geniculate axons. Although other areas have names such as V2, V3, and V4, this numbering scheme should not be taken to mean that the synapses proceed sequentially from one area to the next. The lines connecting these extrastriate visual areas demonstrate extensive convergence and divergence across visual areas. In addition, connections between many areas are reciprocal; areas frequently receive input from an area to which they project.

How a visual area is defined depends on the criteria used. An obvious criterion is that cells within the area respond to visual stimuli; however, if this were the sole criterion, it would be difficult to tell where one visual area begins and another ends. Sometimes neuroanatomy helps. For example, the border between V1 and V2 corresponds to the boundary between Brodmann areas 17 and 18. But boundaries often cannot be identified with anatomical methods. For the physiologist, area 19 has many distinct visual areas. Physiologists depend on criteria different from the ones used by anatomists.

A primary physiological method for establishing visual areas is to measure how spatial information is represented across a region of cortex. Each visual area has a topographic representation of external space in the contralateral hemifield, and the boundaries between anatomically adjacent visual areas are marked by topographic discontinuities (Figure 5.17). The replication of topography within the cortex does not result from independent inputs to each area. As one area projects to another, topography is preserved. Precise spatial information is preserved by these multiple retinotopic maps, at least in early visual areas, reflecting the fact that the system has to link features that emanate from a common location.

**CELLULAR PROPERTIES VARY ACROSS CORTICAL VISUAL AREAS**

Why would it be useful for the primate brain to have evolved so many visual areas? One possibility is that the areas form a hierarchy in which each area successively elaborates on the representation derived by processing in earlier areas, representing the stimulus in a specific way. The simple cells of the primary visual cortex calculate edges. Complex cells use the information from many simple cells to represent corners and edge terminations. In turn, higher order visual neurons integrate information from complex cells to represent shapes. Successive elaboration culminates in formatting the representation of the stimulus so that it matches (or doesn't match) information in memory. As Figure 5.16 shows, though, there is not a simple hierarchy; extensive patterns of convergence and divergence result in multiple pathways.

An alternative hypothesis relates to the idea of visual perception as an analytic process. Although each visual area provides a map of external space, the maps differ with regard to the type of information they represent. For instance, neurons in some areas are highly sensitive to color variation. In other areas, they may be sensitive to movement but not to color. By this hypothesis, neurons within an area not only code where an object is located in visual space but also provide information about the object's attributes. Visual perception is a divide-and-conquer strategy. Rather than all attributes of an object
being represented by all visual areas, each visual area provides its own limited analysis. Processing is distributed and specialized. As we advance through the visual system, different areas elaborate on the initial information in V1 and begin to integrate this information across dimensions to form recognizable percepts.

Extensive physiological evidence supports the specialization hypothesis. Consider cells in area MT, so named because it lies in the middle temporal lobe region of the macaque monkey, a species used in many physiology studies. Physiologists commonly refer to this region as visual area 5 (V5). Single-cell recordings reveal that neurons in this region do not show specificity in terms of the color of the stimulus. These cells will respond similarly to either a green or a red circle on a white background. Even more striking, these neurons
Pioneers in the Visual Cortex

Although they had little difficulty identifying individual cortical cells, the cells failed to respond to the kinds of stimuli that had proved so effective in Kuffler's studies: small spots of light positioned within a cell's receptive fields. Indeed, the lack of consistent responses made it difficult to determine where the receptive field was situated. Hubel and Wiesel had a breakthrough, though, when they switched to dark spots, which they created by placing an opaque disk on a glass slide. Although the cell did not respond to the dark spot, Hubel and Wiesel noticed a burst in activity as the edge of the glass moved across part of the retina. After hours of play with this stimulus, the first organizational principle of primary visual cortex neurons became clear: Unlike the circular receptive fields of ganglion cells, cortical neurons were responsive to edges.

Subsequent work revealed that LGN cells and ganglion cells behave similarly: Both are maximally excited by small spots of light. Such cells are best characterized as exhibiting a concentric center-surround organization. Figure 1 shows the receptive field of an LGN cell.
cell. When a spot of light falls within the excitatory center region, the cell is activated. If the same spot is moved into the surrounding region, the activity is inhibited. A stimulus that encompasses both the center and the surrounding region will fail to activate the cell because the activity from the excitatory and inhibitory regions will cancel. This observation clarifies a fundamental principle of perception: The nervous system is most interested in change. We recognize an elephant not by the homogeneous gray surface of its body, but by the contrast of the gray edge of its shape against the background.

In Figure 2, outputs from three LGN cells with receptive fields centered at slightly different positions are linked to a single cortical neuron. This cortical neuron would continue to have a center–surround organization, but for this cell the optimal stimulus would have to be an edge. In addition, the cell would be selective for edges in a certain orientation. As the same stimulus was rotated within the receptive field, the cell would cease to respond because the edge would now span excitatory and inhibitory regions of the cell. Hubel and Wiesel called these cells simple cells, to connote the fact that their simple organization would extract a fundamental feature for shape perception: the border of an object. The same linking principle can yield more complex cells—cells with a receptive-field organization that makes them sensitive to other features, such as corners or edge terminations.

Orientation selectivity has proved to be a hallmark of neurons in the primary visual cortex. Across a 2 mm x 2 mm chunk of cortex, the receptive fields of neurons are centered on a similar region of space (Figure 3). Within the chunk, the cells vary in terms of their preferred orientation, and they alternate between columns that are responsive to inputs from the right and left eyes. A series of such chunks allows for the full representation of external space, providing the visual system with a means of extracting the visible edges in a scene.

Hubel and Wiesel's studies established how a few organizational principles can serve as building blocks of perception derived from simple sensory neurons. The importance of their pioneering studies was acknowledged in 1981, when they shared the Nobel Prize in Physiology or Medicine.

Continued on the following page

Figure 2  Simple cells in the primary visual cortex can be formed by the linking of outputs from concentric LGN cells with adjacent receptive fields. In addition to signaling the presence of an edge, simple cells are selective for orientation. The simple cell illustrated here is either excited or inhibited by an edge that follows its preferred orientation. It shows no change in activity if the edge is at a perpendicular orientation.
Figure 3  Feature representation within the primary visual cortex. (a) As the recording electrode is moved along the cortex, the preferred orientation of the cells varies in a continuous manner. The preferred orientation is plotted as a function of the location of the electrode. (b) The orientation columns are crossed with ocular dominance columns to form a cortical module. Within a module, the cells have similar receptive fields (location sensitivity), but they vary in terms of input source (left or right eye), and sensitivity to orientation, color, and size. For example, the so-called blobs contain cells that are sensitive to color and finer details in the visual input. This organization is repeated for each module.
Figure 5.17  Physiological methods are used to identify different visual areas. An area is defined by a discontinuity in the retinotopic representation of the cells. Along the continuous ribbon of cortex shown here, seven different visual areas can be identified. However, processing is not restricted to proceeding from one area to the next in a sequential order. For example, axons from V2 project to V3, V4, and V5/MT.

respond weakly when presented with an alternating pattern of red and green stripes whose colors are of equal brightness.

