

THE HUMAN EYE

Differences During World War II, the United States Navy wanted its sailors to see infrared signal lights that were invisible to the enemy. Normally, we humans find it impossible to see infrared radiation because the receptors in our eyes are insensitive to infrared energy—as far as our eyes are concerned, infrared energy might as well not be there. To make infrared signals visible, Navy scientists had to come up with a way to modify the sailors’ night vision. These scientists knew that the eye’s receptors contained specialized light-sensitive molecules, and that a portion of each molecule was derived from vitamin A. Knowing that vitamin A comes in different forms, the Navy scientists wondered whether one of the alternative forms might encourage production of photosensitive molecules sensitive to infrared radiation. To find out, they had human volunteers eat diets supplemented by an alternative form of vitamin A (extracted from the livers of walleyed pikes). Over several months, the volunteers’ vision changed remarkably, boosting their sensitiv-

ity to light extending into the infrared region. The experiment seemed to be working. At the same time, however, other scientists were successfully developing the snooperscope, an electronic device for seeing infrared radiation. This invention made it unnecessary to alter the eye itself, and the experiment was aborted (Rubin and Walls, 1969). Still, the project dramatizes a truth that we’ll document throughout this chapter: the structure of the eye governs what we can, and cannot, see. Altering that structure would literally change your vision of the world.

The visual system of any vertebrate, humans included, consists of three major components: *eyes*, which capture light and convert it into neural messages; *visual pathways*, which modify and transmit those messages from the eye to the brain; and *visual centers of the brain*, which interpret the messages in ways useful for guiding behavior. All three components are crucially involved in seeing, so each component’s structure and function must be understood in order to comprehend how an

organism sees. This chapter and the next one concentrate on the first of these components, the human eye; they discuss its anatomy (structure) and physiology (how it works). These chapters emphasize how the eye is built, how it captures light and how it turns that light into neural messages the brain can interpret. Chapter 4 will discuss the remaining two major sections of the mammalian visual system, the visual pathways to the brain and the brain's visual centers.

Each of these three chapters has features that require special comment. First, we don't spend time talking about anatomy simply because we are fascinated with structure per se. Structure is important because it influences how and what we can see. Second, although mainly interested in the *human eye*, we also consider the eyes of other animals, particularly animals whose environments and lifestyles differ from those of humans. Understanding the diversity of vision—and the uniqueness of human vision—will heighten your appreciation of the processes involved in seeing. Finally, we also consider how various defects impair vision. Besides being fascinating in their own right, visual defects illuminate the intimate connection between structure and function.

In writing these chapters we were very much influenced by Gordon Walls's book *The Vertebrate Eye and Its Adaptive Radiations* (1942). Walls wrote eloquently about the eye, as the following statement demonstrates:

“Everything in the vertebrate eye means something. Except for the brain, there is no other organ in the body of which that can be said. It does not matter in the least whether a liver has three lobes or four, or whether a hand has five fingers or six, or whether a kidney is long and narrow or short and wide. But if we should make comparable changes in the makeup of a vertebrate eye, we should quite destroy its usefulness. Man can make optical instruments only from such materials as brass and glass. Nature has succeeded with only such things as leather and water and jelly; but the resulting instrument is so delicately balanced that it will tolerate no tampering.” (Walls, 1942, pp. iii–iv)

Walls' credo underscores that we cannot understand vision without first understanding the eye's structure. We'll begin our actual discussion of the eye with general questions about the nature of vision—*why* vision took the form that it did.

DESIGNING THE ORGAN OF VISION

The Diversity of Eyes in Nature

Chapter 1 distinguished between near senses and distance senses, putting vision in the latter category. By definition, a distance sense allows you to detect and recognize objects without having to come in immediate proximity to those objects. Both hearing and seeing endow us with this capacity. But vision enjoys a major advantage over hearing: it allows us to sense objects that make no sound. (Hearing offers its own special advantages, including the ability to register the presence of things that cannot be seen—but we'll get to that in Chapters 10 and 11.) Vision also provides important information about objects, information that's simply beyond the reach of hearing. It's sight, not hearing, that can tell us about the color of a piece of fruit, the shape of a cup we're about to grasp, the likelihood of threat posed by an approaching stranger and countless other bits and pieces about objects and events in our environments.

To appreciate vision's power, all we have to do is look at the diversity of creatures that possess eyes. Many species, including varieties of birds and fish, have eyes that follow the same basic design as ours, and are every bit as complicated. Other species, however, possess eyes of a very different structure. Some insects, such as butterflies, have eyes consisting of hundreds of tiny camera-like lenses each focusing light on its own cluster of light-sensitive cells. This array of miniature optical systems is packaged in a single large eye, sometimes mounted on the end of a long stack protruding from the creature's head. These so-called compound eyes work very differently from our eyes, with their single optical system (Figure 2.1). But those species with com-



Differences



FIGURE 2.1

Examples of the variety of eyes in nature.

Cat © PhotoDisc website; Gecko © Gregory G. Dimijian, M.D., 1988/Photo Researchers, Inc.; Owl © PhotoDisc website; Iguana © Stephen Dalton/Photo Reserchers, Inc.

pound eyes allow their owners to see what is important to them. In still other species, including some worms, “eyes” consist of nothing more than a patch of photosensitive pigment somewhere on the surface of the skin. By reacting to the presence of light, these light-sensitive spots tell the creature whether it’s day or night. It’s arguable whether we should refer to this as “vision” but there’s no doubt that these creatures are sensing light. In fact, patches of photosensitive pigment in these kinds of primitive creatures probably represent the precursors for the variety of eyes found in other, more advanced animals.

Take a look at the unusual caterpillar pictured in Figure 2.2. If you didn’t know better, you’d swear it was staring at you. That’s exactly what natural selection—the designer of this caterpillar—wants you to think. This menacing “eye,” however, is nothing more than pigmentation on the caterpillar’s back. Its real eyes are those tiny pinholes located on the side of its body. This adaptive ornamentation serves as a deterrent to potential predators, alerting them to be wary about trying to sneak up on the caterpillar. This caterpillar with its mesmerizing eye brings to mind the title character in Samuel Taylor Coleridge’s “The Rime of the



FIGURE 2.2
Pigmented spots on the back of a caterpillar resemble eyes. (Edward Wester)

Ancient Mariner,” an old seafarer: “He holds him with his glittering eye.” These examples from nature and literature remind us that eyes, besides being the organ of sight, also communicate unmistakable messages to others looking at us.

It is estimated that natural selection has independently “reinvented” the sense of vision dozens of times throughout the animal kingdom (Mayr, 1982). Each time vision has evolved, the process has been lengthy and complicated. Indeed, the complex structure of the human eye has led some to conclude that it simply couldn’t have arisen by natural selection. Instead, they argue, the grand design of the eye reveals the hand of God in creation. We believe, however, that natural selection alone represents the “designer” of eyes (Dawkins, 1996). But our purpose here isn’t to quarrel about the origin of eyes. Rather, we wish to instill in you an appreciation for the quality and service provided by that design. Toward that end, we must start with some basics about light, the messenger

that bridges the distance between you and the objects you see.

Why Have Eyes That Use Light?

Light is just one form of **electromagnetic radiation** (see Figure 2.3). Other familiar varieties include radio waves, infrared and ultraviolet radiation, microwaves, and x-rays. All these forms of energy are produced by the oscillation of electrically charged material. Since virtually all matter consists of oscillating (that is, wavelike) electrical charges, electromagnetic energy exists in abundance. An animal that can sense electromagnetic radiation benefits in two important ways.

First, electromagnetic radiation travels very rapidly (in empty space, at 186,000 miles, or approximately 300,000 kilometers, per second). Any creature able to detect such radiation can pick up information from distant sources with minimal delay. Thus, we receive optical information about objects and events almost instantaneously (except, of course, for extra-terrestrial events transpiring millions of miles away). Second, electromagnetic radiation tends to travel in straight lines. This means that images created by this radiation retain important geometrical characteristics of the objects that reflect that radiation toward the eyes. We’ll return to this point in a moment.

The frequency of electromagnetic radiation depends on the emitting material’s mode of oscillation. In fact, electromagnetic radiation can be scaled (or arranged) along a spectrum according to the frequency of oscillation. This frequency, or oscillation rate, of light energy can be converted into units termed wavelengths. **Wavelength** is defined by how far the radiation travels between oscillations. High rates of oscillation mean that radiation travels a very short distance between oscillations—hence a short wavelength. Figure 2.3 underscores an important point: light, the form of radiation on which we depend for sight, occupies only a very small portion of the

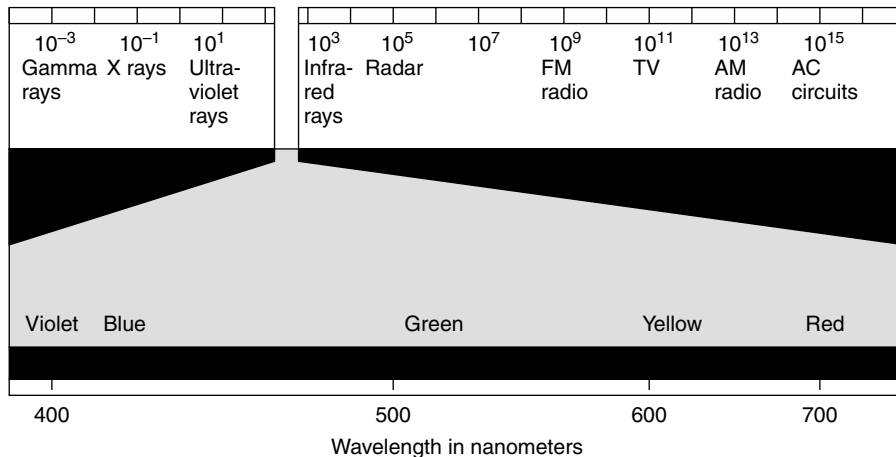


FIGURE 2.3

The spectrum of electromagnetic energy. The region containing visible light is shown enlarged.

electromagnetic spectrum. Actually, radiation from other portions of this spectrum *could* have been used to bridge the distance between the perceiver and objects of visual perception. So why do the eyes rely solely on this one, very narrow portion of the entire electromagnetic spectrum, the part we call light?

One reason the eyes use light as their medium is that there is a lot of it in the world, courtesy of our solar system's sun. That abundance ensures that terrestrial creatures will have ample opportunity to use their light-sensing apparatus. It wouldn't have made much sense for early vertebrate "eyes" to depend on wavelengths in the ultraviolet range, for example, because most (but not all) of sunlight's energy in this short wavelength region of the spectrum is absorbed by molecules in the earth's atmosphere (mainly nitrogen and oxygen). Consequently, most of the sun's ultraviolet rays energy never reach objects in our immediate environment. Second, light is useful as a medium of information about the world because light interacts with the surface molecules of many objects we're interested in seeing. These interactions, in the form of reflec-

tion and absorption, allow light to convey information not only about the presence and absence of objects, but also about the structure of those objects and their surfaces (Gibson, 1966). Energy from outside the "light" portion of the electromagnetic spectrum—because of the length of the constituent waves—interacts very differently with solid objects that are relevant to behaving organisms. For example, longer wavelengths—including microwave energy—penetrate opaque objects rather than being reflected by them. (By penetrating them, microwaves can more evenly heat objects and therefore are useful in cooking.)

To sum up, eyes are a good idea. And eyes that use light are an even better idea.

The Message and the Messenger: Inverse Optics

As we progress through this discussion of vision, keep in mind that light is the *carrier* of visual information. It delivers messages from environmental objects to your eyes. But light itself is seldom the message of interest. It's the objects we want to see.

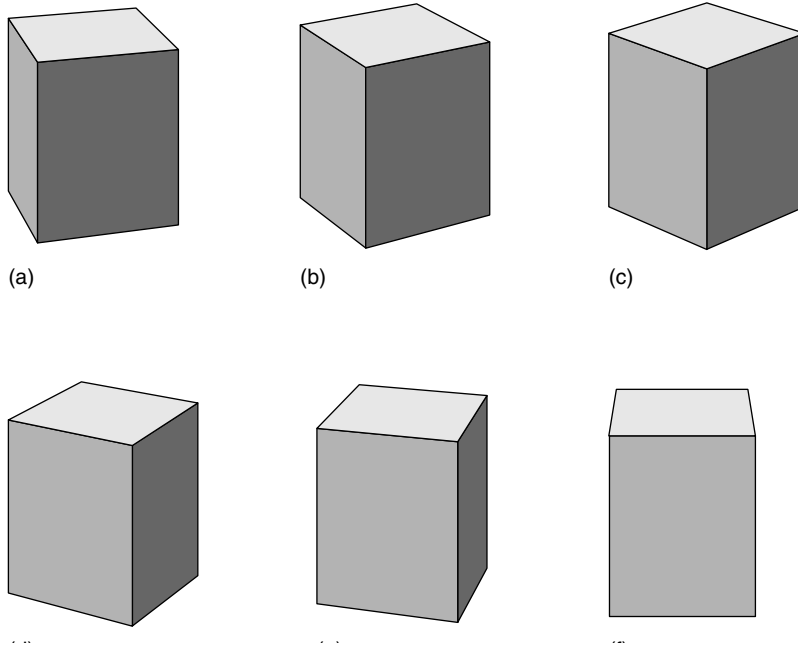


FIGURE 2.4

Images on the back of the eye change when you vary your angle of vision.

In a nutshell, the eye receives patterns of light energy reflected from the surfaces of objects in the environment. Those patterns of energy depend on a host of factors, including the surface properties of objects, the distance from the eye to those objects, and the source of light illuminating the objects. Whenever you change the angle of vision between yourself and an object, for example, you alter the pattern of light energy falling on your eyes (Figure 2.4). Whenever you view an object in natural sunlight and then move indoors to look at it under artificial lighting, you change the wavelength composition of light energy arriving at your eyes (artificial light isn't identical to sunlight). In short, the same object can convey countless different optical messages to your eyes.

So how do the eyes and brain figure out what produced some particular pattern of light energy? That's really what these next three chapters are all about. You will learn how the eye and brain are

able to work backward, or inversely, starting with patterns of light and culminating in descriptions of objects in the world. This backward approach, which vision accomplishes automatically and effortlessly, is called **inverse optics**.

As you will learn, solving the inverse optics problem requires that the eye and brain make some assumptions about the objects and events we're likely to encounter in our environments. Fortunately, those assumptions are fairly safe ones because the physical nature of matter imposes certain constraints on the properties of objects. For example, light cannot pass through opaque objects, which means that when one object partially occludes another, we automatically see the partially occluded one at a greater distance than the occluding object (this constraint is discussed in more detail in Chapter 8). In addition, those constraints don't change over time. Nature has exploited this stability to develop

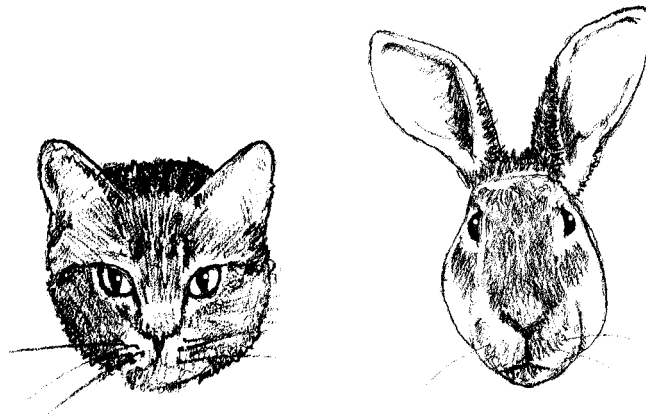


FIGURE 2.5

The placement of the eyes in the head of a cat (frontal eye placement) and in the head of a rabbit (lateral eye placement).

shortcuts for solving the problem of inverse optics. We'll be describing some of these shortcuts throughout this book.

Where Should the Eyes Be Placed?

