

# 6 BASIC ANATOMY AND PHYSIOLOGY OF THE HUMAN VISUAL SYSTEM

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The human eye is a complex structure designed to gather a significant amount of information about the environment around us. It is *the* sensor used by the Warfighter in the visually-rich battlespace. In designing a head/helmet-mounted display (HMD) system for the Warfighter, the human visual system (which begins with the eyes) could be considered as an integral component of the HMD and not as a separate and different system that subsequently is mated with the HMD. It is therefore important that HMD designers have an understanding of both anatomy and function of the human eye itself.

In the following chapter (Chapter 7, *Visual Function*), the functional operations of the human eye, its pointing and tracking mechanisms and the integration into a binocular visual system will be described. In this chapter, the goal is to provide the HMD designer with a basic understanding of the anatomy and physiology of this critical element of the human visual system. This chapter provides a brief overview of the visual system (inclusive of the eye organ itself), beginning at the front surface of the eye and progressing to the primary visual cortex at the back of the brain. Topics include:

- The Protective Structures of the Eye
  - The Orbit
  - The Lids
  - The Sclera
- The Anterior Segment of the Eye
  - The Cornea
  - The Aqueous Humor
  - The Iris
  - The Crystalline Lens and Ciliary Muscle
- The Posterior Segment of the Eye
  - The Retina
  - The Vitreous Humor
- The Visual System Pathways to the Brain
  - The Optic Nerves and Optic Tracts
  - The Lateral Geniculate Nucleus
  - The Visual Cortex

For more detailed discussions of the human eye's anatomy and physiology, the reader should refer to the large volumes of texts available, e.g., *Adler's Physiology of the Eye* (Kaufman and Alm [Eds.], 2003).

## The Protective Structures of the Eye

The two orbits, sometimes referred to as “sockets,” that protect the human eyes are situated at the front of the skull, each with a wider opening to the front narrowing to a small opening at the rear where the optic nerve exits to connect through the visual pathways and the brain. The orbits are angled outward approximately 23° with respect to the midline of the skull. The human eye itself is approximately 24 millimeters (mm) (0.94 inches [in]) in diameter and occupies about 25% of the volume of the orbit, allowing for the extraocular muscles, blood vessels, nerves, orbital fat and connective tissue that surround and support the eye (Figure 6-1). The orbit

surrounds and supports most of the human eye, while the cornea and part of the anterior globe extend somewhat beyond the orbital rims. These structures are protected by the eyelids.

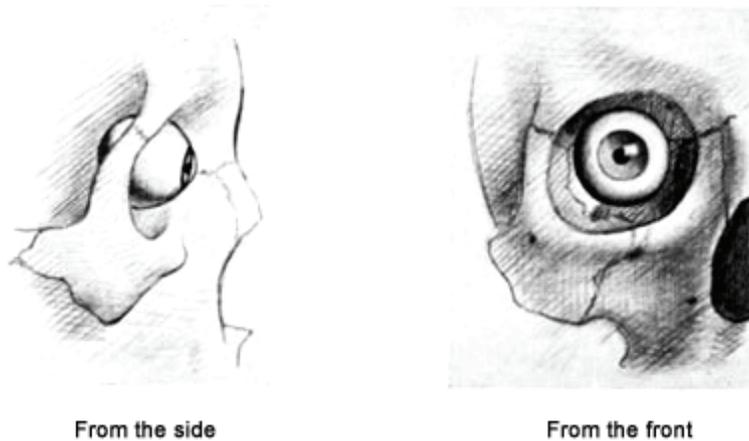


Figure 6-1. The position of the eye in its socket (Wolff, 1933).

The upper and lower eyelids form an aperture that is generally 30 mm (1.2 in) wide and 10 to 12 mm (0.4 to 0.5 in) high when the eye is “open.” The lids themselves have cartilage-like tarsal plates within their structure that provide shape to the lids and additional strength for protection of the eye. Each lid has a row of cilia or eyelashes that are very sensitive to touch or particles near the eye, which when stimulated bring on the blink reflex. The lids also contain the glands responsible for maintenance of the tear layer.

The globe itself is predominately formed of and protected by the sclera that extends from the edges of the clear cornea at the front of the eye (the “limbus”) to the optic nerve at the back of the eye. The sclera is a thick, opaque white tissue that covers 95% of the surface area of the eye. It is approximately 530 microns ( $\mu\text{m}$ ) in thickness at the limbus, thinning to about 390  $\mu\text{m}$  near the equator of the globe and then thickening to near 1 mm (0.04 in) at the optic nerve. At the posterior aspect of the eye, the sclera forms a netlike structure or “lamina cribrosa” through which the optic nerve passes. The sclera also serves as the anchor tissue for the extraocular muscles.