In contrast, MT neurons are quite sensitive to movement and direction, as Figure 5.18 shows (Maunsell & Van Essen, 1983). The stimulus, a rectangular bar, was passed through the receptive field in varying directions. The cell's response was greatest when the stimulus was moved downward and left. In contrast, this cell was essentially silent when the stimulus was moved upward or to the right. Thus, the cell's activity correlates with two attributes of the stimulus. First, the cell is active only when the stimulus falls within its receptive field. Second, the response is greatest when the stimulus moves in a certain direction. Activity in V5 cells also correlates with the speed of motion. The cell in Figure 5.18 responded maximally when the bar was moved rapidly. At lower speeds, the bar's movement in the same direction failed to raise the response rate above baseline.

HUMAN VISUAL AREAS

Single-cell recording studies have provided physiologists with a powerful tool to map out the visual areas in the monkey brain and characterize the functional properties of the neurons within these areas. This work has provided strong evidence that different visual areas are specialized to represent distinct attributes of the visual scene. Inspired by these results, researchers have employed neuroimaging techniques to ask whether a similar architecture can be discerned in the human brain.

Semir Zeki (1993) of University College London and his colleagues at London's Hammersmith Hospital used position emission tomography (PET) to verify that different visual areas are activated when subjects are processing color or motion information. They used subtractive logic by factoring out the activation in a control condition from the activation in an experimental condition. Consider first the color experiment. For the control condition, subjects passively viewed a collage of achromatic rectangles. Various shades of gray, spanning a wide range of luminances, were chosen. The control stimulus was expected to activate neural regions with cells that are contrast sensitive (e.g., sensitive to differences in luminance).

For the experimental condition, the gray patches were replaced by a variety of colors (Figure 5.19a). Each color patch was matched in luminance to its corresponding gray patch. With this setup, neurons sensitive to luminance information should be equally activated in control and experimental conditions. However, the colored stimulus should produce more activity in neural regions sensitive to chromatic information. These regions should be detected if the metabolic activity recorded when subjects viewed the gray stimulus is subtracted from the activity recorded when subjects viewed the color stimulus.

The same logic was used to design the motion experiment. For this study, the control stimulus consisted of a complex black-and-white collage of squares (Figure 5.19b). The same stimulus was used in the experimental condition, except that the squares were set in motion. They would move in one direction for 5 s and then in the reverse direction for the next 5 s.

The results of the two studies provided clear evidence that the two tasks activated distinct brain regions (Figure 5.20). After subtracting activation during viewing of the achromatic collage, investigators found
numerous residual foci of activation when subjects viewed the colored collage. These foci were bilateral and located in the most anterior and inferior regions of the occipital lobe (Figure 5.20a). Although the spatial resolution of PET is coarse, these areas were determined to be in front of the striate (V1) and prestriate (V2) cortex. In contrast, after the appropriate subtraction in the motion experiment, the residual foci were bilateral but near the junction of the temporal, parietal, and occipital cortices (Figure 5.20b). These foci were more superior and much more lateral than the color foci.

Zeki and his colleagues were so taken with this dissociation that they proposed that the nomenclature developed by primate researchers be applied here. They labeled the area activated in the color foci as area V4 and the area activated in the motion task as V5. Note that researchers frequently refer to area V5 as human area MT, even though the area is not in the temporal lobe in the human brain. Of course, with PET data we cannot be sure that the foci of activation really consist of just one visual area.

Note that there are also striking between-species differences in terms of the relative position of the color and motion areas (compare Figures 5.17 and 5.20). Such differences probably result from the fact that the surface area of the human brain is substantially larger, and this
expansion required additional folding of the continuous cortical sheet. Thus, we can ask questions about functional homology (see Chapter 15)—the correspondence in structure and function between species—but we need to be aware that the mapping is unlikely to be straightforward. Indeed, it is quite possible that humans have visual areas that do not correspond to any region in our close primate relatives.

The activation maps in this PET study are rather crude. Many laboratories now employ more sophisticated fMRI techniques to study the organization of human visual cortex. In these studies, a stimulus is systematically moved across the visual field (Figure 5.21). For example, a semicircular checkerboard pattern is slowly rotated about the center of view. In this way, the BOLD response for areas representing the superior quadrant will be activated at a different time than areas representing the inferior quadrant, and in fact, the representation of the entire visual field can be continuously tracked. To compare areas that respond to foveal stimulation and those that respond to peripheral stimulation, a dilating and contracting ring stimulus is used. By combining these different stimuli, the cortical representation of external space is measured.
Figure 5.21  Mapping visual fields with functional magnetic resonance imaging (fMRI). The subject views a rotating circular wedge while fMRI scans are obtained. The wedge passes from one visual quadrant to the next, and the blood oxygenation level dependent (BOLD) response in visual cortex is measured continuously to map out how the regions of activation change in a corresponding manner.

Because of the convoluted nature of the human visual cortex, the results from such an experiment would be indecipherable if we were to plot the data on the anatomical maps found in a brain atlas. To avoid this problem, a flat map representation is constructed. High-resolution anatomical MRI scans are obtained and computer algorithms transform the folded, cortical surface into a two-dimensional map by tracing the gray matter. The activation signals from the fMRI study are then plotted on the flattened surface, with color coding used to indicate areas that were activated at similar times.

Researchers at the Massachusetts General Hospital used this procedure to reveal the organization of the human visual system in exquisite detail. The flattened map from the visual cortex of the right hemisphere shows the activation map when the checkerboard moves across the left side of space (Figure 5.22). Primary visual cortex (V1) lies along the calcarine sulcus and, as found in all physiological studies, the physical world is inverted. Except for the most anterior aspects of visual cortex, areas above the sulcus are active when the rotating stimulus is in the lower quadrant; the reverse is true when the stimulus is in the upper quadrant.

In addition, the activation patterns show a series of repetitions across the visual cortex. The green and blue repetitions in the superior portion of the visual cortex indicate that the lower quadrant of the visual field is represented multiple times. Similarly, the red and purple alternations in the lower portion indicate the upper quadrant of the visual field is represented multiple times. These repetitions indicate separate topographic maps. Following the conventions adopted in the single-cell studies, the visual areas are numbered in increasing order, with primary visual cortex (V1) most posterior and secondary visual areas (V2, V3/VP, V4) more anterior.

Figure 5.22b shows how eccentricity is represented in the ventral part of the right visual cortex. The cortical representation of the fovea, the regions shown in red, is quite large. Visual acuity is much greater at the fovea due to the disproportionate amount of cortex that encodes information from this part of space.

**VISUAL ILLUSIONS**

Imaging studies have opened a new window for studying visual illusions that have puzzled philosophers and psychologists alike for many years. Patterns of brain activation during illusory states can be compared with
those observed during visual stimulation. In this way, areas of overlap can provide insight into the level of processing at which illusions arise, as well as indicate how information is represented in different visual areas.

