Sriwijaya Journal of Ophthalmology
Journal Homepage: https://sriwijayaopthalmology.com/index.php/sjo

Basic Concept of Ophthalmology and Visual Disorder

Liliyanti Fauzi[1*], Tiara Bunga[1]

1 Specialized Residency Training, Department of Ophthalmology, Faculty of Medicine, Universitas Sriwijaya, Palembang, Indonesia

ARTICLE INFO

Keywords:
Visual disorder
Ophthalmology
Impulse
Light receptor
Visual acuity

Corresponding author:
Liliyanti Fauzi

E-mail address:
liliyanti@gmail.com

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/sjo.v5i1.67

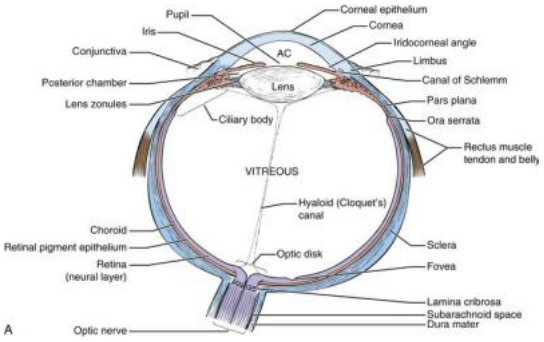
ABSTRACT

The eye is a complex sensory organ that is responsible for vision. Within the protective sheath, each eye has receptors, a lens system for focusing light on receptors, and a nervous system for transmitting impulses from the receptors to the brain. Visual dysfunction can be caused by abnormal eye movements or changes in visual acuity, refraction, color vision, or accommodation. Visual dysfunction may also be a secondary effect of other neurological disorders. This narrative review aims to describe the structure of the eye in general and visual disturbances caused by the aging process and disorders of the protective structure of the eye.

1. Introduction

The external protective structures of the eye include the eyelids (palpebrae), conjunctiva, and apparatus lacrimal. The eyelids are used to control the amount of light that reaches the eye, and the conjunctiva lines the eyelids. Tears released from the lacrimal apparatus moisten the surface of the eye and prevent friction, maintain hydration, and wash away foreign bodies and other irritants.[1,2]

The wall of the eye consists of three layers: the sclera, choroid, and retina (figure 1). The sclera is the thick, white outer layer. It becomes transparent in the cornea, the part of the sclera in the middle anterior region that allows light to enter the eye. The choroid is a highly pigmented middle layer that prevents light from scattering inside the eye. The iris, part of the choroid, has a round opening, the pupil, through which light passes. Smooth muscle fibers control the size of the pupil so that in near and bright light the pupil narrows and in far and dim light the pupil dilates.



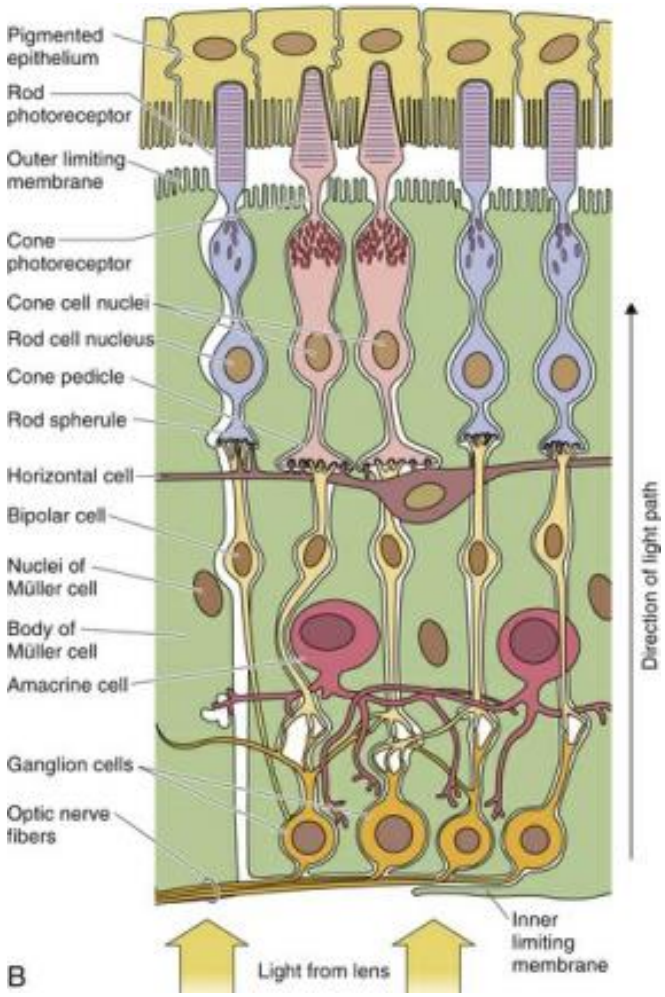


Figure 1. Structure of the eyeball and retinal cell layers. A, Right eye viewed from above (horizontal section). B, Various layers and cells of the retina. AC, anterior chamber.