Recognizing that animals would do very well to have eyes that exploit light, we must now decide *where* those eyes should go. Because embryologically the eyes are an outgrowth of the brain, their most-natural location is in the head, near the brain. But where exactly in the head should the eyes go? Nature has devised several different ways to position the eyes. In vertebrates, there are two popular designs for outfitting the head with a pair of eyes: they can be located in a *frontal* position, as are those of a human being or a cat; or they can be located in a *lateral* position, as are those of a rabbit (see Figure 2.5). Each strategy carries its own advantages: frontal eyes improve depth perception (as discussed in Chapter 8), whereas lateral eyes make it possible to take in more of the visual world at one time. As a rule, predatory animals—those who hunt and eat other animals—have frontally placed eyes. Those who are prey—animals frequently taken as food—

have laterally placed eyes. In other words, those needing excellent depth perception to stalk and capture have considerable binocular overlap of the two visual fields. Those needing a more panoramic view of the environment in order to watch for predators have little binocular overlap.

Why Should the Eyes Be Able to Move?

Because we humans lack panoramic vision, what we see at any given moment is rather limited. (see Figure 2.6). And there is no guarantee that our eyes will always be directed toward things in the environment that we need to see. Fortunately, we can compensate for our relatively narrow field of vision by turning the head and eyes. This is what you do when you look both ways before crossing a street. You also move your eyes because they are not uniformly sensitive. Some parts of the eye provide much better detail vision than other parts. Think for a moment what you do when something suddenly appears out of the corner of your eye. To see exactly what has grabbed your attention, your eyes reflexively move so that you're looking directly at whatever caught your attention.

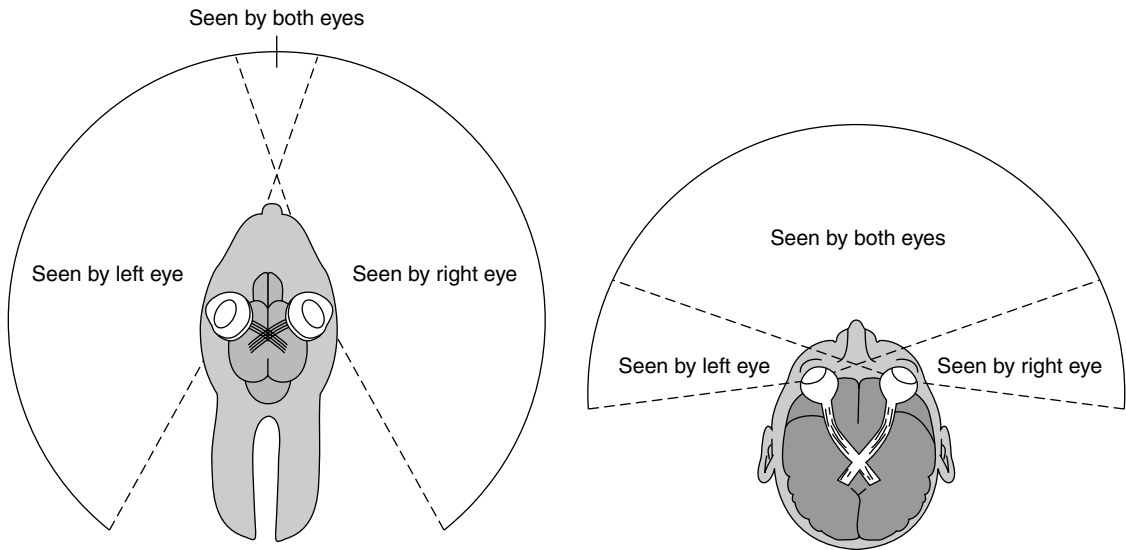


FIGURE 2.6

The extent of visual field of view for a rabbit and for a human. Note that the rabbit has almost completely panoramic vision, whereas the human's field of view encompasses only about 180 degrees.

D

Differences

Animals use various strategies for changing their field of view: they can move their bodies, turn their heads, or merely move their eyes. Some animals—owls for one—have very limited ability to move their eyes (Steinbach and Money, 1973). This comes about because their eyes are large and fit tightly into their sockets (Walls, 1942, p. 212). Rather than depending on eye movements, the owl moves its entire head. From a human standpoint, the owl's method is slow and inefficient.

Not all birds are our inferiors when it comes to eye movements, however. For example, the European starling, now so numerous as to be a nuisance in most North American and European cities, owes some of its success to its eye movements. Most birds, as they forage on the ground, need to look sideways in order to see what they're about to peck at. But starlings, whose skulls are quite narrow and whose eyes are extremely mobile, can turn their eyes far enough forward so that they can actually see between their opened

beaks. Equally important (and amazing) is the starling's ability to instantly swing its eyes up and back to scan the skies behind its head for possible predators (Martin, 1987). Because it can watch for predators without having to turn its head, the starling remains relatively unobtrusive, another element of protection. With eye movements so well designed both for fruitful foraging and self-protection, it's no wonder that in a mere one hundred years the starling population of North America has gone from approximately 100 European imports to more than 200 million birds.

While we humans can't match the starling's ability to shift its eyes without moving its head, we can shift the position of our eyes with enormous speed. For instance, it takes less than one-fifth of a second for our eyes to turn from their extreme leftward position to their extreme rightward position. When we execute this movement, our eyes are briefly traveling 700 degrees per second! (To make this figure more concrete, think of the moon's rotation. If the moon rotated at this

speed, it would complete two revolutions in one second.) Not only do the eyes move rapidly, they move with great accuracy as well. For example, as you read these lines, your eyes skip along from one place of interest to another, alighting with great precision on the desired letter or space. The cooperative interaction among the **extraocular muscles** (six for each eye) makes this rapid and accurate eye movement possible. The extraocular muscles enable movement of the eyes in all directions. Let's consider the mechanical arrangement that makes this possible.

How the Eyes Move Every muscle in your body works by contracting and thereby pulling on the structure or structures to which the muscle is attached. In the case of the extraoculars, each muscle is connected at one end to an immovable structure, the eye socket of the skull, and at the other end to an object that is free to move, the eyeball. So when an extraocular muscle contracts, it pulls on the eyeball and moves it. The *amount* of movement depends on the strength of the muscle's contraction and on the action of the other muscles. The *direction* of movement depends on the place at which the contracting muscle is attached to the eyeball and skull, and on what the other muscles are doing. Because each extraocular muscle is attached to the eyeball at a different position, contraction of any particular muscle turns the eyeball in a characteristic direction. The following is a brief and simplified description of what the extraoculars do.

Each eye's muscles can be divided into two groups, one with four muscles and one with two. The larger group, the **rectus muscles**, run straight back from the eyeball. Muscles in the other, smaller group run obliquely back from the eyeball. We can understand the general principles of the eye's movements by looking just at the muscles in the larger group.

Each rectus muscle is attached to the eyeball at a different location, toward the front of the eyeball (see Figure 2.7). The other end of each rectus muscle is attached to the rear of the bony cav-

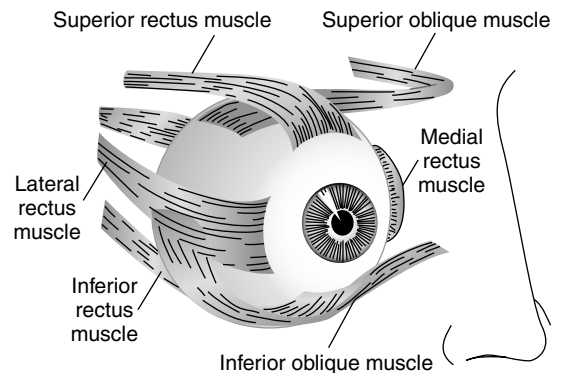


FIGURE 2.7

A view of the eye muscles attached to the right eye. The medial rectus, the muscle closest to the eye, is partially obscured by the globe.

ity holding the eyeball; this is the immovable end of the muscle. Whenever a rectus muscle contracts, it pulls the eyeball toward the place at which that muscle connects to the eyeball. When a rectus muscle relaxes, the eye turns back toward its original position.

One muscle, the *medial* rectus, attaches to the side of the eyeball closest to the nose. When it contracts, the medial rectus rotates the eye toward the nose. Another muscle, the *lateral* rectus, has exactly the opposite effect. It is connected to the side of the eyeball farthest from the nose, so its contraction turns the eyeball laterally, away from the nose. The *superior* rectus muscle connects to the top of the eyeball, and its contraction elevates the eyeball, causing you to look upward. The superior's opposing muscle, the *inferior* rectus, is attached to the lower portion of the eyeball and its contraction lowers the eye, causing you to look down.

Now let's consider how these muscles cooperate to move the eyes. Imagine that while looking straight ahead, you decide to glance leftward. Both eyes must move to the same degree and in the same direction. In order for you to look to the left, the medial rectus of the right eye and the lateral rectus of the left eye must both contract,

while both the lateral rectus of the right eye and the medial rectus of the left eye relax. You should be able to figure out for yourself what will happen if you now decide to glance rightward.

For the eye movements just described, both eyes have moved in the same direction—upward, leftward, and so on. Eye movements of this type are called **conjunctive** eye movements. But the eyes are capable of other types of movements as well. The eyes can move in opposite directions—both may turn inward or both may turn outward. These are called **vergence** eye movements. For example, the left eye can turn rightward while the right eye turns leftward. As a result, both eyes turn inward, toward the nose. This movement aims the two eyes at a very close object straight ahead of you. This particular type of vergence movement is called a convergent eye movement. To accomplish it, the medial rectus muscles of both eyes contract, while the lateral rectus muscles of both eyes relax. If you look at an object at arm’s length and then bring it closer to you, your eyes converge, tracking the object. When the object moves away from you, your eye muscles will engage in the opposite behavior, resulting in a divergent eye movement.



Execute

Much more can be said about eye movements and their role in vision, particularly in reading, and we’ll return to these roles in later chapters. But for now, we want to stress that eye movements alone are insufficient to compensate for our limited field of view. To experience this for yourself, try a simple exercise. Hold your head very steady, close one eye and then look around in all directions by moving your open eye. Notice how limited this monocular field of vision really is. Repeat the exercise using your other eye. When the famous Austrian scientist and philosopher Ernst Mach (1838–1916) performed this exercise, he made a sketch of what he saw. Figure 2.8 shows you what Mach drew. Mach firmly believed that scientists should confine themselves to descriptions of phenomena that can be perceived by the senses. In this particular case, Mach’s senses revealed a seldom realized truth: Because we’re accustomed to moving our eyes

and heads, we seldom realize how limited our actual view of the world is at any given moment. Our grasp on the whole of visual reality comes in bits and pieces assembled over time.

How Should the Eyes Be Protected?

Vertebrate eyes are fragile, complicated devices occupying a very exposed position in the head. Fortunately, various protective measures have evolved to compensate for the eyes’ vulnerability. An outline of these protective mechanisms will provide a good introduction to the overall structure of the eye.

The eye is partially protected by virtue of its location within the **orbit**, a bony depression in the skull. Within the orbit, the eye is cushioned by heavy deposits of fat surrounding each eyeball. Without this orbital fat, blows to the head would be transmitted directly to the eye. But by absorbing such shocks, orbital fat cushions the eye against all but the most severe jolts.

The eye is also protected by eyelids, movable folds of tissue. The position of the upper lid relative to the lower one determines the opening through which the front of the eye is visible. The lids move in concert with one another in various modes. For instance, in a fraction of a second, they can open and close (blink). Such blinks usually occur spontaneously, without thought. Blinks clean and moisten the front of the eye to keep it from drying out. The lids can also execute reflexive blinks that protect the eye when it is touched by a foreign object or when an object is on a collision-course with the head. In addition to involuntary and reflexive blinks, we can also voluntarily blink our eyes or, for that matter, close them entirely when we want to go to sleep.

Under normal circumstances, a blink occurs about once every four seconds (Hart, 1992). This figure varies from person to person, depending on emotional state and the environmental conditions. When the air is very dry, we tend to blink our eyes more often, ensuring that the delicate front surface of the eye doesn’t dry out.



FIGURE 2.8
Ernst Mach's view of his study.

A blink, measured from the instant the lids begin to close until they reopen, takes roughly one-third of a second. For about half this time, the lids are completely closed, reducing the light by over 90 percent (Crawford and Marc, 1976). If the room lights were momentarily dimmed by this amount, the resulting blackout would be very noticeable. Why is it, then, that we never notice the same blackout when it's caused by a blink?

Volkman, Riggs, and Moore (1980) offered an intriguing answer to this question. They hypothesized that when the brain signals the lids to close, it also produces an ancillary neural signal that suppresses, or temporarily shuts off, vision for the duration of the blink. Suppression would keep you from noticing that a blink had occurred. This hypothesis, although intriguing, is difficult to test. The appropriate test would be to measure

the eye's sensitivity to light during a blink without the lid closure actually affecting the intensity of the eye's exposure to that light. Volkmann and colleagues developed the following ingenious way to stimulate the eye so that the light reaching the retina would not be affected by lid closure.

The eyes lie directly above the roof of the mouth. Thus, a strong light focused on the roof of the mouth under one eye can penetrate this tissue and stimulate the retina—you're actually able to see light delivered through the roof of your mouth whether or not your lids are closed. This procedure makes it possible to measure the ability to see a dimming of the light at various times relative to a blink.



Execute

Volkmann and colleagues used a bundle of optical fibers to carry light to the roof of a person's mouth. By abruptly dimming the light, they determined the smallest reduction in light intensity visible to that person. Remember: All the light that could be seen came through the person's mouth, bypassing the lids. At the same time, Volkmann and colleagues used electrodes attached to the skin near the eyelids to monitor when blinks occurred. Since a blink is produced by muscle contractions, it's easy to detect the electrical activity of the lid muscles and know from that activity when a blink begins and ends. These researchers found that dimming was much harder to see *during* a blink than *between* blinks. To be detected during a blink, the light had to be dimmed by an amount 5 times greater than it did between blinks.

These results suggest that the brain suppresses vision just before and during each blink, keeping us from noticing the visual blackouts. Without this suppression, which occurs in tandem with the typical blink rate of 10 to 15 times per minute, we would be bothered by profound blackouts (Riggs, Volkmann and Moore, 1981). To our relief, the visual system uses a temporary, well-timed suppression to protect us from the annoying but necessary behavior of our eyelids.

Our social environment also affects the behavior of our eyelids. We tend to blink more often dur-

ing casual conversation than when we're alone. And during a heated discussion, our blink rate skyrockets. In addition to our blink rate, the size of the opening between the lids provides a reliable clue as to how interested your listener is. Watch a friend's eyes closely; the opening between the lids will average 8 millimeters (about 1/3 inch). You'll probably notice a change in that opening as your friend's attention varies. When interest is high, the opening between the lids increases to about 10 millimeters. Similarly, drowsiness or boredom reduces the size of the opening. And, of course, we immediately recognize the social message conveyed when someone looks directly at us and gives an exaggerated blink with one eye. A wink speaks volumes.

Tears also convey social messages—sadness, pain, great joy, etc. But tears protect the eyes as well. Although usually associated either with emotional states or with slicing raw onions, tears irrigate the eyes' exposed surfaces. Tears are secreted from a gland situated in the upper, front portion of each orbit (under the upper lid). From there, they pass down over the eye's front surface, moistening it, and then drain through small openings in the lower nasal portion of each orbit. Finally, the tears drain onto the mucous membrane lining the nose's inner surface. This nasal membrane acts as an evaporator for the tears, which explains you have to blow your nose when you've been crying. Tears contain an antimicrobial agent that helps protect the eye from bacteria present in the environment. In addition, the regular flow of tears flushes away debris, such as dust. Tears also lubricate the surface of the eyes so that blinking won't abrade the lids or scratch the front of the eyeball. The constant, very thin film of tears over the front of the eye minimizes the wear and tear produced by constant lid movement. In many older adults, the secretion of tears slows down, producing a condition called "dry eyes." Fortunately, the regular application of ophthalmic drops to the eyes provides relief.