Stare at the Enigma pattern shown in Figure 5.23a. After a few seconds, you should begin to see scintillating motion within the blue circles—an illusion created by their opposed orientation to the radial black and white lines. What are the neural correlates of this illusion? We know that moving patterns produce a strong hemodynamic response in V5. Is this same area also activated during illusory motion? Both PET and fMRI have been used to show that viewing displays such as the Enigma pattern do indeed lead to pronounced activity in V5. This activation is selective: Activity in V1 does not increase during illusory motion.

A different illusion is demonstrated in Figure 5.23b. After staring at the bright green circle for about 30 s, shift your gaze to the neighboring gray circle. You should perceive the gray circle as tinged with a reddish purple, the complementary color to green. Scans of subjects while they are perceiving this color illusion reveal a high level of activation in an inferior visual area just anterior to V4 for an extended period of time after a saturated color turns to gray. In a control condition, the color patch alternates between two saturated colors before turning gray, thus negating the illusion. Here, activity quickly returns to baseline when the gray patch is presented. As with the motion illusion, such aftereffects are not seen in V1.

These results show that our perceptual activity is more closely related to activity in higher visual areas than to activity in primary visual cortex. Indeed, illusions can be viewed as an interesting way to link the study of perception and imagery, an issue we return to in Chapter 6. While most of us do not have vivid percepts when we tap into our visual knowledge—for example, think about the color of a banana—imaging studies demonstrate that accessing this knowledge can produce measurable changes of activity in secondary visual areas.

**Deficits in Visual Perception**

Before the advent of neuroimaging, much of what we learned about visual processing in the human brain came from lesion studies. In 1888, Louis Verrey (cited in Zeki, 1993), described a patient who had lost the ability to perceive colors in her right visual field. Verrey reported that the patient had problems with acuity within restricted portions of this right visual field. But the color deficit was uniform and complete. We can guess that this patient’s world looked similar to the drawing in

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**Figure 5.23** The neural basis of visual illusions.

(a) When viewing the Enigma pattern, we perceive illusory motion. Viewing the pattern is accompanied by activation in area MT. (b) Staring at the bright green patch produces color aftereffects. (c) Activation in a visual region anterior to V4 is plotted (see text for details).
DEFICITS IN COLOR PERCEPTION: ACHROMATOPSIA

When we speak of someone who is color-blind, we are usually describing a person who has inherited a gene that produces an abnormality in the photoreceptor system. Dichromats, people with only two photopigments, can be classified as red-green color-blind if they are missing the photopigment sensitive to either medium or long wavelengths, or blue-yellow color-blind if they are missing the short-wavelength photopigment. Anomalous trichromats, in contrast, have all three photopigments, but one has abnormal sensitivity. The incidence of these genetic disorders is high in males: about 8% of the population. The incidence rate is much lower in females: less than 1%.

Much rarer are disorders of color perception that arise from disturbances of the central nervous system. These disorders are called aachromatopsia, taken from the prefix a- ("without") and the stem chroma ("hue"). As is evident in Verrey's description, these patients see the world without color. J. C. Meadows (1974) of the National Hospital for Neurology and Neurosurgery in London described one such patient as follows: "Everything looked black or grey. He had difficulty distinguishing British postage stamps of different value, which look alike, but are of different colors. He was a keen gardener, but found that he pruned live rather than dead vines. He had difficulty distinguishing certain foods on his plate where color was the distinguishing mark" (p. 629).

Patients with aachromatopsia often report that colors have become a bland palette of "dirty shades of gray." The shading reflects variations in brightness rather than hue. Other aspects of vision, such as depth and texture perception, also remain intact, enabling someone with aachromatopsia to see and recognize objects in the world. Indeed, color is not a necessary cue for shape perception. Its subtlety is underscored when we consider the fact that people often do not notice the change from black and white to color when Dorothy lands in Oz in the movie The Wizard of Oz. Nonetheless, when lost forever, this subtlety is sorely missed.

In almost all published cases of aachromatopsia, patients exhibit abnormalities in other areas of visual perception as well. What remains clear is that the deficit in color perception is markedly more severe. Consider the performance of an aachromatopsia patient on tests of hue discrimination and brightness discrimination conducted by Alan Cowey and his colleagues at Oxford University (Heywood et al., 1987). Although we think of colors as differing in hue, they also vary in other properties, such as saturation (e.g., pink versus deep red) and reflectance, the physical property of reflected light that reaches the eye and determines our psychological impression of the brightness of a visual scene. Although there is no objective way to equate a difference in hue with a difference in reflectance, we can determine the psychological equivalence of differences across the dimensions (Figure 5.25a).

On each trial the subject viewed three color patches. Two patches were identical; the third varied in hue or reflectance. The subject was asked to identify the stimulus that was different. As Figure 5.25b shows, the task was much more difficult when stimuli varied in hue. Reflectance differences, for example, were almost always correctly identified with a 4-step difference. For hue differences, the patient scored only 70% correct even with a 10-step difference, which corresponds to a transition from a yellowish green to a deep orange! Though this hue perception deficit is striking, the patient's performance on reflectance differences was also impaired. Healthy subjects rarely made errors with a one-step difference on any of the dimensions.

Note also that this patient had lost visual acuity after his stroke. He could read, but only if the print was enlarged. We should not be surprised that people with aachromatopsia exhibit visual deficits that extend beyond color perception. Neurological disorders such as
Figure 5.25 Assessing sensitivity to different dimensions of color perception in achromatopsia. (a) Psychological scaling techniques can be used with healthy individuals to create stimulus sets in which the similarity of all neighboring pairs is judged as equal. These techniques are used to create norms for the similarity across the different dimensions of a color (hue, saturation, and reflectance), the physical measure that underlies our perception of brightness. (b) Three chips were presented, two that were identical and one that differed either in hue or reflectance. A patient with achromatopsia had difficulty identifying chips that differed in hue, even when the stimuli differed by 10 units. His ability to discriminate brightness, although not normal, was much better. Here, he almost always labeled the stimuli as different when they were separated by at least 4 units.

strokes and tumors do not respect the borders of visual areas. Achromatopsia has consistently been associated with lesions that encompass V4 and the region anterior to V4, but the lesions typically extend to neighboring regions of the visual cortex. Color-sensitive neurons are also orientation selective; as such, many achromatic patients have difficulty with form perception.

This hypothesis linking achromatopsia with deficits in form perception was carefully explored in the case study of a patient who suffered a stroke resulting in a small lesion near the temporo-occipital border in the right hemisphere. The damage was centered in area V4 and anterior parts of the visual cortex (Figure 5.26a). To assess the patient's achromatopsia, a hue-matching experiment was performed in which a sample color was presented at the fovea, followed by a test color in one of the four quadrants of space. The patient's task was to judge if the two colors were the same. The difference between the sample and test color was adjusted until the patient was performing correctly on 80% of the trials, and this difference was measured separately for each quadrant. Regardless of the sample hue, the patient was severely impaired on the hue-matching task when the test color was presented in the upper left visual field (Figure 5.26b). The fact that the deficit was found only in the upper contralateral visual field is consistent with previous reports of achromatopsia.

