2 Types of Vision Impairment Following Acquired Brain Injury and How They Affect Occupational Performance

2.1 Visual Acuity

2.1.1 What is Visual Acuity?

Visual acuity is the ability to see visual details and color and provides the ability to clearly see high and low contrast details at near and far distances. It is an important foundation visual function that adds clarity to vision. By delivering high quality details, acuity contributes to the brain's ability to quickly identify objects and facilitates information processing and decision-making.

Acuity results from a multi-step process that begins with the focusing of light onto the retina. Light rays enter the eye through the pupil and are focused onto the retina via anterior eye structures including the cornea and the lens[152] (see Figure. 2.1). Photoreceptor cells in the retina record the basic components of an image that are relayed via the optic nerve and other pathways to the cortex for perceptual processing.[152] Although the concept is simple, the process is complex, involving multiple structures that communicate via complex pathways to create precisely coordinated action sequences. These pathways link the photoreceptor cells with the thalamus, brainstem, cortex, and cerebellum. The key action sequences of focusing include 1) precisely focusing the image onto the retinal photoreceptors through coordination of the anterior eye structures and eye movements 2) ensuring the photoreceptor cells are sufficiently suffused with light via the pupil 3) maintaining the sharpness of focus over a range of distances through eye movement and accommodation. This visual input is then transmitted via the optic nerve and other pathways through posterior cortical areas that attach language to the image and store it in memory.[137, 152] Any compromise of these structures or pathways may cause blurred vision.

2.1.2 Deficits in Visual Acuity

There are many causes of reduced acuity including congenital or acquired conditions; inherited or acquired imperfections in the eye structures; eye diseases that occur early or late in life; conditions that occur due to other diseases, neurological diseases, and eye and brain injuries.[32, 116] Ghannam et al.[85] identified reduced acuity in up to 70% of patients in the early stages of recovery from stroke. Their impaired acuity occurred from various causes including lost, dirty, or broken glasses. Ciuffreda et al.[48] identified 13 ocular conditions that had an elevated risk of occurring with TBI. Visual acuity deficits caused by brain injury generally involve disruption of one of the three action sequences described in the previous section: ability to focus light onto the retina, ability of the photoreceptor cells to accurately capture image and ability of the optic nerve and other pathways to the transmit the information to the rest of the brain for processing.[218] The next three sections describe types of vision impairment that can occur when brain injury disrupts these action sequences.

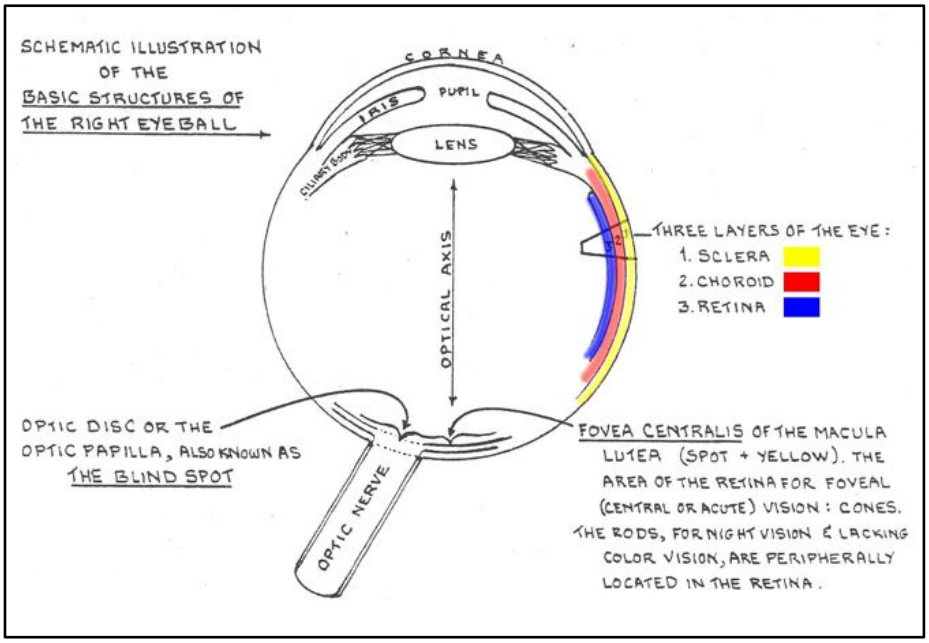

Figure 2.1: Structures of the eyeball. Images pass through the transparent cornea, lens and vitreous to focus on the photoreceptor cells of the retina and then onto the optic nerve. Illustration courtesy of Josephine C. Moore OT, PhD.