## The Anterior Segment of the Eye

The portion of the eye visible to the observer without special instrumentation is considered the anterior (or “front”) segment of the eye. Most of the structures responsible for focusing images onto the retina of the eye are here. The cornea is the primary focusing structure, providing about 75% of the focusing power of the eye. The crystalline lens provides the remaining variable focusing power and serves to further refine the focus, allowing the eye to focus objects at different distances from the eye. The iris controls the aperture or pupil of the eye for different light levels. The iris is actually an extension of the ciliary body, a structure that has multiple functions in the anterior segment, from production of the fluid that fills the anterior segment (aqueous humor) to suspension and control of the shape of the crystalline lens of the eye. Figure 6-2 shows most of the major structures of the human eye, including the components of the anterior segment, the protective sclera and the posterior segment (described in the next section).

### The cornea

The cornea is a unique biological tissue that is transparent to light and contains no blood vessels. This small transparent dome at the front of the eye is approximately 11 mm (0.43 in) in diameter and 500  $\mu\text{m}$  thick in the

center, thickening to around 700  $\mu\text{m}$  at the periphery. At the very edge of the cornea, transparency is slowly lost over a 1-mm (0.04-in) range in an area known as the “limbus”, which is where the cornea integrates into the opaque sclera. The cornea is more curved than the rest of the globe with an average radius of curvature of 7.7 mm (0.3 in), while the radius of curvature of the globe is approximately 12 mm (0.5 in).

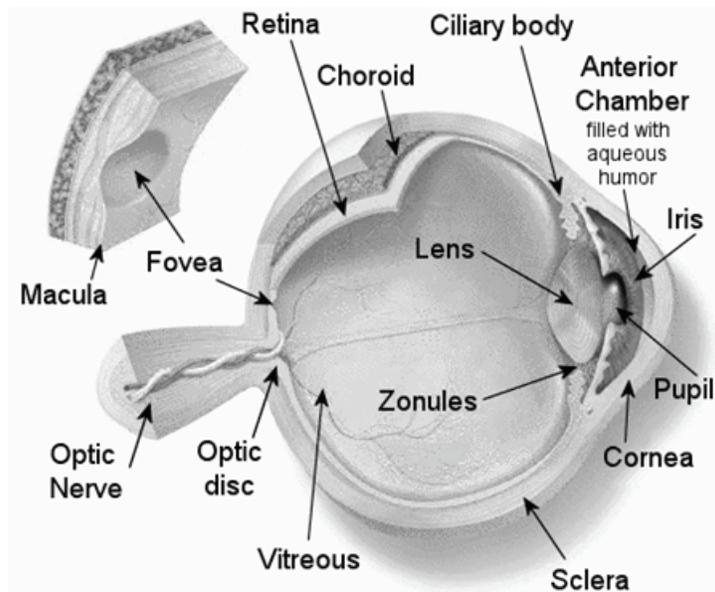


Figure 6-2. Cross-sectional view of the eye ([http://www.gimbeleyecentre.com/images/Cross\\_Section\\_Labelled.gif](http://www.gimbeleyecentre.com/images/Cross_Section_Labelled.gif)).

With the primary function of transmitting and focusing light into the eye, all the structures of the cornea are very specifically arranged (Figure 6-3). About 90% of the cornea is made up of evenly spaced collagen fibrils arranged in sections that crisscross to cover the entire extent of the cornea. This layer is known as the “stroma” and it provides not only transparency, but strength. Four more layers make up the remaining 10% of the cornea, the epithelium and Bowman’s layer at the front of the cornea and Descemet’s membrane and the endothelium at the back of the cornea.

The epithelium of the cornea, much like the epithelium of the skin, serves as a barrier to bacteria or other pathogens. Additionally, the epithelium helps to maintain the stroma at a proper level of hydration by preventing fluid from entering the stroma through its tight cell junctions and the pumping of a small portion of fluid out of the stroma. Bowman’s layer is a very thin (12  $\mu\text{m}$ ) membrane right beneath the epithelium and, in mammals, is only found in primates. Its purpose is not entirely known, although it may aid in protection of the stroma.

At the back or posterior aspect of the cornea is another very thin membrane called Descemet’s membrane that is between 10 to 15  $\mu\text{m}$  thick. It also is felt to have some protective function. The endothelium is a single layer of cells at the very posterior aspect of the cornea. The endothelium is in direct contact with the aqueous humor, the fluid that fills the anterior chamber of the eye. The endothelium pumps nutrients, such as glucose, from the aqueous humor into the cornea while actively pumping fluid out of the cornea. The hydration balance maintained by the endothelium and somewhat assisted by the epithelium is important to the transparency of the cornea, since excess fluid would disturb the regularity of the corneal fibrils and result in increased light scatter. In mild cases of edema, such as may occur when contact lenses are worn too long or under hypoxic conditions, the cornea may become slightly cloudy (Jones and Jones, 2001; Liesegang, 2002; Morris et al., 2007). Under more extreme conditions, such as anoxic conditions, or in cases of endothelial dystrophies, the swelling of the stroma could result in complete opacity of the cornea.