The main focus of the study, however, was to examine form perception. Would the patient show similar deficits in form perception in this quadrant? If so, what types of form perception tasks would reveal the impairment? To answer these questions, a variety of tasks were administered. The stimuli are shown in Figure 5.27. On the basic visual discriminations of contrast, orientation, and motion, the patient's performance was similar for all four quadrants and comparable to the performance of control participants. However, he showed impairment on tests of higher order shape perception, and again, this impairment was restricted to the upper left quadrant. For these tasks, shape information requires combining information from neurons that might detect simple properties such as line orientation. For example, the orientation of the line separating the two semicircles is defined only by the combination of the lengths of the individual stripes and their offset.
Figure 5.26  Color and shape perception in a patient with a unilateral lesion of V4. (a) MRI scans showing a small lesion encompassing V4 in the right hemisphere. (b) Color perception thresholds in each visual quadrant. The y-axis indicates the color required to detect a difference between a patch shown in each visual quadrant (upper left, UL; lower left, LL; upper right, UR; lower right, LR) and the target color shown at the fovea. The target color was red for the results shown in the top panel and green for the results shown in the bottom panel.

Characterizing area V4 as a “color” area is too simplistic. This area is part of secondary visual areas devoted to shape perception. Color can provide an important cue about an object’s shape. V4 may be invaluable for using color information as one cue to define the boundaries that separate the objects that form our visual environment.

Revisiting Patient P.T.  Let’s return to patient P.T., who was left with visual agnosia following a stroke to his right hemisphere. In particular, let’s turn to a striking demonstration of P.T.’s deficit.

During examination, P.T. was shown two paintings, one by Monet depicting a subdued 19th-century countryman dressed in his Sunday suit, and the other by Picasso of a woman with a terrified expression (Figure 5.28). P.T. was asked to describe what he saw in each painting. When shown the Monet, he looked puzzled. He saw no definable forms, but just an abstract blend of colors and shapes. His problem in interpreting the painting was consonant with the deficits he experienced at home. Yet he readily identified the figure in Picasso’s painting and pointed out that it was a woman, or perhaps a young girl. This dissociation is compelling, as most would readily agree that the Monet is more realistic.

Picasso painted the parts of his work as separate units. He used sharp contrasts in brightness and vivid colors to highlight facial regions. Monet opted for a softer approach, in which parts are best seen in a continuous whole, with gradual changes in contrast and color. Can any of these factors account for P.T.’s performance in identifying the figures in Picasso and Monet?
The neurologist evaluating P.T. emphasized that the primary problem stemmed from a deficit in color perception. This hypothesis is in accord with one of the primary differences between the Monet and the Picasso. In the Monet painting, the boundaries between the face and the background are blended: Gradual variations in color demarcate the facial regions and separate them from the background landscape. A deficit in color perception provided a parsimonious account of the patient's problems in recognizing faces and landscapes. The rolling green hills of an Oregon farm can blur into a homogeneous mass if one cannot discern fine variations in color. In a similar way, each face has its characteristic coloration.

It seems equally plausible, however, that the problem stemmed from a deficit in contrast or contour perception. These features are salient in the Picasso and absent in the Monet. Indeed, we know from our recent discussion of V4, that color and form perception are often conflated. What is clear is that the patient's stroke affected primarily the cortical projections of the pathways essential for color and form perception. In contrast, the cortical projections of the pathway involved in motion were intact. The patient had no trouble recognizing his wife as she moved from the stove to the kitchen table; indeed, P.T. commented that her idiosyncratic movement enabled him to recognize her.

DEFICITS IN MOTION PERCEPTION: AKINETOPSIA

In 1983, researchers at the Max Planck Institute in Munich reported a striking case of a woman who had incurred a selective loss of motion perception, or akitopsia (Zihl et al., 1983). For this woman, whom we call M.P., perception was akin to viewing the world as a series of snapshots. Rather than seeing things move continuously in space, she saw moving objects appear in one position and then another. When pouring a cup of tea, M.P. would see the liquid frozen in air, like a glacier. She would fail to notice the tea rising in her cup and would be surprised when the cup overflowed (Figure 5.29). The loss of motion perception also made M.P. hesitant about crossing the street. As she noted, "When I'm looking at the car first, it seems far away. But then, when I want to cross the road, suddenly the car is very near" (Zihl et al., 1983, p. 315).

Examination revealed M.P.'s color and form perception to be intact. Her ability to perceive briefly presented objects and letters, for example, was within the normal range. But her ability to judge the direction and speed of moving objects was severely impaired. This deficit was most apparent with stimuli moving at high speeds. At speeds faster than 20%, M.P. never reported detecting the motion. She could see that a dot's position had changed and hence could infer motion. But her percept was of two static images; there was no continuity from
secondary visual areas in the unimpaired hemisphere. The receptive fields in primate area V5 are huge and have cells that can be activated by stimuli presented in either visual field.

Nonetheless, the application of transcranial magnetic stimulation (TMS; see Chapter 4) over human V5 can produce transient deficits in motion perception. In one such experiment, subjects were asked to judge whether a stimulus moved to the left or right (Beckers & Zeki, 1995). To make the task demanding, the stimulus was visible for only 25 ms. TMS was applied to three locations over the visual cortex, targeted to activate neurons in V5, V1, or, as a control, an extrastriate region lying between these two regions (Figure 5.30). Performance of the motion task was disrupted by stimulation over V5. One advantage of this method is that the timing of the magnetic pulses can be varied to determine the time of maximum disruption in the ability to perform the task. When TMS was applied to V5 just prior to the onset of the stimulus, performance was no better than chance. When the TMS was delayed until 40 ms after stimulus onset, performance was perfect.

These results are puzzling, especially when one considers that only at much longer delays did stimulation over V1 disrupt performance. If V1 provides the gateway to all information processing in the visual cortex, one would expect the optimal time of stimulation over V1 to precede the optimal time of stimulation for V5. Alternatively, it is possible that there are at least two ways for signals to reach V5. The first is a cortical route: the extension of geniculostriate pathways and on to V5. The second route bypasses V1. It may involve direct projections to V5 from the LGN or projections from other subcortical structures that are sensitive to moving stimuli, such as the superior colliculus or pulvinar. Physiological and anatomical studies in primates will likely be needed to address these hypotheses.

**PERCEPTION WITHOUT A VISUAL CORTEX**

Almost all of the ascending axons from the LGN terminate in the primary visual cortex. We would expect an individual with damaged primary visual cortex to be blind and indeed, this is what is observed. The blindness may not be complete, however. If the lesion is restricted to one half of the visual field, the loss of perception will be restricted to the contralateral side of space; such a deficit is referred to as hemianopia. Smaller lesions may produce more discrete regions of blindness, or scotomas. Patients with primary visual cortex lesions are unable to report seeing anything presented within a scotoma. As anatomists have shown,
however, there are not only multiple visual pathways within the cortex, but also prominent subcortical visual pathways. These observations have led to some very surprising findings showing that visual capabilities may persist even in the absence of the primary visual cortex.