Having covered its ancillary features, we are now ready to discuss the structure of the eye itself.

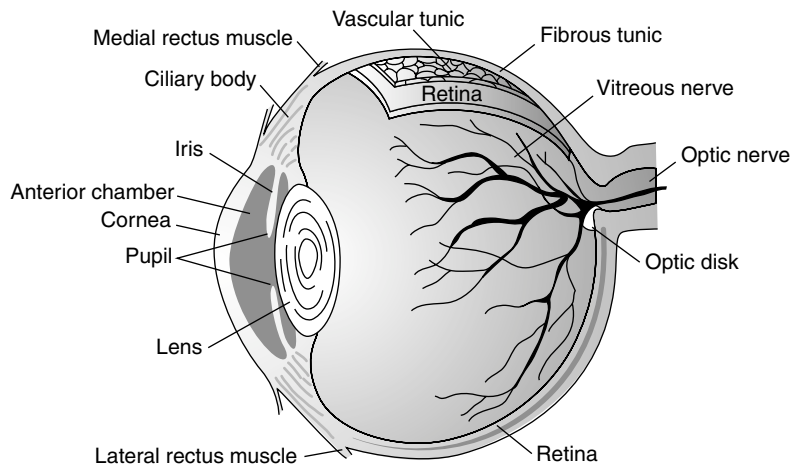


FIGURE 2.9

Cross section of human eye, showing major layers and structures. View is from above the left eye.

THE STRUCTURE OF THE HUMAN EYE

To understand the human eye, let's start with an overview tour of the major features. We'll ignore the details, however, until the second stage of our discussion, by which time you'll have a good idea where those details fit into the eye's grand scheme. In portraying these details, incidentally, people often draw an analogy between the eye and a camera. This analogy is apt, but only to a point. Certainly, both are optical devices designed to record visual images on light-sensitive material (film, in the case of the camera; photoreceptors, in the case of the eye). And the two do have components in common (mechanical, in the case of the camera; biological, in the case of the eye). So where appropriate, we will point out these commonalities. But don't be misled. A camera merely records optical images on film. The eyes do much, much more. Besides recording images, the eyes recode those images, extract biologically meaningful information from the recoded signals, and transmit that information to the brain for interpretation and reaction. In this and the next few chapters, we'll point out other instances where the analogy

fails. For now, let's start with an overview of the eye's major features.

The human eye is very nearly spherical, with a diameter of approximately 24 millimeters (nearly one inch), or slightly smaller than a Ping-Pong ball. It consists of three concentric layers, each with its own characteristic appearance, structure and functions. From outermost to innermost, the three layers are the **fibrous tunic**, which protects the eyeball; the **vascular tunic**, which nourishes the eyeball; and the **retina**, which detects light and initiates neural messages bound for the brain. Figure 2.9 illustrates this three-layered arrangement. In this figure you can see that the eye is partitioned into two chambers, a small anterior chamber and a larger vitreous chamber. Thus the basic layout is three concentric layers and two chambers, plus the iris, pupil and lens.

The Outermost, Fibrous Tunic

When looking directly at someone's eye, you see only about one-sixth of its outer surface. The rest is tucked into the bony orbit, hidden behind the lids and other protective structures. The "white"

of the eye is part of the outermost, fibrous coat. Since this white part is made of tough, dense material, it is called the **sclera**, from a Greek root meaning “hard.”*

The sclera averages about 1 millimeter in thickness, and microscopic inspection reveals that it’s made of tightly packed, interwoven fibers running parallel to the sclera’s surface. These densely packed fibers give the sclera its toughness. Actually, the sclera needs to be tough because pressure inside the eyeball is double that of the atmosphere. If the sclera were more elastic, that pressure differential could cause the eyeball to become deformed. As a result, the quality of one’s sight would be greatly diminished. We’ll return to the importance of the eyeball’s shape later, when we discuss the eye as an optical instrument.

At the very front of the eye, this outer coat loses its white coloring and becomes so transparent that it’s difficult to see it in the mirror. However, if you look at someone else’s eye from the side, you will notice a small bulge on the front of the eye. This bulge is called the **cornea**, from a root word meaning “like a horn.” (The cornea is composed of tissue comparable to that of an animal’s horn.) The cornea’s transparency is crucial for vision. It allows light to enter the eyeball unimpeded. The neat and orderly arrangement of the cornea’s fibers is the primary reason that the cornea is transparent. In addition, greater transparency is made possible because the cornea has no internal blood supply of its own. Since blood and the requisite vessels could reduce the passage of light, the cornea draws its nourishment from the clear fluid in the anterior chamber.

The transparent cornea plays a crucial role in the formation of images on the back of the eye. Anything that disturbs the cornea’s transparency, therefore, will reduce the quality of these images and, hence, the quality of vision. For self-protection, the cornea has extremely high sensitivity to touch. Foreign bodies, such as dust particles or a misguided finger, contacting the cornea trigger a sequence of protective responses, including lid closure and tear production. There’s one reflexive response, however, that should be avoided: as our mothers used to tell us, “Never rub your eye except with your elbow”—these words of wisdom mean leave your eyes alone, for most remedies simply do further damage.

The Middle, Vascular Tunic

For most of its course, the vascular tunic hugs the wall of the eyeball, and only toward the front of the eyeball does it pull away from the wall. We’ll begin by considering the rear two-thirds of this middle layer, the part that fits snugly against the wall of the eyeball.

Most of the middle layer consists of a heavily pigmented, spongy structure called the **choroid**. The choroid averages about 0.2 millimeters in thickness and contains a network of blood vessels, including capillaries. Blood from these capillaries nourishes one particular class of cells in the retina, the photoreceptors that turn light into neural signals. Without their nourishing blood supply, these vital photoreceptor cells would starve to death from lack of oxygen.

The choroid’s heavy pigmentation also reduces light scatter, the tendency for light to be reflected irregularly around inside the eyeball; light scatter would reduce the sharpness of images formed inside the eye. The choroid’s dark pigmentation reduces scatter by harmlessly absorbing light that isn’t captured by the photoreceptors. Incidentally, this is the same reason the inside of a camera is painted flat black. The paint absorbs scattered light and protects the sharpness of images on the film.

*Most parts of the eye have names related to their character or appearance. Knowing the origin of some of these names can help you appreciate the structure of the eye. Our explanations come from a book, *On Naming the Parts of the Human Body*, written in the first century A.D. by Rufus of Ephesus. Ephesus is an ancient city located in what is modern-day Turkey. We have drawn on an excellent partial translation by Stephen Polyak (1941, p. 96).

The Anterior Chamber

Toward the front of the eye, this middle, choroidal layer curls away from wall of the eyeball and runs more or less parallel to the front surface of the eye. Over this part of its course, the middle layer forms a long slender structure called the **ciliary body**. This spongy network of tissue manufactures **aqueous humor**, the watery fluid that fills the smaller, anterior chamber of the eye located behind the cornea and in front of the lens. The aqueous humor serves a number of important maintenance functions. It transports oxygen and nutrients to several of the structures it bathes, and it carries away their waste products. The eye's crucial optical components—the cornea and lens—rely on the aqueous humor as their source of nourishment.

The aqueous fluid also helps maintain the shape of the eyeball. If there were too little fluid in the anterior chamber, the eye would become deformed, like an underinflated soccerball. Constant pressure is maintained, however, because cells in the ciliary body are constantly producing new aqueous to keep the supply of nutrients from becoming exhausted. The creation of new fluid also prevents the buildup of high concentrations of waste products.



Clinical

For pressure to remain constant, of course, a balance must be achieved between the rate at which aqueous fluid is created and the rate at which it is drained from the eye. Sometimes this balance cannot be maintained and too much aqueous accumulates in the eye, which elevates intraocular pressure. Excess aqueous accumulates either because of overproduction or because of improper drainage out of the anterior chamber. Drainage can be blocked or slowed down if the outlets for aqueous (which lie at the junction of ciliary body and cornea) become squeezed shut or clogged. Pressure builds up within the eye, and if the pressure remains high for too long, vision can be impaired permanently. Increased intraocular pressure—the condition called **glaucoma**—is the single most common cause of blinding eye

disease in North America. Fortunately, glaucoma can be treated if the condition is detected early enough. For this reason, eye doctors routinely measure the intraocular pressure in eye examinations these days.

The Iris, Pupil and Lens

The Iris As it curls inward, away from the wall of the eye, the ciliary body gives rise to the **iris**, that circular section of tissue that gives your eye its characteristic color: brown, blue, green, gray, and so forth. This variety of colors makes the name “iris” very appropriate because iris comes from the Greek word for “rainbow.”

The iris actually consists of two layers, an outer layer containing pigment, and an inner layer containing blood vessels. If the outer layer is heavily pigmented, the iris will appear brown. But if this outer layer is lightly pigmented, the inner layer becomes partially visible through the outer one. In this case, the iris will look more lightly colored. Thus, the color of one's eyes results from a combination of the pigmentation of the outer layer and the color of the blood vessels in the inner layer. If the iris's outer layer has no pigment, the inner layer becomes very noticeable, giving the eye a pinkish hue. This condition occurs in albino humans, who, because of a genetic defect, have greatly reduced pigmentation.

The Pupil Looking in a mirror at the center of your own iris, you'll see a round black region, the **pupil**. The pupil is actually an opening, or gap, within two sets of muscles. The inner set runs circularly around the pupil. When this circular band of muscles contracts, the pupil gets smaller. Another set of muscles runs radially out from the edge of the circular muscles, away from the pupillary opening. When the radial muscles contract, the pupil widens, or dilates. These changes in pupil size regulate the amount of light reaching the back of the eye.

The size of the pupil at any given moment depends on several factors. First, it depends on the



Differences

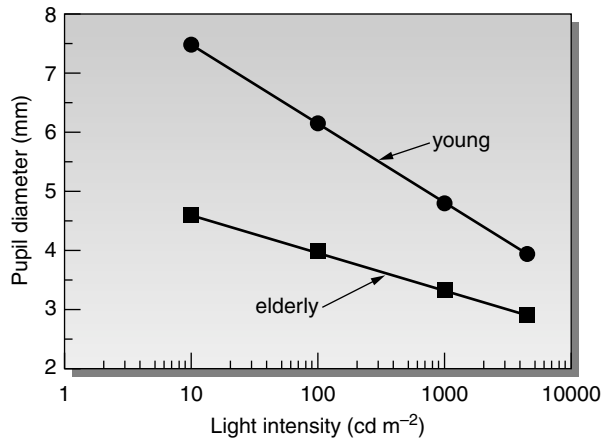


FIGURE 2.10

Pupil size varies according to both light intensity and age. (cd m^{-2} stands for candelas per square meter, a common unit of light intensity.)

light level to which the eye is exposed: the size of the pupil decreases as the level of light increases. In young adults, the pupil diameter varies from about 8 millimeters down to less than 2 millimeters, a fourfold variation in diameter. The amount of light passing through the pupil is proportional to the pupil's *area*, which is itself proportional to the square of pupil's diameter. Thus, as the pupil diameter varies over a range of 4 to 1, the amount of light passing through the pupil varies over a range of 16 to 1. The pupils of older adults change size in response to light in different proportions than their younger counterparts. In dim light, the pupil of an eighty-year-old person is only about half that of a twenty-year-old (see Figure 2.10). As a result, less light reaches the retinas of elderly people. This undoubtedly explains why older people frequently complain about the dim lighting in restaurants.

Besides light level, the size of the pupil also varies in response to events that stimulate the autonomic nervous system. Anything that induces excitement, fear, or sexual interest can change the size of the pupil. Some people, such as seasoned poker players, are quite adept at sensing an opponent's excitement or dismay on the basis of

their opponent's pupil size. An opponent's widened pupils are a dead giveaway that he or she has drawn a potentially winning card. Realizing the potential cue involuntarily provided by their eyes, clever poker players will wear dark glasses to hide their telltale pupils.

Although large pupils allow more light into the eye, smaller pupils can sometimes offer an advantage (Cornsweet, 1970). Suppose you are looking at an object located several meters in front of you. While you're looking at that object, other objects—those much closer or much farther away—will tend to appear somewhat blurred. The range of distances over which objects will appear sharply focused varies inversely with the size of the pupil. This range of sharp vision is called the **depth of field**. The easiest way to demonstrate depth of field is to substitute a camera for your eye. In taking the photographs shown in Figure 2.11, the photographer varied the size of the camera's aperture to simulate the effects of changing pupil size. The photograph on the left was taken with a large aperture; the one on the right, with a small aperture. In both instances, the camera was focused on the object centered in the picture. Note that on the left, the picture taken



FIGURE 2.11

The degree of blur in a picture depends on the size of the aperture of the camera. The sharp photo on the right was taken through a smaller aperture than was used in taking the photo on the left, thus increasing the depth of field in the right-hand photograph. (Glyn Cloyd.)

with the large aperture, very few objects appear sharp. The photograph on the right was taken without changing the focus but with a smaller aperture. Now, more objects appear well focused—the range of distances over which objects appear in sharp focus has increased. This range defines depth of field. Whether we're talking about cameras or about eyes, depth of field is determined by pupil (or aperture) diameter.

The Lens One very important optical element of the eye, the **crystalline lens**, lies right behind the iris. The lens takes its name from its resemblance to a lentil, or bean. In adults, the lens is shaped like a very large aspirin tablet, about 9 millimeters in diameter and 4 millimeters in thickness. The lens consists of three distinct parts: an elastic covering, or *capsule*; an *epithelial layer* just inside the capsule; and the *lens* itself. As you might expect, each of these parts has its own job to do.

In fact, the thin, elastic capsule around the lens has two jobs. First, it moderates the flow of aqueous humor into the lens, helping the lens retain its transparency to light. Second, the elastic capsule molds the shape of the lens—varying its flatness and, thereby, the lens's optical power. This variation in optical power is called **accommodation**.

The lens never stops growing. Throughout your life span, the outer, epithelial layer of your lens continues to produce protein fibers that are added to the surface of the lens. Consequently, those protein fibers nearest the center of the lens are the oldest (some were present at birth), whereas the fibers on the outside are the youngest. Between birth and age 90 years, the lens quadruples in thickness and attains a weight of 250 milligrams (Paterson, 1979). In the center of the lens, the old fibers become more densely packed, producing **sclerosis**, or hardening, of the lens. We'll describe the significance of sclerosis later in this chapter.

For good vision, the lens must be transparent—light must be able to pass through it easily, without loss or deviation. Like the cornea, this transparency depends on the material out of which the lens is made. Of all the body's parts, the lens has the highest percentage of protein, and its protein fibers are lined up parallel to one another, maximizing the lens's transparency to light. Anything that disturbs this alignment—such as excess fluid inside the lens—reduces transparency.



Clinical

An opacity (or reduced transparency) of the lens is called a **cataract**. While some cataracts are minor—barely reducing the transmission of light—others undermine vision to the point of blindness. In some cases, cataracts can be traced to cumulative exposure to ultraviolet radiation present in sunlight (Schein et al, 1994), which helps explain why cataracts are more common in elderly people. But not all young people are spared from cataracts. In fact, certain populations (Arabs and Sephardic Jews, for instance) have a very high incidence of congenital cataracts—lens opacities at birth. These opacities severely degrade the stimulation received by the eye, and this can be serious. At birth, the visual nervous system is immature, and its proper development depends on normal stimulation of the eye. Deprived of that proper stimulation, the immature visual nervous system develops abnormally (Hubel, Wiesel, and LeVay, 1977). Realizing this consequence of visual deprivation, physicians now recommend removal of congenital cataracts as early in life as possible.