2.1.2.1 Disruption of the Ability to Focus an Image onto the Retina

The anterior eye structures must work in tandem with the oculomotor system to focus an image onto the retina long enough for the photoreceptor cells to extract visual details. Sharp focusing of the image onto the retina depends on the integrity of the anterior structures of the eye. The cornea must be smooth and transparent, the iris must be able to quickly contract and relax to change the size of the pupil, and the lens must be transparent and flexible. Light entering the eye passes through four transparent refractive media that help focus the image on the retina: the cornea, aqueous humor, lens, and vitreous humor (see Figure 2.1). Any opacity or irregularity in these structures will prevent light from properly reaching the retinal photoreceptor cells.

Focusing deficiencies from damage to the anterior eye structures and/or the oculomotor system are common following acquired brain injury.[137] Corneal scarring, trauma induced cataract and vitreous hemorrhage are three brain injury related conditions that reduce transparency.[48, 96, 139] Corneal scarring may occur from trauma to the eye incurred during an assault to the head. The damaged cornea forms a scar, creating an irregular surface that refracts the light unevenly and reduces transparency. A forceful blow to the eye can damage the lens and induce the eventual development of a cataract that clouds the lens. Eye trauma can also cause bleeding into the vitreous humor. Blood is opaque and the person experiences floaters, shadows, and darkness as the blood floats in front of the retina. The client's primary complaint from each of these conditions is blurred vision that affects reading and seeing visual

details. Of the three conditions, only vitreous hemorrhage creates a temporary condition that resolves without medical treatment.

The oculomotor system also contributes significantly to acuity by controlling *accommodation*-the ability to zoom in to focus on objects that are close to the face (see section 2.3.1 and illustration 2 in Appendix J).[232] The natural alignment of the anterior eye structures enables effortless focusing when viewing at a distance, but the structures must change and *accommodate* to focus on the short distance required for reading or other activities.[137] Accommodation occurs through a three step process: 1) the eyes *converge* (turn inward) to ensure that the corresponding photoreceptors in the retina are stimulated to capture the image, 2) the lens *thickens* to refract the light rays more strongly and shorten the focal distance, so that the image is focused onto the foveal area of the retina and 3) the pupil *constricts* to reduce scattering of the light rays and sharpen the image. Multiple areas of the brain are involved in coordinating accommodation including the retinal cone photoreceptor cells, the optic nerves, the lateral geniculate nucleus, the occipital lobes, the posterior parietal lobe, the frontal eye fields, the cerebellum, both nuclei of cranial nerve 3 and the oculomotor nerve.[232] Injury to the pathways connecting these areas can cause difficulty achieving and/or sustaining focus during near vision tasks. Accommodative disorders are estimated to occur in nearly half of adult and pediatric clients following traumatic brain injury.[49, 239] Focusing issues are also common in adults experiencing oculomotor impairment from stroke and neurodegenerative diseases like Parkinson's disease and multiple sclerosis.[40, 71] The client's most frequent complaint is difficulty maintaining focus when reading causing the print to blur and sometimes swirl on the page.[96, 121]

2.1.2.2 Disruption of the Ability of the Retina to Process the Image

The retina must be adequately perfused with light to capture high-quality images. Too little or too much light will degrade the image. The pupil regulates the amount of light entering the eye in response to changes in illumination by increasing or decreasing its aperture to ensure optimal perfusion of the photoreceptor cells in the retina.[86] The pupil is controlled through complex pathways involving the eye, brainstem, cortex, and cerebellum.[95, 231] Any condition that affects the responsiveness of the pupil diminishes the client's ability to rapidly adjust to changes in lighting. Difficulty regulating the speed and efficiency of the pupillary response may contribute to the elevated incidence of light sensitivity (photophobia) in clients with traumatic brain injury. Impairment of this reflex also interferes with accommodation as discussed in the previous section.[95, 137]

Retinal photoreceptor cells (see illustration 3 in Appendix J) can also be damaged by injury or disease. Age-related eye diseases (ARED) target the retina and are very common among adults over the age of 80 (see illustration 4 in Appendix J).[116, 251] The three most common ARED are age-related macular degeneration (AMD), diabetic retinopathy (DR) and open angle glaucoma (OAG)-(see section 2.1.2.3 for a discussion of glaucoma). AMD, the most prevalent cause of vision loss in older Americans, does not progress beyond the central visual field. The person never becomes blind, but damage in the central visual field can significantly reduce in visual

acuity. Diabetes can cause several types of vision impairment, the most serious being diabetic retinopathy.[127] Diabetic retinopathy damages the central and peripheral areas of the retina and can cause blindness. Because the incidence of stroke also increases with age, it is not uncommon for an older client with stroke to also experience reduced visual acuity due to ARED. Unless the client discloses that they have an ARED, the vision loss from the eye disease may be overlooked or misdiagnosed as an attentional or cognitive impairment from the stroke. Other causes of retinal damage include stroke of the eye due to an occlusion of the central retinal artery. A central retinal artery occlusion (CRAO) may cause complete or partial blindness in the affected eye that is usually permanent.[145] Partial retinal detachment can occur following trauma to the eye.[139] The person may notice bright flashes of light or light progressing to shadow after the eye injury. Retinal detachment if not immediately treated, will result in permanent visual impairment.