**Cortical and Subcortical Perception in the Hamster**

Many nongeniculate fibers terminate in the superior colliculus. This structure plays a critical role in producing eye movements. If this midbrain structure becomes atrophied, as with supranuclear palsy, the patient will lose the ability to generate eye movements. It is as if the eyes have become paralyzed. Stimulation studies in primates further demonstrate the role of the superior colliculus in eye movement. When this area is stimulated, the animal’s eyes move and the direction of this movement depends on the stimulation site.

Gerald Schneider (1969), working at the Massachusetts Institute of Technology, provided initial evidence of the importance of the colliculus in studies of hamsters. These animals were trained to do the two tasks illustrated in Figure 5.31. In one task, the hamsters were trained to turn their heads in the direction of a sunflower seed held in an experimenter’s hand (Figure 5.31a). The task was easy for hamsters because they have a strong propensity to find sunflower seeds and put them in their cheeks.

The second task presented more of a challenge. Here the animals were trained to run down a two-arm maze and enter the door behind which a sunflower seed was hidden (Figure 5.31b). The task required the animals to
make simple visual discriminations such as to distinguish between black and white doors or between doors with vertical or horizontal stripes. For normal hamsters, the discriminations are not taxing. Within a few trials, they became proficient in selecting the right door in almost all trials.

After training, Schneider divided the hamsters into two experimental groups. One group received bilateral lesions of the visual cortex, including all of areas 17 and 18 (Figure 5.31c). For the second group, the superior colliculus was rendered nonfunctional by the ablation of its input fibers (Figure 5.31d). This strategy was necessary because direct lesions to the colliculus are likely to kill the animals, since the structure borders numerous brainstem nuclei that are essential for life.

The two lesions yielded a double dissociation. Cortical lesions severely impaired the animals' performance on the visual identification tasks. The animals could run down the maze and had sufficient motor capabilities to enter one of the doors, but they could not discriminate black from white or horizontal from vertical stripes. In contrast, the animals with collicular lesions demonstrated no impairment.

The deficits were reversed on the sunflower seed localization task. Animals with cortical lesions were perfect at this task once they had recovered from the surgery. Yet animals with collicular lesions acted as though they were blind. They made no attempt to orient toward the seeds—and not because they were unmotivated or had a motor problem. If the seed brushed
against a whisker, the animal rapidly turned toward it and gobbled it up.

These data provide compelling evidence for dissociable functions of the hamsters’ superior colliculus and visual cortex. The collicular lesions impaired their ability to orient toward the position of a stimulus, and the cortical lesions disrupted visual acuity. For the hamster, one might think of this double dissociation as reflecting two systems: one devoted to spatial orientation, the other devoted to object identification. As we will see in the next chapter, the ability to distinguish between representing properties of an object and representing the location of the object can also be traced to the cortex itself.

**Blindsight: Evidence of Residual Visual Function Following Cortical Blindness**

Inspired by Schneider’s findings that cortically blind hamsters could still locate the sunflower seeds, Lawrence Weiskrantz (1986) at Oxford University tested a hemianopic patient, D.B., to determine whether he could still detect the location of objects presented within his scotoma. Weiskrantz was not satisfied with the usual method of self-report to test for visual function. Rather, he designed a clever behavioral assay for residual function. After presenting a spot of light, the experimenters sounded a tone and D.B. was asked to move his eyes to the location of the stimulus. This task was easy when stimuli were presented in D.B.’s intact right visual field. But when stimuli were presented within the scotoma, the task struck D.B. as utter nonsense. He could not understand how he should know where to move his eyes when he failed to see anything. Nonetheless, the experimenters encouraged D.B. to guess. On control trials, a tone was sounded, requiring D.B. to move his eyes, but it was not preceded by a light. D.B., of course, was not aware of this difference between the control and experimental trials. To him, all of the trials seemed bizarre, in that he was being asked to look at stimuli that he had not been aware of (Figure 5.32).

The results could hardly have been more dramatic. As expected, D.B.’s eye movements on the phantom trials were random. But in the experimental trials, D.B. did much better than chance at localizing the stimuli. When the stimuli were within 20° of the fovea, D.B.’s eye movements were highly correlated with the position of the stimuli.

Weiskrantz named this paradoxical phenomenon **blindsight**. The patient acts and feels as if he is blind yet shows a residual ability to localize stimuli. Although the blindsight phenomenon has been reported in many other patients, the interpretation of the effect remains controversial. It would be premature to conclude that this phenomenon reflects solely processing in subcortical pathways. Information may reach extrastriate visual areas in the cortex, either through direct geniculate projections or via projections from other subcortical structures. Using PET, researchers have shown that extrastriate regions such as human MT can be activated by moving stimuli, even when the ipsilateral striate cortex is completely destroyed (Barbur et al., 1993). Another possibility is that the lesions in the primary visual cortex are incomplete and that blindsight results from residual function in the spared tissue (Fendrich et al., 1992). The representations in the damaged region may be sufficient to guide eye movements, even though they fail to achieve awareness.

**DEFICITS IN OTHER ASPECTS OF VISUAL PERCEPTION**

The case for specific deficits in other elementary aspects of visual perception is more contentious than for achromatopsia and akinetopsia. A report from the early 20th century (Riddoch, 1917) described a patient to whom the world appeared essentially flat. This depth perception impairment was said to exist despite the patient’s ability to perceive variations in color and shading. However, it is not possible to evaluate the selectivity of the deficit, because careful assessments of other visual functions were not performed. In a similar manner, patients who have lost visual acuity after suffering cortical lesions generally have widespread problems in visual perception. Thus, there are no unambiguous reports of selective deficits in form or depth perception.

The visual system is likely to represent form and depth redundantly. Depth perception arises from a multitude of cues. One potent cue, binocular disparity, arises when each eye has a slightly different view of the world. Inputs from the two eyes converge on common cortical neurons. Depth also can be inferred from motion. A moving object will obscure a more distant object or be hidden by a nearer object. In a similar sense, as we move, the change in the retinal image is greater for near objects than for ones far away. All these cues can be used in normal depth perception. This redundancy would be expected to help preserve depth perception even if certain modules were lesioned.

Form perception also stems from multiple sources of information. Indeed, form perception is an essential goal of vision, and all visual processing is devoted to determining what objects are in the visual field and where they are located. Motion perception may be important for a predator anticipating the location of moving prey. Nonetheless, movement also can help to identify prey, to separate it from the background, and
to determine if its motion is characteristic of an animal worthy of pursuit. A hungry frog does not want to flick its tongue at every moving object. Rather, the frog can recognize a potential snack by its specific motion—for example, that produced by a fly. Color perception is important only in that it facilitates form perception. Colors do not exist without forms. Color is a potent cue for discriminating an object or part of one from other regions of space. Unlike borders, which can change as a function of illumination conditions—consider the effects of a shadow falling across an object—color remains invariant as long as sufficient light is available for the cones to fire.

