

The First Steps in Vision: From Light to Neural Signals

WITH THE NAKED EYE WE CAN SEE STARS that are up to about 2000 light-years (almost 6 trillion miles) away, and in a dark winter sky far from city lights the neighboring galaxy, Andromeda, is visible at over 2 million light-years away! This chapter describes the first steps in seeing. To understand how we see, we must first consider a little physics and optics, and then we'll look at how the eye is built to capture and begin to process light.

In Chapters 3–8 we'll see how light information gleaned by the eyes travels back through the head to the brain, and how the brain transforms this information into a meaningful interpretation of the outside world. For a preview of the entire visual process, work through **Web Activity 2.1: Visual System Overview**.

A Little Light Physics

Light is a form of electromagnetic radiation—energy produced by vibrations of electrically charged material. There are two ways to conceptualize light: as a **wave** or as a stream of **photons**, tiny particles that each consist of one quantum of energy. This dual nature of light can be confusing to physics and psychology students alike. In this discussion we'll try to avoid confusion as much as possible by treating light as being made up of waves when it moves around the world, and being made up of photons when it is absorbed.

Although the full spectrum of electromagnetic radiation is very wide, light makes up only a tiny portion of this spectrum. **Figure 2.1** illustrates the electromagnetic spectrum, from gamma rays (which have very short wavelengths) to radio and television waves (which have very long wavelengths). Visible light waves have wavelengths between 400 and 700 nanometers (nm; $1 \text{ nm} = 10^{-9}$ meter), as illustrated on the bottom of the figure. Note that as the wavelength varies in the visible spectrum, the color we observe changes, from violet at about 400 nm through the whole spectrum of the rainbow up to red at about 650 nm. (As we'll discuss in Chapter 5, however, the light waves themselves are not colored; it is only after our visual system interprets an incoming wave that we perceive the light as a specific color.)

Let's consider what happens to light on its way from a star to an eye. (See **Web Activity 2.2: From Sun to Eye**.) In empty space the electromagnetic radiation from a star travels in a straight line at the speed of light (about 186,000 miles per second). Once it reaches the atmosphere, some of the starlight's photons will be **absorbed** by encounters with dust, vaporized water, and so on; and some of the light will be **scattered** (sometimes called diffracted) by these particles. Most of the photons, however, will make it through the atmosphere and will eventually hit the surface of an object.

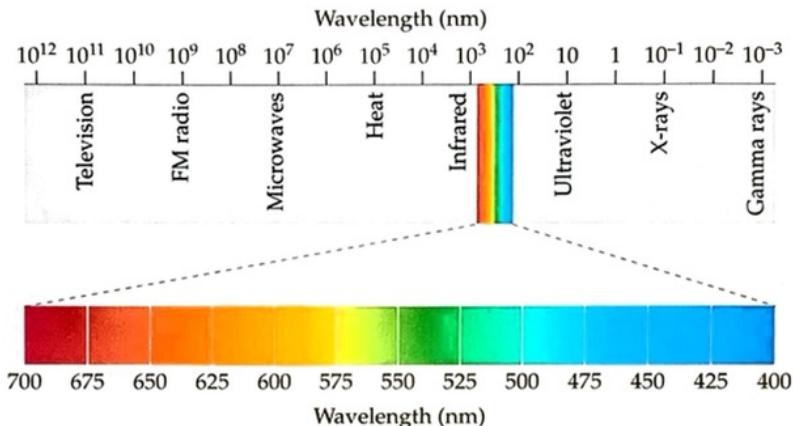
wave An oscillation that travels through a medium by transferring energy from one particle or point to another without causing any permanent displacement of the medium.

photon A quantum of visible light or other form of electromagnetic radiation demonstrating both particle and wave properties.

absorb To take up something—such as light, noise, or energy—and not transmit it at all.

scatter To disperse something—such as light—in an irregular fashion.

FIGURE 2.1 The spectrum of electromagnetic energy (specified in nanometers), with the visible spectrum (400–700 nm) expanded at right. Note that $1 \text{ nm} = 10^{-9} \text{ m}$.



If the ray of starlight were to strike a light-colored surface, most of the light would be **reflected**. Indeed, the fact that most of the light bounces off the surface accounts for that surface's "light" appearance. However, most of the light striking a dark surface is absorbed. Light that is neither reflected nor absorbed by the surface is **transmitted** through the surface. If we are gazing at our star through a window as the light travels from air into the glass, some of the light rays will be bent, or **refracted**, as it is transmitted.

Refraction also occurs when light passes from air into water or into the eyeball. In fact, the part of an eye exam in which the eye doctor checks the patient's prescription is often called a "refraction" because the doctor determines how much the light must be bent by eyeglasses for it to be properly focused on the retina. In the next section we'll see how the optic system of our eyes performs this same kind of focusing.

Eyes That Capture Light

In order to see stars or anything else, we need some type of physiological mechanism for sensing light. Even single-celled organisms such as amoebas respond to light, changing their direction of motion to avoid bright light when it is detected. But eyes go well beyond mere light detection. An eye can form an **image** of the outside world, enabling animals that possess eyes to use light to recognize objects, not just to determine whether light is present and what direction it's coming from.

Before explaining how eyes form images, let's take a tour through the human eye to become familiar with its important parts. Figure 2.2 shows a front-to-back slice through a human eye, with the most important structures labeled. (See [Web Activity 2.3: Eye Structure](#).)

The first tissue that light from the star will encounter is the **cornea**. Contact lenses sit on a thin film of tears in front of the cornea. The cornea provides a window to the world because it is **transparent** (that is, most light photons are transmitted through it, rather than being reflected or absorbed). It is transparent because it is made of a highly ordered arrangement of fibers and because it contains no blood vessels or blood, which would absorb light. The cornea does, however, have a rich supply of transparent sensory nerve endings, which are there to force the eyes to close and produce tears if the cornea is scratched, preserving its transparency. If you have ever scratched your cornea or worn contact lenses too long, you know exactly how painful this can be! Fortunately, the external layers of the cornea regenerate very quickly, so even when a cornea is scratched, it usually heals within 24 hours.

reflect To redirect something that strikes a surface—especially light, sound, or heat—usually back toward its point of origin.

transmit To convey something (e.g., light) from one place or thing to another.

refract 1. To alter the course of a wave of energy that passes into something from another medium, as water does to light entering it from the air. 2. To measure the degree of refraction in a lens or eye.

image A picture or likeness.

cornea The transparent "window" into the eyeball.

transparent Allowing light to pass through with no interruption, so that objects on the other side can be clearly seen.

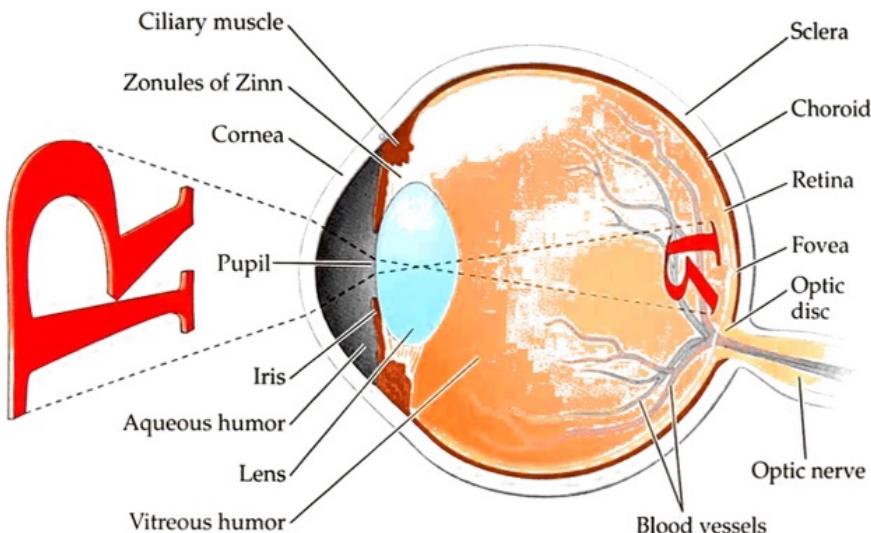


FIGURE 2.2 The human right eye in cross section (viewed from above). Note that the *R* on the retina is reversed right to left, and it is upside down. The “hole” in the retina where the optic nerve leaves the eyeball is the optic disc (where the absence of photoreceptors results in a blind spot). (After Breedlove and Watson, 2013.)

The **aqueous humor**, a fluid derived from blood, fills the space immediately behind the cornea, supplying oxygen and nutrients to, and removing waste from, the cornea and the **lens**. Like the cornea, the lens has no blood supply, so it can be completely transparent; and, as we'll see later, the shape of the lens is controlled by the ciliary muscle.

To get to the lens, the light from our star must pass through the **pupil**, which is simply a hole in a muscular structure called the **iris**. The iris gives the eye its distinctive color, and controls the size of the pupil, and thus the amount of light that reaches the retina, via the pupillary light reflex. When the level of light increases or decreases, the iris automatically expands or contracts to allow more or less light into the eye.

After passing through the lens, our starlight will enter the vitreous chamber (the space between the lens and the retina), where it will be refracted for the fourth and final time by the **vitreous humor**. This is the longest part of the journey through the eyeball; this chamber comprises 80% of the internal volume of the eye. The vitreous is gel-like and viscous (a bit like egg white), and generally transparent. While staring up at the bright blue sky on a lazy sunny day, however, you may have noticed “floaters,” small bits of debris (biodebris) that drift around in the vitreous. Floaters are quite common, and they are usually not a cause for concern.

Finally, after traveling through the vitreous chamber, the light emitted by our favorite star will (hopefully) be brought into focus at the **retina**. To be a bit more precise, only some of the light will actually reach the retina. Much of the light energy will have been lost in space or the atmosphere, because of absorption and scattering, as described already. In addition, a good deal of light becomes lost in the eyeball, so only about half of the starlight that arrives at the cornea actually reaches the retina. The role of the retina is to detect light and “tell the brain about aspects of light that are related to objects in the world” (Oyster, 1999). In other words, the retina is where seeing really begins.