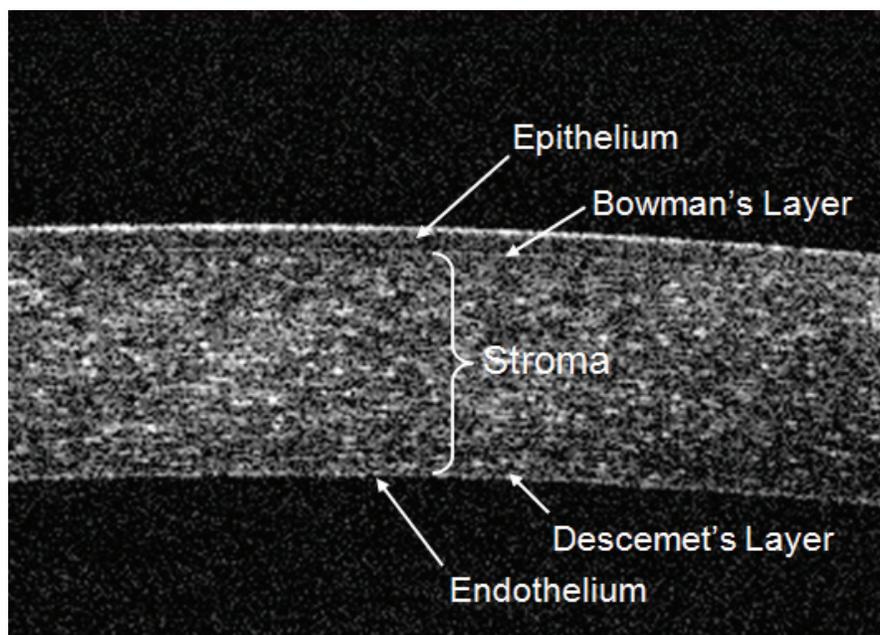
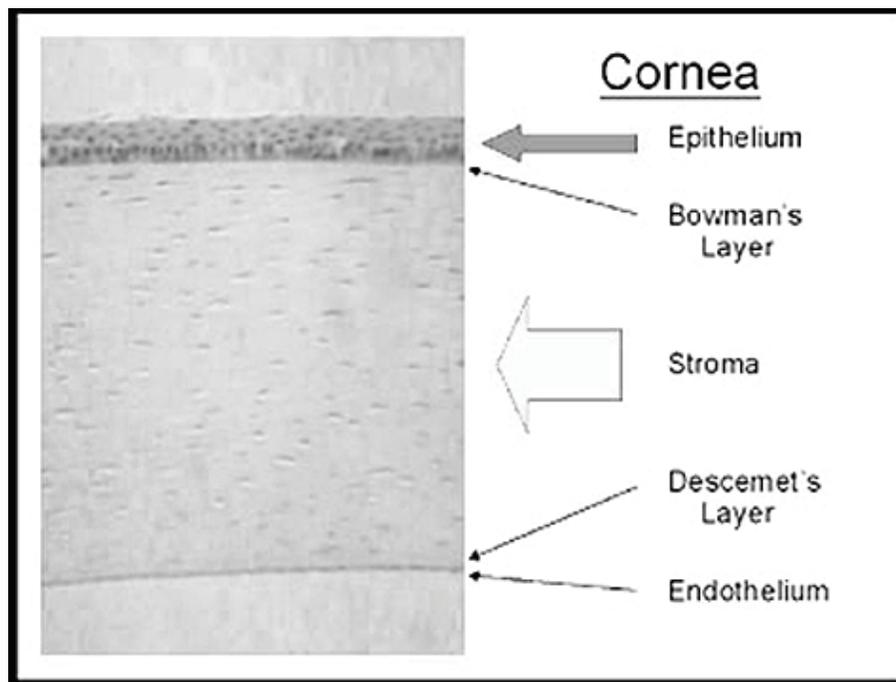


Figure 6-3. Cross-section of the cornea (top) (adapted from <http://www.opt.pacificu.edu/ce/catalog/10603-AS/Cornea.jpg>); actual Optical Coherence Tomography image of a cross-section of the author's cornea.

