4.8 Vision

Learning Objectives

- List the three fundamental steps in vision
- List the three layers of the outer structure of the eyeball
- Describe the function of the iris and ciliary body
- Identify the fluid chambers of the eyeball and describe control of intraocular pressure
- Identify parts of the retina including optic disk, macula lutea, fovea
- Define "near point," "accommodation," "convergence," "refractive index"
- Write Snell's law of refraction and the thin lens formula
- Be able to calculate the refractive power of the lens
- Compare the major features of rods and cones
- List the events in phototransduction
- List the functions of the retinal pigmented epithelium
- Describe the response of bipolar cells to light in their receptive field
- Identify the cells in the retina that produce action potentials
- Distinguish between the magnocellular and parvocellular pathway
- Trace the optic nerve input from retina to cortex and identify the visual cortex

OVERVIEW OF THE VISUAL SYSTEM

Vision can be broken down into three sequential processes. These are:

A. Focus light on the retina
B. Transduce light to a nervous signal
C. Process the nervous signals to form conscious perception of objects.

The structure of the eye provides the means for focusing light onto the retina. The retina contains specialized photoreceptors that absorb light and transduce the light energy into neural signals. The processing of these neural signals begins at the retina and continues along pathways to the occipital lobe of the brain where the visual cortex processes the signals. Since we do not yet understand consciousness, we do not understand how conscious perception occurs. However, we understand something of how the signals are processed to form bits and pieces of the final **percept**.

THE STRUCTURE OF THE EYE ENABLES FOCUSING OF LIGHT ON THE RETINA

The outer structure of the eyeball contains three layers: the **sclera**, the **uvea**, and the **retina** (Figure 4.8.1). The sclera is a tough layer of connective tissue that forms the outermost layer of the eye bulb. Its anterior parts are visible as the "whites" of the eye. It is continuous with the **cornea**, which is a specialized transparent covering of the anterior one-sixth of the eye. The junction of the sclera and the cornea is called the **limbus**. At this transition, the cornea gives rise to the sclera and a delicate membranous tissue, the **conjunctiva**. This structure is reflected back to form the layer of the eyelid that rides over the eyeball.

The **uvea** is the middle layer of the eye that contains the **choroid**, the **ciliary body**, and the **iris**. The iris is shaped like a squashed doughnut. It has a central aperture, the **pupil**, whose size varies from 1 to 8 mm depending on the light conditions. The iris contains a **dilator** and a **sphincter muscle**. Sympathetic nerves control the dilator muscle and parasympathetic nerves activate the sphincter (see Chapter 4.9). Bright illumination of one eye causes constriction of the pupil in a reflex called the **direct pupillary light reflex**. The contralateral pupil also constricts, even if that eye is not illuminated, in the **consensual pupillary light reflex**.

The ciliary body contains the **ciliary muscle**, which controls the shape of the **lens**. The ciliary muscle is a smooth muscle with fibers oriented in various directions. This muscle connects to the lens through **suspensory ligaments**. The ciliary muscle forms folds arranged radially around the lens, and the suspensory ligaments arise from the valleys of these folds in zones (hence the term "**zonules of Zinn**"). Contraction of the ciliary muscle makes its diameter smaller, which loosens the tension on the suspensory ligaments, and the lens becomes thicker.

The ciliary body produces a fluid called the **aqueous humor** with a composition similar to plasma except that its protein content is much lower, $5-15\ \mathrm{mg\ dL^{-1}}$ compared to about $7\ \mathrm{g\ dL^{-1}}$ in plasma. This fluid provides nutrients to the cornea and lens which do not have a blood supply. It flows from the **posterior chamber** between the zonules and the iris, out of the pupil and into the **anterior chamber** between the iris and the cornea (see Figure 4.8.2).

DOI: http://dx.doi.org/10.1016/B978-0-12-800883-6.00041-0

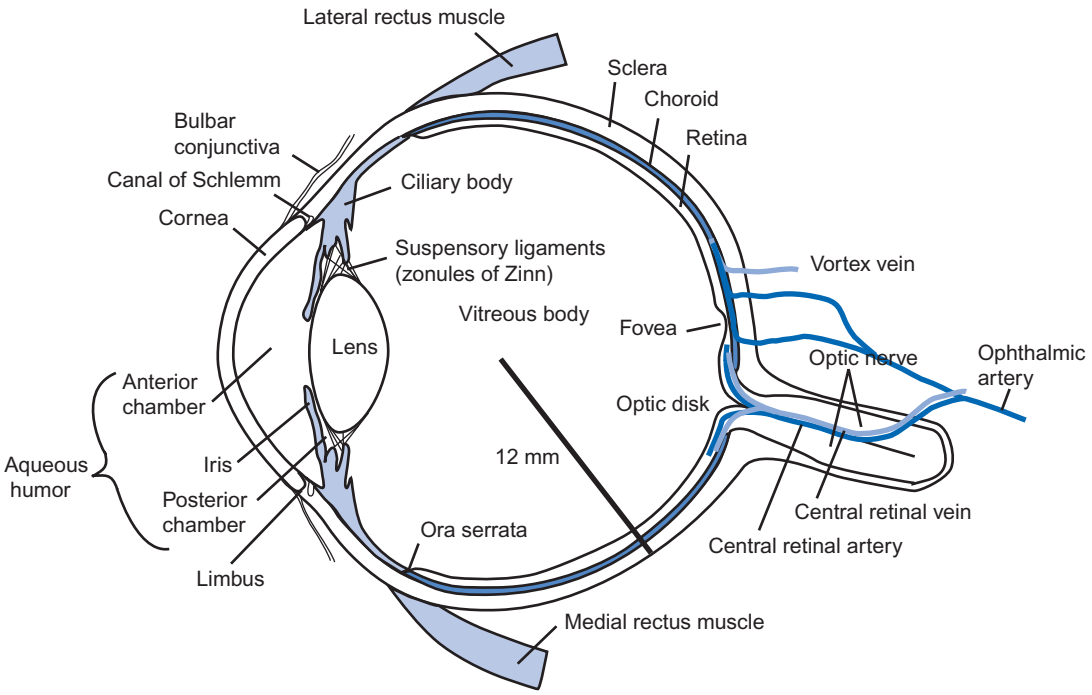


FIGURE 4.8.1 Structure of the eye. The diagram represents a horizontal section taken through the right eye and viewed from above. Individual parts of the eye are described in the text.

Clinical Applications: Glaucoma

Continual production of the aqueous humor ensures adequate intraocular pressure for maintaining the structural integrity of the eyeball. Healthy values of the intraocular pressure range from 13 to 18 mmHg. Because the ciliary body produces a constant flow of aqueous humor of about 2.5 μL min^{-1}, the intraocular pressure is determined largely by the outflow through Schlemm's canal. Large increases in the intraocular pressure damage the eye and cause blindness. This condition is called **glaucoma**, but there is overlap of the intraocular pressures among normal persons and persons with glaucoma. Glaucoma is a heterogeneous class of conditions for which increase in intraocular pressure is a major risk factor.

Intraocular pressures are measured with **tonometers**. Indentation tonometers measure the amount of indentation of the cornea by a known force. Applanation tonometers estimate the intraocular pressure by measuring the force required to flatten a known area of the eyeball. The intraocular pressure is approximately equal to the force required divided by the area that is flattened (the Imbert–Fick law). Most applanation tonometers make contact with the cornea with a plunger, and this contact creates the danger of the transmission of pathogens including hepatitis B and human immunodeficiency virus (HIV). The air-puff tonometer deforms the cornea by a jet of air whose force increases linearly with time. Flattening of the cornea is detected by light reflected off the cornea. The air-puff tonometer avoids corneal abrasion, reaction to the local anesthetics used in the plunger-type applanation tonometers, and reduces the transmission of infectious diseases.

The choroid extends from the **ora serrata** (the junction of the choroid and ciliary body, see Figure 4.8.1) and forms a middle layer over the rest of the eyeball. It consists primarily of blood vessels and connective tissue. It provides nutrients to the outer retina and removes metabolites from the retina.

The retina is the innermost layer of the eye ball, lying between the choroid layer and the **vitreous body**. It extends from the circular edge of the **optic disk**, where the optic nerve leaves the eye, to the ora serrata. The retina transduces incident light into nervous signals. The retina consists of several distinct layers. The outermost layer (the layer closest to the choroid) is the **retinal pigmented epithelium** consisting of pigmented columnar cells arranged in hexagonal close packing. Photoreceptor cells lie adjacent to the retinal pigmented epithelium. Two kinds of photoreceptor cells make up the photoreceptor layer. These are the **rods** and the **cones**. Rods are extremely sensitive and form the basis for **scotopic vision**, vision in dim light. Cones are less sensitive but allow for color vision in **photopic vision**, or vision in bright light. Cones vary in their spectral sensitivity. Red, green, or blue cones respond best to those colors of light. The other layers of the retina consist of nerve cells that process visual information even before it leaves the eye.

The retina possesses two general regions: a peripheral region in which rods predominate and a central region

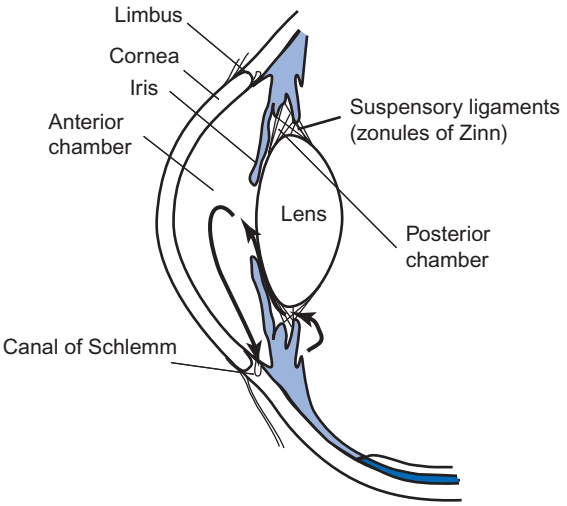


FIGURE 4.8.2 Flow of the aqueous humor. The ciliary body produces about 2.5 μL of aqueous humor each minute. This flows through the zonule fibers, into the posterior chamber between the lens and the iris. From there the fluid flows out of the pupil and into the anterior chamber. There it drains through a trabecular meshwork that feeds into the canal of Schlemm. From there the fluid drains into the veins.