Focusing Light onto the Retina

To focus a distant star on the retina, the refractive power of the four optic components of the eye must be perfectly matched to the length of the eyeball. This perfect match, known as **emmetropia**, is illustrated in Figure 2.3a. The average eyeball is about 24 millimeters (mm) long and has a power, when unaccommodated (focused on a distant object like our star), of about +60 diopters. (We'll explain what accommodation means in a bit.)

aqueous humor The watery fluid in the anterior chamber of the eye.

lens The lens inside the eye that enables the changing of focus.

pupil The dark, circular opening at the center of the iris in the eye, where light enters the eye.

iris The colored part of the eye, consisting of a muscular diaphragm surrounding the pupil and regulating the light entering the eye by expanding and contracting the pupil.

vitreous humor The transparent fluid that fills the vitreous chamber in the posterior part of the eye.

retina A light-sensitive membrane in the back of the eye that contains rods and cones, which receive an image from the lens and send it to the brain through the optic nerve.

emmetropia The condition in which there is no refractive error, because the refractive power of the eye is perfectly matched to the length of the eyeball.

diopter (D) A unit of measurement of the optic power of a lens. It is equal to the reciprocal of the focal length, in meters. A 2-diopter lens will bring parallel rays of light into focus at $\frac{1}{2}$ meter (50 cm).

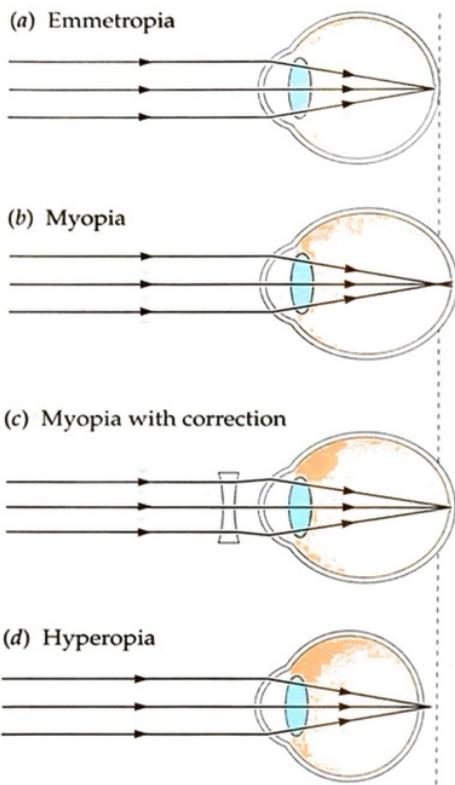


FIGURE 2.3 Optics of the human eye. See the text for details. (After Oyster, 1999.)

Refractive errors occur when the eyeball is too long or too short relative to the power of the optic components. If the eyeball is too long for the optics (**Figure 2.3b**), the image of our star will be focused *in front* of the retina, and the star will thus be seen as a blur rather than a spot of light. This condition is called **myopia** (or “nearsightedness”). Myopia can be corrected with negative (minus) lenses, which diverge the rays of starlight before they enter the eye (**Figure 2.3c**). If the eyeball is too short for the optics (**Figure 2.3d**), the image of our star will be focused *behind* the retina—a condition called **hyperopia** (or “farsightedness”). If the hyperopia is not too severe, a young hyperope can compensate by accommodating, thereby increasing the power of the eye. If accommodation fails to correct the hyperopia, the star’s image will again be blurred. Hyperopia can be corrected with positive (plus) lenses, which converge the rays of starlight before they enter the eye.

On average, the adult human eye is 24 mm long, about the diameter of a quarter. However, eyeballs can be quite a bit longer or shorter and still be emmetropic because eyes generally grow to match the power of the optic components we’re born with. (Most newborns are hyperopic because the optic components of their eyes are relatively well developed at birth compared with the length of their eyeballs.)

The most powerful refracting surface in the eye is the cornea, which contributes about two-thirds of the eye’s focusing power. When the cornea is not spherical, the result is **astigmatism**. With astigmatism, vertical lines might be focused slightly in front of the retina, while horizontal lines are focused slightly behind it (or vice versa). If you have a reasonable degree of uncorrected astigmatism, one or more of the lines in **Figure 2.4** might appear to be out of focus while other lines appear sharp. Lenses that have two focal points (that is, lenses that provide different amounts of focusing power in the horizontal and vertical planes) can correct astigmatism.

So far, we’ve considered the image of a distant object. But what happens when we want to focus on something close by (like the words on this page)? Remember that refraction (light bending) is necessary to focus light rays. Because the cornea is highly curved and has a higher refractive index than air (1.376 versus 1), it forms the most powerful refractive surface in the eye. The aqueous and vitreous humors also help refract light. However, the refractive power of each of these three structures is fixed, so they cannot be used to bring close objects into focus. This job is performed by the lens, which can alter the refractive power by changing its shape—a process called **accommodation**.

Accommodation (change in focus) is accomplished through contraction of the ciliary muscle. The lens is attached to the ciliary muscle through tiny fibers (known as the zonules of Zinn) (see **Figure 2.2**). When the ciliary muscle is relaxed, the zonules are stretched and the lens is relatively flat. In this state, the eye will be focused on very distant objects (like our star). But to focus on something closer—say, a wristwatch—the ciliary muscle must contract. This contraction reduces the tension on the zonules and enables the lens to bulge. The fatter the lens is, the more power it has.

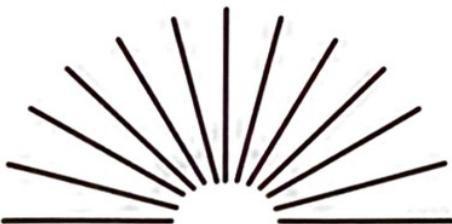


FIGURE 2.4 Fan chart for astigmatism. Take off your glasses (if you wear glasses) and view this “fan.” If you have a significant degree of astigmatism, one or more of the lines will appear to have lower contrast.

FIGURE 2.5 The precipitous drop in amplitude of accommodation with age. Each symbol represents data from a different study. The gray dashed line indicates the amplitude of accommodation required to focus at a distance 40 cm. Each colored symbol represents data from a different “classical” study.

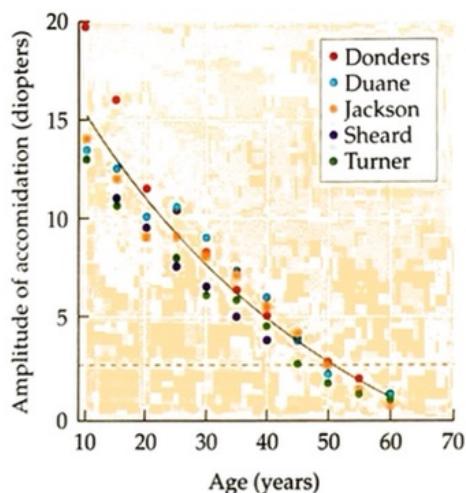
Accommodation enables the power of the lens to vary by as much as 15 diopters. Note that lens power ($P = 1/f$, where f is the focal distance in meters). So, if your unaccommodated eyes were perfectly corrected for distant vision, 15 diopters of accommodation would enable you to read your watch at a distance of about 0.067 meters ($1/15$) or 6.7 centimeters (cm; to convert meters to centimeters, simply multiply by 100). If you can read your watch at 6.7 cm (while wearing your distance correction), you are either very lucky or very young. Our ability to accommodate declines with age, starting from about 8 years old, and we lose about 1 diopter of accommodation every 5 years up to age 30 (and even more after age 30). By the time most people are between 40 and 50 years old, they find that their arms are too short because they can no longer easily accommodate the 2.5 diopters or so needed to see clearly at 40 cm ($1/0.4 = 2.5$). This condition is called **presbyopia** (meaning “old sight”), and it is, like death and taxes, inevitable! Figure 2.5 illustrates the precipitous drop in accommodation with age.

Why do we all have presbyopia to look forward to? The main reason is that the lens becomes sclerotic (harder) and the capsule that encircles the lens (enabling it to change shape) loses its elasticity. Lucky for us, Benjamin Franklin (1706–1790) invented bifocals—lenses that have one power at the top (permitting us to see distant objects) and a different power at the bottom (allowing us to be in focus at a comfortable reading distance).

Like the other optic components of the eye, the lens is normally transparent. It is transparent because the crystallins (a class of proteins that make up the lens) are packed together very densely and therefore are very regular. Anything that interferes with the regularity of the crystallins will result in loss of transparency (areas that are opaque—that is, “opacities”). Opacities of the lens are known as **cataracts**. Cataracts can occur at different ages and take many different forms. Congenital cataracts (present at birth) are relatively rare, but if they are dense (and therefore interfere with retinal image quality), they can have devastating effects on normal visual development if not treated promptly. Most cataracts are discovered after age 50, and the prevalence of cataracts increases with age, so by 70 almost everyone has some loss of transparency. Cataracts can interfere with vision because they absorb and scatter more light than the normal lens does. Fortunately, treatment of cataracts (in which the opacified lens is extracted and replaced with a plastic or silicone implant) has become quite routine.

The Retina

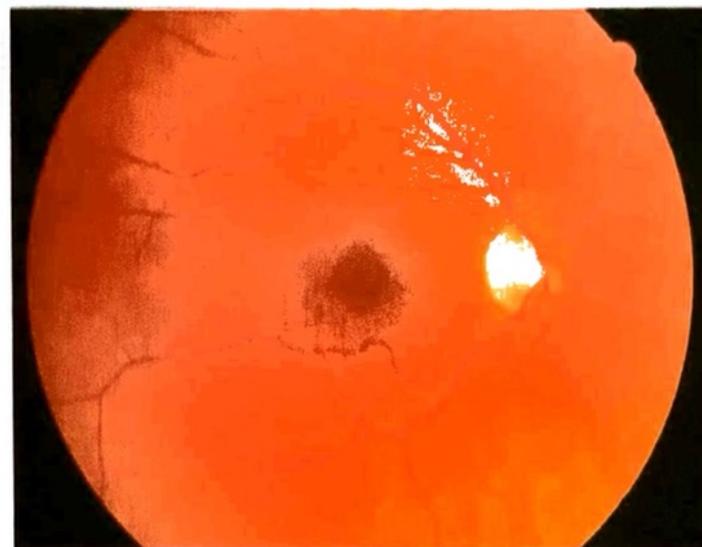
The preceding discussion covered how the human visual system delivers a focused image of our favorite star onto the retina, which is spread across the back of the eyeball. The optics involved (see Figure 2.2) have been likened to those in most cameras, which also include a mechanism for regulating the amount of light (the “stop,” analogous to the iris in human eyes) and a lens for adjusting focal length so that both near and far objects can be focused on the film spread across the back of the camera. However, this is as far as we can take this analogy, because the purpose of a camera is simply to record the image projected onto the film, while the purpose of the human visual system is to interpret this image. This is the difference between taking a picture and seeing a picture. And the process of seeing begins with the retina, where the



presbyopia Literally “old sight.” The age-related loss of accommodation, which makes it difficult to focus on near objects.

cataract An opacity of the crystalline lens.