2.1.2.3 Disruption of the Ability of the Optic Nerve to Relay the Retinal Image

Trauma is a common cause of optic nerve damage in clients with TBI. The injury may occur from a direct penetrating injury to the nerve or indirect trauma from forces transmitted during the impact to the brain.[214, 241] In severe closed head injuries stretching or tearing of the optic nerves can occur during the sudden deceleration of the head, usually resulting in bilateral damage to the optic nerves.[128, 241] Bilateral nerve injury can also occur from compression of the nerves due to increased intracranial pressure. Immediate, sudden, and complete loss of vision due to optic nerve trauma is often unresponsive to medical treatment and results in severe visual impairment or blindness.[128]

Glaucoma and multiple sclerosis are common neurodegenerative diseases that can cause optic nerve damage. Glaucoma is a collection of progressive optic nerve diseases that lead to significant vision impairment.[71] Open angle glaucoma (OAG) is one of the three age-related diseases that cause most of the low vision in older adults.[251] Glaucoma typically starts with vision loss in the mid-peripheral visual field and progresses simultaneously outward towards the periphery and inward towards the central field, eventually resulting in blindness if the disease process is not arrested.[137] The central field deficit significantly decreases visual acuity and the ability to see detail and color. Persons are typically unaware of the disease until it has progressed into the central visual field. Persons with Parkinson's Disease appear to have higher rates of open angle glaucoma although the association between these two diseases is not fully understood.[71] Glaucoma can also develop following blunt trauma to the eye, and this is the form most likely to occur following TBI.[154] Persons with multiple sclerosis may experience optic neuritis-an inflammation of the optic nerve that can occur in one or both eyes. The central visual field is usually affected reducing the ability to see details, color and low contrast and the person often experiences light sensitivity.[40, 54] Early episodes of optic neuritis may only cause temporary vision loss, but the condition can eventually lead to permanent vision loss.[54]

2.1.2.4 Uncorrected Refractive Error

One last cause of reduced acuity must be mentioned. *Uncorrected refractive error* (URE) is a commonly occurring condition in American adults that causes a subtle but significant effect on occupational performance. Although some people enjoy perfect visual acuity throughout their lifetimes, many of us are born with or acquire refractive errors that reduce our visual acuity. Refractive errors (see illustration 5 in Appendix J) alter how light rays are focused onto the retina. They result from imperfections in the shape of the cornea or eyeball or from aging of the lens. Common refractive errors include hyperopia (far-sightedness), myopia (nearsightedness), astigmatism (uneven corneal surface), and presbyopia (age-related loss of lens flexibility).[184] Eye doctors are skilled at identifying and treating refractive errors using lenses or surgery. However, despite the availability of eye care, uncorrected vision impairment is a significant public health issue. It has been estimated that over eight million Americans experience unnecessary vision impairment URE.[247] Older adults make up the largest number of Americans with correctable vision impairment. Medicare covers the cost of an annual eye exam, but many older adults never receive this exam due to health reasons or limited access to eye doctors.[72, 167] Other older adults cannot afford to update their eyeglasses prescription as Medicare does not cover the cost of eyeglasses. These barriers are more likely to affect older adults residing in nursing homes and assisted living facilities where it is estimated that nearly a third of vision impairment can be corrected with glasses or surgery.[72, 167] Owsley et al.[167] found that residents in nursing homes with URE reported increased psychological distress, (e.g., worry, frustration, anxiousness) and depression. A subsequent RTC completed on the study participants showed that residents who received updated eyeglasses reported a decrease in psychological distress, an increase in social participation and fewer depressive symptoms.[168]

2.1.3 Occupational Limitations Caused by Reduced Visual Acuity

Blurred vision is the most common acuity complaint of persons with ABI and difficulty reading is their most common functional complaint.[32, 95, 196] Reading is a key component of many important I-ADLs. People need to read to shop in a store, eat in a restaurant, pay bills, drive a car, participate in a religious service, dial a cell phone, read a text, read a clock face, cash a check, wash a new garment, and measure weight, blood pressure and glucose level. Poor acuity causes more difficulty completing I-ADLS because they are more dependent on good acuity.[19] With some effort, a blindfolded person can complete all basic ADLs including dressing, bathing, toileting, feeding, and oral hygiene, but that same person would not be able to read a bank statement, accurately measure ingredients to bake a cake, or write a letter to a friend. Difficulty seeing small visual details and color also impairs fine motor coordination which can affect occupations such as meal preparation, medication management, financial management, grooming and shopping. Poor acuity may also impair the ability to locate landmarks and obstacles quickly enough to adapt to a dynamic environment limiting participation in community activities such as driving, shopping, and attending social events.