### The aqueous humor

The fluid that fills the anterior chamber of the eye, that area between the cornea and the front surface of the crystalline lens, is called the aqueous humor. Aqueous is produced by the ciliary body that is just posterior to the

root of the iris and extends backwards along the inner globe to the anterior aspect of the retina (Figure 6-2). Aqueous finds its way into the anterior chamber by flowing between the crystalline lens and the iris through the pupil.

Aqueous has two functions; it provides nutrients to the cornea and is part of the optical pathway of the eye. Aqueous humor is basically a fortified blood plasma that circulates in the anterior chamber, providing nutrients to the cornea and the crystalline lens. It is a transparent fluid with an index of refraction of 1.333, which is slightly less than the index of refraction of the cornea (1.376) and less than the index of refraction of the lens (gradient index of 1.406 to 1.386). As is discussed in another chapter, it is these differences in index of refraction between media coupled with the curvature of the various optical surface interfaces that result in the bending of light at each interface.

As nutrients are drawn from the aqueous into the cornea by the endothelium, the aqueous fluid is circulated out of the eye and replaced by newly produced aqueous produced. To move out of the anterior chamber, it flows out of the eye primarily through the trabecular meshwork, a “drainage” system that lies behind the limbus in the angle between the cornea and the anterior iris. There is some resistance to outflow of aqueous at the trabecular meshwork that serves to maintain a pressure within the eye of approximately 15 mmHg. If there were no resistance, the eye would lose its shape and therefore its optical integrity. If there is too much resistance (or too much production of aqueous), the pressure in the eye may exceed the eye’s tolerance and damage to the optic nerve may occur, a condition known as “glaucoma.”

Glaucoma generally results in a loss of mid-peripheral vision with sparing of central vision until the condition has progressed significantly. It is most commonly hereditary with a higher prevalence in certain ethnic groups (Friedman et al., 2004; Leske, 2007; Rivera, Bell, and Feldman, 2008; Wadhwa and Higginbotham, 2005); however, it may occur in individuals without a family history of glaucoma or may result secondarily to blunt trauma to the eye Cavallini et al., 2003; Kenney and Fanciullo, 2005; Sihota, Sood, and Agarwal, 1995). Glaucoma can be slowly progressive, as in the case of *primary open angle glaucoma* (POAG) or *low tension glaucoma* (LTG), and the loss of vision may be initially barely noticeable. A third type of glaucoma, *angle closure glaucoma* (ACG), is more acute and may or may not be accompanied by pain in and around the eye when it occurs (Ang and Ang, 2008; Congdon and Friedman, 2003). During routine eye exams, measurement of intraocular pressure and assessment of visual fields are essential for early detection of glaucoma.

## The iris

The iris is visible through the cornea and is what gives the eye its “color.” All irides have a dark pigmented posterior layer; it is the amount of pigment in the anterior or stromal layer that produces different colors. A “blue” eye results from the selective absorption of long wavelength light by the stroma of the iris and the reflection of short wavelength (blue) light by the posterior pigmented layer. In a “brown” eye almost all visible wavelengths are absorbed by the iris stroma and very little light is left to reflect out of the eye.

The main purpose of the iris, however, is to block excess light from entering the eye and to control the iris aperture or “pupil” for differing amounts of ambient light (Figure 6-2). There are two opposing muscles in the iris; the sphincter muscles that serve to constrict the pupil and the dilator muscles that serve to dilate the pupil. Parasympathetic nerves innervate the sphincter muscles and sympathetic nerves innervate the dilator muscles. It’s because the sympathetic system is heightened relative to the parasympathetic system during “fight or flight” situations that pupils dilate when danger is sensed. Most pupil responses are controlled by a complex set of signals sent through the midbrain (specifically the Edinger-Westphal nucleus) in response to the amount of light striking the retina or as part of the accommodative triad (discussed in Chapter 7, *Visual Function*).

There are very few conditions that affect the iris directly; however, changes in the normal response of the pupil to light or accommodation can result from lesions in the neural pathways or direct trauma to the iris. If the iris does not constrict in response to light, likely the parasympathetic system has been affected by such conditions known as Adie’s tonic pupil or *third nerve palsy*. This lack of constriction may also occur in response to

anticholinergic drugs, such as found in scopolamine patches, or adrenergic drugs, such as found in some eye drops used for “red eye.” If the iris fails to dilate under low light conditions, likely the sympathetic system has been affected by a condition known as *Horner’s syndrome*.