Surgical removal of a cataractous lens has become more or less routine today. Since the lens contributes to the total optical power of the eye, removal of the lens must be accompanied by some form of optical compensation. Powerful spectacles or contact lenses can be worn, or alternatively, a plastic lens can be surgically inserted into the eye, replacing the missing biological lens (Aplegate et al., 1987). None of these alternatives, however, restores the ability to accommodate.

People who have had such surgeries must use different glasses for near versus far vision.

The Vitreous Chamber

The vitreous chamber accounts for nearly two-thirds of the total volume of the eye. This larger of the eye's two chambers is bounded by the lens in front and the retina on the sides and in the rear. This chamber is filled with a transparent fluid called **vitreous**, a substance with the consistency of egg white.

Encased in a thin membrane, the vitreous is anchored to the inner wall of the eyeball. Unlike the aqueous, the vitreous is not continuously renewed, which means that debris can accumulate within it. Sometimes you become aware of this debris, in the form of **floaters**, small opacities that float about in the vitreous (White and Levatin, 1962). If you look at a bright, uniform surface, floaters cast shadows on the back of the eye, producing little dark spots that dart about immediately in front of you. Thanks to gravity, floaters tend to settle to the bottom of the vitreous chamber, out of the line of sight. Although floaters are usually harmless, dense or persistent floaters may be a symptom of retinal detachment, a vision-threatening condition that requires treatment.

The Retina

The innermost of the eye's three layers, the **retina**, resembles a very thin, fragile meshwork, which explains its name—*rete* is Latin for “fisherman's net.” Although no thicker than a postage stamp, the retina has a complex, layered organization. Figure 2.12 shows how a section of the retina would look if magnified greatly and viewed from the side. The arrows denote the direction taken by incoming light. From this perspective, you can see that the cells comprising the retina have a peculiar arrangement: light must pass through a complex network of neural elements before reaching the **photoreceptors**,

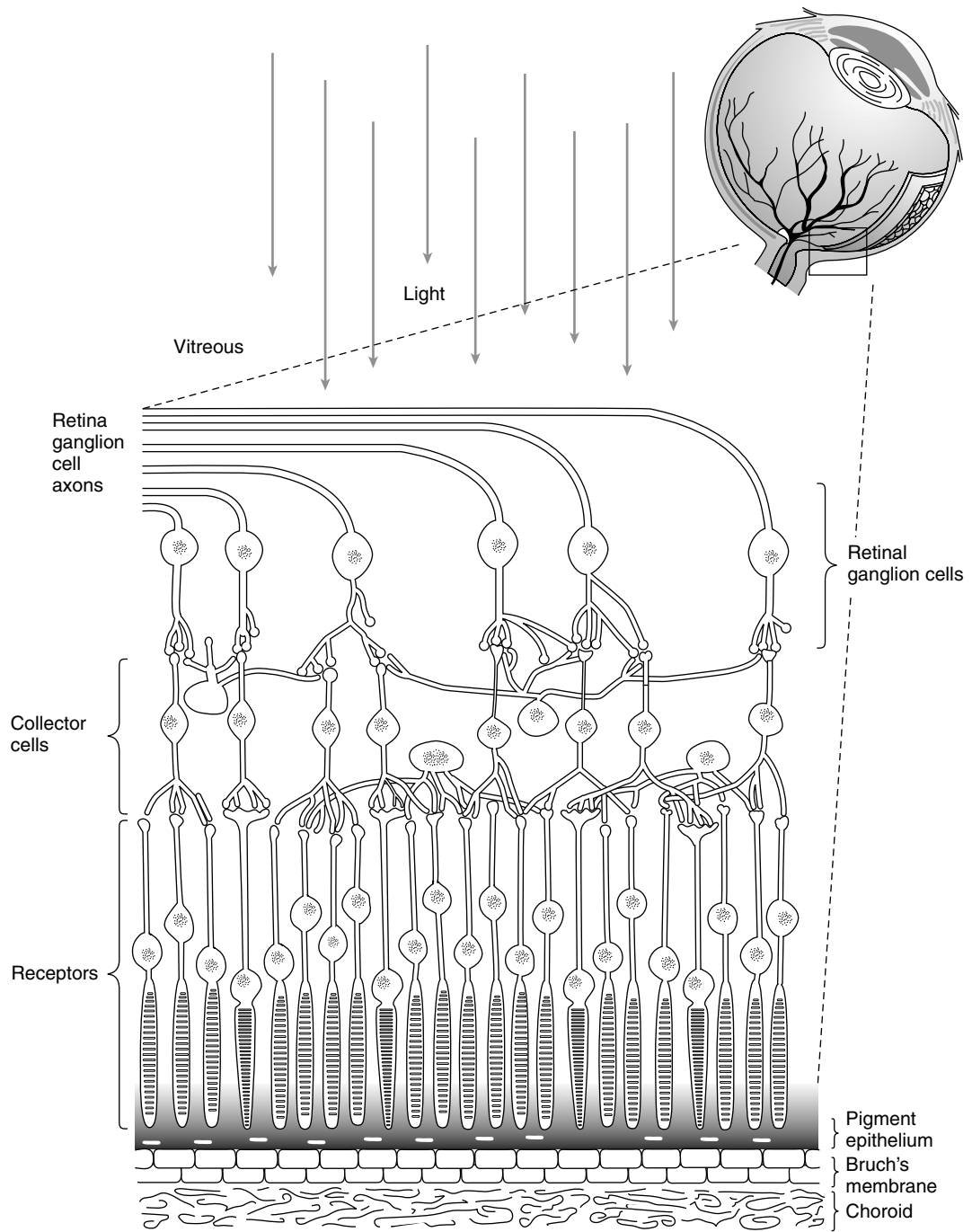


FIGURE 2.12

Cross section of the retina. The small box in the inset at the base of the eyeball shows the region of the eye represented in the enlarged drawing.

which are actually responsible for converting light energy into neural signals. Moreover, the light-sensitive part of each photoreceptor, called the **outer segment**, actually faces away from the incoming light and toward the back of the eye. This seemingly “backward” arrangement has one distinct advantage. Photoreceptor outer segments, which have high metabolic demands, end up snuggled into the choroid, which, as we’ve already learned, contains a rich blood supply.



Neural

The neural signals generated by the photoreceptors, in turn, pass through a network of cells—**bipolar**, **amacrine** and **horizontal cells**—that collect and recombine the photoreceptor signals. These collected, transformed signals are then passed on to the **retinal ganglion cells**, where biologically important information about the distribution of light over space and time is extracted and recoded. The recoded neural messages are then carried by the axons of the ganglion cells out of the eye and into the brain. As you can already begin to appreciate, crucial events underlying visual perception are inaugurated in this complex neural network, the retina.

To a large degree, the retina’s complexity reflects its origins. Embryologically, the retina arises from the same tissue that spawns the billions of cells comprising the brain. So the retina is actually a direct extension of the central nervous system. This affinity with the brain has one unfortunate aspect, though: damaged retinal cells, like damaged brain cells, cannot be repaired. The visual consequences of damage are permanent, which is why it’s so important to protect the eyes from excessive light exposure (by wearing sunglasses), from trauma (by wearing goggles when playing sports), and from disease (by having a routine, periodic eye examination).

The eye is not just a window out, onto the outside world, it also is a window through which another person can look directly inside your body and view your nervous system. In fact, the eye is the *only* place where the nervous system and blood supply can be viewed directly, without sur-

gery. If you were to look directly into another person’s pupil, however, you wouldn’t be able to see anything except a dark hole because your head obstructs the light necessary for seeing the back of the eye. Essentially, you’re looking through a peephole into a dark chamber. Thanks to the nineteenth-century physicist, physician, mathematician and philosopher, Hermann von Helmholtz, it’s possible to illuminate the eye while at the same time looking into it (Figure 2.13). Helmholtz invented a simple, clever device called an **ophthalmoscope**,* which allows us to visualize the interior of the living human eye, including its retina and blood supply. Doctors use this instrument to examine millions of patients every year, usually employing a hand-held, battery-powered model that you’ve probably encountered during a routine vision exam.

Retinal Landmarks and Blood Supply Figure 2.14 illustrates what is seen when looking into a normal human eye using an ophthalmoscope. Because the living retina is virtually transparent, the ophthalmoscope mainly reveals structures lying in front of the retina—such as the central retinal artery—and structures lying behind it—such as the choroid. Although not a complete view of the entire retina, this photograph does highlight several of the retina’s most significant features. Look first at the nearly circular area in the center. Measuring roughly 1.5 millimeters in diameter, this region is called the **macula**. When you look directly at objects such as the words on this page, the images of those objects are centered within the macula of each eye. Vision is most acute in the center of the macula.

Consider another landmark shown in Figure 2.14, the **optic disk**. This is the region of the eye where optic nerve fibers (the axons of the gan-

*Actually, Charles Babbage, who developed a mechanical digital computer in the middle of the eighteenth century, made a working model of an ophthalmoscope some years before Helmholtz but didn’t pursue the project (Rucker, 1971).



FIGURE 2.13

German scientist Hermann von Helmholtz who invented the ophthalmoscope, which is illustrated on the right.

Left: © Bettmann/CORBIS

Right: Courtesy of Welch Allyn, Inc.



FIGURE 2.14

Photograph showing the inside of the back of the human eye.

glion cells) exit the retina, carrying information to the brain. Normally, the optic disk has a pinkish color because of small blood vessels on its surface; these blood vessels nourish part of the optic nerve. Loss of this pink color can signify the presence of a circulatory problem that could eventually starve the optic nerve and impair vision. Fortunately, such changes in color can be detected quite easily with the ophthalmoscope.

The retina has the highest metabolic rate of any part of the body, so its access to blood—for oxygen and nutrition—is vital. To supply the entire retina with blood is a challenge because metabolically active cells need to be in extremely close contact with the capillaries that nourish them. To meet this challenge, the eye contains two blood supplies: one that primarily nourishes the photoreceptors (the choroidal circulation system), and the other that nourishes the remaining cells of the retina (the retinal circulation system).

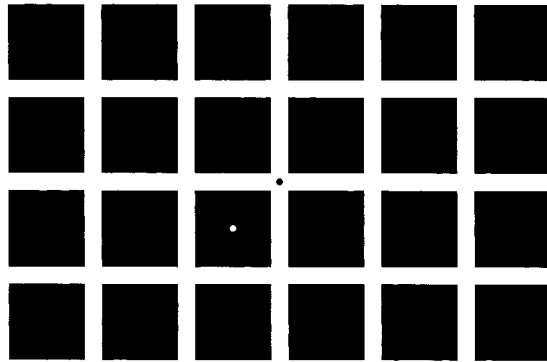
BOX 2.1

EYES THAT NEVER STAND STILL

Contractions of the extraocular muscles pull and tug on the eyeballs, guiding their direction so objects of interest can be fixated. But even when you try to keep your eyes absolutely still, small random contractions of the extraocular muscles keep the eyes moving. These involuntary eye movements are usually too minor for you to be aware of, but they are important for seeing. Before learning why they're important, though, try this simple trick that will let you see your own involuntary eye movements.

The trick requires a pattern like the grid in the accompanying figure (Verheijen, 1963). First carefully fixate on the black dot in the center of the pattern for about 30 seconds, keeping your eyes as still as possible. Then quickly move your eyes to the white dot. Again, keep your eyes as still as possible. You'll see an illusory pattern, called an **afterimage**, that jiggles slightly. The jiggling of the afterimage is caused by the movements of your eyes. You can prove this to yourself by now making large, intentional eye movements; the afterimage follows your eyes. The afterimage itself arises from the differential adaptation of neurons in your retina respond-

ing to the light and dark parts of the figure. Of course, the adapted neurons are fixed in place in the eye's retina; it is as if a negative image of the grid is temporarily imprinted on the back of the eye. Thus, whenever you see the afterimage move, you're in fact witnessing your eyes moving—whether you intended them to or not. The slight jiggling of the afterimage even when you try to fixate is the consequence of your own involuntary eye movements.



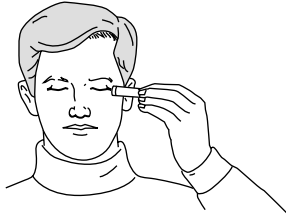
Continued

As you've already learned, the choroid and its blood supply are located behind the photoreceptors and, therefore, out of the path of light that stimulates the photoreceptors. This isn't true, however, for the retinal circulatory system. Notice in Figure 2.14 the large blood vessels that run outward from the optic disk, which are arteries and veins. These vessels make up the retinal circulatory system. They fan out from the optic disk, dispersing into a fine network of capillaries spread throughout the inner part of the retina. Because they're situated in front of the retina, these blood vessels lie directly in the path of incoming

light. This arrangement may strike you as odd, but it's the only way to get the blood vessels in close proximity to the outer portions of the retina that require nourishment.

You may also notice in Figure 2.14, however, the clever rerouting of the arteries and veins around the macula. Within this most important region for seeing, therefore, light is not obstructed on its route to the photoreceptors. But what about the other parts of the retina? Why don't we see the blood vessels and nerve tissue in the other parts through which light must pass? Box 2.1 gives the answer, and also allows you to

BOX 2.1 continued



These small random eye movements are actually very important for vision; when they are eliminated, vision changes dramatically. For example, special optical systems have been developed that move whatever you're looking at in step with the movements of your eyes. This scheme produces a motionless, stabilized image on your retina. Here's one way to produce a stabilized retinal image. A small photographic transparency of some object is mounted on a special high-power contact lens that focuses the image on the retina. Since the contact lens moves along with the eye, the image of the transparency attached to this lens is stabilized on the retina. And the perceptual result is remarkable—within a few seconds, the object begins to fade from view, as if the brightness on a television screen were being reduced. Eventually the object disappears entirely, leaving nothing but a homogeneous gray field. So when the

effects of normal involuntary eye movements are eliminated, vision is eliminated too (Pritchard, Heron and Hebb, 1960; Riggs, Ratliff, Cornsweet and Cornsweet, 1953).

The disappearance of stabilized retinal images is actually quite fortunate. If this didn't happen, you'd constantly be annoyed by the images produced in your eyes by blood vessels. Because they are in the path of light, these blood vessels cast shadows on your retina. Since the vessels move along with your eye, their shadows are stabilized retinal images and, consequently, are invisible to you. However, you can temporarily "destabilize" them by moving a beam of light back and forth across the vessels. This causes their shadow to move back and forth slightly, enough to make them visible. The simplest way to produce this effect is with a small flashlight (a penlight). Looking straight ahead with eyes closed, place the penlight against the corner of your eye—the corner away from your nose, as is shown in the illustration. Gently rock the penlight back and forth. After a second or two you should begin to see what look like the branches of a tree. These are the shadows of blood vessels in your eye. Once you get them into view, stop moving the flashlight and the branches will disappear within a few seconds, as the image returns to its normal, stabilized condition.

see what you've probably never seen before: the blood vessels in your own eye.

Because its metabolic activity is so high, the retina is crucially dependent on normal blood flow. This makes the retina particularly vulnerable to diseases and disorders that interrupt or impede the flow of blood. What sorts of things might interrupt the retina's blood supply? For one thing, feeder arteries can become clogged, blocking the flow of blood to and within the inner retina. This happens in arteriosclerosis ("hardening of the arteries") and sometimes in sickle cell disease, a condition common among Africans and people of African descent.

Impairment of the blood supply to the outer, choroidal system can also have disastrous consequences. The outermost sheath of the retina consists of a single layer of cells, the **pigment epithelium**, which forms a barrier through which choroidal blood must pass in order to nourish the outer segments of the photoreceptors. The pigment epithelium transfers oxygen, nourishment, and vitamin A from the choroidal circulation to the photoreceptors. A steady supply of vitamin A is required for the synthesis of the receptors' light-sensitive pigment, a detail we'll return to later.



Clinical

The pigment epithelium also provides for waste disposal. Molecular garbage shed by the receptors is taken up and recycled within the pigment epithelium. If uncollected debris accumulates, it can eventually impede the transfer of nutrients. So anything that keeps pigment epithelium cells from performing their tasks will cause photoreceptor starvation and, eventually, death.