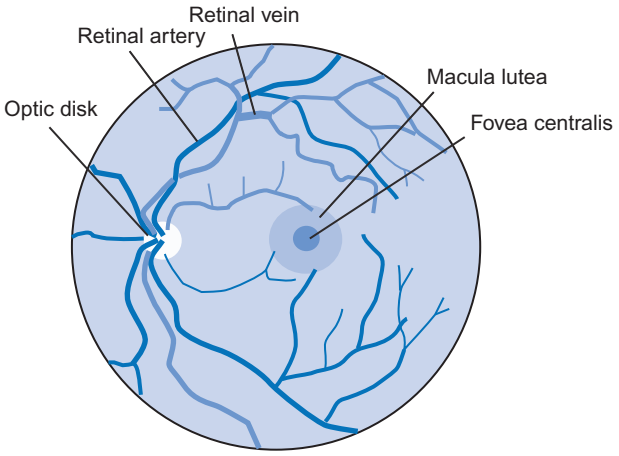


FIGURE 4.8.3 Schematic drawing of a fundus, the inner lining of the eye visible through the pupil. The optic disk, 1.5 mm in diameter, is where nerve and blood vessels enter or exit the eyeball. Branches of the ophthalmic artery form the posterior ciliary artieries that supply the choroid. Anterior ciliary arteries originate from blood vessels supplying the rectus muscles. The inner retina (toward the center of the eye) is supplied by the central retinal artery that branches off the ophthalmic artery and enters the eye along with the optic nerve. Four or five vortex veins drain the choroid, about one vortex vein in each quadrant of the eye. The retinal arteries and veins supply the inner retina with oxygen and nutrients. The macula lutea is a darker area of pigmentation about 5–6 mm in diameter with a central fovea, a depression in the retina in which cones predominate.

dominated by the cones. The peripheral region detects gross form and motion, whereas the central region possesses high resolution. Dim light involving the peripheral retina allows for the identification of objects, but the level of fine detail is poor and color vision is absent. In bright light, the eyes focus light onto the central region of the retina called the **macula lutea**. The macula lutea is about 5–6 mm in diameter. Its most central region is called the **fovea**. This is a central depression in the retina caused by a displacement of the neural cells in the retinal layers, so that the fovea contains only the photoreceptors. There are no photoreceptors in the optic disk, where the nerve fibers from the retina come together and exit the eyeball. Thus light that falls on the optic disk remains undetected. The visual field, the points in visual space that can can be seen, includes a **blind spot** that corresponds to the projection of visual space onto the optic disk. The retina can be viewed through an **ophthalmoscope**, which simultaneously illuminates the retina and projects the reflected light back to the observer. This fundus view provides the only easy observation of the circulatory system. The pattern of blood vessels in the fundus is one possible basis for physical identification of persons. "Red eye" in photographs results from light reflected back to the camera from the fundus (Figure 4.8.3).

THE VITREOUS BODY MAINTAINS EYE SHAPE

The vitreous chamber is filled with a gel-like vitreous body that consists of a solution of salts, soluble proteins, and a mucopolysaccharide called **hyaluronic acid**. A fine meshwork of collagen fibers courses through the vitreous body and ties it to various eye structures. The strongest attachments tie the vitreous body to the ora serrata. Other attachments occur at the posterior lens, optic disk, macula, and retinal blood vessels. Because of its viscoelastic properties, the vitreous body protects the eye by absorbing shocks to it.

THE EYE FOCUSES LIGHT ON THE RETINA BY REFRACTION

Light is focused when all rays emanating from each point of an object are bent to reach a point on the image. The path of light through transparent surfaces such as the cornea and lens depends on the curvature of the surfaces and the relative refractive indices for each material. The equation governing the light path is **Snell's law of refraction** (see Appendix 4.8.A1). This law states that

$$[4.8.1] \qquad n_1 \sin \theta_1 = n_2 \sin \theta_2$$

where n_1 is the refractive index of medium 1, n_2 is the refractive index of medium 2, θ_1 is the angle formed by the incident light ray and a line perpendicular to the surface of medium 1 (its **normal**) and θ_2 is the angle formed by the ray in medium 2 and its normal. When light travels from a region of low n to a region of high n, the light is bent toward a line normal to the surface. Thus the ability to bend light depends on the difference in refractive index at the interface and the curvature of the surface. Light entering the eye sequentially encounters a series of transparent surfaces with different refractive indices:

- Air, $n = 1.00029$
- Thin film of tears, $n = 1.33$
- Cornea, $n = 1.376$
- Aqueous humor, $n = 1.336$
- Lens, $n = 1.386$
- Vitreous body, $n = 1.336$.

EXAMPLE 4.8.1 Calculate the Refractive Power of the Eye at Rest

At rest, the eyes are focused on objects far away, and we can approximate $1/O = 0$. The image forms on the retina about 1.6 cm = 0.016 m away from the nodal plane. Then we can calculate

$$\frac{1}{f} = 0 + \frac{1}{0.016\ \text{m}} = \mathbf{63\ D}$$

This is the refractive power when the lens is most relaxed.

EXAMPLE 4.8.2 Refractive Power at the Near Point

The near point of a young person is about 10 cm. Calculate the refractive power.

According to the thin lens formula, the refractive power is given as

$$\frac{1}{f} = \frac{1}{O} + \frac{1}{I}$$

So calculating the refractive power means identifying O and I. The distance to the object is just the near point: 10 cm = 0.1 m. The distance to the image depends on the size of the eye. We will use 1.6 cm = 0.016 m. Then we have

$$\frac{1}{f} = \frac{1}{0.1\ \text{m}} + \frac{1}{0.016\ \text{m}} = \mathbf{73.5\ D}$$

Bending light more means a reduction in the focal length. The focal length of the eye is the distance from the eye's nodal point to the focused image. In a spherical lens, the nodal point lies in the center of the lens along an equatorial plane. Because there are multiple refractive surfaces in the eye, its nodal point can be considered to lie 1.5–1.7 cm in front of the retina. The **refractive power** in **diopters** is the inverse of the focal length:

$$[4.8.2] \qquad \text{Diopters (D)} = \frac{1}{f}$$

where f is the focal length in m. For any optical system, there is a point beyond which all objects can be considered to be at infinity. For the eye, this distance is about 6 m or 20 ft from the eye. Light rays emanating from such an object can be considered to be parallel to each other and to the optical axis of the eye. The distances from the object and the image obey the thin lens formula (see Appendix 4.8.A1):

$$[4.8.3] \qquad \frac{1}{O} + \frac{1}{I} = \frac{1}{f}$$

where O is the distance from the eye's nodal point to the object, I is the distance from the nodal point to the image, and f is the focal length.

When the lens is relaxed, about two-thirds of the refractive power, 43 D, is contributed by the cornea and about 20 D is contributed by the lens. The reason the cornea provides so much refraction is that its interface with air entails a large difference in refractive index (air has $n = 1.000$, whereas for the cornea $n = 1.376$). Thus, the refractive power results from the interface of a surface with its neighboring structures as well as from its own structure.

THE LENS CHANGES SHAPE TO FOCUS NEAR OBJECTS

Light rays from objects that are close to the eye are not parallel. Therefore, the eye must bend them more in order to focus the rays on the retina. To accomplish this, the lens change shape. Constriction of the ciliary muscle causes its diameter to decrease, which releases tension on the suspensory ligaments or zonule fibers connecting the ciliary muscle to the lens. Because the lens is inherently elastic, the release of tension causes the lens to become rounder, which increases its refractive power (see Figure 4.8.4). The increased curvature of the lens increases its refractive power. This process is called **accommodation**. Accommodation usually also involves **convergence**, when both eyes turn toward the midline to focus on close objects. The increased refractive power allows us to see near objects. The nearest an object can be and still be clearly seen is called the **near point**.

The difference in the refractive power of the relaxed eye and the maximally accommodated eye is the **power of accommodation**. In young persons, this is 10–12 D. The power of accommodation decreases with age as the elasticity of the lens decreases. This causes a recession of the near point with age, or **presbyopia**, as shown in Table 4.8.1. Close inspection of the thin lens formula shows that the power of accommodation is equal to the inverse of the near point when the near point is expressed in m.