### The crystalline lens and ciliary muscle

Like the cornea, the crystalline lens is a transparent structure. Unlike the cornea, it has the ability to change its shape in order to increase or decrease the amount of refracting power applied to light coming into the eye. Transparency is maintained by the regularity of elongated fiber cells within the lens. These cells originate at the equator of the lens and lay down across the surface of other fiber cells while growing toward the anterior portion of the lens and the posterior portion of the lens until they meet at the central sutures. During elongation they pick up crystallins, hence the name “crystalline lens.” It is these crystallins that give the lens a higher index of refraction than the aqueous and vitreous humors. The gradient index of refraction of the lens ranges from about 1.406 through the center to about 1.386 through the more peripheral portions of the lens (Hecht, 2002). This is due to the fiber cells near the surface having a lower index of refraction than deeper cells, which results in a decrease in spherical aberrations and therefore a more refined quality of focus.

The lens is surrounded by an elastic extracellular matrix known as the “capsule.” The capsule not only provides a smooth optical surface, but it provides an anchor for the suspension of the lens within the eye. A meshwork of nonelastic microfibrils or “zonules” anchor into the capsule near the equator of the lens and, much like a suspension system around a trampoline, connect into the ciliary muscle (Figure 6-2). When the ciliary muscle is relaxed, the tension on the zonules is highest and the lens is “pulled” to its flattest curvature. This generally results in focus for a distant object when the eye is emmetropic (e.g. does not have any refractive errors, such as myopia or hyperopia). When the ciliary muscle contracts, it moves slightly forward, but mostly inward towards the center line of the eye. This releases the tension on the zonules and allows the lens to take up its preferred shape, which is more rounded and thereby more powerful. This increases the focal power of the eye to focus on nearby objects.

Since the lens continues to lay down fiber cells throughout life, it becomes denser and less flexible resulting in a loss of the ability to change focus for near objects with age. This process called *presbyopia* will be covered in a later chapter. A *cataract* is a condition in which the crystalline lens starts to develop opacities or lose its transparency. Cataracts can be associated with environmental factors such as smoking, health conditions such as diabetes, or the use of certain medications such as corticosteroids (Delcourt et al., 2000; Rowe et al., 2000). The effect of cataracts on vision is generally a reduction in contrast sensitivity, an increase in glare and halos at night and some shift in color sensitivity due to the “yellowing” of the lens.

### The Posterior Segment of the Eye

The retina lines the interior of the posterior portion of the globe and is where images are formed. Initial processing of the image occurs at this highly specialized sensory tissue. Vitreous is the clear gel that fills the posterior segment and serves to provide for light transmission through the eye and to protect the retina.

#### The retina

The retina is a mostly transparent thin tissue designed to capture photons of light and initiate processing of the image by the brain. The average thickness of the retina is 250  $\mu\text{m}$  and it consists of 10 layers (Figure 6-4). From the surface of the retina to the back of the eye the layers are the inner limiting membrane, the nerve fiber layer (axons of the ganglion cells), the ganglion cell layer, the inner plexiform layer (synapses between ganglion and bipolar or amacrine cells), the inner nuclear layer (horizontal, bipolar amacrine and interplexiform cells, along with the retina spanning glial cells), the outer plexiform layer (synapses between bipolar, horizontal and photoreceptor cells), the outer nuclear layer (photoreceptor cells), the outer limiting membrane, the receptor layer

(outer and inner segments of the photoreceptor cells) to the retinal pigment epithelium (RPE). The RPE is the outmost layer of the retina and serves as the primary metabolic support for the outer segment of the receptor cells and also acts as the final light sink for incoming photons that reduces intraocular glare. Its light absorbing pigmentation is why the pupil appears black.