The retina is the innermost layer of the eye and contains millions of rods and cones, specialized photoreceptors that convert light energy into nerve impulses. Rod Cells mediate peripheral and dim light vision and are most dense at the periphery. The cone cells, densely packed in the center of the retina, are both color receptors and detail receptors. Rod and cone photoreceptors are distributed throughout the retina, except where the optic nerve leaves the eyeball. The lack of rods and cones in this area forms the optic disc or blind spot. Lateral to each optic disc is the macula, a yellow disc that absorbs ultraviolet light. The fovea centralis is a small central area of the macula that contains only the cones and provides the greatest visual acuity.[3,4]

The optic nerve (second cranial nerve) consists of the axons of retinal cells. As shown in Figure 2, nerve impulses pass through the optic nerve after leaving the retina. At the optic chiasm, fibers from the inner (nasal) retina cross to the opposite side, where they join with fibers from the outer (temporal) retina to form the optic tract. The optic tract fibers synapse in the dorsal lateral geniculate nucleus, and from there the geniculocalcarine fibers pass the optic radiation (or geniculocalcarine tract) to the primary visual cortex in the occipital lobe of the brain. Some fibers terminate in the suprachiasmatic nucleus (located above the optic chiasm) and are involved in regulating the sleep-wake cycle.[5]

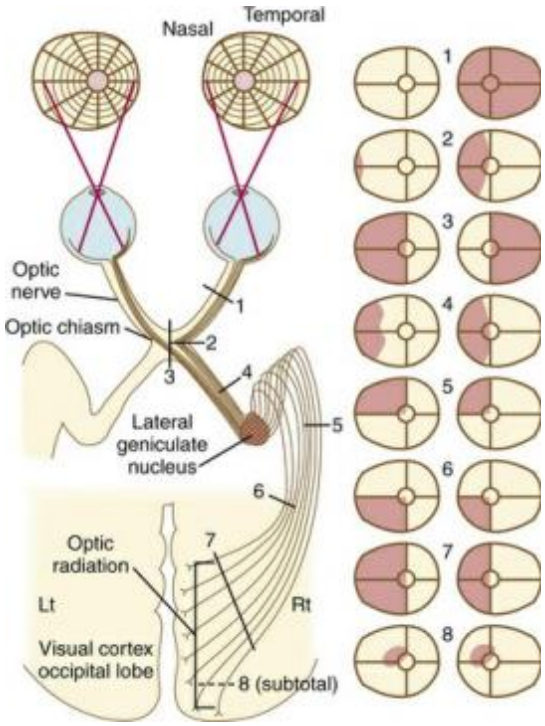


Figure 2. Visual Field and Accompanying Defects in Right Visual Pathway. 1, Optic nerve: blindness. 2, Lateral optic chiasm: highly mismatched homonym, incomplete (contralateral) hemianopsia. 3, Central optic chiasm: bitemporal hemianopsia. 4, Optic tract: inappropriate and incomplete homonymous hemianopsia. 5, Optic radiation temporal loop: homonymous superior partial or complete quadrantanopia (contralateral). 6, Parietalprojection (superior) of optical radiation: congruent partial or complete homonymous inferior quadrantanopia. 7, Complete parietooccipital interruption of optical radiation: homonymous hemianopsia that is as complete as a psychophysical shift of the foveal point, often sparring of central vision and resulting in "macular sparring." 8, Incomplete (subtotal) damage to visual cortex: congruent homonymous scotoma, usually interferes at least acutely with central vision.

Light entering the eye is focused on the retina by the lens—a flexible, biconvex, crystalline structure. In youth, the lens is transparent and has the consistency of a hardened jelly. The flexibility of the lens allows a change in curvature with contraction of the ciliary muscle. This is called accommodation, and it allows the eye to focus on objects at different distances. Anterior to the lens is the iris and aqueous space, which is filled with aqueous humor. Aqueous humor helps maintain pressure inside the eye and provides nutrition to the lens and cornea. Aqueous humor is a free-flowing fluid, secreted by the ciliary processes and reabsorbed into the canal of Schlemm. If the drainage is blocked, the pressure inside the eye increases (as is the case with glaucoma). Behind the lens is the vitreous space, which is filled with a gel-like substance called vitreous humor. The vitreous humor helps prevent the eyeball from collapsing inward.