2.2 Contrast Sensitivity

2.2.1 What is Contrast Sensitivity?

Contrast sensitivity-also called low contrast acuity-is the ability to reliably distinguish the borders of objects as they degrade in contrast from their backgrounds.[166] Because much of the visual environment is made up of objects with low contrast borders and features, it could be argued that reduced contrast sensitivity contributes more to our ability to perceive and visually adapt to the environment than visual acuity. Common examples of low contrast features include unpainted curbs, curb cuts and steps; glass doors, water spilled on the floor or rising in a sink or bathtub, and facial features. Sub-optimal lighting conditions also reduce the contrast of objects in the environment.[116] Normal aging causes a progressive decline in contrast sensitivity largely due to optical changes in the eye.[116] A large longitudinal study on older adults without eye disease found that a 70-year-old adult required twice as much contrast as a 20-year-old to identify a faint object/feature and a 90-year-old needed 6 times as much contrast to identify the same object.[31] ARED generally causes a significant reduction in contrast sensitivity adding to the client's difficulty completing ADLs and increasing the risk for falls.[114 ,166]

2.2.2 Deficits in Contrast Sensitivity

Reduced contrast acuity can occur with stroke and TBI.[35, 85] Ciuffreda et al.[48] reported that approximately 20% of persons with mild TBI experience reduced contrast sensitivity. Bulens et al.[35] reported that nearly two thirds of participants with stroke affecting the posterior visual pathways demonstrated reduced contrast sensitivity. Reduced contrast acuity from cerebral lesions is thought to be due to destruction of neurons in the cortical areas that process visual information along with a decreased sensitivity in the surviving neurons due to disruption of neuronal interaction.[28] Decreased contrast sensitivity also occurs with neurodegenerative diseases including Alzheimer's Dementia, Parkinson's Disease, and multiple sclerosis.[54, 116, 132, 166, 221]

2.2.3 Occupational Limitations Caused by Reduced Contrast Sensitivity

Reduced contrast sensitivity is more likely to occur following stroke and TBI than reduced high contrast visual acuity. It can also occur without a change in high contrast acuity, and often occurs in conjunction with visual field deficits (hemianopia and macular scotoma).[35, 66, 125] When present, it interferes with the ability to complete a diverse number of daily occupations including reading, recognizing faces, locating objects, driving, navigating safely through environments, and using tools.[66, 166, 271] Persons with reduced contrast acuity also have difficulty completing activities in suboptimal lighting conditions and significantly benefit from enhanced illumination.[116, 125]

2.3 Oculomotor Control

2.3.1 What is Oculomotor Control?

Oculomotor control entails the ability to move the eyes together to provide a clear single image to the brain. The sole job of eye movement is to place an object of interest onto the fovea and maintain fixation as long as needed to accomplish the desired goal.[48, 137] This process is known as foveation and the need to foveate objects drives the oculomotor system.[137] This is a daunting task because human beings are mobile creatures who interact with dynamic environments. An image focused on the fovea is always in danger of slipping off when the head or target moves. Eye movements keep the target stabilized on the fovea during fixation, gaze shift, and head movement. Several oculomotor systems interact to provide this control including the vestibular-ocular-cervical responses, the optokinetic system, the saccadic system, the smooth pursuit system, and the vergence system (see illustration 7 in Appendix J).[137]

- The *vestibular-ocular-cervical responses* hold images of the seen world steady on the fovea during *transient* movement of the head and body. Even when completely stationary, we experience significant involuntary head movement from varied sources. Examples include smaller vibrations transmitted from the heartbeat, the support surface, and minute postural sway, and larger forceful head movements during activities like walking, running, or riding in a car. To counter this threat, the brain combines input from the vestibular, cervical, and ocular systems to produce compensatory eye movements. These eye movements occur automatically in response to head movement to keep the image on the fovea. The vestibular ocular reflex (VOR) is the primary reflex used to stabilize gaze during transient head movement supplemented by the cervical ocular reflex (COR).[137]
- The *optokinetic system* augments the vestibular-cervical-ocular responses to maintain foveal stabilization during *prolonged* head movement. Head movement lasting longer than 30 seconds results in adaptation of the VOR preventing it from eliciting compensatory eye movements. The optokinetic reflex relies on a continuous (e.g., tonic) signal from the retina rather than a short term (e.g., phasic) signal from the inner ear labyrinth to detect head movement and initiate compensatory eye movements. This ability enables the optokinetic reflex to take over the function of the VOR during sustained head movement.[137]