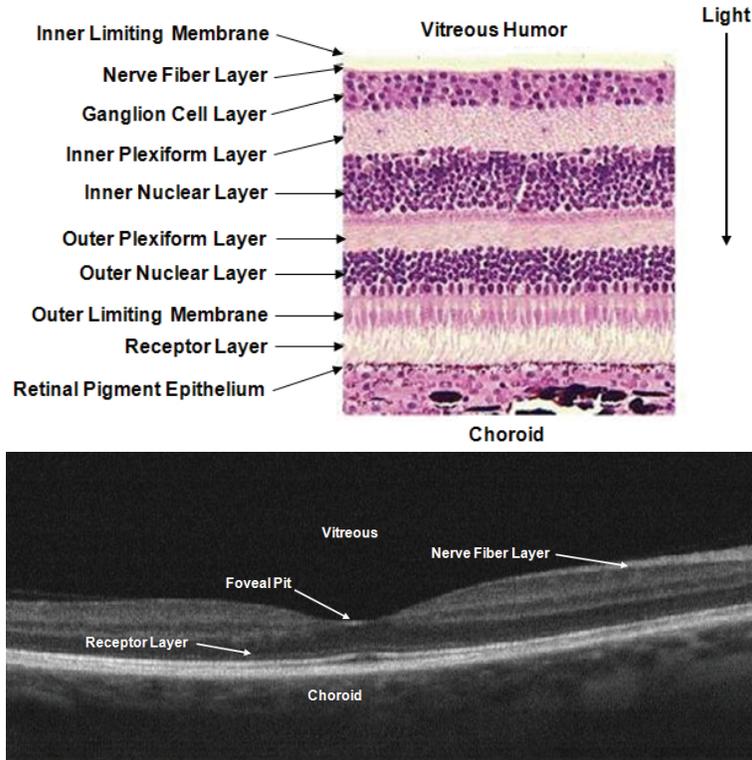


Figure 6-4. Cross-section of the retina (top) (adapted from <http://www.opt.pacificu.edu/ce/catalog/12059-PS/Fig1N.jpg>); actual Optical Coherence Tomography image of a cross-section of the author's retina.

The fact that the receptor layer is deep within the retina means that photons of light actually must pass through most layers of the retina before reaching the receptors. The receptors absorb and convert photons to neural signals, which are then processed through the network of bipolar, horizontal, amacrine and ganglion cells. The output axons of the ganglion cells form the nerve fiber layer that collects at the optic nerve to exit the eye. It's the intricate interconnections of the various neural cells in the retina that complete the first processing of the visual information being sent to the brain.

There are two types of receptors in the receptor layer, rods and cones, essentially named for their shape. The outer segment of the receptor cells contain the light sensitive visual pigment molecules called "opsins" in stacked disks (rods) or invaginations (cones). There are approximately 5 million cones and 92 million rods in the normal adult retina. Cones provide the ability to discern color and the ability to see fine detail and are more concentrated in the central retina. Rods are mainly responsible for peripheral vision, vision under low light conditions and are more prevalent in the mid-peripheral and peripheral retina.

At the most posterior aspect of the retina, where most of the light that the eye receives is focused, is a region called the macula lutea. The macula is an area approximately 5 to 6 mm in diameter which has a greater density of pigments (lutein and zeaxanthine). These pigments help to protect the retinal neural cells against oxidative stress. Within the macular area is the fovea centralis, the small region at the center of the retina where vision is most acute. In this small 1.5 mm (0.06 in) diameter area there are no rods, only cones and the overlying neural layers

are effectively swept away so that there is a depression in the retina. The average thickness of the retina drops to around 185  $\mu\text{m}$  in this “foveal pit.” The area immediately outside the fovea is called the parafoveal region and is where there is a transition from cone-dominated to rod-dominated retina.

The retina receives its nourishment from two sources, the retinal vasculature serves the inner layers of the retina and the choroidal vasculature, which lies between the RPE and the sclera, serves the metabolically active RPE and outer layers of the retina. In order to maximize photon capture in the central retina, the retinal capillary system does not extend in to the fovea centralis, an area known as the foveal avascular zone. This area depends on the blood supply provided by the choriocapillaris.

One of the most common conditions that can affect the retina is age-related macular degeneration (ARMD), in which there is a loss of vision in the center of the visual field (Klein et al., 2004; Nicolas et al., 2003; van Leeuwen et al., 2003). In ARMD, the ability of the retinal pigment epithelium to remove the waste produced by the photoreceptor cells after processing light coming into the eye is reduced. As a result, waste builds up in the form of “drusen.” These drusen further disrupt the metabolic process and eventually the retina starts to deteriorate. If blood vessels from the choriocapillaris break through (“wet ARMD”) the condition can become significantly worse. ARMD is generally hereditary and early signs are detectable through routine eye exams.

### The vitreous humor

The vitreous body is a gel-like structure that fills the posterior portion of the globe. Vitreous humor is comprised of collagen fibrils in a network of hyaluronic acid and is a clear gel (Kaufman and Alm [eds.], 2003). The vitreous body is loosely attached to the retina around the optic nerve head and the macula and more firmly attached to the retina at the ora serrata just posterior to the ciliary body. The connections at the anterior portion of the vitreous body help to keep the anterior and posterior chamber fluids separated. The connections around the optic nerve and macula help to hold the vitreous body against the retina.

With aging, the vitreous starts to liquefy and shrink. When this happens, aqueous from the anterior chamber can get into the posterior chamber of the eye. Additionally, there can be increased tugging at the attachment points on the retina causing a release of cells that the individual sees as “floaters.” If there is significant traction at the attachment points, the retina can be pulled away from the inner globe and a retinal tear or detachment can result.

### The Visual System Pathways to the Brain

The neural signals initially processed by the retina travel via the axons of the ganglion cells through the optic nerves, dividing and partially crossing over into the optic chiasm and then travelling via the optic tracts to the lateral geniculate nucleus (LGN). From the LGN, the signals continue to the primary visual cortex, where further visual processing takes place (Figure 6-5).