The central retinal artery supplies blood to the inner retinal surface. Nutrients and oxygen are supplied to the outer surface of the retina by the choroid, the layer of blood vessels that lies between the retina and the sclera. Six extrinsic eye muscles, which are attached to the outer surface of each eye, allow coarse eye movements and allow the eyes to follow moving objects (Figure. 3).[5]

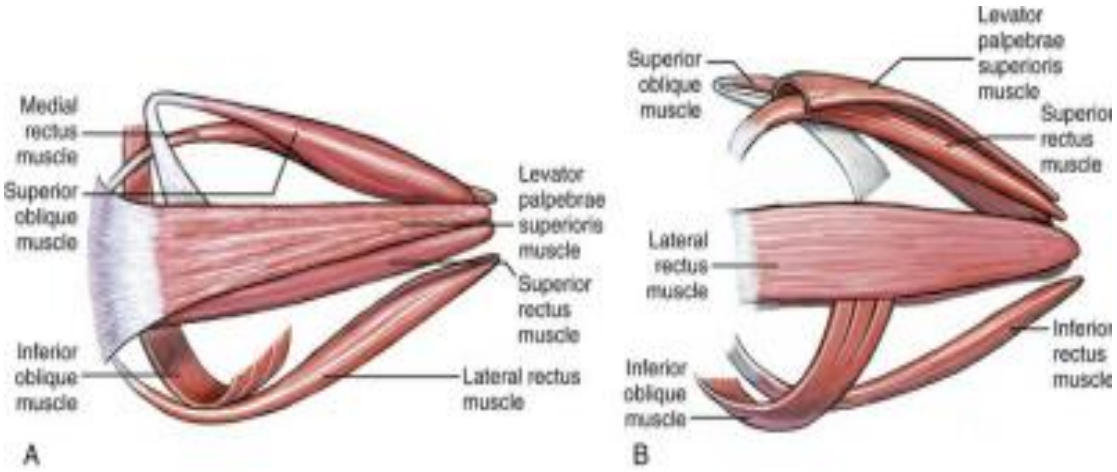


Figure 3 Extrinsic Muscle of the Right Eye. A. Looks superior. B, Looks inferior

Disorders of the supporting structures of the eye

Infection and inflammatory response are the most common conditions affecting the supporting structures of the eye. Blepharitis is inflammation of the eyelids caused by *Staphylococcus* or seborrheic dermatitis. Redness, edema, tearing, and itching are common symptoms. An external hordeolum (anterior blepharitis) is an infection of the sebaceous glands of the eyelid, and an internal hordeolum is an infection of the eyelid margin. A chalazion (posterior blepharitis) is an infectious lipogranuloma of the meibomian and may be associated with an internal hordeolum. This condition presents with redness, swelling, and tenderness and is treated symptomatically. Entropion is a common eyelid malposition in which the eyelid margin turns inward against the eyeball. In ectropion, the eyelids protrude away from the eye. Trichiasis is an abnormally positioned eyelash that grows back toward the eye. There are surgical and non-surgical treatments to reposition the eyelid margins.[6,7]

Conjunctivitis

Conjunctivitis ("pink eye") is inflammation of the conjunctiva (the mucous membrane that covers the front of the eyeball). Conjunctivitis can be caused by bacteria, viruses, allergies, or chemical irritants. The inflammatory response produces photophobia, blurred vision, redness, edema, pain, and lacrimation. Treatment is related to the cause. Acute bacterial conjunctivitis (pink eye**) is** highly contagious and is often caused by gram-positive organisms *(Staphylococcus, Haemophilus, Streptococcus pneumoniae,* and *Moraxella catarrhalis),* although other bacteria may be involved. The onset is acute, characterized by mucopurulent drainage from one or both eyes. In children younger than 6 years, infection *Haemophilus* often causes otitis media (conjunctivitis-otitis syndrome). It is important to prevent the spread of organisms by washing hands thoroughly and using separate towels. The disease is often self-limiting and resolves spontaneously in 10 to 14 days. Antibiotic eye drops are usually effective.[8]

Viral conjunctivitis is caused by adenoviruses. Again, it is contagious with watery symptoms, redness, and photophobia. Some viruses cause conjunctivitis and pharyngitis (pharyngoconjunctival fever), and others cause keratoconjunctivitis. Both diseases are contagious, with watering, redness, and photophobia. Treatment is symptomatic.

Allergic conjunctivitis is associated with various antigens, including pollen. Itching of the eyes is associated with photophobia, burning, and a gritty sensation in the eyes. Treatment is symptomatic and may include antihistamines, low-dose corticosteroids, mast cell stabilizers, and vasoconstrictors.