NEAR-SIGHTEDNESS AND FAR-SIGHTEDNESS ARE PROBLEMS IN FOCUSING THE IMAGE ON THE RETINA

In the normal resting eye, parallel light rays are focused on the retina. This condition is called **emmetropia**, from the Greek *en* meaning *in* and *metron* meaning *measure*. In some cases, the resting eye does not focus light on the retina. In **myopia**, or nearsightedness, the image is formed in front of the retina. Usually an abnormally long eyeball causes nearsightedness but sometimes abnormally powerful refractive power causes myopia. Lenses that diverge light (indicated by a − sign) correct myopia. In **hypermetropia**, or farsightedness, the image forms behind the retina because the eyeball is

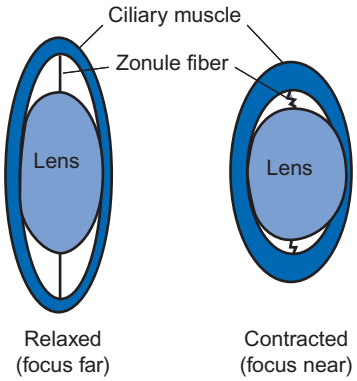


FIGURE 4.8.4 Accommodation of the eye. Far objects are focused on the retina by a combination of the refraction of the cornea and lens. Focusing near objects requires an increase in the refractive power of the eye, which is accomplished by changing the shape of the lens. The lens shape is controlled by the ciliary muscles. Contraction of the muscles removes tension on the zonule fibers that suspend the lens. Removal of tension allows the lens to recoil to a rounder shape.

too short. Converging lenses (+ lenses) correct hypermetropia.

PHOTORECEPTOR CELLS IN THE RETINA TRANSDUCE LIGHT SIGNALS

Rod-shaped cells and cone-shaped cells in the retina detect light. Their structures are shown schematically in Figure 4.8.5. The rod-shaped cells are more highly sensitive to light because they contain larger numbers of visual pigments and their effects are more efficiently transduced to changes in rod cell membrane potential. The overall mechanism for transduction in rod cells is shown in Figure 4.8.6. Neither rods nor cones produce action potentials. Instead, they alter the membrane potential of another kind of retinal cell, the bipolar cell, which also does not produce action potentials.

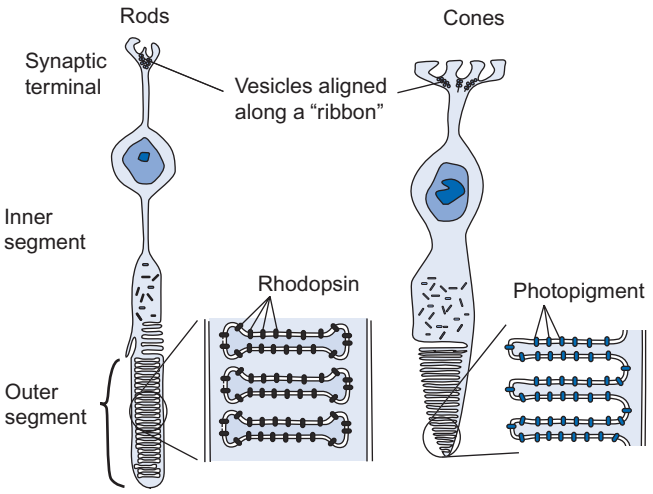
The outer segments of the rods contain discrete disks of membranes that do not contact the surface membrane. Each rod has some 700–2000 of these disks, and each contains some 50,000 molecules of **rhodopsin**, consisting of the protein **opsin** and its chromophore, **11-*cis*-retinal**, a derivative of vitamin A. Rhodopsin maximally absorbs light at a wavelength of 507 nm. The visual pigments in the cones reside in folds of the surface membrane of the cone outer segment. There are three types of cones that differ in the protein that binds 11-*cis* retinal. A single cone contains only one of three different visual pigments. Cones are maximally sensitive to red, green, or blue light, and they maximally absorb light at 555, 530, and 426 nm, respectively.

THE RETINA CONSISTS OF SEVERAL LAYERS AND BEGINS PROCESSING OF VISUAL SIGNALS

The outermost layer of the retina is the **retinal pigmented epithelium**. This cell layer consists of cuboidal cells with tentacle-like processes at the apical surface that extend between photoreceptor cells. These cells contain **melanin granules**, a high molecular weight, insoluble polymer of oxidized tyrosine molecules. These absorb stray light, preventing light scatter between photoreceptor cells. The photoreceptor cells do not divide, but they continually renew themselves by making new layers in the outer

TABLE 4.8.1 The Effect of Age on the Near Point and Power of Accommodation

Age (years)	Near Point (m)	Power of Accommodation (D)
10	0.07	14
20	0.10	10
30	0.13	7.7
40	0.19	5.3
50	0.54	1.9
60	0.83	1.2



Long outer segment	Shorter outer segment
Large synaptic terminal	Larger synaptic terminal
High sensitivity	Low sensitivity
Scotopic vision	Photopic vision
Saturates in daylight	Saturates only in intense light
Lower temporal resolution (20 Hz)	High temporal resolution (70 Hz)
Rhodopsin	Erythrolabe Chlorolabe Cyanolabe
Low spatial acuity	High spatial acuity
Color blind	Color sensitive

FIGURE 4.8.5 Schematic diagram of the structures of rods and cones in the human retina. Both rods and cones consist of an outer segment that contains the light-sensitive chemicals and an inner segment that contains the nucleus and an abundance of mitochondria. Rods and cones differ in their structure, sensitivity to light intensity, sensitivity to the wavelength of light, and saturability. Rods are concentrated outside the fovea, whereas cones are concentrated within the fovea. Cones give rise to color vision in bright light (photopic vision), whereas rods allow vision in dim light (scotopic vision).

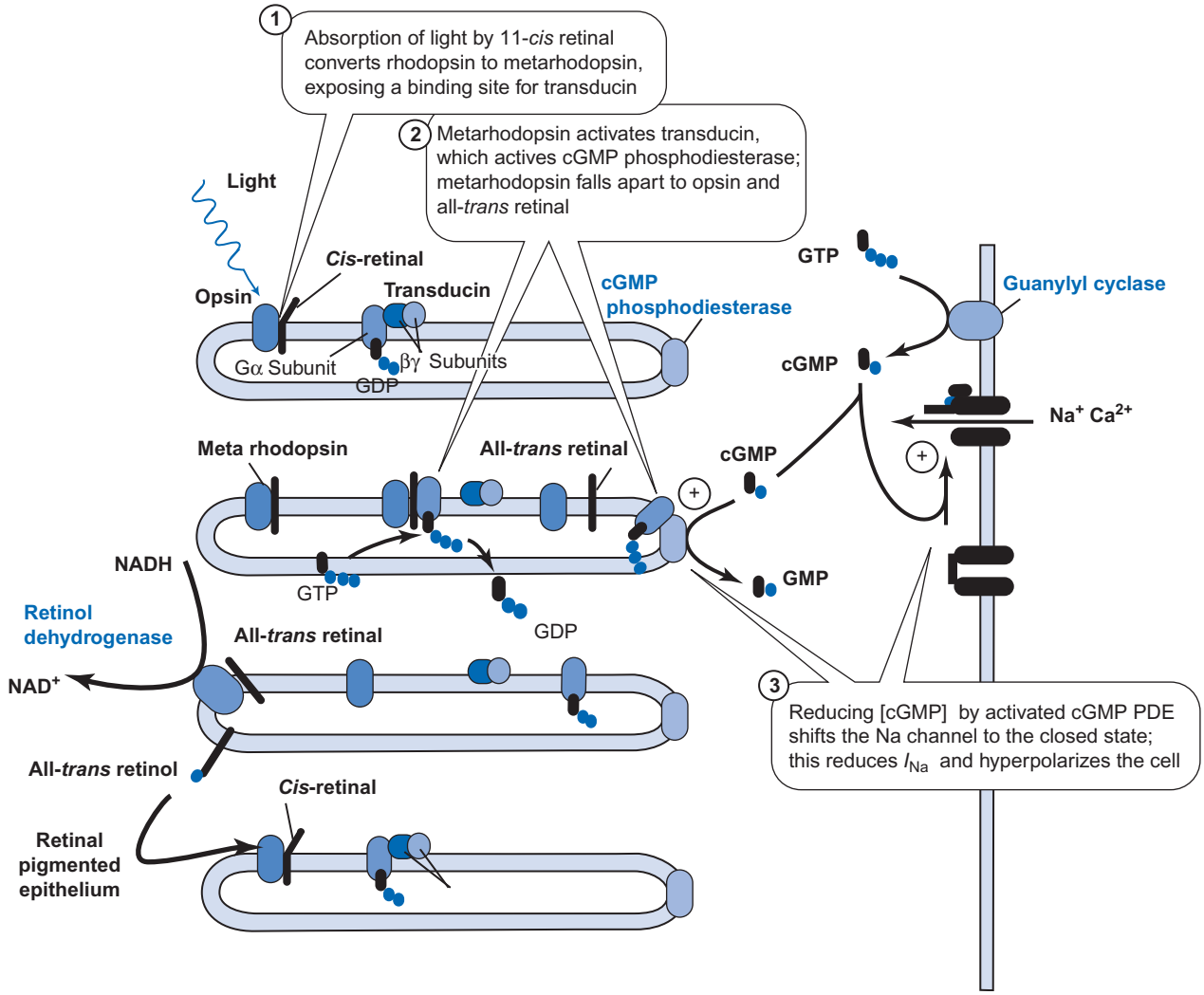


FIGURE 4.8.6 Photobleaching of rhodopsin and mechanism of photodetection. Rhodopsin is a complex of the protein, opsin, with its chromophore, 11-*cis* retinal. Absorption of a photon of light causes 11-*cis* retinal to isomerize to all-*trans* retinal. The complex of opsin with all-*trans* retinal is converted rapidly to metarhodopsin II. This metarhodopsin interacts with a heterotrimeric G-protein, transducin, whose α subunit activates a cGMP phosphodiesterase. The cGMP phosphodiesterase lowers the concentration of cGMP in these cells. cGMP sets the equilibrium between the open and closed state of a channel on the surface membrane that conducts both Na^+ and Ca^{2+}. Reducing [cGMP] favors the closed state, which reduces the inward I_{Na}, hyperpolarizing the cell. Meanwhile, the metarhodopsin dissociates to form opsin and all-*trans* retinal. Regeneration of rhodopsin begins with conversion of all-*trans* retinal to all-*trans* retinol inside the rods. The all-*trans* retinol is exported to the retinal pigmented epithelium where it is converted back to 11-*cis* retinal, which recombines with opsin to form rhodopsin again.

segments and shedding the old layers. The retinal epithelial cells phagocytose the shed outer segments. Photoreception produces **all-*trans* retinal** from **11-*cis* retinal** (see Figure 4.8.6), and the photoreceptor cells convert this to all-*trans* retinol. The retinal pigmented epithelial cells take up the all-*trans* retinol and convert it to 11-*cis* retinal which is taken back up by the photoreceptor cells. The retinal pigmented epithelium also synthesizes proteins that form the interphotoreceptor matrix and aid in the exchange of nutrients and metabolites between the photoreceptor cells and blood (Figure 4.8.7).