One natural enemy of the pigment epithelium is aging. Age-related macular degeneration, a condition that causes a progressive loss of vision, is the leading cause of impaired vision in the Western world and accounts for about one-half the cases of blindness among the elderly. Macular degeneration can seriously impair a person's ability to read, to drive an automobile and even to get around on foot (Hazel, et al., 2000). Currently, a minority of cases of age-related macular degeneration can be arrested, but not reversed, if treated early by means of laser surgery.

Diabetes is another common disease that can affect the retina's blood supply. This disease is marked by disordered insulin metabolism that causes too much sugar to accumulate in the diabetic's blood. For reasons not completely understood, the excess sugar can promote the development of a cataract in the eye's crystalline lens. Cataracts, as you've learned, can blur vision to the point of blindness. Besides cataracts, diabetes can have another serious consequence for vision. In some diabetics, the retina's blood supply is severely reduced. Sensing that it is being starved for oxygen (carried in the blood), the retina generates a chemical that stimulates the growth of new, larger blood vessels. Growing new blood vessels may sound like an excellent solution to the problem, but this growth produces devastation of its own. The thick new vessels grow out of control, blocking light and eventually causing blindness. In the last decade, lasers have been used very successfully to stop the growth of these new vessels.

THE EYE AS AN OPTICAL INSTRUMENT

Obviously, to see objects in the environment around you, your eyes must capture light reflected by those objects. But simply capturing the light itself isn't sufficient. The pattern of light reaching the retina must mirror the distribution of light in the scene being viewed. This light distribution, or **retinal image** as it's called, is the raw material on which the retina works. The fidelity of the retinal image depends on the ways that the patterns of light arriving at the eye interact with various ocular structures that influence the passage of that light to the back of the eye where the photoreceptors are located. To appreciate the workings of the eye's retina, then, requires understanding something about the image it receives.

Light conveys to the eyes information about objects in the environment. But how does light acquire that information in the first place? Initially, light originates from a source such as the sun or a light bulb. This is called emitted light, and it's certainly a necessary antecedent for vision: we cannot see in the dark. But emitted light *per se* isn't the stimulus for vision. Rather, we are interested in seeing *objects*, and for that to happen our eyes must pick up and register light reflected off the surfaces of those objects. It is reflected light that conveys biologically important information about the appearance—and hence the identity—of objects. How does this come about?

Surfaces absorb some, but not all, of the light shining on them; the portion of light not absorbed is reflected by the surface. Objects with high reflectance usually appear "light," whereas objects with low reflectance appear "dark." For example, this page has a reflectance of about 80 percent, while the print on this page has a reflectance of about 10 percent. Abrupt changes in reflectance usually signal discontinuities in a surface, such as the letters on this page or the edges and corners that demarcate an object's boundaries (see the top drawing in Figure 2.15). More gradual changes in

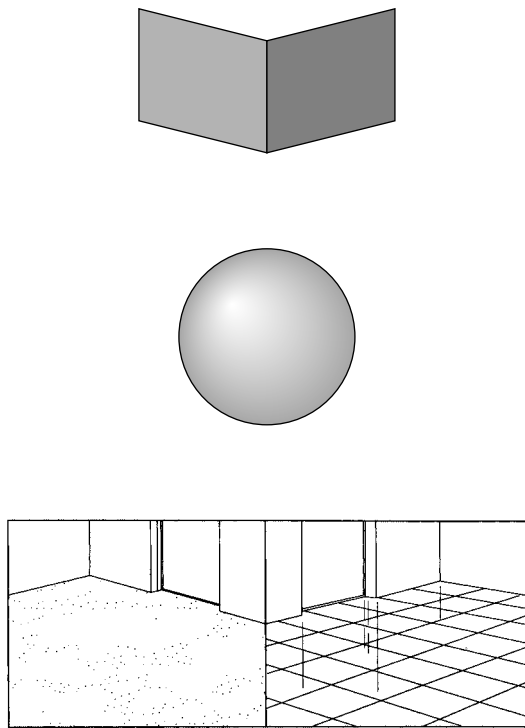


FIGURE 2.15

The top drawing illustrates how abrupt changes in reflectance signal the presence of an edge. The middle drawing illustrates how gradual changes in reflectance signal the presence of a smooth surface. The bottom drawing shows how reflected light specifies the texture of a surface.

reflectance usually correspond to curved surfaces (see the middle drawing in Figure 2.15).

Surfaces can “sculpt” the patterns of reflected light in other ways, too. For example, some surfaces reflect light evenly in many different directions. Because such surfaces lack highlights, they appear dull or matte. Other surfaces reflect light strongly in one narrow range of directions, giving those surfaces highlights and making them appear smooth and glossy (Greenberg, 1989). (The differences between dull and glossy surfaces are depicted in the bottom drawings in Figure 2.15.)

So, reflected light is structured by objects in the environment, and this optical structure conveys potential information about those objects and their textured surfaces. But before that potential can be realized, three prerequisites must be satisfied. First, the light must be sufficiently intense to penetrate the eye, reaching the photosensitive material in the retina. In fact, about 50 percent of all light striking the cornea is reflected or absorbed before reaching the retina (Cornsweet, 1970, p. 24).

Second, the distribution of light imaged on the retina must be properly focused. Consider the sharp contours forming this pair of letters: GO. To produce a sharp image of these letters, light reflected from that region of the page must form small, well-defined replicas of those contours on the retina. Blurred replicas would be created if each small, sharp contour were imaged as a larger, spread-out distribution of light on the retina. As a result, different parts of the retinal images of the contours would overlap, blurring one another’s boundaries and making it difficult to see separate, individual letters, in which case reading would be impossible. Some effects of blur are illustrated in Figure 2.16.

Third, the pattern of light falling on the retina must preserve the spatial structure of the object from which it is reflected. If that spatial structure is preserved, light arising from two adjacent regions in space—from neighboring parts of an object, for instance—will fall on adjacent parts of the retina. A distribution of light that preserves the spatial ordering of locations in space is called an **image**. If the light distribution on the retina were scrambled or spatially random, it would be useless as a source of information about the structure and layout of objects.

Image Formation in The Human Eye

The sharpness of images formed on the retina depends mainly on two factors. The first is the

In a nutshell, the eye receives patterns of light energy reflected from the surfaces of objects in the environment. Those patterns of energy depend on a host of factors, including the surface properties of objects, the distance from the eye to those objects, and the source of light illuminating the objects. Whenever you change the angle of vision between yourself and an object, for example, you alter the pattern of light energy falling on your eyes (Figure 2.4). Whenever you view an object in natural sunlight and then move indoors to look at it under artificial lighting, you change the wavelength composition of light energy arriving at your eyes (artificial light isn't identical to sunlight). In short, the same object can convey countless different optical messages to your eyes.

able to work back patterns of light of objects in the which vision effortlessly, is call
 As you will b problem requires some assumption we're likely to c Fortunately, tho ones because th poses certain con jects. For exam opaque objects, ject partially occ see the partially

FIGURE 2.16

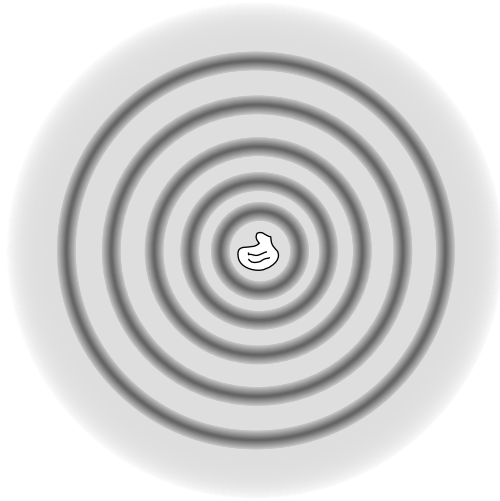
Effect of blur on the legibility of text. (For a legible version, you can find these paragraphs on page 38.)

optical power of the cornea and crystalline lens (where “optical power” means ability to bend, or refract, light). The other factor controlling image sharpness is the size of the eyeball, particularly the eyeball’s length from front to back. In a camera, a good picture requires that the film be just the right distance from the lens. In the eye, the same thing holds true: the retina must be the right distance from the crystalline lens. Some eyes are too short, others are too long; either condition impairs vision.

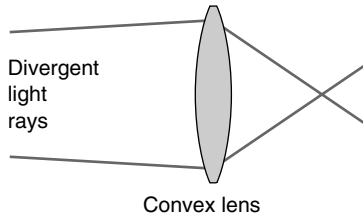
The optical power of the eye is not constant, though. By changing its shape somewhat, the crystalline lens automatically changes its optical power. This automatic change, called accommodation, helps one see objects clearly, regardless of their distance from the eye. To appreciate how these components of the eye contribute to vision, we’ll have to consider the behavior of light and its interaction with these components. To simplify our analysis of image formation we’ll begin with a very small object: a single point in space that emits light. The same analysis works for other, more complex visual objects, since we can

think of them as consisting of a large set of points. But dealing with just one point will simplify our explanation of the rudiments of image formation.

In the eighteenth century, Thomas Young showed that light seemed to behave as though it consisted of waves. Upon dropping a pebble into a pond, you’ll see wavefronts spreading out from the place where the stone hit the water. The stone corresponds to our point of light, and radiating out from that point is a set of spherical wavefronts (see Figure 2.17). Light that spreads out in this way is said to be **divergent**. Divergent light cannot form a well-focused image—a point—unless something is done to reverse its divergence. Let’s examine how the eye accomplishes this feat. Certain optical devices can counteract light’s tendency to diverge. One such device is a convex lens, which gets its name from its shape. Once a diverging wavefront passes through a strong convex lens, the paths of neighboring points on the wavefront get progressively closer together and eventually converge to a single point. After passing through this point, light di-

**FIGURE 2.17**

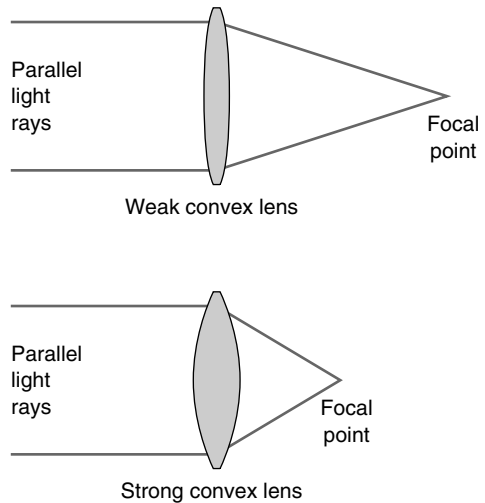
Light waves radiate out from a source of light in a way that resembles the ripples produced when a pebble is dropped into a pond.

**FIGURE 2.18**

A convex lens focuses diverging light.

verges once again. Figure 2.18 illustrates this effect of a convex lens.

Lenses differ in their ability, or power, to converge light. A highly convex lens converges light more strongly than does a mildly convex lens. As Figure 2.19 shows, rays that pass through a convex lens of lower power are focused at some distance farther from the lens, whereas rays that pass through a convex lens of high power are focused close to the lens. The distance at which a lens brings light to focus depends on both the power of the lens itself and the degree of light divergence

**FIGURE 2.19**

Convex lenses of different power bring light to focus at different distances. If the light rays striking the lens are parallel, the spot at which the light converges to a point is called the *focal point*.

striking the lens. To converge the light, a convex lens must overcome, or null, the light's divergence. This is demonstrated in Figure 2.20: a lens of constant power is shown converging light of three different degrees of divergence. The most strongly divergent light comes from the source positioned closest to the lens, while the most weakly divergent light comes from the source farthest from the lens. In addition, each object is focused at a different distance from the lens: the most divergent light is focused farthest from the lens, and the least divergent light is focused closest to the lens.

With these optical principles in mind, consider a human eye that is looking at an object sufficiently far away that light coming from that object has essentially zero divergence (in this case we say the object is located at "optical infinity"). To form a useful image, light from the object must be focused on the retina. Since cornea and crystalline lens both contribute to image formation, let's lump them together, calling the combination "the optics of the eye." How

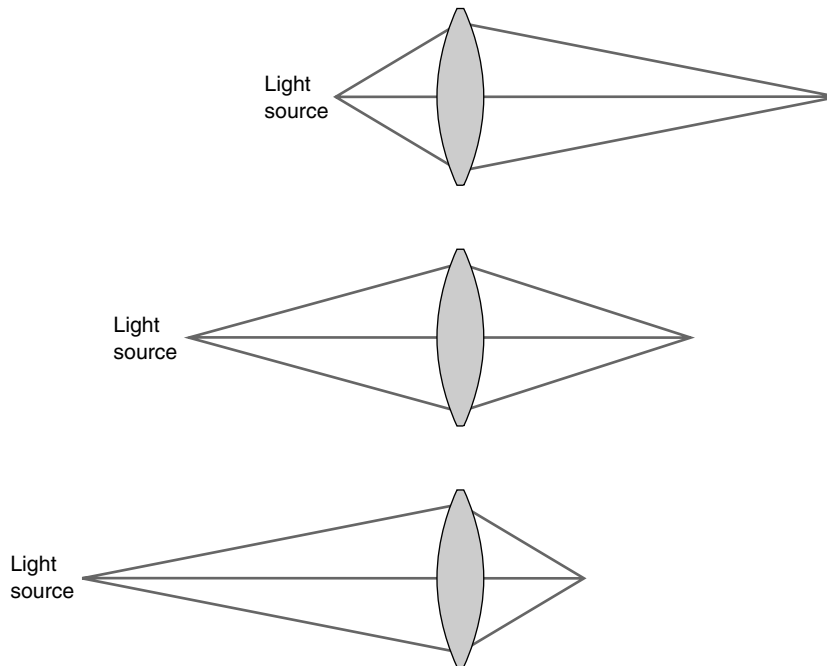


FIGURE 2.20

The spot at which a convex lens brings light to a point depends on the degree of divergence of the light arriving at the lens.

powerful should those optics be in order to produce a sharp retinal image of that distant object? For the retinal image to be sharply focused, the optics' power must match the length of the eyeball—specifically, the distance from the lens to the retina.

This idea is illustrated in Figure 2.21. The top eyeball is just the right length, given the power of its optics. As a result, the distant object is brought to focus exactly on the retina. Such an eye is described as **emmetropic** (meaning “in the right measure or size”).

The middle panel shows an eye that is too long, given the strength of its optics. Although an image *is* formed, that image is formed in front of the retina, rather than on it. In fact, the rays have begun to diverge again by the time they reach the retina, so the image on the retina is blurred. Such

an eye is described as **myopic**, or nearsighted, because near objects will be in best focus.

The bottom panel in Figure 2.21 shows an eye that is too short for its optics; an image is formed on the retina, but it, too, is not well focused and hence the image is blurred. Actually, for this eye the best-focused image would lie behind the retina—if light were able to pass through the retina. Such an eye is described as **hyperopic**, or farsighted, because far objects will be in best focus.

What are the perceptual consequences of a mismatch between an eye's length and its optics? You've seen that in myopic or hyperopic eyes, light does reach the retina, but it is not sharply focused. When an eye of the wrong size looks at a distant point, the resulting image on the retina will be a circular patch, not a point. Thus the point in space will appear blurred, or indistinct.