FIGURE 2.6 Fundus of the right eye of a human. (From Rodieck, 1998.)



transduce To convert from one form of energy to another (e.g., from light to neural electrical energy, or from mechanical movement to neural electrical energy).

fundus The back layer of the retina: what the eye doctor sees through an ophthalmoscope.

light energy from our star is **transduced** into neural energy that can be interpreted by the brain.

What the Doctor Saw

Eye doctors use an instrument called an ophthalmoscope to look at the back surface of their patients' eyes, which is called the **fundus** (plural *fundi*). (You probably remember all too well having that bright light shining into your eye while the doctor examined your fundus.) **Figure 2.6** shows a normal fundus. The white circle is known as the optic disc. This is the point where the arteries and veins that feed the retina enter the eye, and where the axons of ganglion cells (which we will get to shortly) leave the eye via the optic nerve. This portion of the retina contains no photoreceptors, and consequently it is blind. You can experience your own blind spot, corresponding to the optic disc, by closing your left eye, fixating on the *F* in **Figure 2.7a** with your right

FIGURE 2.7 To experience your blind spot, close your left eye, fixating on the *F* in part (a) with your right eye. Hold the book about 15 cm away from your eye, and adjust the distance of the book from your eyes until the red circle disappears. This is your blind spot. Ordinarily you are not aware of it, because the visual system "fills in" the blind spot with information from the surrounding area. If you fixate on the *F* in part (b) with your right eye and again adjust the distance, when the gap in the line falls in your blind spot, it will fill in and you will see a continuous red line.

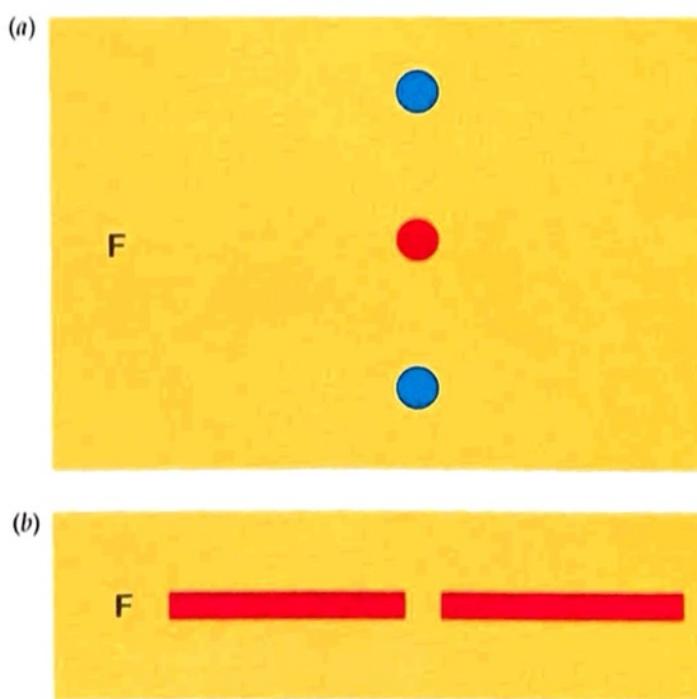


FIGURE 2.6 Fundus of the right eye of a human. (From Rodieck, 1998.)



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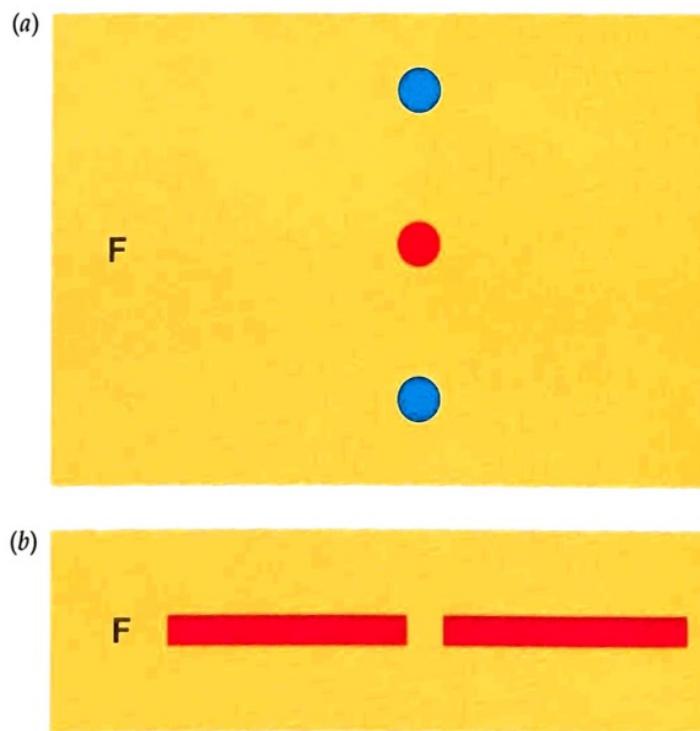
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eye, and adjusting the distance of the book from your eyes until the red circle disappears. The reason you don't normally notice this large blind spot in your visual field is that the visual system "fills it in" with information from the surrounding area (Figure 2.7b).

The fundus is the only place in the body where one can see the arteries and veins directly, so it provides doctors with an important window on the well-being of the body's vascular system. The vascular "tree" spreads out across the retina in a characteristic way but stops short of the fovea (the center of the brownish spot near the center of the fundus in Figure 2.6).

You can see your own vascular tree by using a simple trick that requires only a penlight. In a dark room, close your eyes and place the penlight against the outside corner of one eye. Holding the penlight against the eye, gently move the light around (up and down, and back and forth). Within a few seconds you should see the shadows cast by your blood vessels looking like the branches of a tree. We don't normally see them because the blood vessels move with our eyes, so their shadows are stabilized retinal images and, as with the blind spot, the visual system fills in behind them. The motion of the penlight makes the shadows move, enabling us to see them.

Even when viewed through an ophthalmoscope with a lot of magnification, the fundus does not provide a detailed view of the retina. To get a good look at the structure of the retina, we need a photomicrograph (Figure 2.8), which reveals that the retina is a layered sheet of clear neurons, about half the thickness of a credit card (Rodieck, 1998), with another layer of darker cells, the pigment epithelium, lying behind the final layer.

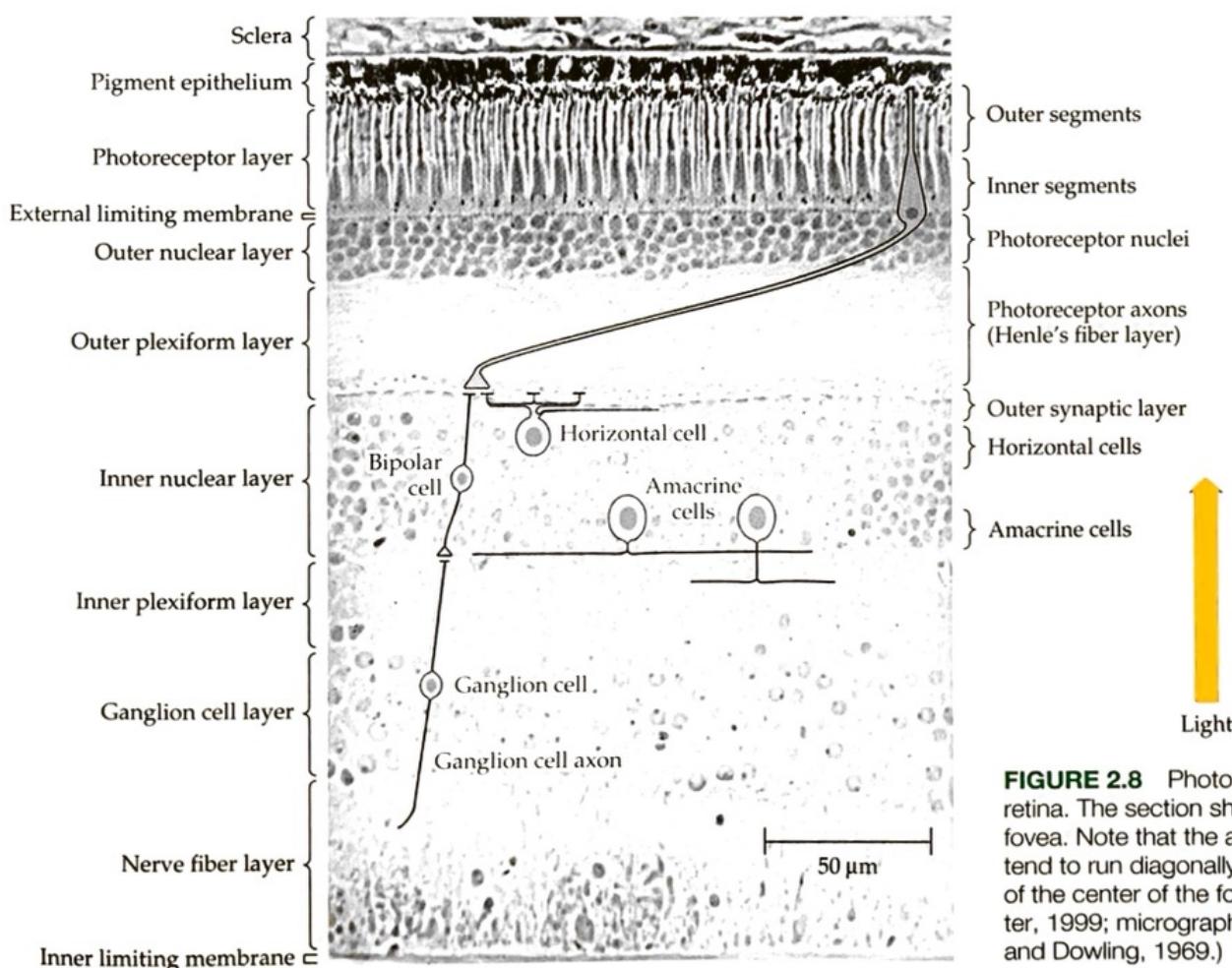


FIGURE 2.8 Photomicrograph of the retina. The section shown is near the fovea. Note that the axons in this region tend to run diagonally to get them out of the center of the fovea. (From Oysterman, 1999; micrograph from Boycott and Dowling, 1969.)