The **photoreceptor layer** contains the parts of the photoreceptor cells that absorb light and begin transduction between light absorption and neural signal. The retina contains some 80–110 million rod cells and some 4–5 million cones. The number of rod cells per unit area of retina is greatest in the retinal area immediately surrounding the fovea and decreases with distance toward the ora serrata (the line where the retina ends). The fovea contains a high density of tightly packed cones, with 200,000 cones mm^{-2}.

The **outer nuclear layer** contains the nuclei of the photoreceptor cells.

The **outer plexiform layer** contains synaptic contacts between photoreceptor cells, **bipolar cells**, and **horizontal cells**. The photoreceptor cells end in a **synaptic terminal** that makes contact with processes from **bipolar cells**. The bipolar cells derive their name from the fact that their cell bodies have two major processes. One extends to the outer plexiform layer and the other to the inner plexiform layer. Each bipolar cell receives inputs from a set of rod photoreceptor cells or from a set of cones, but not from both. **Rod bipolar cells** collect signals from 15 to 20 rod cells; **midget**

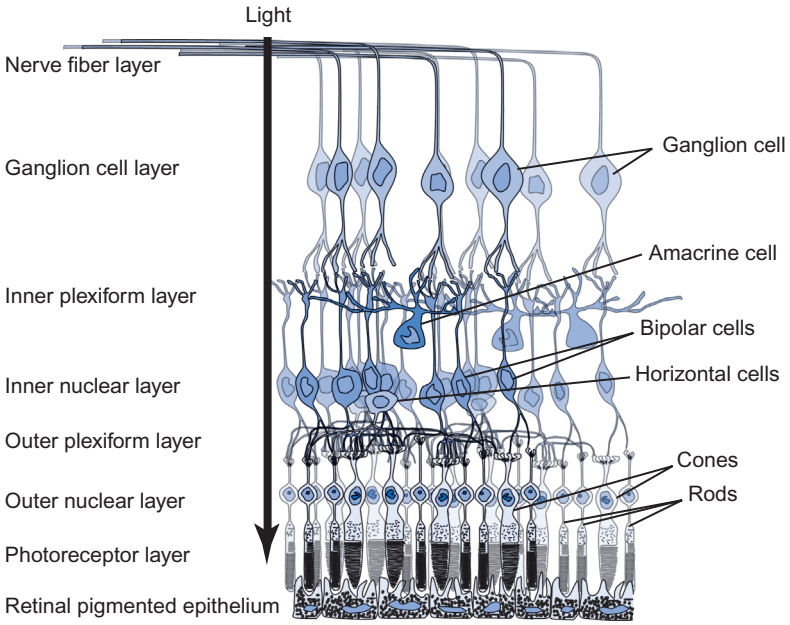


FIGURE 4.8.7 Schematic diagram of the retina. The outermost part of the retina is the retinal pigmented epithelium that absorbs scattered light and participates in the maintenance of the photoreceptors and in recycling bleached photopigments. This is the layer furthest away from incoming illumination. The photoreceptor layer is immediately adjacent to the retinal pigmented epithelium. The signal arising from the photoreceptors converge on several different bipolar cells. The bipolar cells, like the photoreceptor cells, exhibit graded changes in membrane potential but do not produce action potentials. A second line of signal processing occurs at the inner plexiform layer. Ganglion cells produce action potentials that travel down their axon. These collected axons make up the optic nerve.

bipolar cells receive inputs from a single cone; **flat bipolar cells** have inputs from 5 to 20 cones. As mentioned above, there are some 80–110 million rod cells and about 4–5 million cones. There are about 36 million bipolar cells. Thus there is convergence of inputs from the photoreceptors onto the bipolar cells. The **horizontal cells** are interneurons that receive inputs from many photoreceptor cells and make synapses back onto other photoreceptor cells and with bipolar cells.

The **inner nuclear layer** contains the nuclei of retinal interneurons including the bipolar cells, horizontal cells, and **amacrine cells**. The **inner plexiform layer** contains synaptic contacts between bipolar cells, amacrine cells, and **ganglion cells**.

The **ganglion cell layer** forms the output of the retina. The retina contains about 1–2 million ganglion cells. They form nerve fibers that make up the major parts of the **optic nerve**. The ganglion cells are the only retinal cells that produce action potentials. The photodetector cells continually secrete glutamate neurotransmitters in the dark. Illumination closes Na^+ channels in the outer segments, through transducin activation of cGMP phosphodiesterase, which hyperpolarizes the photoreceptor cell and diminishes release of glutamate neurotransmitter. Thus the photoreceptor cells have a graded membrane potential but do not produce action potentials. Horizontal, bipolar cells and amacrine cells also do not produce action potentials. Their response is a graded potential. Ganglion cells, however, produce action potentials that travel along their axons down the optic nerve.

The **nerve fiber layer** contains the axons of ganglion cells.

BIPOLAR CELLS ARE OFF-CENTER OR ON-CENTER

Bipolar cells receive inputs from a set of photoreceptor cells that define the bipolar cell's **receptive field**. **The neurotransmitter released from all photoreceptor cells is glutamate**. Because glutamate release is *decreased* upon exposure to light, a bipolar cell that responds to glutamate by excitation will be excited when the light is off. These are called **off-center bipolar cells** because they **are active when the light is off** in the center of their receptive field (Figure 4.8.8).

Some bipolar cells respond to glutamate by hyperpolarization, or inhibition. In the dark, the photoreceptors maximally release glutamate and these bipolar cells are maximally hyperpolarized. In the light, the photoreceptors are hyperpolarized and they release less glutamate. These bipolar cells are called **on-center bipolar cells** because they **are active when the light is on**.

THE OUTPUT OF BIPOLAR CELLS CONVERGE ONTO ON-CENTER AND OFF-CENTER GANGLION CELLS

As information flows centrally, the number of cells carrying the information decreases. Thus, there are many more photoreceptor cells (80–110 million rods, 4–5 million cones) than bipolar cells (36 million), and there are many more bipolar cells than ganglion cells (1–2 million ganglion cells per eye).

GANGLION CELLS IN THE PARVOCELLULAR PATHWAY RECEIVE INPUT FROM A FEW BIPOLAR CELLS

Some ganglion cells receive inputs that are equivalent to a single or just a few bipolar cells. This small receptive field allows the ganglion cell to encode fine details of the visual field. The axons from these ganglion cells segregate in the optic nerve and the segregation remains intact all the way up the neural pathways to the visual cortex. These ganglion cells are small, and therefore they are named **parvocellular** or P cells from the root meaning "small." These cells project to the four dorsal layers of the **lateral geniculate nucleus** (two ipsilateral