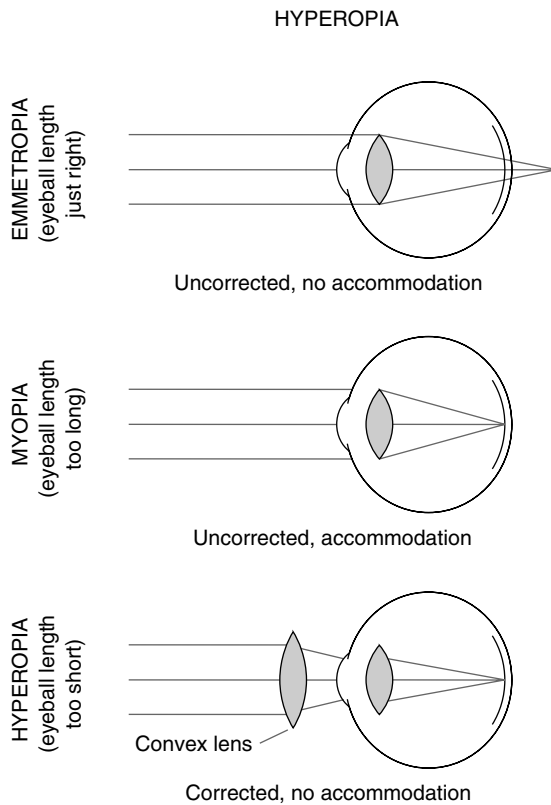
**FIGURE 2.21**

Image formation in emmetropic, myopic, and hyperopic eyes.

The degree of blur depends on the extent to which the eye is too short, or too long: the greater the mismatch between the eye's optics and its length, the worse the blur. The photographs in Figure 2.22 illustrate how the world might appear to a properly focused eye (panel A), to an eye that is only one-third of a millimeter too long (panel B), and to an eye that is 2 millimeters too long (panel C). Remember that when we describe an eye as “too long” or “too short,” we mean this in relative terms. “Too long” and “too short” are defined relative to the power of the eye's optics.

The blur in panels B and C is so striking that it is hard to imagine that many people actually suffer unwittingly for years with that much blur or

**FIGURE 2.22**

Focus influences the quality of the image. Panel A simulates the image formed by an emmetropic eye; panel B, by a mildly myopic eye; and panel C, by a more severely myopic eye. (Glyn Cloyd.)

more. They go through their entire childhood and adolescence never realizing that their vision is defective. For reasons that are explained below, if their vision is blurred from myopia, they may have trouble seeing the blackboard clearly. If the blur comes from hyperopia, they may have trouble reading for prolonged periods. Unfortunately, the difficulties they experience in school may be mistakenly attributed to poor learning abilities rather than to poor vision.

Since about half the human race is afflicted with these problems, it's worth our time to consider hyperopia and myopia, and what steps can be taken to correct those problems.

D

Differences

Hyperopia Imagine an eye whose length and optical power are properly matched. For that eye, a distant object would be in proper focus on the retina. Recall that proper focus demands an object just far enough from the eye so that wavefronts from the object, when they strike the eye, will be diverging at the right rate. If that same eye were shortened—making it hyperopic—by even a fraction of a millimeter, the distant object would no longer be focused as well on the retina. The plane in which the image would be focused best would lie behind the retina, not right on it (see the top panel of Figure 2.23). In its shortened state, this eye's optics would be too weak even for light coming from an object so distant that it is not diverging at all. If it has to deal with light from closer objects (even more strongly diverging), the eye will misfocus the light all the more.

A person with hyperopia—a hyperope—can alleviate this problem by accommodating, increasing the eye's optical power. This enables the hyperope to produce focused images of objects, provided the eye is not too hyperopic and provided the objects are not too close. Accommodation makes the lens more convex, thereby increasing its power and allowing well-focused images to be formed on the retina. The middle panel of Figure 2.23 shows how accommodation helps the hyperope bring an object into better focus on the retina.

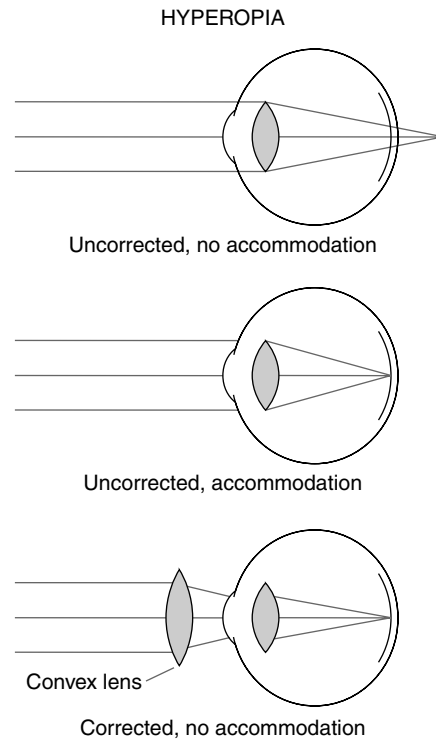


FIGURE 2.23

Mild hyperopia can be overcome either by accommodation or by placing a convex lens in front of the eye.

But the hyperope pays a price for constant accommodation. First, there are limits to the amount of accommodation that the human eye can produce. As a result, if the eye is much too short and the resulting hyperopia substantial, the hyperope may be unable to accommodate enough for close work, such as reading. Second, even if the hyperope *could* accommodate enough to read, accommodation requires maintained muscular effort. The very strong, prolonged accommodation, as might be required in order to read, triggers eye-strain, headaches and nausea (Daum, 1983).

Fortunately, there is an alternative. Since the hyperopic eye cannot make incoming light converge rapidly enough, the light is not focused on the plane of the retina. The problem can be cor-

rected by placing a convex lens in front of the cornea. With such a lens, the total optical power of the eye will be approximately the sum of the eye's own inherent optical power plus the power of the supplementary lens. This increased total optical power allows distant objects to be focused on the retina with little or no accommodation. As a result, the hyperope will need less accommodation when doing close work and will suffer less accommodative strain. The bottom panel of Figure 2.23 illustrates how this added convex lens helps the hyperope see near objects without accommodation.

Myopia Next let's consider an eye that is too long for its optical power. Such an eye is myopic, meaning that an object at optical infinity would be sharply focused somewhere in front of the retina, not at the plane of the retina (top panel, Figure 2.24). This situation could be rectified if we moved the object closer to the myopic person, or conversely, if we moved the myopic person closer to the object. Sometimes, of course, neither of these approaches is practical. Nor is it possible for the blur to be corrected by accommodation. In fact, increasing the power of the crystalline lens increases the overall optical power of the eye, worsening the blur. Fortunately, there is an effective solution to myopia: alter the effective optical power of the eye by placing the proper spectacle lens in front of, or the proper contact lens on, the eye.

What sort of corrective lens would the myopic eye need? We could correct the myopic eye by adding a concave lens (which causes light to *diverge*, combating the myopic eye's tendency to make light *converge* too much). The bottom panel in Figure 2.24 shows how such a lens helps focus an otherwise misfocused target on a myope's retina.

In many countries, myopia is so common that it poses a costly public health problem. For example, in the United States about 25 percent of the adult population is myopic and cannot see distant objects in sharp focus without glasses or

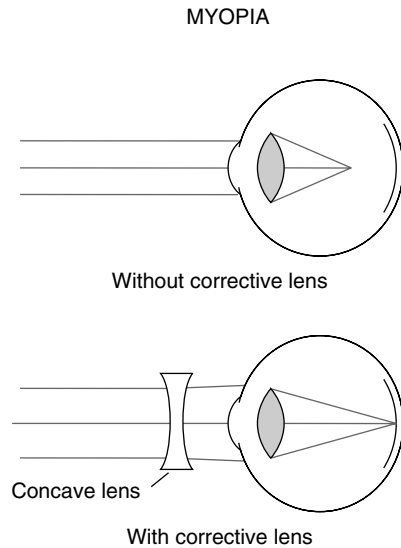


FIGURE 2.24

A concave lens in front of the eye can correct myopia.

contact lenses (NRC, 1989). It is important to note, however, that this prevalence is not uniform across different populations. For example, in Hawaii nearly 80 percent of people of Chinese descent are myopic (Baldwin, 1981).

Myopia also tends to run in families, suggesting that genetic factors are important (Zadnik, 1997). But genetics doesn't tell the entire story. For example, one recent report implicates the use of night lights during infancy as a possible contributor to myopia.

The prevalence of myopia also varies with environmental factors, including the visual demands of a person's occupation. People who do a great deal of near work, for instance, show an increased incidence of myopia. Submariners are an excellent case in point. They are cooped up for months on end in very small quarters, with little or no opportunity for distance vision (Kinney et al., 1980)—myopia is one consequence of this enforced diet of near vision. Closer to home, a number of studies have documented the slow but steady development of myopia among



Clinical



Plasticity

college students (NRC, 1989). The importance of environmental factors in producing myopia is clinched by a study in which monkeys spent their waking time on the equivalent of near work. In order to simulate near work, newborn monkeys were reared so that there was nothing

for them to see more than 50 centimeters from their eyes. Over a 3-year period, these rearing conditions led to severe, permanent myopia (Young, 1981). As Box 2.2 describes, certain conditions produce a temporary, reversible form of myopia.

BOX 2.2

MYOPIA AS A SOMETIME THING

Some conditions have the power to diminish even the best vision, converting emmetropes into myopes, though only temporarily. Because this error in the eye's optical power is temporary, it differs from myopia that we discussed earlier. But as you'll see, the transitory nature of this condition, called **anomalous myopia**, does not reduce its potential seriousness.

It's been known for two hundred years that some people who are blessed with perfectly good eye sight during the daytime become quite nearsighted at night or at twilight. You may have experienced this yourself: nearby objects are seen clearly, but distant ones are not. There exist wide differences among individuals in the severity of anomalous myopia—some people show virtually no myopia at low light levels, while others become extremely nearsighted.

Low light is not the only condition that elicits a temporary myopia. Even in daylight, many individuals become myopic in featureless environments, such as a large open field, or when viewing a clear, cloudless sky. The amount of nearsightedness an individual experiences in such a featureless environment is strongly related to the nearsightedness experienced at twilight. One suspects then that both "open field myopia" and twilight myopia arise from a common cause. Both are subsumed under a single general rubric, anomalous myopia.

What causes this condition? Thanks to the efforts of Herschel Leibowitz and Fred Owens (1975), we know the answer. At low light levels or in open fields, there is no powerful stimulus to control the amount of accommodation. Freed from stimulus control, accommodation returns to a preferred neutral, or resting, level. The eye becomes myopic because that resting level is more suited to seeing near

objects rather than very distant ones. In other words, the resting state of accommodation in many people does not produce a perfectly relaxed lens with minimal optical power. Instead, the resting state of accommodation leaves the lens focused for relatively nearby distances. Using laser-based instrumentation, Leibowitz and Owens confirmed that in the dark, most people's eyes tended to focus at some intermediate distance, not optical infinity. The actual distance varied widely among individuals, with an average viewing distance of about 0.67 meters.

The existence of anomalous myopia poses a potentially serious safety threat (Owens, 1984). Imagine you are the pilot of an airplane flying through the nighttime sky or the driver of a car speeding over a dark country road. In both conditions, accommodation will revert to its resting level. This will tend to blur and diminish the visibility of any object that appears within the field of view at a distance other than that associated with the resting level. If you are a pilot, the blurred object might be another aircraft; if you are a driver, it could be a pedestrian.

Like other refractive errors, anomalous myopia can be remedied optically. Leibowitz and Owens (1976) measured the resting level of accommodation for various individuals and then fitted them with concave corrective lenses of the appropriate power. These glasses, although inappropriate for daylight, nonmyopic conditions, produced impressive improvements in drivers' nighttime vision. Perhaps, sometime in the future, drivers and pilots will be routinely outfitted with individually prescribed glasses for nighttime use. The benefits, in lives saved, could be substantial.



Presbyopia Myopia and hyperopia affect many individuals. But the eye's focus can be disturbed in yet another way that *everyone* who lives long enough will experience sooner or later.

As people get older, their ability to accommodate decreases. As Figure 2.25 shows, the trend begins very early in life and continues until about age 70 (Carter, 1982). For the average 20- or 30-year-old, this loss has no practical consequence; people that age still have sufficient ability to accommodate. But upon reaching the mid-forties, the average person can no longer accommodate sufficiently to bring very close objects into focus. Reduced accommodation arises from various sources, including sclerosis of the lens and reduced elasticity of the lens's capsule (Koretz and Handelman, 1988). Severely diminished ability to accommodate is called **presbyopia**, meaning "old sight." In addition, an old lens is very sluggish in executing even the small shape changes of which it is still capable. This lengthens the time required to change gaze from near to distant objects, and vice versa, causing potential problems in driving and other tasks that entail rapid shifts in focus at different distances.

You have probably seen signs of presbyopia in people who are beyond 40 years of age but have yet to wear glasses or contact lenses. To see clearly, these individuals have to hold their reading material at arm's length, and some eventually complain that their arms need to grow longer. A relatively easy solution is readily available. A convex lens can be placed in front of the eye to substitute for the crystalline lens's own diminished ability to become sufficiently convex.

Benjamin Franklin was about 47 years old when he found that he could no longer read without spectacles. However, his reading glasses made distant objects too blurred. Constantly having to switch from one pair of glasses to another annoyed him, so Franklin invented bifocals—glasses having two separate lenses in front of each eye, with the more strongly convex lens filling the bottom of the frame. Looking down at reading material, Franklin could take advantage

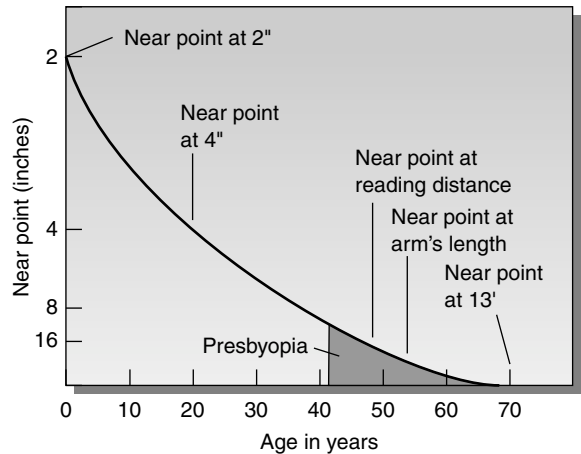


FIGURE 2.25

The *near point*—the closest distance at which an object can be seen without blur—increases with age.

of the extra help given by that lens. Looking slightly upward, he could see the world through a less powerful convex lens, allowing him clear vision of distant objects.

Astigmatism So far, our discussion of image formation in the eye has emphasized the role of the crystalline lens. The cornea, though, contributes more to the formation of sharp images on the retina than the lens does. To be more precise, the cornea contributes about two-thirds of the eye's total optical power, with the crystalline lens contributing the rest (see Box 2.3). As in the case of the lens, the cornea's shape determines its power—the more spherical the cornea, the more strongly it will converge incoming light. When the cornea is misshapen, the retinal image will be distorted. The most common distortion of shape produces a visual problem called **astigmatism**.

You can test yourself for astigmatism by using the spoke pattern in Figure 2.26. Look at the center of the spoke pattern. Without shifting your gaze, note whether some lines look fainter or have fuzzier edges than others. If they do, you may have astigmatism. This condition is not uncommon. It's

SEEING UNDER WATER

The next time you go swimming, try this experiment. Hold your hand under water and, with your head out of the water, look at your hand. Now, keeping your hand where it was, put your face into the water with your eyes open. When you look at your hand, you'll notice it doesn't look as sharp and clear as it did when your eyes were out of the water. The reason is that the cornea has effectively been eliminated as part of the eyes' optical system.

When light moves from air into the cornea, the path it travels is altered. This alteration, or bending, of light is called **refraction**. The amount of refraction depends on the difference between air and the material out of which the cornea is made. Under water, light enters the eye not from air but from the water itself. Because of the strong similarity between water and the material out of which the cornea is constructed—a large percentage of the cornea is water—light from the water is bent very little as it enters the cornea. Thus, when under water, the eye has effectively no cornea. Putting your face under water has reduced the optical power of your eye by about two-thirds, so it's no wonder you can't see so well. Of the eye's usual optical system, only the crystalline lens remains functional. But steps can be taken to restore the effectiveness of your cornea under water. A transparent diving mask keeps water away from direct contact with your corneas, allowing them to work just as they did with your face out of water.