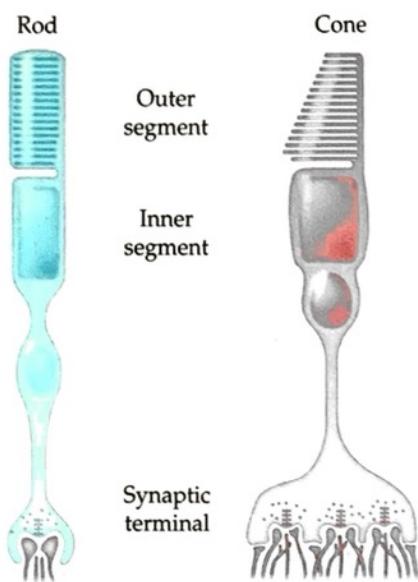


FIGURE 2.9 Rod and cone. (From Rodieck, 1998.)

As we'll see in the next section, together these neurons constitute a mini-computer that begins the process of interpreting the information contained in visual images. The transduction of light energy into neural energy begins in the backmost layer of the retina, which is made up of cells called **photoreceptors** (see Figure 2.8). When photoreceptors sense light, they can stimulate neurons in the intermediate layers, including bipolar cells, horizontal cells, and amacrine cells. These neurons then connect with the frontmost layer of the retina, made up of ganglion cells, whose axons pass through the optic nerve to the brain.

Before we describe the function of these layers, we should address an obvious question regarding the structure of the retina (see Figure 2.8): Why are the photoreceptors at the back—that is, in the last layer? This arrangement requires light to pass through the ganglion, horizontal, and amacrine cells before making contact with the photoreceptors. However, these neurons are mostly transparent, whereas cells in the pigment epithelium, which provide vital nutrients to the photoreceptors, are opaque. Once we see that the photoreceptors must be next to both the pigment epithelium and the other neurons, the layering order makes much more sense.

Retinal Geography and Function

The retina contains roughly 100 million photoreceptors. These are the neurons that capture light and initiate the act of seeing by producing chemical signals. The human retina contains at least two types of photoreceptors: **rods** and **cones**. These two types not only have different shapes (which is how they earned their names; see Figure 2.9), but they have different distributions across the retina and serve different functions. Because human retinas have both rods and cones, they are considered to be **duplex** retinas. Some animals, such as rats and owls, have mostly rod retinas; others (e.g., certain lizards) have mostly cone retinas.

Humans have many more rods (about 90 million) than cones (about 4–5 million), and the two types of cells have very different geographic distributions on the retina (Figure 2.10). Rods are completely absent from the center of the fovea, and their density increases to a peak at about 20 degrees and then declines again. The cones are most concentrated in the center of the **fovea**, and their density drops off dramatically with retinal **eccentricity** (distance from the fovea). The fovea is the “pit” in the inner retina that is specialized for seeing fine detail.

As the photographs of photoreceptors at different eccentricities in Figure 2.10 illustrate, cones are also smaller and more tightly packed in the foveal center (0.0 in Figure 2.10). This “rod-free” area (about 300 square micrometers [μm^2] on the retina) subtends a visual angle of about 1 degree, and it is directly behind the center of the pupil. So if we look directly at an object whose image

photoreceptor A light-sensitive receptor in the retina.

rod A photoreceptor specialized for night vision.

cone A photoreceptor specialized for daylight vision, fine visual acuity, and color.

duplex In reference to the retina, consisting of two parts: the rods and cones, which operate under different conditions.

fovea A small pit, near the center of the macula, that contains the highest concentration of cones, and no rods. It is the portion of the retina that produces the highest visual acuity and serves as the point of fixation.

eccentricity The distance between the retinal image and the fovea.

TABLE 2.1
Properties of the fovea and periphery in human vision

Property	Fovea	Periphery
Photoreceptor type	Mostly cones	Mostly rods
Bipolar cell type	Midget	Diffuse
Convergence	Low	High
Receptive-field size	Small	Large
Acuity (detail)	High	Low
Light sensitivity	Low	High

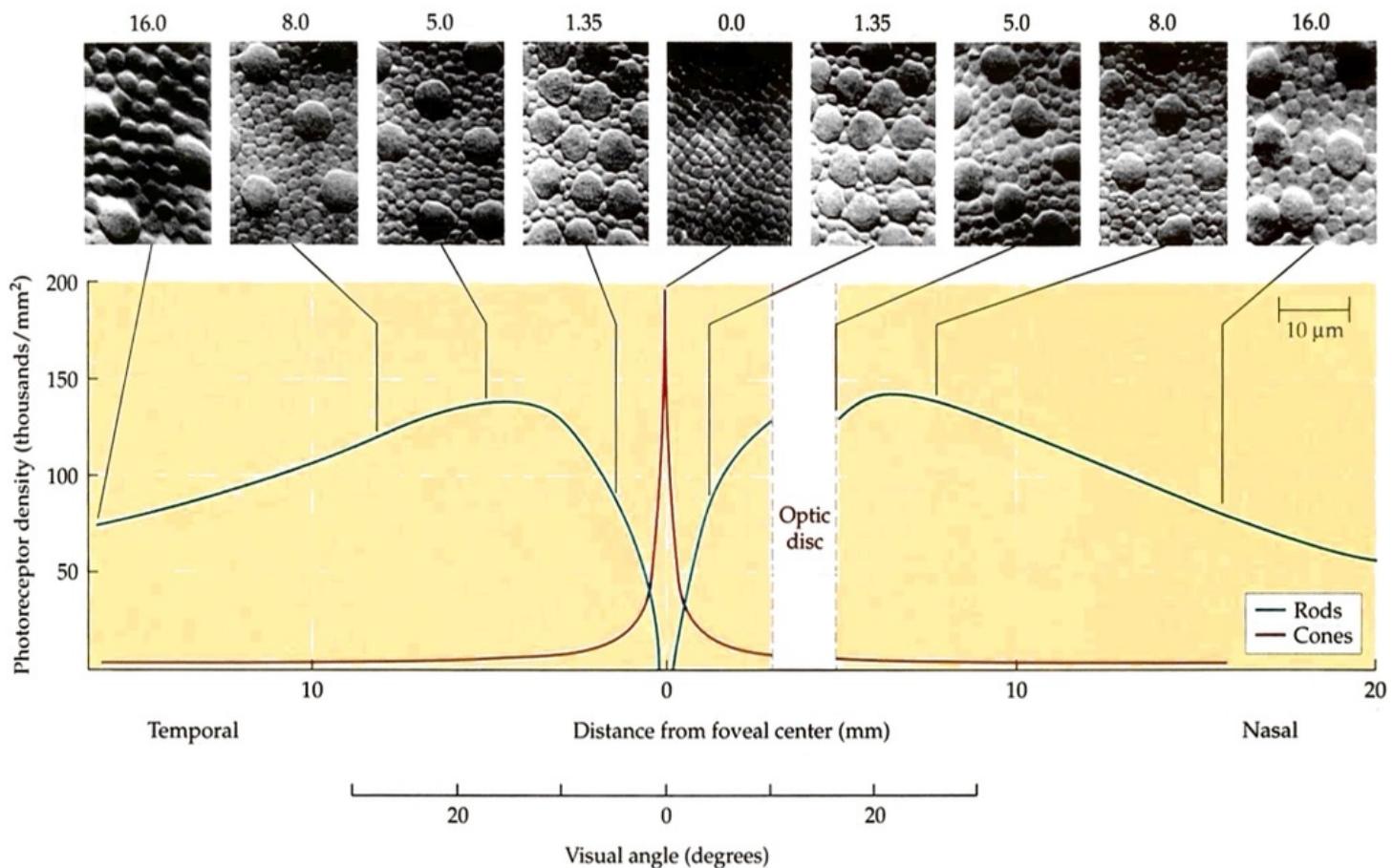


FIGURE 2.10 Photoreceptor density across the retina. The top panels show slices through the photoreceptor inner segments at different eccentricities (distances from the fovea). The graph shows the density of rods and cones plotted as a function of distance from the fovea. Note that in the peripheral slices, the cones are always the larger cells. (After Oyster, 1999; micrographs from Curcio et al., 1990.)

is smaller than 1 degree, the image will land on a region of the retina that has only cones. (How big is 1 degree? Here's a rule of thumb, illustrated in Figure 2.11: your thumb, when viewed at arm's length, subtends an angle of about 2 degrees on the retina, assuming your thumb is about 2 cm across and your outstretched arm extends about 57 cm from your eye). Table 2.1 illustrates some of the fundamental differences in the properties of the fovea compared

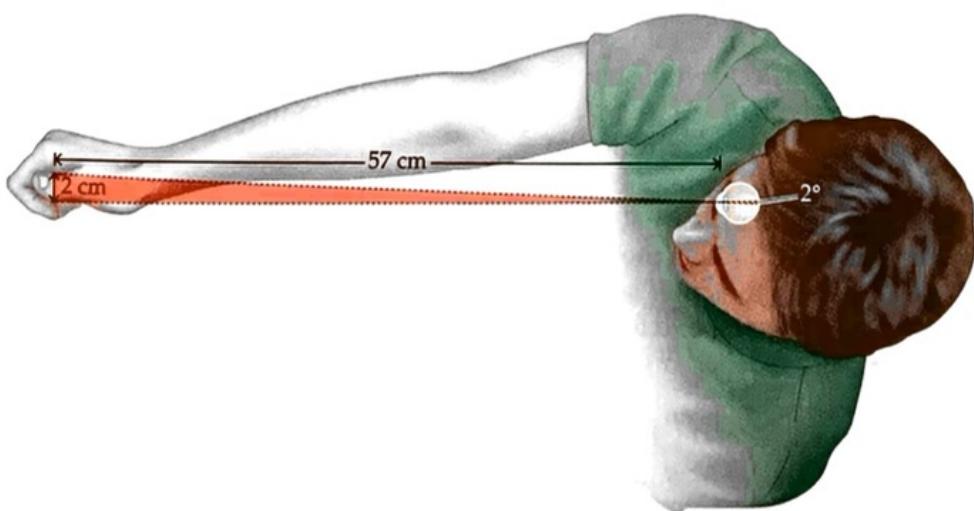


FIGURE 2.11 The "rule of thumb": when viewed at arm's length, your thumb subtends an angle of about 2 degrees on the retina.

with the peripheral retina. Most important for us, the fovea has high acuity and we use it to identify objects, to read, and to inspect fine detail. On the other hand, we use the periphery when detecting and localizing stimuli that we aren't looking at directly (e.g., seeing a moving truck out of the "corner of the eye").