Therefore, it is not surprising that we have no clinical reports of patients who are "form-blind." It is reasonable to suppose that the many visual pathways provide multiple inputs to systems devoted to object recognition, one of the primary computational goals of sophisticated perception. In the next chapter we will explore how these higher level aspects of perception operate.
Each sense provides us with unique information about the world in which we live. Color is a visual experience; pitch is uniquely auditory. However, even though the information that each sense provides is distinct, the resulting representation of the surrounding world is not one of disjointed sensations, but of a unified multisensory experience. Indeed, we are often more accurate or efficient at sensory tasks when information from more than one sense is provided.

How is information from different sensory systems integrated in the brain? What mechanisms allow for sensory summation?

Multimodal Processing in the Brain

The first brain candidates for multisensory interaction are the areas where information from two or more senses converges. Neuropsychological methods are especially useful here: once the electrode has been placed in the targeted brain region, the animal can be presented with stimuli along different sensory channels. For example, some cells in the superior temporal sulcus (STS) of anesthetized monkeys respond to visual, auditory, and somatosensory stimuli. In one study, recordings were made from over 200 cells in this region, although 50% of these cells were unimodal (meaning that they responded only to one of the three sensory modalities), over 20% were bimodal or trimodal; the remaining cells did not respond to any of the stimuli (Hikosaka et al., 1988). Multisensory areas are not limited to the temporal lobe. Other brain regions showing similar sensory integration include various regions of the parietal and frontal lobes, as well as the hippocampus.

One particularly well studied multimodal site is the superior colliculus, a subcortical midbrain region. The superior colliculus participates in the control and orienting of movements. It contains orderly topographic maps of the environment in visual, auditory, and even tactile domains. These maps are integrated in the deepest layers of the colliculus (Figure 5.33).

Does the Whole Equal More Than the Sum of the Parts?

Many cells in the superior colliculus combine information from different sensory channels and integrate that information so that the sum of the multisensory input is more useful than information that could be obtained from any single modality alone. Barry Stein at Wake Forest University found that the response of individual cells in the superior colliculus was greater to a combined visual, auditory, and somatosensory stimulus than to any of the three stimuli presented alone. This phenomenon is called multisensory integration (N. P. Holmes & Spence, 2005).

Such enhanced responses are most effective when the unimodal stimuli fail to produce a response on their own. In this way, the combination of weak, even subthreshold unimodal signals can be detected and cause the animal to orient toward the stimulus. Integration effects require that the different stimuli be coincident in both space and time. For example, if a visual event is spatially and temporally synchronous with a loud noise, the resulting multisensory response will be enhanced. But if the sound originates from a different location than the light, or is not temporally synchronized with the light, the response of the collicular cell will be lower than if either stimulus were presented alone.

One interesting form of multimodal processing for humans is lip-reading. With practice, people are capable of learning to understand speech simply by watching the movements of the lips and face. Less appreciated but perhaps even more important for normal speech
comprehension, visual cues improve our ability to understand speech. When talking with someone in a noisy café, people find it much easier to understand what is being spoken if they look directly at the speaker instead of glancing about the room.

Using fMRI, Gemma Calvert and colleagues (1997), then at Oxford University in England, discovered that silent lip-reading activated areas of the auditory cortex. To explore whether this activity reflected multisensory integration, Calvert constructed speech segments in which the sounds were either matched or mismatched with the movement of the lips. In comparison to a baseline condition in which the sounds were presented alone, a region within the superior temporal sulcus of the left hemisphere became more active when the visual and auditory segments matched and became less active when the segments mismatched. Thus, she concluded that the left STS integrates information from the two sensory channels (visual and auditory), presumably using the combined inputs to derive a stronger representation of the stimulus.

**Synesthesia**

J.W. experiences the world differently than most people. He tastes words. The word *exactly*, for example, tastes like yogurt; and the word *accept* tastes like eggs. Most conversations are pleasant tasting, but when J.W. is tending bar, he cringes whenever Derek, a frequent customer, shows up. For J.W. the word *Derek* tastes of earwax!

This phenomenon, in which the senses are mixed, is known as *synesthesia*, from the Greek *syn-* ("union" or "together") and *aesthesia* ("sensation"). Synesthesia is characterized by an idiosyncratic union between (or within) sensory modalities. Tasting words is an extremely rare form of synesthesia. More common are synesthesias in which people hear words or music as colors, or see achromatic lettering (as in books or newspapers) as colored. The frequency of synesthesia is hard to know given that many individuals are unaware that their multisensory percepts are odd. Estimates range from as rare as one in 2,000 to as high as one in 200. Synesthesia tends to recur in families, indicating that at least some forms have a genetic basis (Baron-Cohen et al., 1996; Smilk et al., 2005).

Colored-grapheme synesthesia, in which black or white letters or digits are perceived in assorted colors is the best-studied form of synesthesia. A synesthete might report "seeing" the letter A as red, the letter B as yellow, and so forth for the entire set of characters, as in the example shown in Figure 5.34. The appearance of color is a feature of many forms of synesthesia. In colored hearing, colors are experienced for spoken words or for sounds like musical notes. Colored touch and colored smell have also been reported. Much less common are synesthetic experiences that involve other senses. J.W. experiences taste with words; other rare cases have been reported in which touching an object induces specific tastes.

The associations are idiosyncratic for each synesthete. One person might see the letter B as red, another as green. Although the synesthetic associations are not consistent across individuals, they are consistent over time for an individual. A synesthete who reports the letter B when tested the first time in the lab will have the same percept if retested a few months later.

The sensory stimulus that triggers the synesthetic experience is referred to as the *inducer*, and the resultant synesthetic response is the *concurrent*. In colored-grapheme synesthesia, the letter is the inducer and the color the concurrent. Inducers may be real, such as an object, a printed letter, or a musical note. They can also be imagined. Just the thought of a letter or a musical note may elicit the associated concurrent. Inducers, either real or imagined, are reported as being seen either in the synesthete's "mind's eye" or projected out to a specific location in visual space. Sometimes the concurrents for different inducers are arranged in a distinct spatial pattern (Figure 5.35).

Given that synesthesia is such a personal experience, researchers have had to come up with clever methods to verify and explore this unique phenomenon. One approach with colored-grapheme synesthesia is to create modified versions of the Stroop task. As described in Chapter 4, the Stroop task requires a person to name the color of written words. For instance, if the word *green* is written in red ink, the subject is supposed to say "red." In the synesthetic variant of the Stroop task with a colored-grapheme synesthete, the stimuli are letters and the key manipulation is whether the colors of
the letters are congruent or incongruent to the individual’s synesthetic palette. For the example in Figure 5.34, when the letter A is presented in red, the physical color and synesthetic color are congruent. However, if the A is presented in green, the physical and concurrent colors are incongruent. Synesthetes are faster to name the colors of the letters when the physical color matches the concurrent colors for the particular letter (Mattingley et al., 2001). People without synesthesia, of course, do not show this effect. To them, any color–letter pairing is equally acceptable.

It is interesting to consider the stage of processing at which the synesthetic experience emerges. Look at the array of numbers shown in Figure 5.36a. You have to scan the image for some time to see that the 2s, embedded among the 5s, form a rectangle. But suppose you have synesthesia and the color associated with 2 is different from that associated with 5. For these individuals, the perception of the square is immediate (as it is in Figure 5.36b for nonsynesthetes). Effects like this led Vilayanur Ramachandran of the University of California, San Diego, to argue that the inducer and the concurrent are associated at an early stage in perceptual processing (Ramachandran & Hubbard, 2001a, 2003).