- The *smooth pursuit system* ensures that the target stays on the fovea during fixation and when the target is moving.[137] When the eyes and the target both are stationary, the smooth pursuit system supplies continuous small eye movements that move back and forth over the boundaries of the target to ensure that the image stays fresh on the retina and does not fade. If the viewer remains stationary as the target moves away, the target will eventually slip off the fovea and the image will blur. To prevent this, the smooth pursuit system initiates eye movement in the direction of the moving target. The eye movement increases in velocity until it matches the speed of the target, ensuring that the image stays focused on the fovea.

- The *saccadic system* directs the fovea towards an object of interest. Saccadic eye movements rapidly redirect attention to new objects and keep the brain informed of all possible threats and resources within the field of view.[137] Saccades are initiated under *involuntary control* via the brainstem when a possible threat appears in the environment and *voluntary control* via the prefrontal cortex to purposely search for a desired object. The eyes and head usually move together when searching the environment. We use a saccadic eye movement to initiate search but quickly supplement with smooth pursuit and head movement if the target lies in our periphery.[80, 137] The field of vision we can perceive when using head and eye movement together is wider than that achieved by eye movement alone. Therefore, our ability to completely scan our surroundings, depends more on our gaze stability and full neck range of motion than the ability to move each eye through its full range of motion.

- The vergence system helps to maintain foveation as objects move close to and away from the body. *Convergence* (moving the eyes inward towards the nose) occurs in conjunction with pupil constriction and thickening of the lens to produce accommodation (see section 2.1.2.1 and illustration 6 in Appendix J). During accommodation, convergence keeps the image aligned on corresponding photoreceptors in the two eyes to ensure that the person continues to clearly see only one image (known as sensory fusion) as the object moves closer. When the object moves away from the body, divergence (moving the eyes back to primary gaze) occurs to ensure that only one image continues to be seen.[137]

The parietal, temporal, occipital and prefrontal lobes, plus the thalamus, cerebellum, and brainstem form a complex network to ensure that eye movements are able to attain and maintain foveation.[137, 232] These divergent brain areas exert their control over eye movement using the same motor pathway comprised of three pairs of cranial nerves (CN): CN 3-oculomotor nerve; CN 4-trochlear nerve and CN 6-abducens nerve. Altogether these cranial nerves innervate seven pairs of striated extraocular muscles that move the eye and the internal eye muscles (iris and ciliary muscles) that control pupil size and the shape of the lens.[137] The muscles of the two eyes must work in tandem to produce the precise coordinated movements needed for binocular vision (see illustration 7 and 8 in Appendix J). The extensiveness and complexity of the pathways controlling eye movements makes them vulnerable to damage from many types of brain injury.[137, 233]

2.3.2 Deficits in Oculomotor Control

The prevalence of eye movement disorders from acquired brain injury ranges from 50% to 90% in studies.[21] Stroke and TBI cause the highest percentage of oculomotor impairment: up to 90% of persons with TBI and 87% of adults with stroke.[49] Most impairment occurs from damage to pathways and structures within the cortex, brainstem, and cerebellum. Accommodative disorders and convergence insufficiency are the most reported oculomotor conditions in persons with TBI.[48] Cranial nerve lesions and convergence insufficiency are the most reported

oculomotor conditions in persons with stroke. Oculomotor impairment is also common in persons with Parkinson's Disease, multiple sclerosis, and Alzheimer's dementia.[137]

2.3.2.1 Oculomotor Impairment from Paralytic Strabismus

Cranial nerve lesions account for approximately 20% of oculomotor impairment from TBI or stroke.[197, 241] Damage to one of cranial nerves 3,4,6 can weaken or paralyze the extraocular muscle(s) that it innervates, and cause *acquired paralytic strabismus*.[137, 201, 202] *Strabismus* is a visual condition where the eyes do not align with one another because of muscle imbalance. *Paralytic* means that the muscle imbalance is due to paralysis of one or more of the extraocular muscles. *Acquired* means that the person was not born with this condition but acquired it from injury or disease. Restricted movement of one or more extraocular muscles causes a misalignment of the eyes as the person focuses on an object. The client has difficulty fusing the two images from the eyes into a single image and may complain of a double image that splits vertically or laterally, a ghosting image (like poor TV reception), a blurred image, or even a crooked or distorted image. The diplopia increases in the gaze direction controlled by the paralytic muscle and decreases in gaze directions away from the action of the paralytic muscle (called incomitant strabismus).[137] To minimize the diplopia, the adult client often assumes a head position that avoids the action of the paralyzed muscle. If a single image can't be attained, the client may use a head position that either allows the nose to occlude the second image or increases the disparity between the images to make it easier to identify the false image.