The cones become larger and more sparse away from the foveal center, and the small cells that appear outside the fovea (e.g., 1.35 in Figure 2.10) are rods, which are about the same size as the foveal cones. In all of the micrographs except for 0.0, the large cells are always the cones.

Rods and cones operate best under different lighting conditions: Rods function relatively well under conditions of dim (scotopic) illumination (which is why animals with all-rod retinas are nocturnal), but cones require brighter (photopic) illumination (e.g., sunlight or room lights) to operate efficiently. Having an area at the center of the fovea with no rods means that under dim illumination the central 1 degree or so around the fovea is effectively blind! Indeed, practiced stargazers know that it is often easier to spot a dim star by looking out of the corner of one's eye than by looking directly at it. We will revisit photopic and scotopic vision again in Chapter 5.

Rods and cones differ functionally in another important way. Because all rods have the same type of photopigment, they cannot signal differences in color. Each cone, on the other hand, has one of three different photopigments that differ in the wavelengths at which they absorb light most efficiently. Therefore, cones can signal information about wavelength, and thus they provide the basis for our color vision.

Retinal Information Processing

The retina contains five major classes of neurons: photoreceptors, horizontal cells, bipolar cells, amacrine cells, and ganglion cells mentioned in the previous section (see Figure 2.8). Let's take a closer look at the functions of each of these cell types. (See **Web Activity 2.4: Retinal Structure**.)

Light Transduction by Rod and Cone Photoreceptors

Both types of photoreceptors consist of an **outer segment** (which is adjacent to the pigment epithelium), an **inner segment**, and a **synaptic terminal**. Molecules called visual pigments are made in the inner segment (which is like a little factory, filled with mitochondria) and stored in the outer segment, where they are incorporated into the membrane. Each visual pigment molecule consists of a protein (an opsin), the structure of which determines which wavelengths of light the pigment molecule absorbs, and a **chromophore**, which captures light photons. The chromophore is the part of the molecule responsible for its color, and it selectively absorbs specific wavelengths of light. The chromophore, known as retinal, is derived from vitamin A, which is in turn manufactured from beta-carotene, which is why your mother told you to eat your carrots! The opsin and chromophore are connected. Each photoreceptor has only one of the four types of visual pigments found in the human retina. The pigment **rhodopsin** is found in the rods, concentrated mainly in the stack of membranous discs in the outer segment. Each cone has one of the other three pigments—which respond to long, medium, and short wavelengths, respectively.

Recent evidence suggests that there may be a third type of photoreceptor—one that "lives" among the ganglion cells and that is involved in adjusting our biological rhythms to match the day and night of the external world (Baringa, 2002). These photoreceptors are sensitive to the ambient light level and contain the photopigment **melanopsin**, and they send their signals to the

outer segment The part of a photoreceptor that contains photopigment molecules.

inner segment The part of a photoreceptor that lies between the outer segment and the cell nucleus.

synaptic terminal The location where axons terminate at the synapse for transmission of information by the release of a chemical transmitter.

chromophore The light-catching part of the visual pigments of the retina.

rhodopsin The visual pigment found in rods.

melanopsin A photopigment that is sensitive to ambient light.

suprachiasmatic nucleus (SCN), the home of the brain's circadian clock, which regulates 24-hour patterns of behavior and physiology.

When a photon from our favorite star makes its way into the outer segment of a rod and is absorbed by a molecule of rhodopsin, it transfers its energy to the chromophore portion of the visual pigment molecule. This process, known as **photoactivation**, initiates a biochemical cascade of events eventually resulting in the closing of channels in the cell membrane that normally allow ions to flow into the rod outer segment. Closing these channels alters the balance of electrical current between the inside and outside of the rod outer segment, making the inside of the cell more negatively charged. This process is known as **hyperpolarization**. Hyperpolarization closes calcium channels at the synaptic terminal, thereby reducing the concentration of free calcium inside the cells. The lowering of the calcium concentration, in turn, reduces the concentration of neurotransmitter (glutamate) molecules at the synaptic terminals, and this change signals to the bipolar cell that the rod has captured a photon. The entire sequence of events takes only a matter of milliseconds. Cones act in a qualitatively similar fashion. You can learn more about phototransduction at sites.sinclair.com/neuroscience5e/animations11.02.html.

The amount of glutamate present in the photoreceptor–bipolar cell synapse at any one time is inversely proportional to the number of photons being absorbed by the photoreceptor. Thus, unlike most other types of neurons, photoreceptors do not respond in an all-or-nothing fashion. They pass their information on to bipolar cells via **graded potentials**, which vary in size, instead of all-or-none action potentials or spikes, which are found throughout the nervous system (see Chapter 1).

The three cone photopigments are not distributed equally among the cones (**Figure 2.12**). Short wavelength–sensitive cones (S-cones) constitute only about 5–10% of the total cone population, and they are essentially missing from the center of the fovea. Thus, the foveal center is dichromatic (it has only two color-sensitive cone types). We also know that there are more long wavelength–sensitive cones (L-cones) than medium wavelength–sensi-

photoactivation Activation by light.

hyperpolarization An increase in membrane potential such that the inner membrane surface becomes more negative than the outer membrane surface.

graded potential An electrical potential that can vary continuously in amplitude.

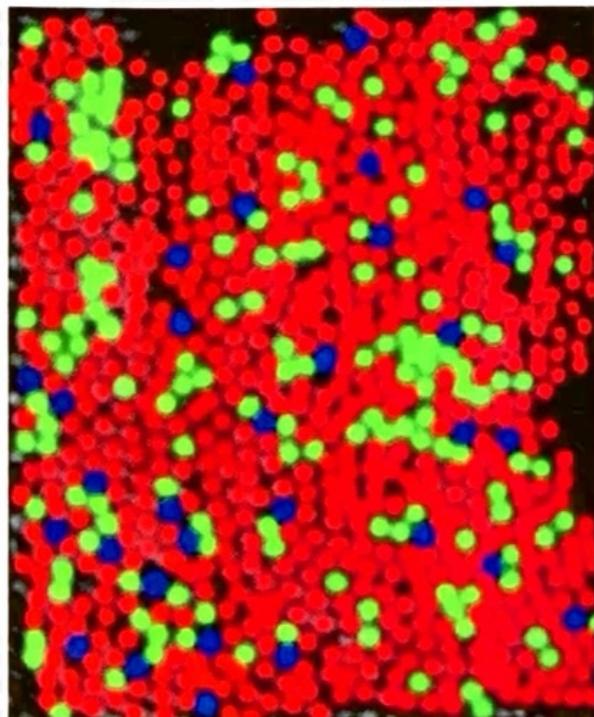


FIGURE 2.12 Blue, green, and red represent the S-, M-, and L-cones, respectively, of a living human being in a patch of retina at 1 degree from the fovea. The pseudocolor image was made by the use of adaptive optics to measure and bypass the aberrations of the eye, and of selective bleaching to isolate the different photopigments. (From Roorda and Williams, 1999; courtesy of Austin Roorda.)

TABLE 2.2
Properties of human photopic and scotopic vision

Property	Photopic system	Scotopic system
Photoreceptors	4–5 million cones	90 million rods
Location in retina	Throughout retina, with highest concentration close to fovea	Outside of fovea
Acuity (detail)	High	Low
Sensitivity	Low	High

tive cones (M-cones); it has been estimated that there are, on average, about twice as many L-cones as M-cones, although the ratio of L- to M-cones varies enormously among individuals. Table 2.2 illustrates some of the fundamental differences in the properties of the photopic (high-illumination) and scotopic (low-illumination) visual systems.

When photoreceptors capture light, they produce chemical changes that start a cascade of neural events ending in a visual sensation. Photoreceptors send their signals by way of the synaptic terminals, specialized structures for contacting other retinal neurons. Figure 2.8 shows examples of rod and cone synaptic terminals. The synaptic terminals contain connections from the neurons that photoreceptors “talk to”: the horizontal and bipolar cells.

FURTHER DISCUSSION of cones and color detection can be found in Chapter 5 on page 124.

Lateral Inhibition through Horizontal and Amacrine Cells

As the name implies, **horizontal cells** run perpendicular to the photoreceptors, making contacts between nearby photoreceptors. These lateral connections play an important functional role in the form of **lateral inhibition**, which enables the signals that reach retinal ganglion cells to be based on differences in activation between nearby photoreceptors. Lateral inhibition plays an important role in visual perception, and in several visual illusions (e.g., Mach bands and the Hermann grid—see **Web Essay 2.1: Seeing Illusory Stripes and Spots**). We will have more to say about lateral inhibition in the “Center-Surround Receptive Fields” section below.

Amacrine cells are also part of the lateral pathway. Like horizontal cells, amacrine cells run perpendicular to the photoreceptors in the inner layers of the retina, where they receive inputs from bipolar cells and other amacrine cells and send signals to bipolar, amacrine, and retinal ganglion cells. Amacrine cells come in many flavors, by some estimates as many as 40 (Rodieck, 1998). Although amacrine cells have been implicated in both contrast enhancement and temporal sensitivity (the detection of changes in light patterns over time), their precise function remains unclear.

Convergence and Divergence of Information via Bipolar Cells

If horizontal and amacrine cells form a lateral pathway in the retina, then photoreceptors, bipolar cells, and ganglion cells can be considered to form a vertical pathway (see Figure 2.8). Bipolar cells are the intermediaries. There are various types of bipolar cells, and their wiring determines the information that is passed from the photoreceptors to the ganglion cells. For example, in

horizontal cell A specialized retinal cell that contacts both photoreceptor and bipolar cells.

lateral inhibition Antagonistic neural interaction between adjacent regions of the retina.

amacrine cell A retinal cell found in the inner synaptic layer that makes synaptic contacts with bipolar cells, ganglion cells, and other amacrine cells.

peripheral vision a **bipolar cell** receives input from as many as 50 photoreceptors, pools this information, and passes it on to a ganglion cell. This convergence of information from many photoreceptors to a single bipolar cell (known as a **diffuse bipolar cell**) is a characteristic of the rod pathway, and the same sort of convergence also occurs in the cone pathway in the peripheral retina.