Other creatures also need to use their eyes under water but don't have access to face masks. How do they do it? If an animal spends all of its time under water there's no problem; its eye is designed so that the cornea contributes little optical power anyway. The lens is strong enough to do all the necessary light bending. Most fish eyes have extremely powerful convex crystalline lenses—a perfect adaptation to their aquatic world.

But what about animals who spend some time above water and some below? They face the same problem that humans do. We'll consider two particularly interesting creatures who solve this challenge in different ways. Think about the problem that a diving bird faces. Flying along, it looks for fish swimming near the surface of the water below. When it spots a fish, the bird dives into the water and tries to snatch the fish. But as soon as it enters the water, the bird's cornea will lose its optical power, handicapping the bird visually. Some diving birds avoid this effect by using the equivalent of an adjustable face mask. The cormorant, for instance, has thick but partially transparent eyelids that close when the bird enters the water. Keeping water from coming into contact with the bird's corneas, the lids preserve much of the cornea's optical power.

But some animals face a situation that is even more optically demanding. Instead of going into and out of the water, these creatures are simultaneously both in and out of the water. The most famous of these creatures is *Anableps anableps*, a freshwater fish found in South and Central America. Because some of its food supply consists of insects above the water, anableps swims along the surface of the river, its eyes half under water and half above water. Anableps has a rather interesting adaptation to this peculiar environmental niche. The upper portion of anableps's eye (the part that is exposed to air) is distinct from the lower portion (the part that is exposed to water). Anableps has two pupils in each eye (one below and the other above the water), but relies more on aerial vision than on aquatic (Albensi and Powell, 1998). In addition, the lower half of its lens is more powerful than the upper half (Sivak, 1976). Anableps is commonly referred to as *cuatro ojos* (Spanish for "four eyes"), but having four eyes rather than two suits Anableps's lifestyle very well indeed.



Differences

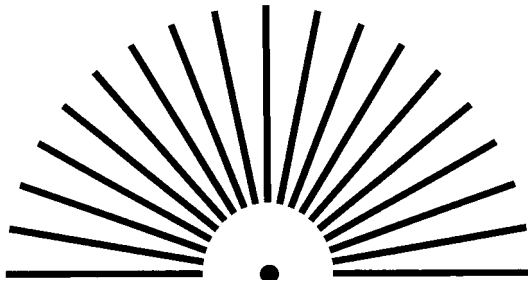


FIGURE 2.26
Chart for testing astigmatism.

caused when the cornea is more sharply curved along one axis than it is along another. These differences in curvature mean that the cornea cannot sharply focus two different line orientations simultaneously on the retina. You may be wearing glasses that correct an astigmatism. To find out, remove your glasses, look through just one lens and slowly rotate the glasses through a 90-degree angle. If the lines on the test chart change in appear-

ance, your glasses contain a correction for astigmatism. People with astigmatism who wear contact lens automatically have their vision corrected. The contact lens itself is perfectly spherical, and it is cushioned snugly against the eye by a thin film of tears. The contact lens thus compensates for any irregularity in the cornea's shape and, thereby, nullifies any astigmatism.

Almost all eyes have some degree of astigmatism because the cornea is almost never perfectly shaped. But for some people the astigmatism may be severe enough to interfere with perception. Figure 2.27 illustrates how severe astigmatism can distort the appearance of a common, everyday scene. Astigmatism can be corrected by providing a lens that compensates for the cornea's distortion by an equal and opposite distortion of its own.

The previous section detailed the optical components responsible for forming images on the back of the eye. Now we're ready to resume looking at the back portion of the eye—the



FIGURE 2.27
The photograph at the right was taken using a lens that simulates astigmatism. (Glyn Cloyd.)

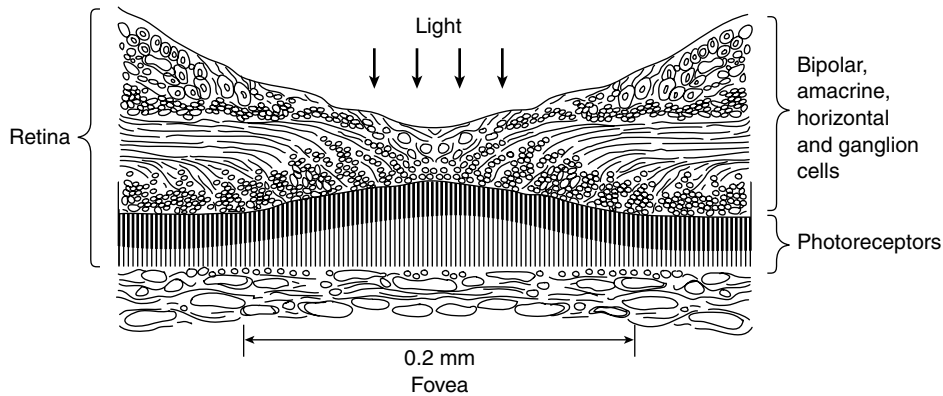


FIGURE 2.28

A sideways view of the central portion of the human retina. (Redrawn from Polyak, 1941.)

retina—which serves as the screen on which the image is cast. As mentioned earlier, this screen is actually located *behind* a complex network of nerve cells. The carefully formed image must pass through this network before the image can be registered by the photoreceptors. Let’s take a look at this odd but obviously successful arrangement.

A SIDEWAYS LOOK AT THE RETINA

Figure 2.28 shows a thin slice of the retina viewed from its side. To create this slice, the retina was carefully removed from the eye, stretched out on a flat surface, and cut downward, through the thickness of the retina. (Special chemical dyes applied to this tissue make different types of retinal cells visually conspicuous under a microscope.)

The cross section in Figure 2.28 was taken from the center of the retina, the region known as the macula. This cross section is arranged so that if it were actually in an animal’s eye, incoming light would first pass through the *top* part of the cross section. Notice that the photoreceptors are situated toward the *bottom* of the drawing. This drawing again reminds us that before light can reach the photoreceptors and initiate the re-

sponses that eventuate in vision, light must traverse the entire thickness of the retina.

Not all regions of the retina have the same thickness. Note in Figure 2.28 that the retina thins out in the center of the macula, forming a pit called the **fovea** (in Latin, *fovea* means “pit”). The retina in the foveal neighborhood is thin because some overlying structures have been pushed to the margins of the foveal pit. There’s a good reason for this small clearing around the fovea. As light passes through the retina, some of it is absorbed or scattered before it can reach the photoreceptors. The thinness of the retina’s fovea minimizes absorption and scatter, thus eliminating the obstacles to the passage of light to this particularly important region of the retina (Hughes, 1977).

With this overview of image formation and the retina firmly in mind, let’s now consider how the photoreceptor cells in the retina sense the presence of light and initiate the process of seeing.

THE PHOTORECEPTORS

The human eye contains two major classes of photoreceptors: **rods** and **cones**. In humans, each eye contains about 100 million rods and approximately 5 million cones (Curcio and Hendrickson,

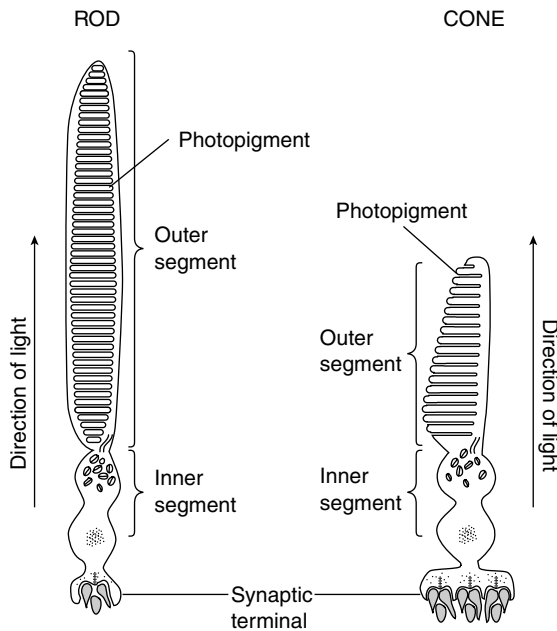


FIGURE 2.29
A single rod photoreceptor and a single cone photoreceptor, magnified approximately 1,500 times.

1991; Wandell, 1995). Figure 2.29 shows an example of rod and cone photoreceptors. The two types derive their names from their appearance. The tip of a cone is tapered, resembling a teepee or an upside down ice cream cone. In contrast, the tip of a rod has straighter sides and a blunt end—it looks like rod. But their differences go well beyond shape. In fact, functional differences between the two types of photoreceptors determine the lifestyles of their owners. Creatures who have a preponderance of rods in their retinas, such as the owl, tend to be most active at night. (We call such creatures “nocturnal” animals.) Creatures who have a preponderance of cones, such as the squirrel, tend to be active only during daylight hours. (We call them “diurnal.”) We humans have duplex retinas, containing both rods and cones; we’re called “arhythmic” since our activities are not limited to just one part of the day/night cycle.

Think back to the analogy between the eye and a camera. The eyes’ photoreceptors would be analogous to the camera’s film. Because human eyes contain two types of photoreceptors, they resemble a camera that holds two different kinds of film at once. The type of film in a camera—color versus black and white—determines what kind of pictures are produced. Our eyes are simultaneously loaded with two different kinds of “film” (rods and cones). The duplex nature of the human eye produces some interesting idiosyncrasies in the way that we see. These idiosyncrasies will become apparent as we survey the differences between the rods and cones.

The Distribution of Rods and Cones

Rods and cones are not uniformly distributed throughout the retina: cones predominate in central vision, and rods predominate in peripheral vision. This differential distribution is shown in Figure 2.30, which plots the density of rods and cones in samples taken from different parts of the retina. In the very center of the macula, only cones are found. There, the cone’s tips are thinner than elsewhere and they’re very tightly packed. About 150,000 cones occupy an area 1 millimeter square (about the size of this letter: o). As we move away from the macula, the number of cones decreases while, at the same time, the number of rods increases. In fact, about 7 millimeters away from the fovea—moving along the retina in a direction toward the nose—rods reach a density approximately the same as that found for cones in the center of the macula. This difference in geographical distribution of rods and cones means that central and peripheral parts of the retina are useful for different aspects of vision. We’ll get to those differences in a moment, but first let’s consider a small, inconspicuous island of complete blindness found within each and every eye.

Notice the interruption in the plots shown in Figure 2.30. This gap denotes the complete absence of photoreceptors at the optic disk.

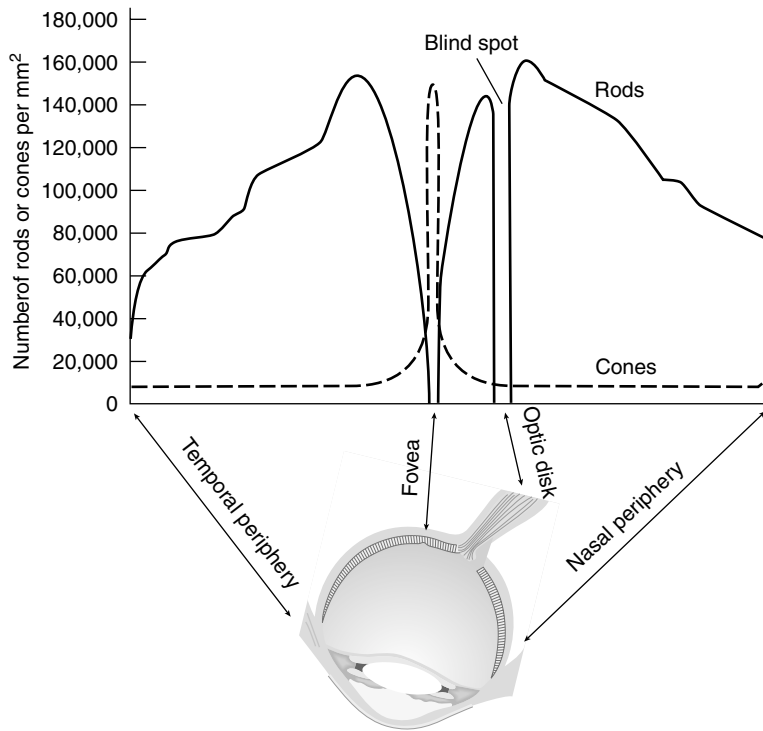


FIGURE 2.30

Distribution of rods and cones over the extent of the retina of the right eye, as seen from above. In the left eye, the nasal and temporal areas of the retina would appear reversed, but the relative distributions of rods and cones would be the same. Note the complete absence of rods within the fovea, where cones abound.

Because you cannot see without photoreceptors, you're actually "blind" to any image falling within this area of the retina devoid of receptors. We referred earlier to an analogy between the photoreceptors and photographic film. Following this analogy, the optic disk would correspond to a defective area of the film, on which the factory neglected to put any light-sensitive chemical. If that defective film were used in a camera and developed, the resulting picture would have a noticeable blank region. Surprisingly though, we almost never notice the large gap that the optic disks create in our retinas. Box 2.4 will help you see what you've been missing all these years.

Let's now return our attention to those parts of the retina that contain photoreceptors. The array

of approximately 100 million photoreceptors in each eye converts the optical image on the retina into a neural image that will be transmitted to the brain. But as Figure 2.31 shows, the density of photoreceptors varies regionally throughout the retina. This means, therefore, that the optical image is not being uniformly sampled by the photoreceptors. Again using our film analogy, it is as if our camera were loaded with film in which the light-sensitive chemical emulsion were unevenly spread over the film's surface. As a result, parts of our photograph would contain sharp detail (where the emulsion was dense) while other parts would appear grainy and blurred (where the emulsion was sparse). Photographic film isn't made this way, of course, but the retina is: it does

A GAP IN YOUR VISUAL FIELD

The optic disk—that place where the axons of the retinal ganglion cells come together to form the optic nerve—contains no photoreceptors. Consequently, it cannot support vision—it is literally a blind spot. Note that we’re distinguishing between a region defined anatomically, the optic disk, and a region defined perceptually, the blind spot. Before we go on about the blind spot, you may want some proof that it actually exists.

Of course you cannot *see* a blind spot (though you can see an optic disk, using an ophthalmoscope). What you can see are the consequences of your blind spot—an object imaged within this blind region of your retina will be invisible. The figure below will help you see the consequences of this blind spot. Making sure that the book is propped up at right angles to the tabletop, view the figure from a distance of about 60 centimeters (about 2 feet). Close your left eye and, using only your right eye, stare at the fixation cross in the figure. At this viewing distance, the black disk to the right of the cross should fall on your optic disk, and will therefore disappear. Since the location of the optic disk varies from one person to the next, you may have to stare at a point slightly different from the fixation cross.

The demonstration of the existence of a blind spot represented a milestone in understanding the eye. Edmé Mariotte, the French scientist who discovered the blind spot in 1668, did not simply stumble upon it by accident (Mariotte, 1668/1948). Instead, his dissection of human eyes suggested to him that vision might be impaired in the region of the optic disk. This was the first time that anyone had predicted a previously unknown perceptual phenome-

non simply from an anatomical observation. From the geometry of the eyeball, including the location of the optic disk, Mariotte correctly predicted where stimuli would have to be placed relative to a fixation point in order for the image to fall on the optic disk. Mariotte also confirmed that there were individual variations in the precise location of the blind spot, corresponding to individual variations in the optic disk itself.

It’s been claimed that England’s “merry monarch,” Charles II, exploited the retina’s blind spot to “behead” symbolically members of his court who were in disfavor (Rushton, 1979). After placing them at the right distance from his throne, Charles would adjust his gaze so that the head of his “victim” was imaged on the optic disk. Although this is an intriguing story, the more so because Charles I, the father of Charles II, had been beheaded, Adam Reeves (1982) describes the story as “a baseless canard” against Charles II. Frankly, we’re not sure who’s correct.