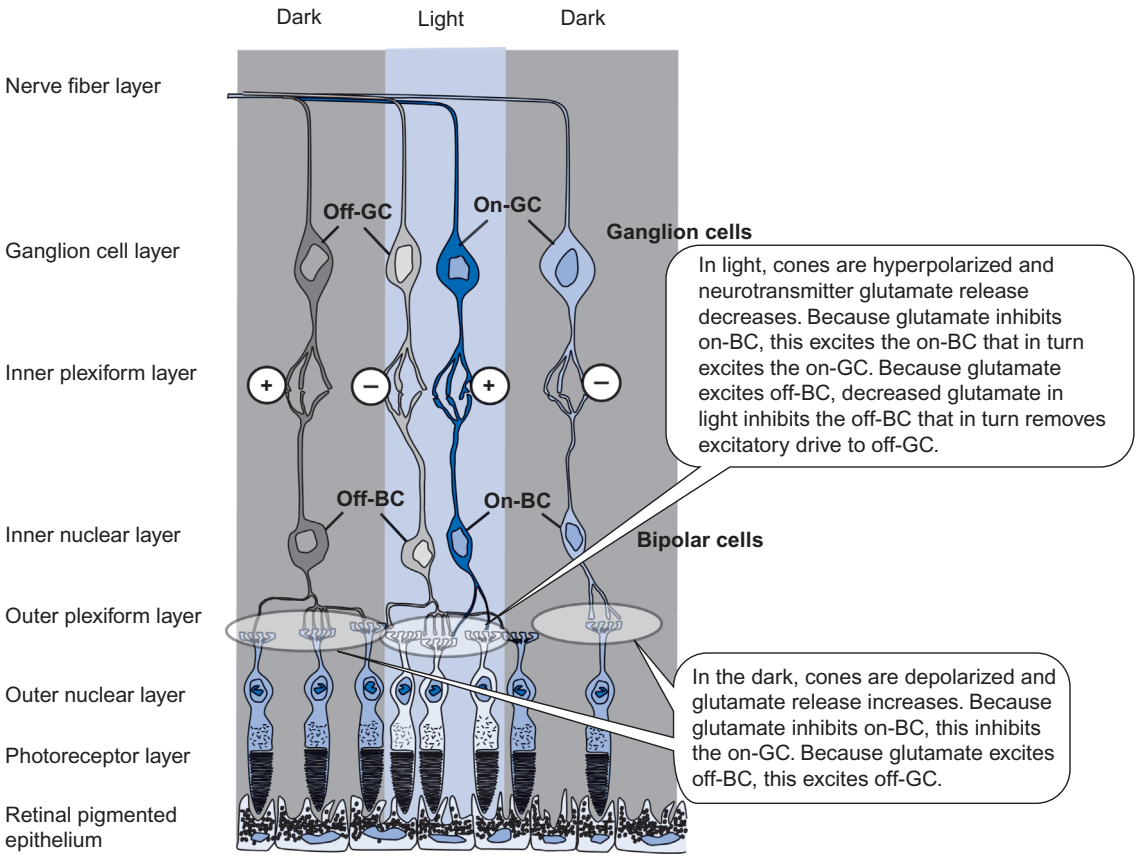


FIGURE 4.8.8 On-center and off-center bipolar cells and ganglion cells. In the dark, photoreceptors are depolarized (dark gray color) and increase their release of glutamate neurotransmitter. Light causes these photodetectors to hyperpolarize and decrease their glutamate release (light blue color). **Glutamate inhibits on-BCs and excites off-BCs**. In the light, decreased glutamate relieves the inhibition of on-BCs. Thus the on-BCs in the light are depolarized (dark blue) which increases their release of neurotransmitter. This activates on-GCs (dark blue). Thus on-GCs are activated when the light is on. By similar arguments, the off-GCs are activated when the light is off (dark gray).

and two contralateral) forming the parvocellular division. These cells carry detailed visual information.

GANGLION CELLS IN THE MAGNOCELLULAR PATHWAY RECEIVE INPUT FROM HUNDREDS OF BIPOLAR CELLS

Another type of ganglion cell are much larger and are called magnocellular or M cells. These cells receive inputs from many bipolar cells and so produce less visual acuity. Their segregation, like that of the parvocellular division, remains intact through the visual pathway. They project to the two ventral layers of the lateral genicular nucleus, one from the ipsilateral retina and one from the contralateral retina.

SURROUND EFFECTS ON GANGLION CELLS ARE DUE TO CONVERGENCE OF BIPOLAR CELLS AND LATERAL PROCESSING BY HORIZONTAL CELLS AND AMACRINE CELLS

The response of ganglion cells to light patterns on the retina is more complicated than a simple "on-center" or "off-center." Illumination or lack of illumination of the surrounding areas of the retina also influences ganglion cell response. This effect arises from convergence of input from bipolar cells onto ganglion cells and is partly produced by integrative functions of the horizontal cells. The rod bipolar cells receive inputs from up to 50 photodetectors, and they also have on-center, off-surround types of receptive fields. Thus the ganglion cells do not create a receptive field pattern; they relay a receptive field organization that is already present at the level of the bipolar cells. The response to light of ganglion cells with on- or off-centers is shown in Figure 4.8.9.

SIGNALS FROM THE TWO EYES CROSS OVER DURING THE CENTRAL VISUAL PATHWAYS

Each retina possesses some 1–2 million ganglion cells that are distributed throughout the retina. The retina itself is divided into four quadrants: the superior and inferior halves are divided into nasal and temporal quadrants. The image of the external world on the retina is inverted and reversed. Thus, illumination of the left peripheral visual field projects to the nasal retina of the left eye, and the right peripheral visual field projects to the temporal retina of the left eye. Accordingly, the output of the ganglion cells represents particular areas

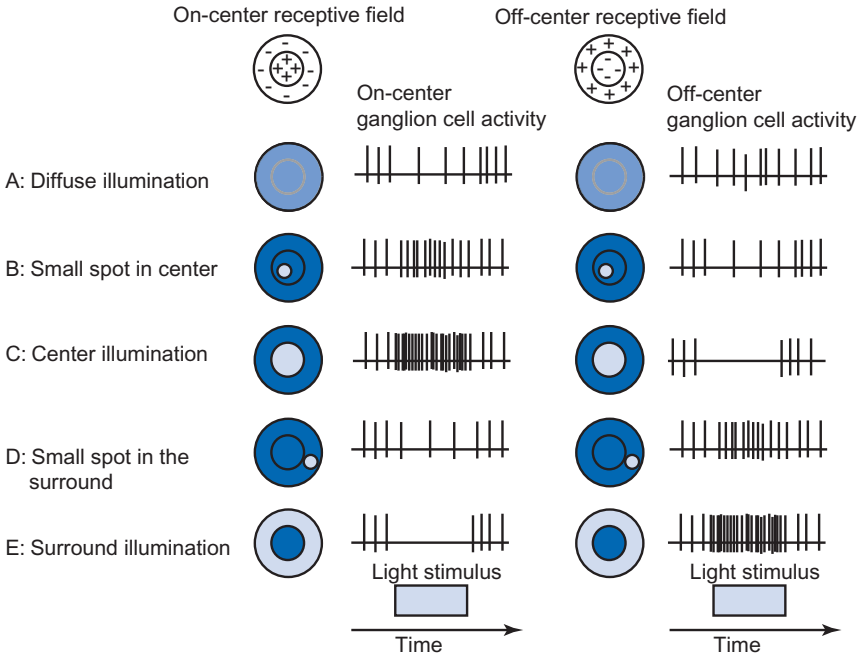


FIGURE 4.8.9 Effect of the size and position of a light stimulus on the response of on-center ganglion cells and off-center ganglion cells. Diffuse illumination (stimulus A) produces conflicting inputs to on-center ganglion cells: the center excites the ganglion cell while the surround inhibits it. Because the surround has a larger area, the result is mild inhibition. Off-center ganglion cells, on the other hand, are inhibited by light in the center and excited by light in the surround. On-center ganglion cells respond most vigorously to light that fills the center and dark in the surround (stimulus C). Off-center ganglion cells respond most vigorously to dark in the center and light that fills the surround (stimulus E). (Source: *Adapted from J. Beatty,* The Human Brain, *Sage Publications, Inc. 2001.*)

of the visual field. Each of these travels along the optic nerve. To produce a single, coherent percept of the visual world, the outputs of these ganglion cells must be merged.

Nerve fibers from ganglion cells from all parts of the retina come together at the **optic chiasm** and from there travel on to the **lateral geniculate nucleus** or the **superior colliculus**. The destination of the optic nerve fibers depends upon their location in the eye. The parts in the left retina of both eyes pass on to the left brain, and those on the right retinas go to the right brain. Thus, the ganglion cells that correspond to the same parts of the visual field mingle together (Figure 4.8.10).

The magnocellular pathway includes fibers from large ganglion cells that receive convergent inputs from many bipolar cells. These fibers from the nasal quadrants cross over at the optic chiasm and project to the **ventral lateral geniculate nucleus** in the thalamus. The parvocellular ganglion cells receive inputs from fewer bipolar cells and therefore these cells are smaller. They project to the **dorsal lateral geniculate nucleus**. These fibers synapse on cell bodies that form 6 layers in the lateral geniculate nucleus, as shown in Figure 4.8.10. These layers are numbered 1–6 from the most ventral to the most dorsal. Layers 3–6 constitute the parvocellular input, and the inputs to these layers alternate between the contralateral and the ipsilateral eye. Layers 1 and 2 form the magnocellular pathway, and these have also ipsilateral and contralateral input.

SOME GANGLION CELLS PROJECT TO OTHER AREAS OF THE BRAIN

Some ganglion cells in the magnocellular pathway do not project to the lateral geniculate nucleus, but project instead to the **superior colliculus**, part of the tectum or roof of the midbrain. The superior colliculus makes no contribution to the conscious visual percept but provides information for visual orientation, focusing of the image, and control of **saccades**, the rapid movement of the eyes used in tracking objects in visual space.

ADDITIONAL PROCESSING OF VISUAL IMAGES OCCURS IN THE VISUAL CORTEX

The cells in the lateral geniculate nucleus project to the visual cortex through fibers called **optic radiations** that spread out like a fan traveling laterally and inferiorly around the horn of the lateral ventricle. These make connections in the visual cortex at the back of the brain in the occipital lobe. This region of the brain is often called the **striate cortex** because a layer of myelinated fibers is found there. The processes from the lateral geniculate nucleus connect primarily to layer 4 of the visual cortex. Like much of the cortex, the visual cortex is organized into 6 layers. The visual cortex has additional levels of organization. Alternate columns of cortex receive inputs primarily from one eye. These alternating bands form **ocular dominance columns**.