Pooling of information from many photoreceptors is a very important mechanism for increasing visual **sensitivity**. Indeed, the fact that most rods communicate with ganglion cells through diffuse bipolar cells largely accounts for the ability of the rod system to function well in dim lighting conditions. A diffuse bipolar cell may fire at the same rate in response to a single point of bright light or several spots of dim light, so a ganglion cell listening to the diffuse bipolar cell will be unable to tell which pattern of light is present. The high degree of neural convergence in peripheral vision has important consequences for **visual acuity**, which falls off rapidly with eccentricity (see Table 2.1), which is discussed further in Chapter 3.

In contrast, in the fovea, **midget bipolar cells** receive input from single cones and pass this information on to single ganglion cells. The fact that one-to-one pathways between cones and ganglions exist only in the fovea accounts for why images are seen most clearly when they fall on this part of the retina. The high degree of convergence in the retinal periphery ensures high sensitivity to light but poor acuity. The low degree of convergence in the fovea ensures high acuity but poor sensitivity to light. You can explore this trade-off of sensitivity and acuity in **Web Activity 2.5: Acuity versus Sensitivity**.

Each foveal cone actually contacts two bipolar cells (representing a divergence of information): one responds to an increase in light captured by the cone and is called an **ON bipolar cell**; the other responds to a decrease and is called an **OFF bipolar cell**. The fact that there are both ON and OFF bipolar cells provides information about whether the retinal illumination increased or decreased, and as we will see, the ON/OFF distinction built into the anatomical structure of the retina is present at many levels of the visual pathway.

Communicating to the Brain via Ganglion Cells

By the time signals arrive at the final layer of the retina (the **ganglion cells**), there has already been a lot of information processing. Some information has been pooled through convergence; some has been enhanced by lateral pathways. Ganglion cells receive their input from bipolar and amacrine cells, process this input further, and send messages off to the brain through their axons, which gather in the back of the eyeball and emerge together as the optic nerve (see Figure 2.2).

By now you are probably getting the idea that each cell type comes in many varieties, and ganglion cells are no exception. The human retina contains about 1,250,000 ganglion cells, about one-hundredth the number of photoreceptors. Midget bipolar cells send their signals to small ganglion cells, which are widely referred to as **P ganglion cells** because they feed the parvocellular ("small cell") layer of the lateral geniculate nucleus (LGN) (discussed in Chapter 3). P ganglion cells constitute about 70% of the ganglion cells in the human retina. Diffuse bipolar cells project to ganglion cells that are known as **M ganglion cells** (Figure 2.13) because they feed the magnocellular ("large cell") layer of the LGN. The dendrites of these ganglion cells spread out much more than those of the P ganglion cells, giving them an umbrella-like appearance. About 8–10% of ganglion cells in the human retina are of the M variety. The dendrites of both P and M ganglion cells increase in size with retinal eccentricity, but at all eccentricities the P ganglion cells have much smaller dendritic trees than do the M ganglion cells.

bipolar cell A retinal cell that synapses with either rods or cones (not both) and with horizontal cells, and then passes the signals on to ganglion cells.

diffuse bipolar cell A bipolar retinal cell whose processes are spread out to receive input from multiple cones.

sensitivity 1. The ability to perceive via the sense organs. 2. Extreme responsiveness to radiation, especially to light of a specific wavelength. 3. The ability to respond to transmitted signals.

visual acuity A measure of the finest detail that can be resolved by the eyes.

midget bipolar cell A small bipolar cell in the central retina that receives input from a single cone.

ON bipolar cell A bipolar cell that responds to an increase in light captured by the cones.

OFF bipolar cell A bipolar cell that responds to a decrease in light captured by the cones.

ganglion cell A retinal cell that receives visual information from photoreceptors via two intermediate neuron types (bipolar cells and amacrine cells) and transmits information to the brain and midbrain.

P ganglion cell A small ganglion cell that receives excitatory input from single midget bipolar cells in the central retina and feeds the parvocellular layer of the lateral geniculate nucleus.

M ganglion cell A ganglion cell resembling a little umbrella that receives excitatory input from diffuse bipolar cells and feeds the magnocellular layer of the lateral geniculate nucleus.

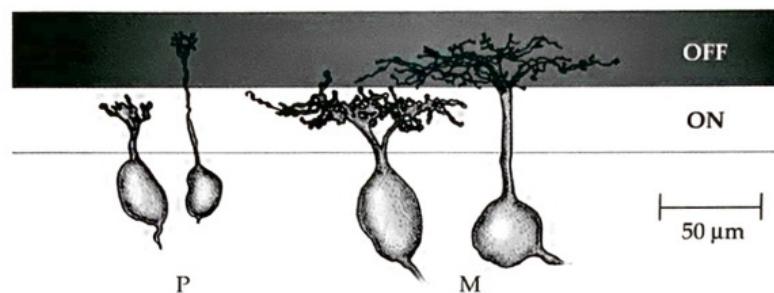


FIGURE 2.13 Different types of retinal P and M ganglion cells. Shown are ganglion cells in section. (After Oyster, 1999.)

The astute reader may have noticed that M and P ganglion cells together constitute about 80% of all ganglion cells. Other ganglion cell types, known as **koniocellular cells**, project to koniocellular layers in the LGN. Some of these, with input from S-cones, may be part of a “primordial” blue-yellow pathway (see Chapter 5), while yet other ganglion cells that project to the koniocellular layers are thought to correspond to “nonblue” koniocellular cells.

CENTER-SURROUND RECEPTIVE FIELDS Much of what we know about how retinal ganglion cells work comes from painstaking physiological studies in which tiny electrodes are used to study the electrical changes in individual ganglion cells. Ganglion cells fire action potentials spontaneously, at about one spike per second, even in the absence of visual stimulation. However, each ganglion cell has a small window on the world known as its **receptive field**. The receptive field is the region on the retina in which visual stimuli influence the neuron’s firing rate. This influence can be either excitatory, increasing the ganglion’s firing rate, or inhibitory, decreasing the ganglion’s firing rate.

FURTHER DISCUSSION of receptive fields can be found in Chapter 3 on pages 70–74.

Work on horseshoe crabs and frogs provided some of our earliest information on the receptive fields of retinal neurons (Hartline, 1940). But it was Stephen Kuffler who first mapped out the receptive fields of individual retinal ganglion cells in the cat, using small spots of light (Kuffler, 1953). **Figure 2.14** illustrates Kuffler’s main findings, which also apply to primate retina, and provides some important insights into how the retina processes visual information. Kuffler’s experiments are simulated in **Web Activity 2.6: Ganglion Receptive Fields**.

Let’s consider Figure 2.14b. Kuffler’s visual stimulus was a small spot of light, which he moved about on the retina, turning it on and off while recording impulses from a single retinal ganglion cell. When the spot was placed on a specific small region of the retina, the ganglion cell *increased* its firing rate when the light was turned on (this response is indicated by a plus sign in the figure). This area of the retina is called the “center” of the ganglion cell’s receptive field. When the spot was moved to an adjacent area of the retina, the ganglion cell *decreased* its firing rate when the light was turned on (indicated by a minus sign). It is interesting that turning the light *off* in this area surrounding the receptive-field center led to a brief surge in the cell’s firing rate, after which the cell settled down to its spontaneous rate.

koniocellular cell A neuron located between the magnocellular and parvocellular layers of the lateral geniculate nucleus. This layer is known as the koniocellular layer.

receptive field The region on the retina in which visual stimuli influence a neuron’s firing rate.

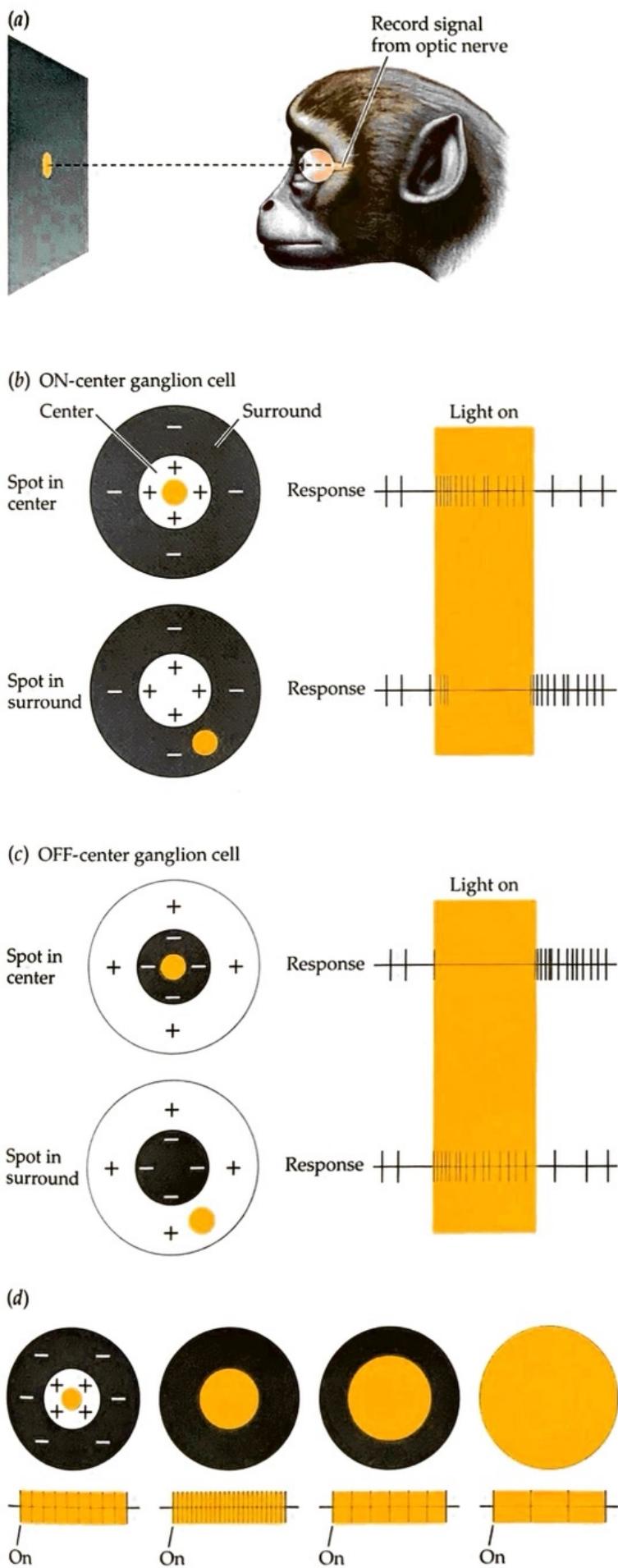


FIGURE 2.14 Retinal ganglion cell receptive fields. (a) Mapping retinal receptive fields. (b) ON-center field. In each image on the left, the small white circle illustrates the region on the retina where the retinal ganglion cell increased its firing rate when the spot (small yellow circle) was turned on. The large gray circle illustrates the region on the retina where the retinal ganglion cell decreased its firing rate when the spot was turned on and increased its rate when the spot was turned off. The plots on the right illustrate the spikes fired by the associated retinal ganglion cell. (c) OFF-center field. In each image on the left, the large white circle illustrates the region on the retina where the retinal ganglion cell increased its firing rate when the spot was turned on. The small gray circle illustrates the region on the retina where the retinal ganglion cell decreased its firing rate when the spot was turned on and increased its rate when the spot was turned off. The plots on the right illustrate the spikes fired by the associated retinal ganglion cell. (d) The effect of varying the spot size. Note that firing is fastest when the spot just fills the RF center. Increasing the spot size further results in reduced firing due to lateral inhibition from the surround (Part c, d after Kuffler, 1953.)