Chronic conjunctivitis is the result of persistent conjunctivitis. The cause requires identification for

effective treatment. Trachoma (chlamydial conjunctivitis) is caused by *Chlamydia trachomatis.* It is often associated with poor hygiene and is the leading cause of preventable blindness in the world. The severity of the disease varies but can involve inflammation with scarring of the conjunctiva and eyelids leading to distorted eyelashes eroding the cornea, causing corneal scarring and blindness. Chlamydial organisms are sensitive to local or systemic antibiotics. The World Health Organization aims to eliminate trachoma as a public health problem by 2020 using the SAFE Strategy: Surgery for inverted eyelashes, Antibiotics, Facial hygiene, and Environmental improvement.[8]

Keratitis

Keratitis is an inflammation of the cornea that is not contagious or caused by bacteria, viruses, fungi, or amoeba. Bacterial infections often cause corneal ulceration and require intensive antibiotic treatment. *Staphylococcus aureus* is the most common bacterial infection. Herpes simplex virus type 1 can involve the cornea and conjunctiva. Predisposing factors include contact lens use, trauma, and penetrating keratoplasty (corneal graft). *Acanthamoeba is* associated with contact lens wear and is a severe, vision-threatening infection of the cornea. Common symptoms include photophobia, pain, and lacrimation. Severe ulceration with residual scar tissue requires a corneal transplant.[9]

Aging and Vision

Changes in visual and motor components of the eye caused by aging begins at an early age, especially on the eyepiece. Changes caused by aging are summarized in Table 1. Structural changes combined with chronic disease, including dementia and diabetes mellitus, resulting in decreased visual acuity and extraocular motor function.[10]

Table 1. Changes in the eye due to aging

Structure	Changes	Consequence
Cornea	Thicker and less curved; decreased sensitivity to touch Formation of a gray ring at the edge of the cornea (arcus senilis)	Increased astigmatism Does not impair vision
Anterior chamber	Decrease in size and volume caused by thickening of the lens	Sometimes puts pressure on Schlemm's canal and can cause increased intraocular pressure and glaucoma
Lens	increase thickness and opacity (yellowing)	Decreased refraction with increased light scattering and decreased color vision (green and blue); delayed dark adaptation; cataracts
	Loss of elasticity	Loss of accommodation (presbyopia: loss of focus for near objects)
Ciliary muscle	Decreased pupil diameter; atrophic dilatation of the radial muscle	Persistent constriction (senile miosis); decrease in critical blink frequency*
Retina	Reduction in the number of rods in the periphery; loss of stem cells and associated nerve cells	The increased minimum amount of light required to see objects
Macula	Atrophy (age-related macular degeneration)	vision loss
VitreousVitreous	liquefaction and decreased gel volume	Posterior vitreous release causes "floaters;" risk of retinal detachment

*The degree to which successive visual stimuli can be presented and still considered separately.

Visual Dysfunction

Changes in Ocular

Abnormal eye movements occur as a result of dysfunction of the oculomotor, trochlear, or abducens cranial nerves (Table 1). The three types of eye movement disorders are strabismus, nystagmus, and individual extraocular muscle paralysis. Strabismus is the deviation of one eye from the other when one looks at an object; it fails in both eyes to simultaneously focus on the same image and therefore loss of binocular vision. The deviation may be upward, downward, inward, or outward, resulting from a weak or hypertonic muscle in one eye. Strabismus can be caused by neuromuscular disorders of the eye muscles, diseases involving the hemispheres of the brain, or thyroid disease.[11]

The main symptom of strabismus is diplopia (double vision). Strabismus in children requires early intervention to prevent the development of amblyopia (reduced vision in the affected eye without ocular pathology and with full optical correction). Surgery can help children and adults with strabismus.

Nystagmus is an involuntary unilateral or bilateral rhythmic eye movement and can occur in infants (congenital) or adults (acquired). It may be present at rest, or it may occur with eye movement. The two main forms of nystagmus are pendular nystagmus and nystagmus *jerk*. Pendular nystagmus is characterized by regular forward and backward eye movements in which both phases of movement are of equal length. Innystagmus *jerk*, one phase of eye movement is faster than the other. Nystagmus can be caused by an imbalance in the normal coordinated reflex activity of the inner ear, the vestibular nucleus (connects the vestibular nerve to the vestibulospinal tract), the cerebellum, the medial longitudinal fasciculus (connects the mesencephalon with the upper part of the spinal cord), or the nuclei of the oculomotor, trochlear cranial nerves. , and abducens. Medications, retinal disease, and diseases involving the cervical cord can also cause nystagmus.