### The optic nerves and optic tracts

The optic nerve of each eye consists of a bundle of approximately 1 million retinal ganglion cell axons. The nerve connects to the posterior aspect of the eye in a position that is about 15° nasal to the macula. The connection is referred to as the optic nerve head and is visible when looking into the eye using an ophthalmoscope. The optic nerve head is approximately 1.8 mm (0.07 in) in diameter. Since there are no photoreceptors (rods or cones) overlying the optic nerve head, there is a small blind spot or “scotoma” of approximately 5° in size about 15° temporal to fixation in the visual field of each eye. When both eyes are open, the blind spot of each eye is “filled in” by the visual field of the other eye.

The optic nerves of each eye continue posteriorly and then meet at the optic chiasm. It is here that axons of neurons from the nasal retina (temporal visual field) cross to the opposite or “contralateral” optic tract (e.g. axons

from the right eye temporal visual field cross to the optic tract on the left side of the brain). Axons of neurons from the temporal retina (nasal visual field) continue along the same side or “ipsilateral” optic tract (same side of the brain). This means that visual signals from the right side of the visual field are traveling to the brain via the left optic tract and signals from the left visual field are traveling via the right optic tract. Each optic tract terminates at its LGN.

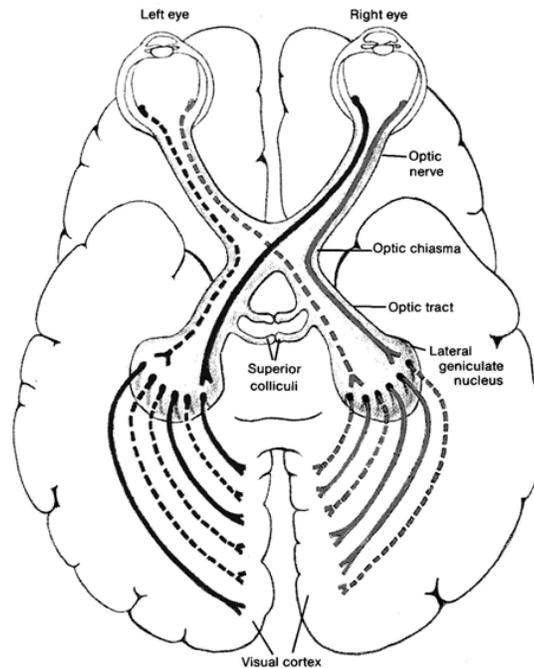


Figure 6.5. Visual system pathway (<http://www.skidmore.edu/~hfoley/images/Brain.top.jpg>).

If a stroke, aneurism or tumor causes damage along the visual pathway, it is often possible to diagnose the exact location of the insult by measuring the visual field. For instance, a pituitary tumor would appear near the optic chiasma and the impact on the visual field would be on the fibers that are crossing to the other side of the brain. Since these fibers are from the nasal retina of each eye, the loss of vision would be in both temporal visual fields or a bitemporal visual field defect (Figure 6.5). Whereas an insult to one of the optic tracts would result in a loss of vision to the opposite or contralateral side of the visual field. For instance, a defect to the right optic tract would cause a loss of the left visual field of both eyes (the temporal visual field of the left eye and the nasal visual field of the right eye).

### The lateral geniculate nucleus (LGN)

The LGN is a paired structure located at the dorsal thalamus. It is here that visual information to the brain, specifically the visual cortex, appears to be regulated and the first stage of coordinating vision from both eyes begins. Each LGN has six layers, three receiving input from the right eye and three receiving input from the left eye. Because of the way the retinal ganglion cell axons are distributed through the chiasm and on to the optic tracts, the information processed in any one layer of the LGN represents specific areas of the visual field for one eye.

Four of the layers are composed of the Parvocellular (small) ganglion cells from the retina that are primarily from the fovea. These cells are most sensitive to color and fine detail. Two of the layers are composed of the

Magnocellular (large) ganglion cells from the retina. These cells are mostly from the perifoveal and more peripheral retina and are largely responsible for the processing of motion.

The LGN then sends forward neurons via the *optic radiations* to the *primary visual cortex*.

## The Visual Cortex

The visual cortex in the occipital lobe of the brain is where the final processing of the neural signals from the retina takes place and “vision” occurs. The occipital lobe is at the most posterior portion of the brain. There are a total of six separate areas in the visual cortex, known as the V1, V2, V3, V3a, V4 and V5.

The *primary visual cortex* or V1 is the first structure in the visual cortex where the neurons from the LGN synapse. In V1, the neural signals are interpreted in terms of visual space, including the form, color and orientation of objects. V1 dedicates most of its area to the interpretation of information from the fovea. This mapping is known as “cortical magnification” and is typical in primates and animals that rely on information from the fovea for survival. The signals then pass through to V2 where color perception occurs and form is further interpreted.