ON-center cell A cell that depolarizes in response to an increase in light intensity in its receptive-field center.

OFF-center cell A cell that depolarizes in response to a decrease in light intensity in its receptive-field center.

filter An acoustic, electrical, electronic, or optic device, instrument, computer program, or neuron that allows the passage of some frequencies or digital elements and blocks the passage of others.

contrast The difference in luminance between an object and the background, or between lighter and darker parts of the same object.

The cell just described is known as an **ON-center cell**. It increases its firing rate when a light is turned on in the center of its receptive field, and it decreases its firing rate when the light is turned on in the surround. However, nearly as many ganglion cells do exactly the opposite: their firing rates decrease when a light is turned on in a spot in the center of the receptive field, and increase when a light is turned on in a spot in the surround. These are known as **OFF-center cells** (Figure 2.14c). Most retinal ganglion cells have one of these two types of concentric center-surround organization.

An important finding of Kuffler's was that the spatial layout of the ganglion cell's receptive field is essentially concentric; that is, a small circular area in the center responds to an increase in illumination, and a surrounding ring responds to a decrease in illumination, and size matters! The ganglion cell fires fastest when the size of the spot of light matches the size of the excitatory center, and it reduces its firing rate when the spot of light begins to encroach on its inhibitory surround (Figure 2.14d). This interaction between the antagonistic center and surround is known as lateral inhibition.

The center-surround organization has two important functional consequences. First, as noted above, each ganglion cell will respond best to spots of a particular size (and will respond less to spots that are either bigger or smaller). In this way, retinal ganglion cells act as a **filter** by responding best to stimuli that are just the right size, and less to stimuli that are larger or smaller. Second, ganglion cells are most sensitive to *differences* in the intensity of the light in the center and in the surround, and they are less affected by the average intensity of the light. This is a useful quality because the average intensity of the light falling on the retina varies a lot, depending on whether you are indoors or outdoors, whether it is daytime or nighttime, how far away you and the objects you're looking at are from the source of illumination, and so on. But the **contrast**—the difference in luminance or brightness between adjacent bits of the scene—will be roughly the same regardless of lighting conditions.

The center-surround antagonism, or lateral inhibition, also has other important perceptual consequences—resulting in the illusions of stripes and spots (Figure 2.15). (See **Web Essay 2.1: Seeing Illusory Stripes and Spots**.)

Phenomenologically, we have the impression that our eyes work like video cameras, capturing faithful snapshots of the world around us. Note, though, that the rest of the visual system sees only what the retinal ganglion cells show

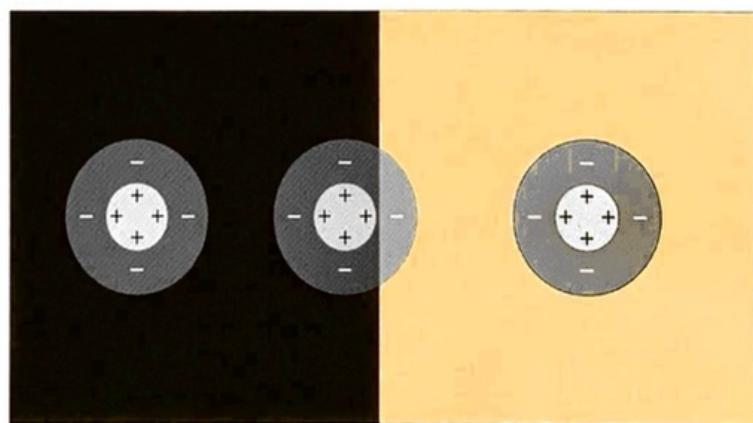


FIGURE 2.15 “Cartoon” showing a classic neuronal explanation for Mach bands. See the text for details.

it, and the ganglion cells are not content simply to pass along the raw images encoded by the photoreceptors. Instead, the ganglion cells, together with the bipolar, amacrine, and horizontal cells, act as an image filter, transforming the raw image into a new representation. This new representation highlights certain important information, such as contrast, and largely discounts other types of less useful information, such as ambient light intensity. In fact, the whole visual system can be considered a long series of filters, with each stage in the system responsible for extracting a particular aspect of the visual world and passing this aspect on to the next stage.

P AND M GANGLION CELLS REVISITED As already mentioned, retinal ganglion cells come in several types; for humans, the most important of these are the P and M cells. The receptive fields of these two types of ganglion cells differ in some important ways. First, at all eccentricities P cells have smaller receptive fields than M cells have. This isn't too surprising, because the size of the receptive field is probably determined by the size of its dendritic field (see Figure 2.13): since M cells listen to more photoreceptors (via bipolar, horizontal, and amacrine cells) than P cells do, M cells respond to a larger portion of the visual field.

An additional consequence of the differing sizes of M and P receptive fields is that M cells are much more sensitive—better able to detect visual stimuli—than are P cells under low-light conditions (e.g., at night). However, the smaller receptive fields of P cells enable them to provide finer resolution (greater acuity) than M cells can, if there is enough light for the P cells to operate. See **Web Activity 2.5: Acuity versus Sensitivity** for more on this trade-off.

P and M ganglion cells also differ in their temporal responses. P cells tend to respond with sustained firing while light shines on their excitatory regions. M cells tend to respond more transiently: an M cell will respond with a brief burst of impulses when the spot is turned on, and then it will quickly return to its spontaneous rate, even if the spot remains lit. Thus, M and P ganglion cells signal different information to the brain. P cells provide information mainly about the contrast in the retinal image, and M cells signal information about how the image changes over time.

Finally, P and M cells differ in what they say to the brain about the color of the light they detect (see Chapter 5).

Dark and Light Adaptation

When you emerge from a dark room into bright light (e.g., coming out of a movie theater), your pupil constricts to reduce the amount of light arriving at your retina. This automatic reflex is nothing to sneeze at, but there's a good chance that you will! Sneezing in response to being exposed to a bright light—the “photic sneeze reflex”—is not yet understood, even though it has intrigued some of history’s greatest minds. Aristotle (384–322 BCE) thought the heat of the sun on the nose might be responsible. However, Francis Bacon (1561–1626) showed that Aristotle was wrong. Bacon stepped into the sun with his eyes closed and did not sneeze; the heat was still there, but the sneeze was not. Bacon guessed that the sun’s light makes the eyes water, and that moisture (“braine humour,” in his words) then seeps into and irritates the nose. We now know that the sneeze takes place too soon after light exposure to be the result of the comparatively slow formation of tears. Current thinking suggests that the photic sneeze reflex is a result of crossed wires in the brain.

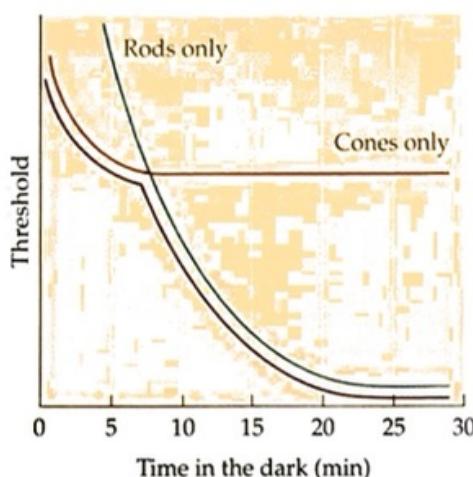


FIGURE 2.16 Dark adaptation curve. The purple curve shows the threshold light intensity required to detect a peripheral spot following several minutes of adaptation to a bright light. The red curve illustrates the rapid adaptation of the cones. The blue curve shows the slower recovery of the rods to much lower threshold intensities (that is, greater sensitivity). The purple curve represents the most sensitive of the two at any given time.

When you enter a dark room from bright sunlight, the number of photons of light entering your eye might be reduced by a factor of several billion. Initially you will have trouble seeing anything, but after about 30 minutes in the dark you will be able to detect even just a few photons. The purple curve in Figure 2.16 illustrates the change in the threshold light intensity needed to detect a peripheral spot. Initially the threshold is very high, indicating low sensitivity. But over 20 minutes or so the threshold is greatly reduced (meaning sensitivity is increased). And when you emerge from the dark and return to the sunlight, you will be able to see almost instantly. How does the visual system alter its sensitivity over such a large operating range?

There are four main ways in which the visual system adjusts to changes in illumination: pupil size, photopigment regeneration, the duplex retina, and neural circuitry.

Pupil Size

When a flashlight is shone in someone's eye in a dimly lit room, the pupil quickly constricts. The diameter of the pupil can vary by about a factor of 4, from about 2 mm in bright illumination to about 8 mm in the dark (Figure 2.17). Because the amount of light entering the eye is proportional to the area of the pupil, the 4-fold increase in diameter accounts for a 16-fold improvement in sensitivity. In other words, 16 times as many quanta can enter the eye when the pupil is completely dilated, compared with when it is constricted. Although this adaptive ability certainly helps, pupil dilation accounts for only a small part of the visual system's overall ability to adapt to light and dark conditions.