Other researchers, however, propose that synesthesia arises at later processing stages. Jason Mattingley and his colleagues (Mattingley et al., 2001) at the University of Melbourne used a priming version of the synesthetic Stroop task to test this hypothesis. On each trial, the participant saw a letter followed by a color patch. The key question was this: In people with synesthesia, would seeing the letter interfere with naming the color of the color patch? For example, if the letter A were perceived as red, would it take longer to name a subsequent color patch that was blue? The answer was yes. When the letter was presented for 500 ms, allowing the letter to be clearly perceived, the interference effect was observed. However, when the exposure duration was reduced to 56 ms, a duration at which conscious perception was precluded, the interference effect was eliminated. Importantly, in a control task the same primes presented for the subthreshold level of 56 ms showed interference for letter naming, indicating that the letters were in fact processed consciously.

Further support that synesthesia arises at a late processing stage comes from the fact that attention can
modulate the synesthetic experience. When colored-grapheme synesthetes are presented with a large letter (say, B) made up of different small letters (say, As) (Figure 5.37), they report that the perceived color changes as attention shifts between the global form (B) and the local forms (As). Moreover, colored-grapheme synesthetes typically experience the same color across a wide range of print forms. If B is seen as green, this will hold for all Bs written in all type fonts or even if the lowercase version of the letter is used. Thus, the synesthetic bond appears to link colors to abstract representations rather than to specific shapes.

Brain-imaging studies indicate that the multisensory experience of synesthesia arises and is manifest at various stages along the visual pathway. Jeffrey Gray at King's College in London performed an fMRI study with a group of individuals who had colored-hearing synesthesia (Nunn et al., 2002). When listening to words, these individuals reported seeing specific colors; when listening to tones, they had no visual experience. Compared to control participants, the synesthetes showed increased activation in V4, similar to what we have seen in other studies of illusory color perception and in the STS, one of the brain regions associated with multimodal perception. Other studies have shown recruitment of the left medial lingual gyrus (a higher color-processing area previously implicated in color knowledge) in synesthetes during the perception of colored-grapheme synesthesia (Rich et al., 2006).

Michael Esterman and his colleagues (2006) at the University of California, Berkeley, used transcranial magnetic stimulation to test the role of different neural regions in the induction of synesthesia. They reasoned that if the concurrent colors were bound to their inducers by an attentional process (see Chapter 12), then a virtual lesion of this process might reduce the synesthetic experience. Before TMS, the participants were given a synesthetic Stroop test, and as found previously, the synesthetes were slower to respond when the physical color of the letter did not match their own concurrent. Stimulating the right posterior parietal cortex with 1-Hz repetitive TMS abolished the interference effect for about 5 minutes. A similar effect was not found when the TMS was targeted to left parietal cortex or primary visual cortex. These results indicate that, even though the concurrent percepts are well established in synesthetes, their association with the inducers can be transiently disrupted by stimulation over neural regions thought to play a role in multidimensional feature integration.

**Perceptual Reorganization**

As we have just seen, people with synesthesia provide a dramatic example of how the brain is able to link information between distinct sensory systems. The extent of the connectivity between sensory systems is also revealed by studies on people who are deprived of input from one of their senses. When a person is blind, what happens to those regions of the brain that are usually used for visual perception? Might this unused neural tissue be able to reorganize to process other information? Is the situation for individuals who have been blind since birth different from that of individuals who became blind after having had vision?

The results of a PET study suggest a remarkable degree of functional reorganization, or what neuroscientists refer to as cortical plasticity (Sadato et al., 1996). The subjects for this study included people with normal vision and people who were congenitally blind—that is, blind from birth. The subjects were scanned under two experimental conditions. In one condition the subjects were simply required to sweep their fin-
gers back and forth over a rough surface covered with dots. In the second condition they were given tactile discrimination tasks such as deciding whether two grooves in the surface were the same or different. Blood flow in the visual cortex during each of these tasks was compared to that during a rest condition in which the subjects were scanned while keeping their hands still.

Amazingly, changes in activation in the visual cortex were in opposite directions for the two groups of subjects. For the sighted subjects, a significant drop in activation was found in the primary visual cortex during the tactile discrimination tasks. Analogous decreases in the auditory or somatosensory cortex occurred during visual tasks given to normal subjects; therefore, as attention was directed to one modality, activation (as measured by blood flow) decreased in other sensory systems. In contrast, in blind subjects the activation in the primary visual cortex increased—but only during discrimination tasks, and not when they swept their fingers over the surface without having to actively use tactile information. Interestingly, a second group of subjects who had become blind early in childhood (before their fifth year) also showed the same recruitment of visual cortex when performing the tactile discrimination task.

A second experiment explored the same issue but used a task that is of great practical value to the blind: reading Braille. The subjects explored strings of eight Braille letters and had to decide whether the strings formed a word. In accord with the results of the first study, activation of the primary and secondary visual cortex increased during Braille reading in comparison with the rest state, but only in the blind subjects.

Of course the term visual cortex is a misnomer with blind individuals. These results indicate that tissue that, during normal development, will become sensitive to visual inputs can be exploited in a radically different manner when the environmental context is changed—for example, when all visual input is lost. At present, it is unclear how tactile information ends up activating neurons in the visual cortex of blind people. One possibility is that somatosensory projections to thalamic relays spread into the nearby lateral geniculate nucleus, exploiting the geniculostriate pathway. This hypothesis is unlikely, since the activation changes in the blind subjects’ visual cortices were bilateral. Somatosensory inputs to the thalamus are strictly lateralized. Because the subjects performed the tactile tasks with the right hand, the blood flow changes should have been restricted to the left hemisphere. A more viable hypothesis is that a massive reorganization of corticocortical connections follows peripheral blindness. The sensory-deprived visual cortex is taken over, perhaps through back-projections originating in polymodal association cortical areas.

Although this study provides a dramatic demonstration of cortical plasticity, the results also suggest a neurobiological mechanism for the greater nonvisual perceptual acuity exhibited by blind people. Indeed, Louis Braille’s motivation to develop his tactile reading system was spurred by his belief that vision loss was offset by heightened sensitivity in the fingertips. One account of this compensation focuses on nonperceptual mechanisms. Though the sensory representation of somatosensory information is similar for blind and sighted subjects, the former group is not distracted by vision (or visual imagery). If the focus of attention is narrowed, somatosensory information can be used more efficiently. The imaging results reviewed here, though, suggest a more perceptual account. Sensitivity increases because more cortical tissue is devoted to representing nonvisual information.