As the neural signals continue further into other areas of the visual cortex, more associative processes take place. In the portions of the visual cortex that make up the *parietal visual cortical areas*, motion of objects, motion of self through the world and spatial reasoning occur. In the *temporal visual cortical areas*, including the *middle temporal* (V5) area, recognition of objects through interpretation of complex forms and patterns occurs. The final psychological and perceptual experience of vision also includes aspects of memory, expectation/prediction and interpolation subserved by other apparently non-visual areas of the brain.

## References

- Ang, L.P., and Ang, L.P. (2008). Current understanding of the treatment and outcome of acute primary angle-closure glaucoma: An Asian perspective. *The Annals, Academy of Medicine, Singapore*, 37(3), 210-215.
- Cavallini, G.M., Lugli, N., Campi, L., Pagliani, L., and Saccarola, P. (2003). Bottle-cork injury to the eye: a review of 13 cases. *European Journal of Epidemiology*, 13(3), 287-291.
- Congdon, N.G., and Friedman, D.S. (2003). Angle-closure glaucoma: impact, etiology, diagnosis, and treatment. *Current Opinion Ophthalmology*, 14(2), 70-73.
- Delcourt, C., Cristol, J.P., Tessier, F., Leger, C.L., Michel, F., and Papoz, L. (2000). Risk factors for cortical, nuclear, and posterior subcapsular cataracts: the POLA study. *Pathologies Oculaires Liees a l'Age. American Journal of Epidemiology*, 151(5), 497-504.
- Friedman, D.S., Wilson, M.R., Liebmann, J.M., Fechtner, R.D., and Weinreb, R.N. (2004). An evidence-based assessment of risk factors for the progression of ocular hypertension and glaucoma. *American Journal of Epidemiology*, 138(3 Suppl), S19-31.
- Hecht, E. (2002). *Optics*. Reading, MA: Addison-Wesley.
- Jones, L.W., and Jones, D.A. (2001). Non-inflammatory corneal complications of contact lens wear. *Contact Lens and Anterior Eye*, 24(2), 73-79.
- Kaufman, P.L., and Alm, A. (Eds.). (2003). *Adler's Physiology of the Eye* (10th ed.). St. Louis: Mosby.
- Kenney, K.S., and Fanciullo, L.M. (2005). Automobile air bags: friend or foe? A case of air bag-associated ocular trauma and a related literature review. *Optometry*, 76(7), 382-386.
- Klein, R., Peto, T., Bird, A., and Vannewkirk, M.R. (2004). The epidemiology of age-related macular degeneration. *American Journal of Epidemiology*, 137(3), 486-495.
- Leske, M.C. (2007). Open-angle glaucoma – an epidemiologic overview. *Ophthalmic Epidemiology*, 14(4), 166-172.
- Liesegang, T.J. (2002). Physiologic changes of the cornea with contact lens wear. *Contact Lens Association of Ophthalmologists Journal*, 28(1), 12-27.

- Morris, D.S., Somner, J.E., Scott, K.M., McCormick, I.J., Aspinall, P., and Dhillon, B. (2007). Corneal thickness at high altitude. *Cornea*, 26(3), 308-311.
- Nicolas, C.M., Robman, L.D., Tikellis, G., Dimitrov, P.N., Dowrick, A., and Guymer, R.H. (2003). Iris colour, ethnic origin and progression of age-related macular degeneration. *Clinical and Experimental Ophthalmology*, 31(6), 465-469.
- Rivera, J.L., Bell, N.P., and Feldman, R.M. (2008). Risk factors for primary open angle glaucoma progression: what we know and what we need to know. *Current Opinion Ophthalmology*, 19(2), 102-106.
- Rowe, N.G., Mitchell, P.G., Cumming, R.G., and Wans, J.J. (2000). Diabetes, fasting blood glucose and age-related cataract: the Blue Mountains Eye Study. *Ophthalmic Epidemiology*, 7(2), 103-114.
- Sihota, R., Sood, N.N., and Agarwal, H.C. (1995). Traumatic glaucoma. *Acta Ophthalmol Scand*, 73(3), 252-254.
- van Leeuwen, R., Klaver, C.C., Vingerling, J.R., Hofman, A., and de Jong, P.T. (2003). Epidemiology of age-related maculopathy: a review. *European Journal of Epidemiology*, 18(9), 845-854.
- Wadhwa, S.D., and Higginbotham, E.J. (2005). Ethnic differences in glaucoma: prevalence, management, and outcome. *Current Opinion Ophthalmology*, 16(2), 101-106.
- Wolff, E. (1933). *Anatomy for Artists*. London: Lewis, H.K.