Infantile nystagmus syndrome has unknown pathogenesis. This syndrome develops in the first 6 months of life and is more common in boys. Ocular motor control and disturbances of the anterior visual pathway are being investigated. One disease-causing gene has been identified, X-linked *FRMD7*. There may be associated strabismus, amblyopia, torticollis, or visual disturbances. Untreated nystagmus can lead to loss of visual acuity.[5,11]

Paralysis of certain extraocular muscles can cause a variety of abnormalities, including limited abduction, abnormal closing of the eyelids, ptosis (drooping eyelids), and diplopia. Abnormalities occur as a result of unopposed muscle activity. Trauma or pressure in the area of the cranial nerves can cause paralysis of certain extraocular muscles. Diseases such as diabetes mellitus and myasthenia gravis can also affect certain extraocular muscles.[5]

Changes in visual acuity

Visual acuity is the ability to see objects in sharp detail. With age, the lens of the eye becomes less flexible and less adjustable, and visual acuity decreases. Visual acuity can also change or decrease for many other reasons. Specific causes of altered visual acuity include: (1) amblyopia, (2) scotoma, (3) cataract, (4) papilledema, (5) dark adaptation, (6) glaucoma, (7) retinal detachment, and (8) macular degeneration.[12]

Amblyopia (lazy eye) is a reduction or dimming of vision associated with changes in the development of the visual cortex and is the most common cause of monocular blindness in childhood. It is not the result of a change in refraction (i.e., the deviation of light rays) or from a change seen in the eye. Amblyopia associated with strabismus, anisometropia (refractive error in one eye differs from the other), ametropia (severe refractive error in both eyes), stimulus deprivation (congenital cataract or orbital lesion); with diseases such as diabetes mellitus, kidney failure, and malaria; and with toxic substances, such as alcohol and tobacco. Early detection is needed to restore vision. Treatment includes patching the

unaffected eye for a long time to ensure a period of use of the affected eye or administering atropine eye drops (blurred vision). Research on brain plasticity with bilateral treatment approaches, including visual perception learning and video games, holds promise for visual recovery in both children and adults. Refractive errors are treated with corrective lenses.[12]

A scotoma is a limited defect of the central field of vision. It can be caused by a central retinal lesion or a sequel to demyelinating optic neuritis, an inflammatory lesion of the optic nerve that is often associated with optico-spinal multiple sclerosis. Age-related macular degeneration is associated with scotoma. Less common causes include compression of one optic nerve by a retro-orbital tumor, neuromyelitis optica (autoantibody-associated inflammation of the optic nerve and spinal cord), pernicious anemia, and toxic or metabolic causes such as methyl alcohol poisoning and tobacco use. The exact mechanism by which this condition causes scotoma is uncertain, but the result is always serious visual acuity impairment.[5]

A cataract is a cloudy or opaque area of the ocular lens and leads to loss of vision when it is on the visual axis. The incidence of cataracts increases with age as the lens enlarges. Cataracts develop due to changes in metabolism and the transport of nutrients within the lens. Although the most common form of cataract is degenerative, it can also occur congenitally or as a result of infection, radiation, trauma, drugs, or diabetes mellitus. Cataracts cause decreased visual acuity, blurred vision, glare, and decreased color perception. Cataracts are treated with removal of the entire lens and replacement with an intraocular artificial lens.

Dark adaptation also affects visual acuity. Low lighting causes impaired visual acuity, especially in older adults. The average 80-year-old needs more than twice as much light as a 20-year-old to see equally well. Changes in the quantity and quality of rhodopsin, a substance found in rods and responsible for low-light vision, are thought to be responsible for reduced dark adaptation in older adults. Vitamin A deficiency can cause the same phenomenon in individuals of all ages.

Glaucoma is the second leading cause of blindness and is characterized by intraocular pressure greater than 12 to 20 mmHg, with the death of retinal ganglion cells and optic nerve axons. Family history is a risk factor, and several glaucoma-related genes have been identified. Types of glaucoma are summarized in Table 2 and Figure 4. Most forms of glaucoma are associated with resistance to the outflow of aqueous humor.

Primary open-angle glaucoma is the most common and is associated with changes in the trabecular meshwork. Chronic increased intraocular pressure causes retinal ganglion death and optic nerve degeneration with loss of peripheral vision, followed by central vision impairment and blindness. Very high pressure can cause blindness within days or hours. Visual acuity loss results from pressure on the optic nerve, which is believed to block the flow of cytoplasm from the nerve bodies in the retina to the peripheral optic nerve fibers that enter the brain. Lack of nutrition, ischemia, oxidative stress associated with mitochondrial failure, inflammatory cytokines, excessive apoptosis, and altered immune mechanisms can lead to the death of the neurons involved. Initially, there were no symptoms. With increasing pressure, acute pain may occur, and there is loss of peripheral vision and progression to blindness. Early detection and treatment prevent optic neuropathy and visual disturbances. Screening programs help in identifying this silent disease. Glaucoma is often treated with eye drops to reduce secretion or increase the absorption of aqueous humor. Surgery may be needed to open the trabecular space and reduce intraocular pressure. Neuroprotective therapy is being evaluated.