The client's performance limitations depend on whether the diplopia is constant or intermittent and whether it occurs at near or far distances. Milder CN injury may weaken but not paralyze the extraocular muscles. The affected muscle can still help move the eye so that the person can maintain sensory fusion when concentrating on a task. However, the weakened muscle often fatigues quickly causing the client to complain of intermittent diplopia, eye strain, headache, and poor concentration. More severe CN lesions cause significant or complete paralysis of the eye muscles resulting in constant diplopia and often a noticeable eye turn of the involved eye (see Illustration 8 in Appendix J). Eye doctors use specific medical terms to describe the extent of the strabismus. *Phoria* describes milder paralysis where the brain's need to maintain sensory fusion keeps the affected eye aligned with the other eye when focusing on an object.[202] *Tropia* describes moderate to severe paralysis where there is a noticeable deviation of the involved eye when focusing.[202] These terms are combined with four prefixes that describe the direction of the deviation: *eso*-the eye turns inward towards the nose; *exo*-the eye turns outward towards the temple; *hypo*-the eye turns downward; and *hyper*-the eye turns upward. For example, *esotropia* indicates a constant inward deviation of the eye during focus (e.g., a crossed eye) whereas *esophoria* indicates inward turning of the eye only when the eye muscle becomes fatigued.[202]

2.3.2.2 Oculomotor Impairment from Traumatic Brain Injury

Most oculomotor impairment results from damage to neural centers that coordinate eye movements.[9] TBI is a common cause of such injuries.[9, 49, 137] The damage occurs from shock

waves transmitted through the brainstem and cerebrum during the TBI. The shock waves cause diffuse axonal damage to the pathways connecting the brain areas controlling eye movements and disrupt communication between these areas [9] Even mild head trauma (concussion) can cause pathway damage.[9, 49] Co-impairments may accompany TBI. They result from pathway damage sustained during the TBI and commonly include light sensitivity, post-traumatic headache, fatigue, and sleep disturbance.[6] Accommodative disorders-specifically convergence insufficiency-have been found to intermix with the other co-impairments from the TBI.[6, 153] Some co-impairments like light sensitivity tend to persist and may still be present up to two years following injury[146] (see section 2.3.3.3). Moderate to severe head injury often causes a combination of pathway and structural damage from focal lesions.[241] Damage to CN 3,4,6 for example can occur from focal lesions, tearing, contusion and compression from the head injury.[137, 241]

Oculomotor impairment can also occur from eye trauma sustained during the TBI. A penetrating eye injury can lacerate one of the extraocular muscles. A blow to the eye can also cause an orbital blow-out fracture. This injury occurs when the thin skeletal structure of the eye socket is fractured; it can entrap the inferior rectus and oblique muscles, restricting ocular motility. The client may experience enophthalmos, vertical diplopia and ptosis along with secondary complications from choroidal rupture, retinal hemorrhage, and glaucoma.[137, 154, 241]

TBI is also a frequent cause of visuo-vestibular dysfunction that causes inability to stabilize gaze during movement of the body and/or environment. Impairment can occur from damage to peripheral vestibular structures or central vestibular pathways.[137] Peripheral injuries (labyrinth of the inner ear; CN 8) can occur from displacement of inner ear structures due to rapid acceleration/deceleration of the head during the TBI, or from a skull fracture involving the temporal or petrous bones.[106, 137] Central vestibular injuries can occur from trauma to the brainstem or cerebellum. Oscillopsia, the perception of constant swirling and movement of the peripheral environment, is a common complaint among persons with visuo-vestibular dysfunction.[137, 236] The illusion of movement occurs from excessive slipping of visual images on the retina due to inadequate vestibular, cervical, and ocular responses during head movement.[137] Because visual stability is necessary for postural control, clients with oscillopsia and visuo-vestibular dysfunction often have impaired balance and postural control.[49, 54]

2.3.2.3 Oculomotor Impairment from Stroke

Stroke also causes oculomotor impairment.[49, 137, 197] Ciuffreda et al.[49] reported that 86.7% of participants with stroke demonstrated some type of oculomotor impairment-most commonly: strabismus, cranial nerve (CN) injury and convergence insufficiency. Rowe et al.[197] found that 54% of participants in large study of stroke survivors had oculomotor impairment. Within this group-18% had cranial nerve lesions involving CN 3,4,6. Vascular disease including diabetes, hypertension, and atherosclerosis can cause also cause discrete lesions of individual cranial nerves.[137]