While you were looking for your own blind spot, you may have noticed something else strange. When the black disk disappeared, you weren’t left with a “hole” in your visual field. Instead, the background appeared uniformly white. This phenomenon is called *completion* or “filling in.” Blind spot completion has been cleverly exploited by Ramachandran (1992) to study the filling-in phenomenon in more detail. His article, which contains some delightful demonstrations, concludes that completion of vision across the blindspot is just one instance of a more general process called interpolation, an aspect of perception we shall take up in Chapter 7.



Fixation cross

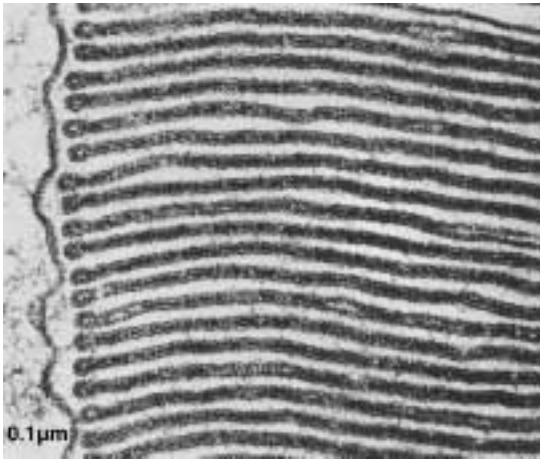


FIGURE 2.31

Highly magnified photograph of an outer segment of a rod photoreceptor from a monkey's eye. The array of thin vertical stripes are stacks of tightly packed disks that house the photosensitive molecules responsible for generating electrical signals when stimulated by light. (John Dowling.)

unevenly register the spatial detail in the image formed on it. In the fovea where cones alone exist, the image is very finely sampled, while out in the periphery, sampling is coarse. You can easily preview the consequence of these sampling differences by trying to read small text out of the corner of your eye—the image of that text is not registered with sufficient detail to convey the indentities of individual letters.

If one knows how densely the photoreceptors are packed, it's possible to calculate the finest pattern that any particular array of receptors could reproduce (Yellott, 1982). In the foveas of most young adults, the photoreceptors are packed very densely, in an orderly arrangement that produces a near-constant distance between neighbors. As one progresses away from the center of the retina, the distances increase between neighboring receptors and the orderliness of the packing diminishes (Hirsch and Miller, 1987). When the photoreceptors are photographed end-on, they suggest a carefully laid mosaic, such as one would make out of small, hexagonal tiles. In the center of the retina, this remarkably orderly and

tightly packed mosaic allows the photoreceptor array to reproduce the finest details that the eye's optics can transmit to the retina.

We've commented admiringly on the exquisite packing of photoreceptors in the central retina of the human adult and on the contribution of that packing to the excellence of vision. The situation is quite different, though, for young infants. It takes about four years for an infant's retina to reach its final, adult state. During that time, photoreceptors migrate toward the center of the retina from the periphery, a migration that creates the pit known as the fovea. Even as late as 15 months of age, the distance between neighboring photoreceptors of the central retina is twice the comparable value in the adult eye (Yuodelis and Hendrickson, 1986). Although some of the infants' poor visual acuity arises from the immature state of their brains, the lion's share results from immaturity at the retinal level (Banks and Bennett, 1988; Teller and Movshon, 1986).

An analogous, though far less dramatic, change occurs at the other end of the life span. In old age, the packing of receptors in the central retina changes because of cell death and other factors (Weale, 1986). Undoubtedly, this contributes to the drop in acuity that accompanies aging (Owsley, Sekuler and Siemsen, 1983).

Although retinal anatomy and retinal function follow parallel courses, in some humans, retinal anatomy and retinal function are permanently arrested at the infant stage. These humans, whose skin, hair and eyes lack pigmentation, have a genetic disorder known as albinism (Abadi and Pascal, 1989). Melanin, the pigment that gives color to skin, hair and eyes, is particularly abundant in the macular region of the normal retina. The melanin promotes the inward migration of photoreceptors and the formation of the foveal pit. The albino eye, lacking melanin, does not have much receptor migration and never develops a foveal pit. As a result, the number of photoreceptors per unit area in the central retina of the albino eye is far below that of pigmented adults. Hugh Wilson and colleagues have studied the vision of



Differences



Clinical

human albinos and liken their central retinal anatomy and function to that of a normal 10-month-old (Wilson et al., 1988). All these unusual cases—infants, the aged, and albinos—reinforce the idea that the retina’s photoreceptor mosaic sets important limits on the information that can be extracted from the retinal image.

THE FIRST STEP TOWARD SEEING



Neural

Light registers its presence on the retina by interacting with special light-sensitive molecules contained within the photoreceptors. These molecules, called **photopigments**, are contained in the membrane portions of an array of discs that are tightly packed within the outer segment of each and every photoreceptor (see Figure 2.31). Light passing through these discs triggers a cascade of biochemical events that culminate in the generation of electrical signals within the photoreceptor. We’ll start by considering the chemical basis of the photopigment molecules.

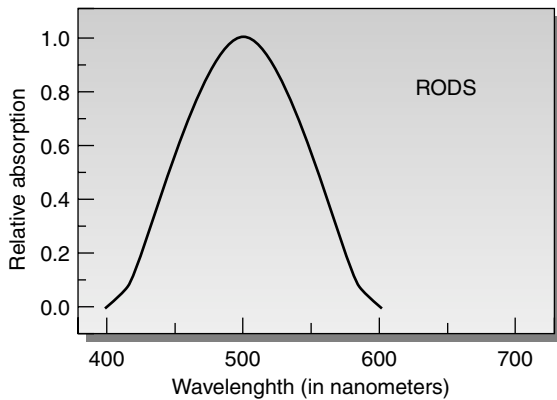
Each photopigment molecule consists of two components: a very large protein, *opsin*; and another component, *retinal*, derived from vitamin A. Normally, the two components are tightly connected, producing a stable molecule that won’t break up spontaneously. But when light strikes one of these molecules, some of the light’s energy is imparted to the molecule, causing it to change shape, or *isomerize*. Once this shape change occurs, the photopigment molecule is no longer stable and its two components undergo a series of changes, eventually splitting apart.

This change in shape alters the flow of electric current in and around the photoreceptor. In the absence of light, electrical current flows into the stack of discs within the photoreceptor’s outer segment. This inward current flow, then, signifies that the eye is in darkness. Photopigment isomerization, triggered by the presence of light, briefly reduces this flow of current. That change in current flow signifies the presence of light. The greater the amount of light, the larger the number of molecules isomerized and the greater the resulting change in electrical potential.

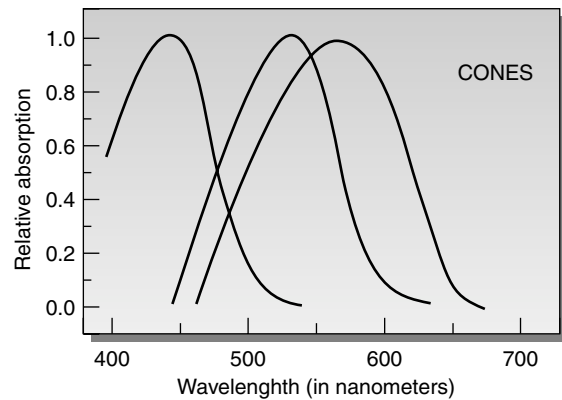
This change in current moves down the length of the photoreceptor to its terminal end, where specialized chemical messengers, called **transmitter substances**, are housed. In the photoreceptors, these messengers are glutamate molecules. In the absence of light, glutamate is continuously released by the photoreceptor into the very small gap that separates the photoreceptor from the cells with which they are in chemical communication. This gap is called the **synapse**. The presence of light—through its effect on the photopigments and the electrical current flow—reduces the rate of glutamate released into the synapse. Light, in other words, turns *down* the release of chemical messages by the photoreceptors. Like the photoreceptors pointing backward into the eye, this is another one of those counterintuitive ways that nature constructs biological mechanisms. But obviously it works, and very efficiently. The entire chain of events—from absorption to isomerization to current flow is called **phototransduction**. It occurs in less than a thousandth of a second. In the next chapter, we’ll learn how the cells in communication with the photoreceptors make sense of these odd messages.

Not all light wavelengths are equally effective in triggering phototransduction. While retinal is the same for all human photopigments, opsin comes in four different molecular forms, each having its own unique sensitivity to lights of different wavelengths. These different molecular forms are genetically determined, and genetic mutation can and does alter the form of the opsins in some individuals. The various sensitivities of opsins to light wavelengths is fundamentally important for vision, but we’ll postpone discussing that until the next chapter. For now, let’s survey the different types of photoreceptors as defined by their wavelength sensitivity.

Exposing a photoreceptor to light from various regions of the wavelength spectrum makes it possible to measure how much of that light is actually absorbed and, therefore, how much that wavelength stimulates the photoreceptor. When this is done, one finds that for any given receptor there is one wavelength of light that most

**FIGURE 2.32**

This graph shows how the amount of light absorbed by rod photoreceptors varies with the wavelength of the light.

**FIGURE 2.33**

This graph shows how the amount of light absorbed by each of three types of cone photoreceptors varies with wavelength.

strongly stimulates the receptor. There is, in other words, one wavelength to which the receptor is most sensitive. Rods give their biggest response when stimulated with approximately 500 nanometers (the wavelength of light is measured in **nanometers**, billionths of a meter); shorter or longer wavelengths give a diminished response. A summary of the rod's spectral sensitivity is graphically illustrated in Figure 2.32. To appreciate the stimulus to which rods are most sensitive, you should know that under daylight conditions, light of 500 nanometers looks bluish-green.

The corresponding story for cones is more complicated, since the wavelength at which sensitivity is optimum depends on *which* type of cone is being studied. There are three distinct classes of cones. One is maximally responsive to light of about 440 nanometers, a second class responds best to light of 530 nanometers, and a third class has its peak response at 560 nanometers. The responses of these three classes of cones are shown in Figure 2.33 as functions of the wavelength of stimulating light. Again, to give you some reference points, under daylight conditions, light of 440 nanometers looks violet, light of 530 nanometers looks green, and light of 560 nanometers looks yellow.

The curves shown in Figures 2.32 and 2.33 underscore an important feature of human vision. The curves separate electromagnetic radiation that one *can* see ("light") from electromagnetic radiation that one *cannot* see. For example, one cannot see infrared radiation (wavelengths longer than 700 nanometers) because human photopigments do not respond to wavelengths that long. (Recall the story about the World War II sailors whose vision was altered by extending the range of wavelengths to which their photopigments were responsive.)

To actually see, though, requires more than just a change in a photoreceptor's own electrical state. Messages about the presence of light must be transmitted from the receptors to the intermediate network of bipolar, amacrine and horizontal cells and then to the retinal ganglion cells, which communicate the outcome of all this neural analysis to the brain. The first three boxes in Figure 2.34 summarize the events that we've been describing. The remaining boxes represent the concerns of the next two chapters.

Box 2.5 provides tantalizing clues for the existence of a third type of photoreceptor in the eye, one whose job is entirely different from that of the rods and cones.

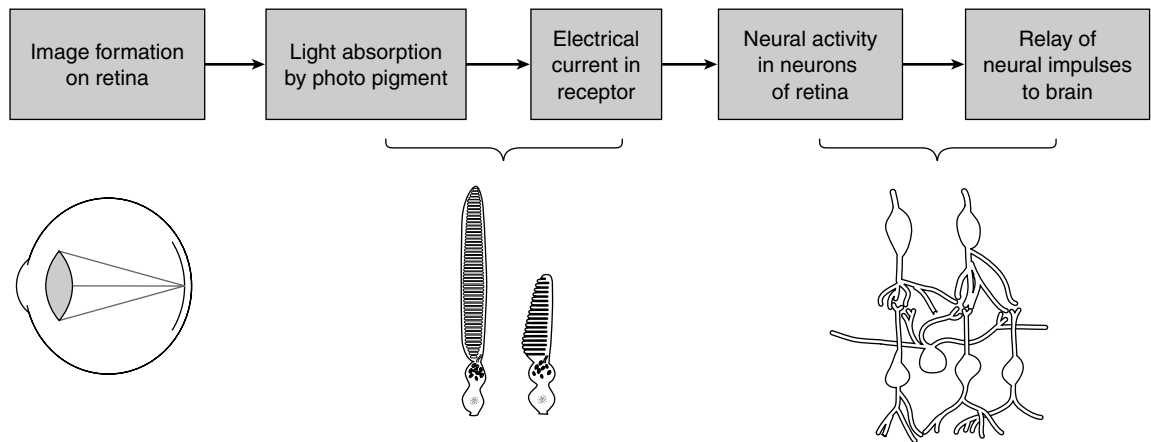


FIGURE 2.34

The three boxes at the left of this figure summarize the events discussed in this chapter. The remaining boxes represent the topics of the next two chapters.

BOX 2.5

YOUR DAILY DOSE OF LIGHT

The text stresses that vision isn't concerned with seeing light but, instead, with seeing objects and events. Thus, light is simply the messenger that carries optical information from the environment to our eyes. There is one aspect of vision, however, where light itself *is* the message. We don't normally stop to think about this aspect of vision. Light calibrates the internal, neural clock that our brains use to regulate our sleep/wake cycles, our appetites and our hormone levels. It's this clock that temporarily gets out of whack when we travel overseas.

Called **circadian rhythms**, these periodic activities and bodily functions are normally coupled to our 24-hour light/dark cycle. In the absence of light, however, the clock that regulates circadian rhythms can drift out of synch. Thus, rats placed in complete darkness for weeks at a time, or humans who voluntarily live in dark caves for several months slip into sleep/wake cycles that deviate significantly from the

normal solar cycle. Once these light-deprived individuals return to environments with normal light exposure, their clocks quickly resynchronize. The eyes are critical for this resetting of the clock.

One would naturally expect either rod or cone photoreceptors to be responsible for setting the daily clock, but recent evidence suggests this is not the case. Mice lack both rod and cone photoreceptors, and yet they maintain normal activity cycles and their light-dependent hormone levels remain normal (Freedman et al., 1999). Evidently, the eye contains a third class of light-sensitive neural elements especially designed for internal clock control. Speculation currently centers around a protein found in the eye called cryptochrome (Barinaga, 1999). But the cells housing this time-keeper protein remain unidentified, as do the ganglion cells that communicate the photoreceptive signals to those regions of the brain responsible for regulating circadian behaviors.

SUMMARY AND PREVIEW

This chapter has laid out the basic design of the human eyeball, emphasizing the good fit between its structure and the job it must do. Because vision depends on an interaction between light and the eye, we also considered how light itself manages to capture information about the environment, information that is conveyed by light. This led us to a discussion of the eyeball's optical characteristics and various common imperfections in those characteristics. The chapter ended

with the capture of light by photopigment molecules and the first step toward seeing—photoreceptor responses that are communicated to other neurons in the retina and eventually to the brain. The next chapter follows these messages as they pass from one retinal neuron to the next. You already know that your vision mirrors the properties of your photoreceptors; the next chapter will show you how other elements in the retina also control what you see.

KEY TERMS

accommodation	emmetropic	photoreceptors
afterimage	extraocular muscles	phototransduction
amacrine cells	fibrous tunic	pigment epithelium
anomalous myopia	floaters	presbyopia
aqueous humor	fovea	pupil
astigmatism	glaucoma	rectus muscles
bipolar cells	horizontal cells	refraction
cataract	hyperopic	retina
choroid	image	retinal ganglion cells
ciliary body	inverse optics	retinal image
circadian rhythms	iris	rods
collector cells	macula	sclera
cones	myopic	sclerosis
conjunctive	nanometers	synapse
cornea	ophthalmoscope	transmitter substances
crystalline lens	optic disk	vascular tunic
depth of field	orbit	vergence
divergent	outer segment	vitreous
electromagnetic radiation	photopigments	wavelength