Table 2. Types of glaucoma

Types	Pressure enhanced mechanism
Open-angle	Obstruction of aqueous humor outflow in the trabecular meshwork or Schlemm's canal; myopia may be a risk factor
Normal or low tension	Forms of open-angle glaucoma with asymptomatic optic nerve damage and gradual vision loss when intraocular pressure is within the normal range (12-20 mmHg)
Narrow-angle (angle closure)	Forward shift of the iris toward the cornea with narrowing of the angle iridocorneal and outflow obstruction of aqueous humor from the anterior chamber
Acute	angle-closure iridocorneal angle closure with a sudden increase in intraocular pressure, causing pain, redness, and visual disturbances
Chronic angle	closure Progressive and permanent closure of the anterior chamber angle
Secondary	open-angle obstruction or closed due to, for example, uveitis, hemorrhage, lens rupture, or tumor
Congenital glaucoma	Trabecular meshwork malformations and extracellular matrix excess in the outer meshwork

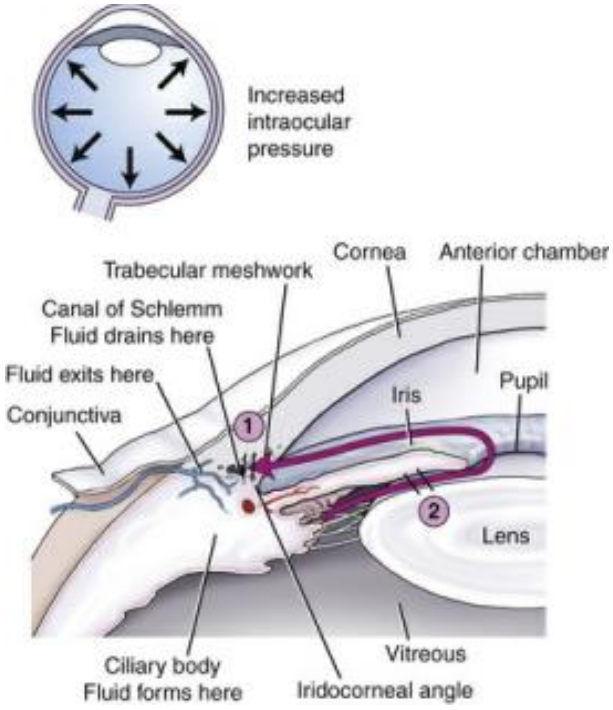


Figure 4. Glaucoma. 1, Open-angle glaucoma. The obstruction to aqueous outflow lies in the trabecular meshwork. 2, Angle-closure glaucoma. The iris presses against the lens, inhibiting aqueous outflow into the anterior chamber and increasing intraocular pressure.

Retinal detachment is a common cause of impaired vision and blindness. Risk factors include retinal holes and vitreoretinal traction. Fluid (exudate, hemorrhage, or vitreous) separates the photoreceptors from the retinal pigment epithelium. The separation deprives the outer retina of oxygen and nutrients as the diffusion distance increases. Communication is also

impaired between the pigment epithelium and photoreceptors. Rhegmatogenous retinal detachment (full-thickness retinal rupture caused by vitreoretinal traction) is the most common form of retinal detachment. Causes include intracapsular cataract extraction, severe myopia, age-related lattice degeneration, vitreoretinal traction, and trauma. Contraction of the fibrous membrane can cause traction separation from the retinal layer as occurs in proliferative diabetic retinopathy. Treatment involves immediate surgical retinal reattachment.

Age-related macular degeneration (AMD) is a severe and irreversible loss of vision and the leading cause of central blindness in older individuals. Risk factors include older age, hypertension, smoking, diabetes mellitus, previous cataract surgery, and a family history of AMD. Degeneration usually occurs after the age of 60 years.