The responses of specific cells in the visual cortex were first described by Hubel and Wiesel, who earned a Nobel Prize in 1981. They described **simple cortical cells** that have receptive fields that are basically elongated on-center, off-surround receptive fields. These simple cortical cells respond when bars of light stimulate the on-center and the off-surround is dark. These cells respond preferentially to bars of light with a particular orientation, and the same stimulus at 90° elicits a poor response. These cells respond to bars only in the correct position; movement of a bar into the off-surround results in inhibition rather than stimulation. Hubel and Wiesel also found **complex cortical cells**

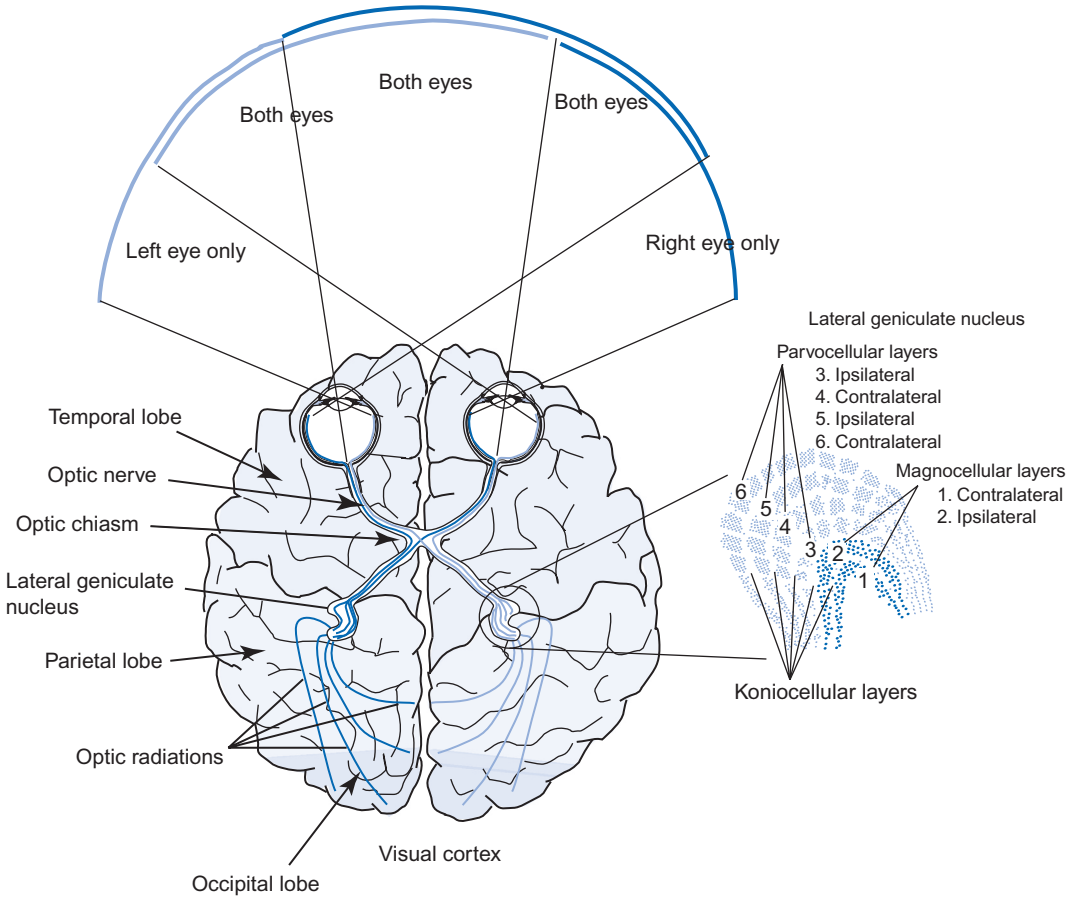


FIGURE 4.8.10 The visual pathway. Areas of the external world constitute the visual field. The optics of the eye focus light from these areas on areas of the retina and the light alters the firing patterns of retinal ganglion cells. The axons of these cells travel over the optic nerve to the optic chiasm, where the fibers from the nasal quadrants cross over to the opposite side of the brain. The fibers from all parvocellular ganglion cells and most magnocellular ganglion cells travel to the lateral geniculate nucleus, where the signals are relayed to the cerebral cortex that lines the occipital lobe. There the visual cortex further processes the visual image. Thus, visual signals from the temporal retina are ipsilateral and those from the nasal retina are contralateral.

that respond to bars of light with the proper orientation anywhere in their receptive field. These remain sensitive to orientation but not position. Another type of cell in the visual cortex responds to bars that have ends to them. These "end-stopped cells" may be corner detectors.

Edge detectors can be constructed by selectively connecting some on-center, off-surround ganglion cells in an array. The retina produces some million of these kinds of receptive fields. A bar along the on-centers of a number of ganglion cells excites them all, so that a cortical cell having excitatory connections to this set of ganglion cells will be excited optimally when the bar is aligned with their centers.

THE VISUAL CORTEX SENDS OUTPUT TO THE TEMPORAL AND PARIETAL LOBES

The visual cortex sends fibers along two distinct output pathways: the **dorsal stream** and the **ventral stream**. The dorsal stream feeds information into the parietal lobe which appears to be necessary for using visual information in forming movements. The ventral stream feeds visual information into the temporal lobe where details of the objects are identified. The ventral stream is the "what" pathway, whereas the dorsal stream is the "how" pathway.

WE STILL DO NOT KNOW HOW WE PERCEIVE VISUAL IMAGES

Despite the remarkable progress in identifying all of those processes involved in focusing light, transducing the signal, and interpreting the signal at the cellular level, we still do not understand exactly what it is that perceives and how that perception is accomplished. It is clear that the visual system is hierarchical; each level builds upon processing accomplished at a lower level. Is it likely that there is a "Grandmother cell" whose firing tells us, whoever the "us" is, that we are seeing our Grandmother? Or is it more likely that there is some delocalized set of neurons, which by their nature are a fleeting occurrence, that leads to the interpretation of "Grandmother" in our visual field? In the final analysis, visual perception remains mysterious.

Clinical Applications: Cataracts

The transparency of the lens depends on lens proteins called **crystallins**. These water-soluble proteins comprise 90% of the total lens protein. The remaining 10% consist of water-insoluble proteins including membrane proteins and cytoskeletal proteins. The lens contains about 66% water and 33% protein, double the protein content of most other tissues. This high protein content causes the high refractive index of the lens. These proteins last longer than any other proteins in the body. Most lens cells lose the ability to make new proteins and so their lens proteins last a lifetime. Because cells are added to the outside layers of the lens, the crystallin concentration varies from 15% in the cortex to 70% in the interior of the lens. This concentration gradient of lens proteins produces a gradient in refractive index.

An opaque area in the lens is called a **cataract**. The opacity interferes with light transmission to the retina and produces glare during night driving, blurred or distorted vision, reduction in visual acuity, and double vision in the affected eye. The opacity may occur in different zones of the eye, and the cataract is classified accordingly. Cataracts may result from trauma, medications, radiation, inflammation, metabolic diseases, and simple aging.

Poor vision caused by cataracts can be improved by surgical removal of the lens. Assessment of the potential for improved vision requires the use of a potential acuity meter (PAM) or laser interferometer. The PAM projects a miniature Snellen eye chart (the chart used to measure visual acuity in the typical physicians's office) on the retina through a 0.1 mm pinhole aperture through a clear area of the lens, thus bypassing the cataract. The interferometers project an interference pattern on the retina. Removal of the lens may be followed by insertion of an intraocular lens implant. Intracapsular cataract extraction removes the entire lens; extracapsular cataract extraction leaves the posterior lens capsule to support an implant.

SUMMARY

The visual system has three problems: focus light on the retina, transduce the light signal to neural signals, and process these neural signals to form a conscious perception. Light passing from the external environment to the retina sequentially passes through a thin film of tears, the cornea, the aqueous humor, the lens, and the vitreous humor. Light is refracted at each interface according to Snell's law:

$$n_1 \sin \theta_1 = n_2 \sin \theta_2$$

where n_1 and n_2 are the refractive indices of media 1 and media 2 and θ_1 and θ_2 are the angles between the incident light ray and surface normal. The purpose of the refraction is to bend light so as to focus the light on the retina. The refractive power of the eye is given by the thin lens formula:

$$\frac{1}{O} + \frac{1}{I} = \frac{1}{f}$$

where O is the distance from the nodal point to the object, I is the distance to the image, and f is the focal length. The refractive power is $1/f$. At rest, the refractive power is about 63 D, most of which is provided by the cornea. The refractive power of the eye depends on the shape of the lens, which is controlled by the ciliary muscle. Contraction of the muscle releases tension on suspensory ligaments of the lens, causing the lens to become rounder because of its inherent elasticity. Accommodation is the ability to change refractive power to focus on nearby objects. This is generally 10−12 D in young people and decreases with age. The gradual loss of accommodation with age is presbyopia.

Retinal photoreceptors are mainly rods and cones. Both release glutamate onto bipolar cells and reduce the amount of release when exposed to light. Rods are highly sensitive but saturate easily, have lower spatial acuity, and do not detect color. Cones are responsible for color vision and vision in bright light.

The photoreceptors contain a chromophore, 11-*cis*-retinal, complexed to proteins that confer sensitivity to different wavelengths of light. Absorption of light converts 11-*cis*-retinal to all-*trans* retinal and uncovers a binding site for a heterotrimeric G-protein called transducin. After excitation, transducin exchanges GTP for GDP and activates a cGMP phosphodiesterase. This reduces [cGMP] which closes a Na^+ channel, leading to hyperpolarization and less release of glutamate.

Glutamate excites off-bipolar cells, so these off-BC cells are excited when the light is off. Glutamate inhibits on bipolar cells; they are excited with the light is on. These connect and converge onto ganglion cells that produce action potentials that are relayed to the lateral genicular nucleus and then to the visual cortex in the occipital lobe. Fibers from the nasal quadrants cross over to the opposite side at the optic chiasm; fibers from the temporal quadrants remain ipsilateral. The magnocellular ganglion cells convey general information about shape; parvocellular cells convey detailed visual information.

The visual cortex has 6 layers and is organized into ocular dominance columns that have information from one eye. Specific cells in the cortex respond to higher-order patterns in the visual field. Simple cells respond to a bar of light with the proper position and orientation. Complex cells respond to bars of light of the proper orientation but regardless of position. Other cells respond to more complex features in the visual field.

REVIEW QUESTIONS

1. What part of the eye causes the most refraction? What part of the eye is most responsible for accommodation? What muscles are responsible for it?