2.3.2.4 Oculomotor Impairment from Neurodegenerative Diseases

Persons with Parkinson's Disease often experience convergence insufficiency early in the course of the disease and develop diplopia and difficulty moving the eyes as the disease progresses.[137, 207, 269] Eye movement disorders are also common in persons with multiple sclerosis (MS) due to the inflammatory demyelinating nature of the disease.[54] Persons with MS may experience blurred vision, diplopia, nystagmus and oscillopsia.[54, 137] They can also develop a perplexing condition-*internuclear ophthalmoplegia (INO)*-from a lesion along the medial longitudinal fasciculus in the brainstem.[137] INO causes an inability to voluntarily adduct the eye on the affected side during horizontal gaze that is accompanied by nystagmus with abduction of the other eye. However, the person can adduct the eye when converging during accommodation. Persons with Alzheimer's dementia can also experience difficulty executing and controlling saccades due to changes in attention.[269]

2.3.3 Occupational Limitations Caused by Impaired Oculomotor Function

2.3.3.1 Convergence Insufficiency

Studies show that nearly half of all adults referred for visual assessment post brain injury complain of difficulties related to accommodation and focusing.[9, 96, 234] The most common focusing disorder is convergence insufficiency.[49, 96, 234] Persons with this condition have difficulty achieving or sustaining adequate focus during near vision tasks. The client often complains of fatigue, eye pain, or headache after a period of sustained viewing of near tasks especially reading.[32, 137, 191] Eye muscle fatigue from the exertion of sustaining convergence during reading breaks down sensory fusion and the client may experience odd visual phenomena such as the print swirling and moving on the page or the page going blank.[121] Because most persons with convergence insufficiency have normal cranial nerve function, their reading complaints may be mis-interpreted as perceptual impairment, inattention, or lack of effort, instead of oculomotor impairment.

2.3.3.2 Diplopia

Diplopia creates perceptual distortion that may significantly affect eye-hand coordination, postural control, and binocular use of the eyes. The client's functional limitations depend on whether the diplopia occurs at near or far focal distances. Diplopia that occurs at near distances (from 3rd or 4th CN lesion) can disrupt reading and activities that require eye hand coordination such as pouring liquids, writing, and grooming. Diplopia that occurs when viewing at a distance (from 6th or 4th CN lesion) can affect walking, driving, television viewing, and sports like golf and tennis. Persons with severe paralytic strabismus often assume an altered head position to maintain sensory fusion.[202] For example, a client with a left lateral rectus palsy (CN 6 lesion) may turn the head towards the left to avoid the need to abduct the eye; a client with paralysis of the right superior oblique muscle (CN 4 lesion) may tilt the head to the right to avoid the downward action of that muscle.[202] Without careful assessment of oculomotor function, there

is a risk that altered head position is mistakenly attributed to instability of the neck or trunk rather than a functional adaptation used to achieve single vision.

2.3.3.3 Influence on Participation

Oculomotor impairment influences the client's willingness to participate in occupations. Its presence typically doesn't prevent the client from independently completing an occupation, but even mild oculomotor impairment makes it harder to use vision to complete occupations. The client must put more effort into focusing and moving the eyes which can increase visual stress, slow visual processing, and cause fatigue. The added stress and effort combined with co-impairments like light sensitivity can trigger post traumatic headache and make it difficult to participate long enough to complete activities. To reduce the stress, the client may begin to avoid environments and activities that place significant demand on the visual system. Reading is particularly stressful because it requires sustained focusing and attention and which is why difficulty reading is the most common complaint reported by persons with oculomotor impairment.[232] Activities in dynamic environments with lots of pattern and movement-such as driving or navigating community environments are also stressful.[96]

2.4 Visual Fields

2.4.1 What is the Visual Field?

The visual field is the area of the visual world that you can see when looking straight ahead. It reflects the functioning of the retinal photoreceptor cells and is analogous to the dimensions of a picture imprinted on the film in a camera (with the retina representing the film). The normal visual field extends approximately 60 degrees superiorly, 75 degrees inferiorly, 60 degrees to the nasal side and 100 degrees to the temporal side.[220] At the very center of the retinal visual field is the fovea-an area approximately 8 degrees in diameter and completely packed with cone photoreceptors.[220] We use the fovea to capture the visual details needed to identify objects. The fovea is embedded in the macula, and together they comprise the central visual field which is approximately 30 degrees in diameter.[220] The macula contains a mixture of mostly cone and some rod photoreceptor cells; it also contributes to object identification.[152] The peripheral field makes up the rest of the visual field and contains only rod photoreceptor cells that detect general shapes and movement in the environment. The peripheral visual field provides the background vision needed to orient within the environment. The blind spot lies on the border between the central and peripheral visual field on the temporal side of the field. The optic disc is located here and there are no photoreceptor cells in this area. Figure 2.2 illustrates the divisions and dimensions of the visual fields. Most of the visual field is binocular and is seen by both eyes. A small portion of the peripheral temporal field in each eye-known as the lateral or monocular crescent-can be seen only by one eye because vision in the other eye is blocked by the bridge of the nose.[152] When viewing Figure 2.2-note that field diagrams are drawn "as the client sees it" so that the field diagram on the right represents the client's right eye and vice versa.