There are two forms: atrophic (dry, nonexudative-geographic atrophy) and neovascular (wet, exudative). The atrophic form is more common and slowly progressive with inflammation and accumulation of lipofuscin (lysosomal pigmented residue) and drusen (a waste product of photoreceptors) in the retina. Symptoms include limited night vision and difficulty reading. Neovascular forms include accumulation of drusen and lipofuscin, abnormal choroidal vessel growth, leakage of blood or serum, retinal detachment, fibrovascular scarring, loss of photoreceptors, and more severe and rapid loss of central vision. Treatment for wet AMD includes injections of antivascular endothelial growth factor (anti-VEGF); New treatments are being investigated. Two carotenoids, lutein, and zeaxanthin, are antioxidants that selectively accumulate in the macula. Lutein and zeaxanthin supplements may improve vision in early dry AMD.[1-3]

Changes in Accommodation

Accommodation is the process by which the thickness of the lens changes with the contraction of the ciliary muscle. Accommodation is required for clear vision and is mediated via the oculomotor nerve (cranial nerve III). Pressure, inflammation, and disease of the oculomotor nerve can alter accommodation. Symptoms include diplopia, blurred vision, and headaches. Loss of accommodation in adults starting at the age of 45 to 50 years is called presbyopia. a condition in which the lens of the eye becomes larger, firmer, and less elastic in response to contraction of the ciliary muscle. The main symptom is reduced near vision, causing the individual to hold reading material at arm's length. Presbyopia correction is accomplished through reading glasses or bifocal lenses, accommodative intraocular lenses, or surgical treatment.[12]

Changes in Refraction

Changes in refraction are the most common visual problem. Refractive errors are caused by irregularities in the curvature of the cornea, the focusing power of the lens, and the length of the eye. The main symptoms of refractive error are blurred vision and headaches. The three types of refractive error are myopia, hyperopia, and astigmatism (Figure. 5).

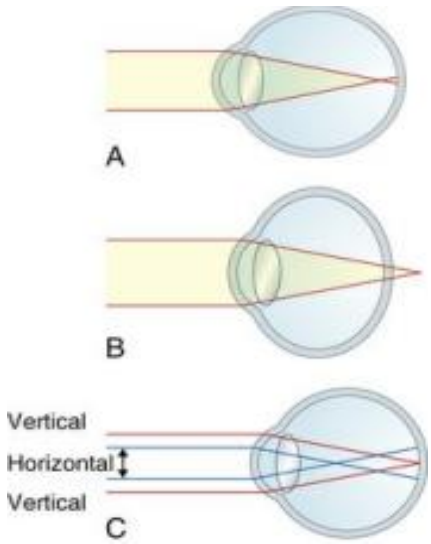


Figure 5. Change of Refraction. A. Myopic eyes. Parallel rays of light are brought into focus in front of the retina. B. Hyperopic eyes. Parallel light rays come to a focus behind the retina in the nonaccommodative eye. C, Simple myopic astigmatism. Vertical light bundles are focused on the retina; Horizontal light is focused in front of the retina and the image is blurred.

In myopia (nearsightedness), the axis of the eyeball is elongated and light rays are focused in front of the retina when a person looks at distant objects, resulting in blurred vision. The cause is unknown. A concave lens is required for correction. Myopia requires frequent eyeglass changes as the eyeball lengthens in childhood. Myopia is a risk factor for retinal detachment, cataract formation, and glaucoma.

In hyperopia (farsightedness), the axis of the eyeball is too short and light rays are focused behind the retina when a person looks at nearby objects. Astigmatism is caused by an unequal curvature of the cornea. In astigmatism, light rays are bent unevenly and do not reach a single focus on the retina. Astigmatism can coexist with myopia, hyperopia, or presbyopia. Hyperopia is corrected with a convex lens or laser refractive surgery.[12]

Changes in color vision

Normal sensitivity to color decreases with age due to the progressive yellowing of the lens that occurs with age. All colors become less intense, although color discrimination for blue and green is most affected. Color vision deteriorates more rapidly for individuals with diabetes mellitus than for the general population. This decline is thought to be an accelerated version of the decline in color vision in the elderly.[3]

Abnormal color vision can also be caused by color blindness, an inherited trait. Color blindness is generally an X-linked recessive characteristic that affects 6% to 8% of the male population and 0.5% of the female population. Although many forms of color blindness exist, most often affected individuals are unable to distinguish red from green. In its most severe form, the individual sees only shades of gray, black, and white.

Neurological disorders causing visual dysfunction

A variety of neurological disorders can cause visual dysfunction. Vision can be impaired at many points along the visual pathway, causing various defects in the field of vision. Visual changes do not always cause disability or blindness throughout the visual field; Hemianopia is a term that describes the vision that is damaged in half of the visual field. (Fig. 2 illustrates the many areas along the visual pathway that may be damaged and associated visual changes.) Due to the anatomy of

the optic nerve, optic nerve injury causes ipsilateral (same side) but contralateral normal (opposite) blindness. side) field of view. Injury to the optic chiasm (X-shaped optic nerve junction), often caused by atherosclerotic ischemia or external compression from trauma or aneurysm, can cause a variety of disabilities, depending on the site of injury. This defect is variable because at the optic chiasm, nerve fibers from the medial half of each retina separate from the lateral half and enter the opposite optic tract.