The five basic sensory systems of audition, olfaction, gestation, somatosensation, and vision allow us to interpret the environment. Each sense involves unique pathways and processes to translate external stimuli into neural signals that are interpreted by the brain. Within each sense, specialized sensory mechanisms have evolved to solve computational problems to facilitate and enhance our perceptual capabilities. As shown in neuroimaging and neuropsychological studies, specialization is found across the sensory cortices of the brain; thus, people may retain the ability to see, even in the absence of cortical mechanisms for color or motion perception. In extreme situations of sensory deprivation, the cortical systems for perception may become radically reorganized. Even in people with intact sensory systems, the five senses do not work in isolation, but rather work in concert to construct a rich interpretation of the world. It is this integration that underlies much of human cognition and allows us to survive, and indeed thrive, in a multisensory world.
achromatopsia
akinetopsia
area MT
area V4
blindsight
chemical senses
cochlear nucleus
corpuscle
cortical visual areas
extrastriate visual areas
glomeruli
hemianopia
inferior colliculus
interaural time
lateral geniculate nucleus (LGN)
medial geniculate nucleus (MGN)
multisensory integration
nociceptors
odorant
photoreceptors
primary auditory cortex (A1)
primary gustatory cortex
primary olfactory cortex
primary somatosensory cortex (S1)
primary visual cortex (V1)
proprioception
retina
scotoma
secondary somatosensory cortex (S2)
superior colliculus
synesthesia
tastant
thalamus

KEY TERMS

General
• Much of the brain is involved in the representation of information acquired from the different sensory receptors.

Audition
• Signal transduction from sound wave to neuronal signal begins at the eardrums. Signals are processed in the hair cells and basilar membrane of the cochlea, and the cochlea sends its information in the form of neuronal signals to the inferior colliculus and the cochlear nucleus. Information then travels to the medial geniculate nucleus of the thalamus and on to the primary auditory cortex.
• Sound localization is aided by the processing of differences in interaural time and interaural sound intensity, which are each coded separately in the brain.

Olfaction
• Signal transduction from odorant to neuronal signal begins when the odorant attaches to an odor receptor in the olfactory epithelium. The signal is then sent to the olfactory bulb through the olfactory nerve, which synapses on the primary olfactory cortex. Signals are also relayed to the orbitofrontal cortex, a secondary olfactory processing area.
• The primary olfactory cortex is important for detecting a change in external odor, and secondary olfactory cortex is important for identifying the smell itself.
• Similar to the importance of sampling sound from two ears, we use our two nostrils to obtain different olfactory samples, varying the rate of air flow through each nostril and thus altering the rate of absorption.
• The anatomical proximity and neural interactions between olfactory cortex and the limbic system likely account for why smells can trigger vivid memories.
• The olfactory pathway is the only sensory pathway that does not send information to the thalamus.

Gustation
• Gustation and olfaction are known together as the chemical senses because the initial response is to molecules (chemicals) in the environment.
• The five basic tastes are salty, sour, bitter, sweet, and umami. The perception of more complex tastes arises from the complex cortical processing of these individual tastes in areas of the brain such as the secondary gustatory cortex in the orbitofrontal region.
• Orbitofrontal cortex is also involved in processing the reward value of food and the resulting motivation to eat food.

Somatosensation
• Corpuscles located in the skin respond to somatosensory information such as touch, pressure, and temperature.
• Nociceptors (free nerve endings) respond to pain information.
• Nerve cells at the junctions of muscles and tendons provide proprioceptive information.
• Primary somatosensory cortex (S1) contains a homunculus of the body, with the more sensitive regions encompassing relatively larger areas of cortex.
• Bilateral secondary somatosensory cortex (S2) receives sensory information from both sides of the body, allowing for cross talk of sensory information.

**Vision**

• Photoreceptors (rods and cones) in the retina translate light into neural signals.
• The three types of cones are sensitive to different regions of the visible spectrum and are used for color perception; rods are very sensitive even under low levels of illumination but are not color sensitive.
• The fovea, at the center of the retina, is densely packed with cones; the periphery of the retina contains mostly rods.
• Light hits the retina and travels within the retina from rod or cone to bipolar cell to ganglion cell. The optic nerve is formed from the axons of the ganglion cells, some of which decussate at the optic chiasm. Axons in the optic nerve synapse on the LGN and from the LGN become the optic radiations that are sent to V1. Note that 10% of the fibers from the retina innervate non-LGN subcortical structures, including the pulvinar and superior colliculus.
• Visual cortex is made up of many distinct regions, which carry out specialized processing functions. For instance, cells in area V4 are sensitive to color information and cells in V5 are sensitive to motion information.
• Achromatopsia, the inability to perceive color results from lesions to areas in and around human V4. However, these regions do not just represent color, but also are important for shape perception. Color is one attribute that facilitates the perception of shape.
• Akinetopsia, the inability to process motion, results from lesions to area V5 (human MT).
• Superior colliculus lesions impair the ability of an animal to orient toward the position of a stimulus (which is important for spatial orientation); visual cortex lesions impair visual acuity (which is important for object identification).
• In some cases, patients with lesions of the visual cortex exhibit blindsight, the ability to locate the position of a stimulus even when they are unaware of its presence. Blindsight may occur because the information reaching the superior colliculus is sufficient to indicate position or extrastriate regions are activated without input from the primary visual cortex.

**Multimodal Perception**

• Some areas of the brain, such as the superior colliculus and superior temporal sulci, process information from more than one sensory modality, integrating the multimodal information to increase the sensitivity and accuracy of perception.
• When multisensory information is presented coincidently in time and space, the multisensory neural response is enhanced. The reverse is also true; that is, when multisensory information is not presented coincidently in time and space, the multisensory neural response is depressed.

**Synesthesia**

• People with synesthesia experience a mixing of the senses, like colored hearing, colored graphemes, or colored taste.
• The stimulus that triggers synesthesia is the inducer, and the synesthetic response is the concurrent.
• There is some debate about where along the processing stream synesthesia is processed—either early (preconsciously) or late (postconsciously).

**Perceptual Reorganization**

• Following sensory deprivation, the function of sensory regions of the cortex may become reorganized, or exhibit what is called plasticity. For instance, in blind individuals, areas of the brain that are usually involved in visual function may become part of the somatosensory cortex.

**THOUGHT QUESTIONS**

1. Imagine watching a short video segment in which a large, purple dinosaur appears briefly in the left visual field. Trace the flow of information about this stimulus and its separate features (color, shape, luminance, motion, position) from the eye through the secondary visual areas.

2. Compare and contrast the functional organization of the visual and auditory systems. What are the computational problems that each system must solve, and how are these solutions achieved in the nervous system?

3. A person arrives at the hospital in a confused state and appears to have some impairment in visual perception. As the attending neurologist, you suspect that the person has had a stroke. How would you go about examining the patient to determine the level in the visual pathways at which the damage has occurred? Emphasize the behavioral tests you would
administer, but feel free to make predictions about what you expect to see on MRI scans.

4. Define the physiological concepts of receptive field and visual area. How is the receptive field of a cell established? How are the boundaries between visual areas identified? Can either receptive fields or visual areas be studied noninvasively in humans?

5. This chapter has focused mainly on salient visual properties such as color, shape, and motion. In looking around the environment, do these properties seem to reflect the most important cues for a highly skilled visual creature? What other sources of information might an adaptive visual system exploit?

SUGGESTED READING