2. What is the near point? At what point is accommodation maximal? What is the name for loss of accommodation with age?
3. What produces the aqueous humor? Where does it drain? What happens if intraocular pressure gets too high?
4. Where are the cones concentrated? Where are the rods? What differences explain their relative functions?
5. How do photoreceptor cells transduce light into a neural signal? What transmitter is used?
6. What neurons connect directly to the photoreceptors? Do they make action potentials? What is an "on-bipolar cell" and an "off-bipolar cell"?
7. How do "on-ganglion cells" respond to light within their receptive fields? How do "off-ganglion cells" respond? Do ganglion cells make action potentials?
8. What is the blind spot? Where do nerve fibers go after they leave the eyeball in the optic nerve?
9. What is the difference between parvocellular and magnocellular ganglion cells?
10. What is the lateral geniculate nucleus? Where is the visual cortex? What do simple cortical cells do? What do complex cortical cells do?

APPENDIX 4.8.A1 REFRACTION OF LIGHT AND THE THIN LENS FORMULA

LIGHT INTERACTS WITH MATTER AS IT PASSES THROUGH

Electromagnetic Radiation Induces Oscillation of Electrically Charged Particles in Matter

Light is an oscillating electromagnetic field. An oscillating, electrically charged particle generates an oscillating electromagnetic field that propagates away in all directions at right angles to the direction of oscillation. Similarly, the oscillating electromagnetic wave of light interacts with charged particles in matter, causing them to oscillate at the frequency of the incident light and to reradiate electromagnetic waves at right angles to their motion. This is equivalent to Huygens' principle, which states that every point on an advancing wave at speed v is the origin of a secondary wavelet that propagates at the same speed. This phenomenon of reradiation of incident light is called **scatter**. When the oscillating charged particles have appropriate spatial separation, the reradiated electromagnetic waves undergo destructive interference in all directions except in the direction of the original incident light. This is the basis of **transparency**. In transparent substances, only the forward scatter survives the destructive interference among the secondary waves produced by the moving charged particles within the material. The transparency of the eye lens depends on the dense packing of scattering elements within the lens, and this is maintained by its relatively dry state. Cataracts are a disruption of this structure so that the lens scatters light in all directions.

The Oscillating Particles Re-emit Light After a Delay, Slowing the Speed of Light Within Matter

The speed of light in a vacuum, c, is $3 \times 10^9\ \mathrm{m\ s^{-1}}$. When light passes through a transparent medium, it passes between the atoms at the speed c. However, each interaction with charged particles in the medium delays the phase of the light, so that its speed within the medium, v, is reduced. The ratio of the speed of light in vacuum to the speed of light in the medium is called the **refractive index** of the medium, denoted by n:

$$n = \frac{c}{v} \qquad [4.8.A1.1]$$

THE DIFFERENT SPEED OF LIGHT IN TWO MEDIA CAUSES REFRACTION, THE BENDING OF LIGHT AT THEIR INTERFACE

According to Huygens' principle, every point on a wave front traveling at speed v is the origin of a secondary wavelet that also propagates at speed v. Consider Figure 4.8.A1.1, which shows the interface between air and a liquid transparent medium. The refractive index of air is 1.00029, so the speed of light in air is very near to its speed in a vacuum. The refractive index of water, however, is 1.33, so that light is significantly slowed when it enters the water. We assume here that the incident light approaches the interface obliquely at some angle, θ_i, the angle of incidence. As the wave front enters the water, it is slowed compared to the remaining wave front that still travels through the air. The result is that the wave front's direction is changed to θ_r, the angle of refraction. During some time t, the wave front advances $v_i t$ in the incident medium, where v_i is its speed in this medium. During that same time, the wave front advances a shorter distance, $v_r t$, in the second medium. We can choose t so that the geometry in Figure 4.8.A1.1 occurs. The arrows in the figure are at 90° to the wave fronts, so that $\angle$ACD and $\angle$ABD are both at right angles. The arrows correspond to the rays that are at right angles to the wave fronts. Thus we can write

$$\sin\theta_i = \frac{v_i t}{\mathrm{AD}} \qquad \sin\theta_r = \frac{v_r t}{\mathrm{AD}} \qquad [4.8.A1.2]$$

These two equations can be combined, by taking their ratios and canceling the like factors, to obtain

$$\frac{\sin\theta_i}{\sin\theta_r} = \frac{v_i}{v_r} \qquad [4.8.A1.3]$$

Since $v_i = c/n_i$ and $v_r = c/n_r$, by Eqn [4.8.A1.1], we rewrite Eqn [4.8.A1.3] as

$$n_i \sin\theta_i = n_r \sin\theta_r \qquad [4.8.A1.4]$$

This equation is **Snell's law of refraction**.

It is often more convenient to deal with refraction in terms of **light rays** rather than the wave fronts. Light

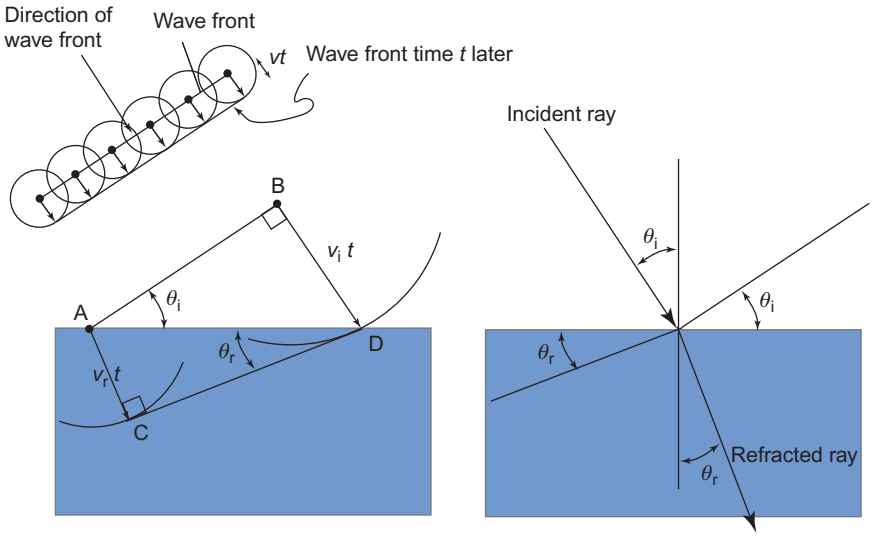


FIGURE 4.8.A1.1 Refraction of light at an interface between two media. The wave front can be imagined as proceeding by the propagation of wavelets at each point along the wave front. Only the leading edge of the wavelets contain energy. When the wave front obliquely enters a surface with a higher refractive index, the wave front is slowed while the wave front remaining in the incident medium is not slowed. The consequence of this slowing is a change in the angle of propagation. The right-hand side of the figure shows the light ray representation of the light, with the same conclusion: movement of light from a region of lower to higher refractive index bends the light toward a line normal to the surface. (Source: *Adapted from J.T. McIlwain,* Introduction to the Biology of Vision, *Cambridge University Press, 1996.*)

rays are normal to the wave fronts and give the direction of propagation of the light. The geometry of the light rays is such that Snell's law still holds, except that the angles are defined as the angles made between the rays and a line drawn perpendicular to the surface of the interface, rather than between the wave front and the surface itself.

REFRACTION DEPENDS ON THE ANGLE OF INCIDENCE AND THE REFRACTIVE INDICES IN THE TWO MEDIA

According to Snell's law, the angle of refraction, θ_r, depends on the angle of incidence, θ_i, and the ratio of the refractive indices of the two media that make up the interface. If the media have identical refractive indices, there is no refraction and the angle $\theta_r = \theta_i$. Similarly, light that enters the interface normal to its surface is not deviated, because $\theta_i = 0°$ and $\sin\theta_i = 0$ and therefore $\theta_r = 0°$ by Eqn [4.8.A1.4].

CONVEX LENSES CONVERGE LIGHT; CONCAVE LENSES DIVERGE IT

Consider a piece of glass with surfaces consisting of three planes that intersect a bundle of parallel light rays, as shown in Figure 4.8.A1.2. The refractive index of the glass is higher than that of the surrounding air, and so incident light is bent toward the normal at all surfaces. However, each surface presents a different angle to the incident light, so that light is bent toward the middle of the glass in its convex arrangement and away from the midline in its concave arrangement. This is the basis for the formation of lenses and the focusing of light.

THE THIN LENS FORMULA LINKS REFRACTIVE POWER AND IMAGE FORMATION

Lenses Can Refract Light to Intersect at a Single Point Called the Focal Point

Figure 4.8.A1.2 indicates that a crude convex lens causes light rays to converge. It is possible to construct a lens in which all of the parallel incident rays intersect at a single point. Here it is clear that the incident rays must deviate more strongly the further from the axis of the lens, and this increased deviation must occur gradually rather than abruptly as in the crude lens. That is, the surfaces of the lens must smoothly curve from the center toward the edge of the lens. This is accomplished practically by making spherical lenses in which the two curved surfaces subtend a solid angle of a sphere, as shown in Figure 4.8.A1.3. These spherical lenses refract parallel incident light so that, for practical purposes, they intersect at a single point. This single point is called the focal point. If the radii of curvature of the two surfaces are large compared to the thickness of the lens, then the lens may be considered to be a "thin lens" for which the following equations are valid.

The Focal Length Is a Measure of the Refractive Power of a Lens

The focal length is the distance from the focal point to the lens. Specifically, the focal length is the distance from the focal point to the plane that passes through the equator of the lens, as shown in Figure 4.8.A1.3. In physiological optics, the strength of the lens is expressed in **diopters**. This is the reciprocal of the focal length, f. We write

$$\text{Diopters (D)} = \frac{1}{f} \qquad [4.8.A1.5]$$

A lens has a single focal length, regardless of whether parallel incident light strikes it from the right or from the left. This is illustrated in Figure 4.8.A1.3.