Due to the normal structure of the visual pathway, damage to one optic tract leads to homonymous hemianopsia (total loss of vision on the inside of one eye and the outside of the other). Thus, if there is an injury to the left optic tract, the individual experiences blindness in the medial (inner) plane of the right eye and the lateral (outer) plane of the left eye. If the compression of the optic tract is asymmetrical, a mismatched (or uneven) homonym will occur. Injury to one optic radiation (ocular pathway in the internal capsule, temporal lobe, or occipital lobe) also causes a homonymous (same plane) defect. Major injury to optic radiation causes homonymous hemianopsia. Minor injuries can cause an upper quadrant homonym defect. Generally, the defect is the same size in both eyes. When homonymous hemianopsia is caused by a lesion of the occipital lobe, the area of hemianopsia is split. Although visual acuity may remain uninterrupted, reading is difficult because of the inability to group words.

Papilledema is edema of the optic nerve at the point of its entry into the eyeball. Papilledema is caused by increased intracranial pressure (e.g. brain tumor, intracranial hemorrhage, hydrocephalus). The subarachnoid space of the brain is continuous with the sheath of the optic nerve. When the cerebrospinal fluid (CSF) pressure increases, the pressure is transmitted to the optic nerve, and the optic nerve sheath compresses the nerve and inhibits axoplasmic transport. This results in the accumulation of axoplasmic substance at the level of the lamina cribrosa (the net-like structure in the sclera where the retinal nerve exits the eye and forms the optic nerve), resulting in characteristic swelling of the optic disc. Removal of the physiologic cup (a bright area usually located in the center of the optic disc) follows. Then the optic disc becomes elevated above the level of the surrounding retina, and its edges become blurred and indistinct. Severe swelling, bleeding, and patches of white exudate (caused by nerve infarction) surround the disc rim. Edematous nerves compress the small retinal veins, causing venous stasis and swelling. Headaches are common, and there may be no visual changes, blurred vision, or narrowing of the visual field.

2. Conclusion

The external protective structures of the eye include the eyelids (palpebrae), the conjunctiva, and the lacrimal apparatus. The wall of the eye consists of three layers: the sclera, choroid, and retina. Infection and inflammatory response are the most common conditions affecting the supporting structures of the eye. Changes in the visual and motor components of the eye caused by aging begin at an early age, especially in the lens of the eye. Structural changes combined with chronic disease, including dementia and diabetes mellitus, result in decreased visual acuity and extraocular motor function.

3. References

1. Sridhar MS. Anatomy of cornea and ocular surface. Indian J Ophthalmol. 2018; 66(2): 190-4.
2. Pekel G, Yagci R, Acer S, Ongun GT, Cetin EN, et al. Comparison of corneal layers and anterior sclera in emmetropic and myopic eyes. Cornea. 2015; 34(7): 786-90.
3. Hoon M, Okawa H, Santina LD, Wong ROL. Functional architecture of the retina: development and disease. Prog Retin Eye Res. 2015; 42: 44-84.
4. Saloblar-Garcia E, Hoz R, Ramirez A, Lopez-Cuenca I, Rojas P, et al. Changes in visual function and retinal structure in the

progression of Alzheimer's disease. PLoS One. 2019; 14(8): e0220535.
5. Furlanetto RL, Teixeira SH, Gracitelli CPB, Lottenberg CL, Emori F, et al. Structural and functional analyses of the optic nerve and lateral geniculate nucleus in glaucoma. PLoS One. 2018; 13(3): e0194038.
6. Putnam CM. Diagnosis and management of blepharitis. Clin Optom (Auckl). 2016; 8: 71-8.
7. Auw-Hädrich C, Reinhard T. Blepharitis component of dry eye syndrome. Ophthalmologe. 2018; 115(2): 93-9.
8. Azari AA, Arabi A. Conjunctivitis: A Systematic Review. J Ophthalmic Vis Res. 2020; 15(3): 372-95.
9. Austin A, Lietman T, Rose-Nussbaumer J. Update on the management of infectious keratitis. Ophthalmology. 2018; 124(11): 1678-89.
10. Grossniklaus HE, Nickerson JM, Edelhauser HF, Bergman LAMK, Berglin L. Anatomic alteration in aging and age-related diseases of the eye. Invest Ophthalmol Vis Sci. 2013; 54(14): ORSF23-7.
11. Hermenean A, Trotta MC, Gharbia S, Hermenean AG, Peteu VE, et al. Changes in retinal structure in the aged mice correlate with differences in the expression of selected retinal miRNAs. Front Pharmacol. 2020; 11: 593514.
12. Marsden J, Stevens S, Ebri A. How to measure distance visual acuity. Community Eye Health. 2014; 27(85): 16.