Photopigment Regeneration

A second mechanism for achieving a large sensitivity range is provided by the way photopigments are used up and replaced in receptor cells. In dim

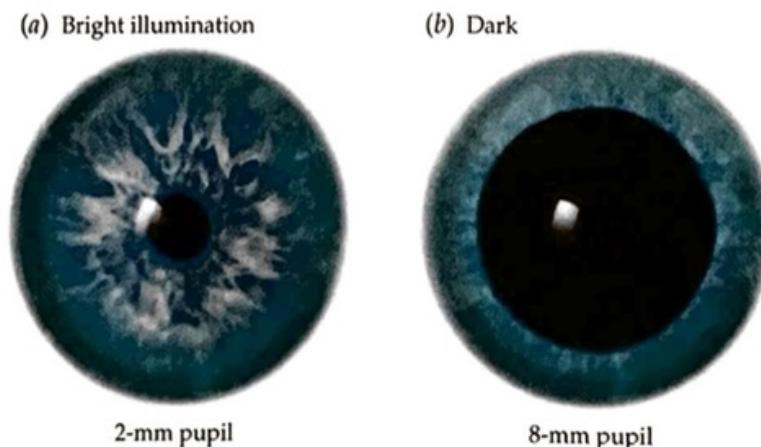


FIGURE 2.17 The black spots in the middle of these two irises show the possible range of pupil sizes as we go from bright illumination (a) into the dark (b).

lighting conditions, plenty of photopigment is available, and rods and cones absorb and respond to as many photons as they can. As already noted, rods provide better sensitivity in such situations than do cones. Indeed, the rod system is capable of detecting a single quantum of light! (See **Web Essay 2.2: How Many Quanta Does It Take?**) After a photopigment molecule is bleached (used to detect a photon), the molecule must be regenerated before it can be used again to absorb another photon.

As the overall light level increases, the number of photons starts to overwhelm the system: photopigment molecules cannot be regenerated fast enough to detect all the photons hitting the photoreceptors. This slow regeneration is a good thing for increasing our sensitivity range. If photons are scarce, we use them all to see; if we have an overabundance, we simply throw some of them away and use the leftovers.

The Duplex Retina

This light compensation mechanism is enhanced by humans' duplex retinas. Rods provide exquisite sensitivity at low light levels, but they become overwhelmed when the background light becomes moderately bright, leading to a loss in information quality. Cones are much less sensitive than rods (they function poorly under very dim light), but their operating range is much larger, stretching from about ten photons per second (just enough light to see color) to hundreds of thousands of photons per second (e.g., a snowcapped mountain in bright sunlight). So we use rods to see when the light is low, and the cones take over when there is too much light for the rods to function well. After adapting to a bright light, cones recover sensitivity quickly (red curve in Figure 2.16) and then saturate. They are not very sensitive to very dim light. Rods recover more slowly (blue curve in Figure 2.16), but after 20 minutes or so they are very sensitive to dim light.

Neural Circuitry

Although pupil size, photopigment regeneration rates, and the rod/cone dichotomy all play a role in dark and light adaptation, the most important reason we are not bothered by variations in overall light levels has to do with the neural circuitry of the retina. As we learned earlier, ganglion cells fire at their maximum rate when the centers of their receptive fields are brightly lit while the surrounds are completely dark (or vice versa) (see Figure 2.14). But the cells will still fire at an above-spontaneous rate when light falls on the entire receptive field, as long as the light is *brighter* in the ON portion than in the OFF portion of the receptive field. Therefore, as long as the photoreceptors feeding the ganglion cells are not completely saturated, the ganglion cells will encode the pattern of relatively light and relatively dark areas in the retinal image. And the pattern of illumination, not the overall light level, is the primary concern of the rest of the visual system.

To sum up, the answer to the question of how the visual system deals with such large variations in overall light levels has two parts. First, we reduce the scale of the problem by regulating the amount of light entering the eyeball, by using different types of photoreceptors in different situations, and by effectively throwing away photons we don't need. Second, by responding to the contrast between adjacent retinal regions, the ganglion cells do their best to ignore whatever variation in overall light level is left over.

Sensation & Perception in Everyday Life

When Good Retina Goes Bad

Millions of people around the world suffer from blinding diseases in which the rods and/or cones degenerate. These include **age-related macular degeneration (AMD)** and **retinitis pigmentosa (RP)**. (See **Web Essay 2.3: Clinical Case: The Man Who Couldn't Read** and **Web Activity 2.7: Simulated Scotoma**.) At present, there are no effective cures to prevent the progressive degeneration of the photoreceptors that occur in these diseases. For patients with AMD this may lead to an inability to read or recognize faces. For patients with long-standing RP, this leads inevitably to irreversible blindness.

Fortunately, there are several exciting new technological developments that provide hope for these patients. These are all based on the notion that while the photoreceptors are dead or dying, post-receptor neurons and their connections are largely intact. One such approach is to substitute an electronic prosthesis (an artificial device to replace or augment a missing or impaired part of the body) into the retina. Typically the prosthesis uses a camera to convert light into energy; an array of electrodes implanted in the retina generates an electrical stimulation pattern based on the light pattern on the camera and delivers this stimulation pattern to the intact post-receptor neurons (**Figure 2.18**). Unfortunately, while these retinal prostheses can restore some sight, there are technical challenges to implanting them, and they suffer low spatial resolution (Weiland, Cho, and Humayun, 2011), allowing only perception of spots of light and very high-contrast edges.

Another approach that has had some early success in animal models is to use gene therapy to express light-activated channels in surviving photoreceptors using adeno-associated viral (AAV) vectors. This approach has been successfully used in several clinical trials in patients.

A third strategy is to chemically modify endogenous channels in retinal ganglion cells to make them light-sensitive. This approach essentially adds a synthetic small molecule "photoswitch" to confer light sensitivity onto retinal ganglion cells, and it has been shown to reinstate light sensitivity in blind mice (Tochitsky et al., 2014).

Each of these approaches provides promise for new treatments for patients with blinding retinal disorders.

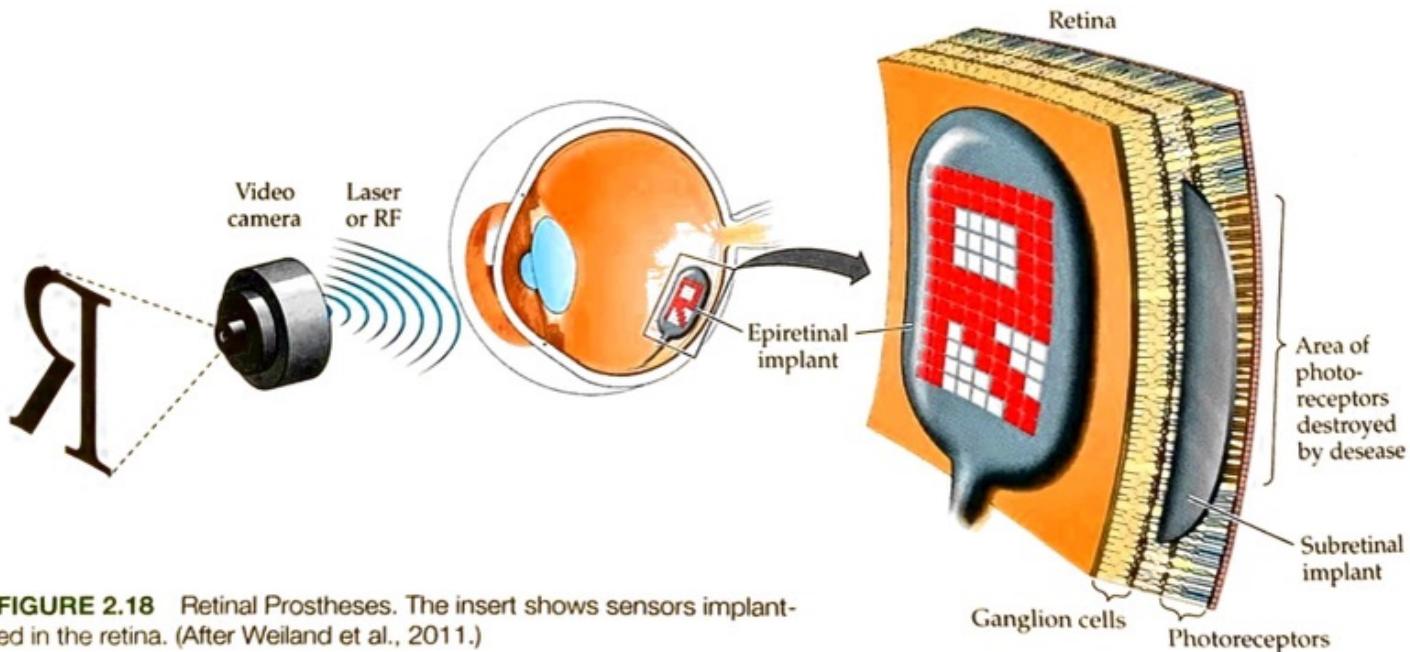


FIGURE 2.18 Retinal Prostheses. The insert shows sensors implanted in the retina. (After Weiland et al., 2011.)

Summary

1. This chapter provided some insight into the complex journey that is required for us to see stars and other spots of light. The path of the light was traced from a distant star through the eyeball and to its absorption by photoreceptors and its transduction into neural signals. In subsequent chapters we'll learn how those signals are transmitted to the brain and translated into the experience of perception.
2. Light, on its way to becoming a sensation (a visual sensation, that is), can be absorbed, scattered, reflected, transmitted, or refracted. It can become a sensation only when it's absorbed by a photoreceptor in the retina.
3. Vision begins in the retina, when light is absorbed by rods or cones. The retina is like a minicomputer that transduces light energy into neural energy.
4. The retina sends information to the brain via ganglion cells, neurons whose axons make up the optic nerves. Retinal ganglion cells have center-surround receptive fields and are concerned with changes in contrast (the difference in intensity between adjacent bits of the scene).
5. The visual system deals with large variations in overall light intensity by (a) regulating the amount of light entering the eyeball, (b) using different types of photoreceptors in different situations, and (c) effectively throwing away photons we don't need.
6. Age-related macular degeneration (AMD) is a disease associated with aging that affects the macula. The leading cause of visual loss among the elderly in the United States, AMD gradually destroys sharp central vision, making it difficult to read, drive, and recognize faces.
7. Retinitis pigmentosa (RP) is a family of hereditary diseases characterized by the progressive death of photoreceptors and degeneration of the pigment epithelium. In the most common form of the disease, patients first notice vision problems in their peripheral vision and under low-light conditions—situations in which rods play the dominant role in collecting light.
8. A number of exciting new developments are aimed at restoring sight in individuals with blinding retinal disease.

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