Spherical Lenses Do Not Exactly Focus at a Single Point

As mentioned above, the refraction of light depends on the angle of incidence and the refractive indices of the two media. Because of its spherical curvature, light passing through the extreme edges of a spherical lens does not focus at exactly the same point as light that passes through the more central parts of the lens. This phenomenon is called **spherical aberration**. The refractive

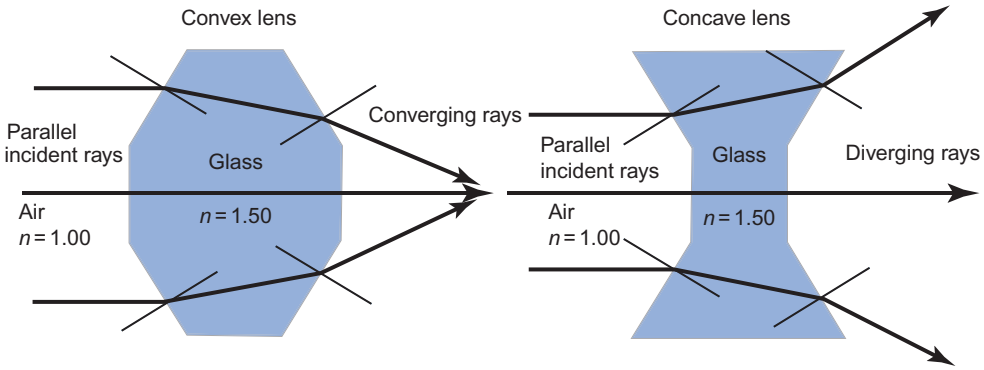


FIGURE 4.8.A1.2 Convergence of light rays by a convex lens (left) and divergence of light rays by a concave lens (right). The refraction of light rays depends not only on the relative refractive indices of the two media but on the angle of incidence.

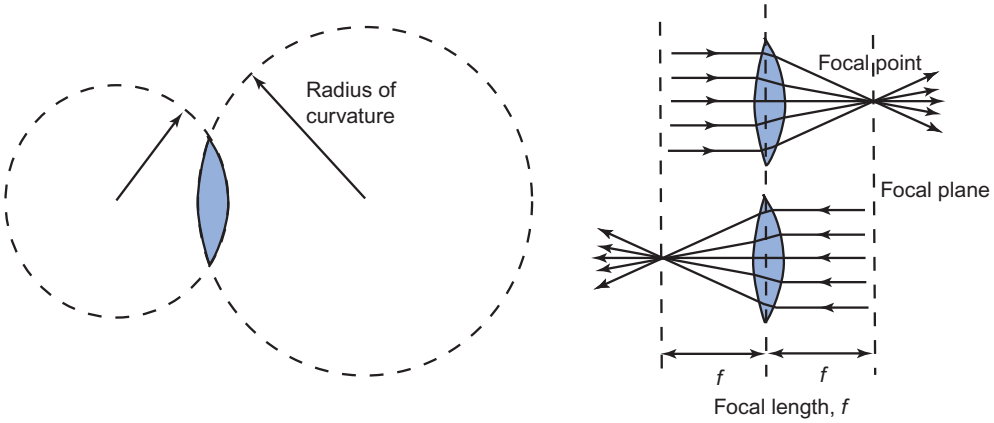


FIGURE 4.8.A1.3 Spherical lenses focus parallel incident light at the focal point. The focal length, *f*, is the same for light approaching the lens from either direction.

index also varies with the wavelength of light, a phenomenon having to do with how light is retarded by its interaction with charged particles within the medium. Blue light is bent more than yellow light, which is bent more than red light. This dependence of refractive index on the wavelength of light causes the separation of white light into its component colors by a prism. The result of this variation in refractive index is that white light produces separate images for each color that makes up the white light. This separation of images is called **chromatic aberration**. Spherical aberration and chromatic aberration are shown schematically in Figure 4.8.A1.4. The eye possesses some degree of both spherical aberration and chromatic aberration.

A SPHERICAL LENS FORMS AN IMAGE OF AN OBJECT THAT IS RELATED TO THE DISTANCE FROM THE OBJECT AND THE FOCAL LENGTH OF THE LENS

The image formed by a thin lens can be located by drawing three rays: (1) a ray which passes through the center of the lens is unchanged because the lens faces are parallel here and the lens is assumed to be thin. This ray is normal to the surface of the lens; (2) a ray parallel to the lens axis is refracted to pass through the focal point on the opposite side; (3) a ray which passes through the focal point on the side of the object emerges from the lens parallel to the lens axis. These three rays are shown in Figure 4.8.A1.5. The image is inverted and found on the far side of the focal point away from the lens. The distance from the object to the lens' nodal plane is O and the distance from the image to the lens' nodal plane is I. The height of the real object is h_O and the height of its image is h_I. The object is oriented at a right angle to the lens axis, and so is the image. The geometry of the situation allows us to identify two sets of similar triangles:

$$\begin{aligned} &\Delta AJD \sim \Delta HGD \\ &\Delta BDF \sim \Delta HGF \end{aligned} \qquad [4.8.A1.6]$$

These triangles are similar by the angle–angle–angle theorem of geometry. Because the ratio of corresponding parts of similar triangles are equal, we write

$$\frac{h_O}{h_I} = \frac{O}{I} \qquad [4.8.A1.7]$$

From the second set of similar triangles, we observe that

$$\frac{\overline{DF}}{\overline{FG}} = \frac{\overline{BD}}{\overline{HG}} \qquad [4.8.A1.8]$$

From the geometry, we can identify these line segments as

$$\begin{aligned} \overline{DF} &= f \\ \overline{FG} &= I - f \\ \overline{BD} &= h_O \\ \overline{HG} &= h_I \end{aligned} \qquad [4.8.A1.9]$$

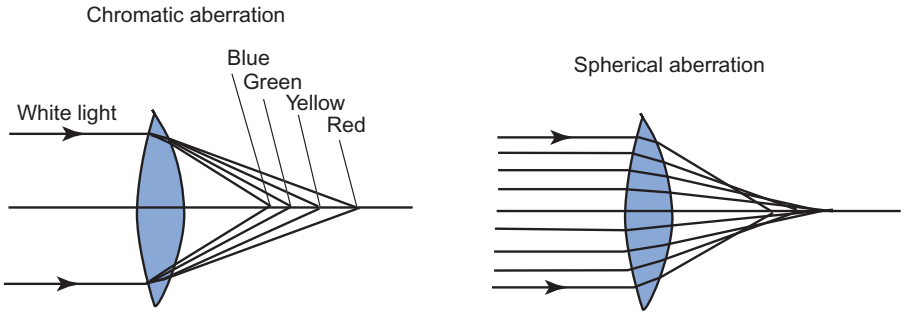


FIGURE 4.8.A1.4 Schematic diagram of the light paths taken and the resulting abnormal focus in lenses with chromatic aberration (left) and spherical aberration (right). In chromatic aberration, light of short wavelengths is bent more than light with long wavelengths, and the result is a different focus for light of different colors. In spherical aberration, light at the periphery of the lens is bent more, resulting in a different focus for light near the optical axis compared to light at the edge of the lens.

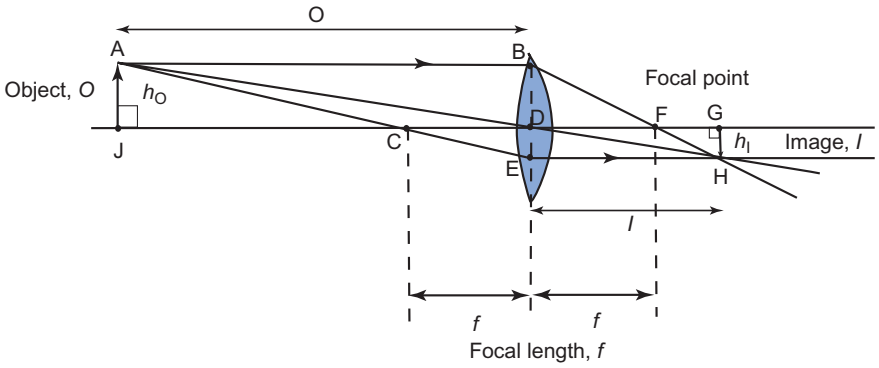


FIGURE 4.8.A1.5 Formation of an image by a spherical thin lens. The position of the image is determined by the three rays shown. One is a construction between the object and the image that passes roughly through the center of the lens. Parallel light from the object is refracted through the focus of the lens. A light ray emanating from the image and parallel to the optical axis of the lens intersects the object at its extreme. From the geometry, the thin lens equation may be derived.

Inserting these values into Eqn [4.8.A1.8], we find

[4.8.A1.10] $$\frac{f}{I-f} = \frac{h_O}{h_I} = \frac{O}{I}$$

Equation [4.8.A1.10] can be rearranged to give:

[4.8.A1.11] $$\frac{f}{O} = \frac{I-f}{I}$$

$$\frac{f}{O} = 1 - \frac{f}{I}$$

$$\frac{1}{O} = \frac{1}{f} - \frac{1}{I}$$

This last equation can be rearranged to give the **thin lens formula**:

[4.8.A1.12] $$\frac{1}{O} + \frac{1}{I} = \frac{1}{f}$$

Once again, here O is the distance from the nodal plane of the lens to the object, I is the distance from the nodal plane of the lens to the image, and f is the focal length. This equation clearly meets the condition when $O \rightarrow \infty$; under these conditions, the light entering from the object is parallel and the image is found at the focal point ($I = f$ in Eqn [4.8.A1.12]).