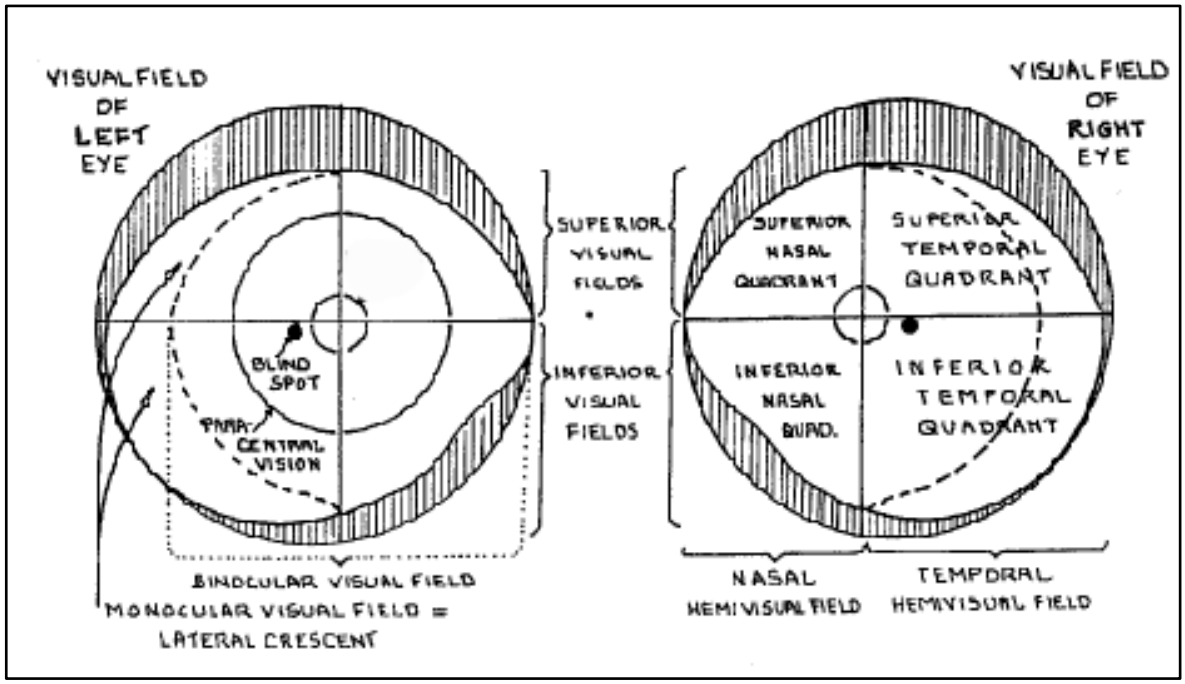

Figure 2.2: Visual field diagram illustrating the divisions of the visual fields as the person see them. Illustration courtesy of Josephine C. Moore OT, PhD.

The visual field is often depicted as a “hill of vision” (see Figure 2.3-next page) to describe its relationship to visual acuity.[180] The fovea lies at the peak of the hill. The fovea is the visual field area with the greatest acuity. The cone receptor cells within this area of the field can perceive even small and dimly lit targets. Visual acuity incrementally decreases as the field expands away from the fovea towards the peripheral visual field. The peripheral visual field is capable only of perceiving larger and brighter targets. The downward sloping of the hill represents this progressive decrease in visual acuity.

To successfully complete daily occupations, you must be able to keep track of the key objects in the environment that can provide either assistance or harm. For example, to successfully drive a car to a destination without an accident, you must see the vehicles directly ahead of you as well as those moving on the side. You must also see informational signs overhead and on the sides of the road along with pedestrians, bicyclists, animals, and other objects that may enter the path of your car. The visual field provides this panoramic picture. We use our visual field not only to see the “big picture” but also to see the details of objects. In reading, for example, the foveal field enables us to clearly see approximately 9 characters with each fixation.[182] This field information is enough to enable us to identify most English words quickly and accurately during a single fixation and to fluently read with good speed and comprehension.[182]

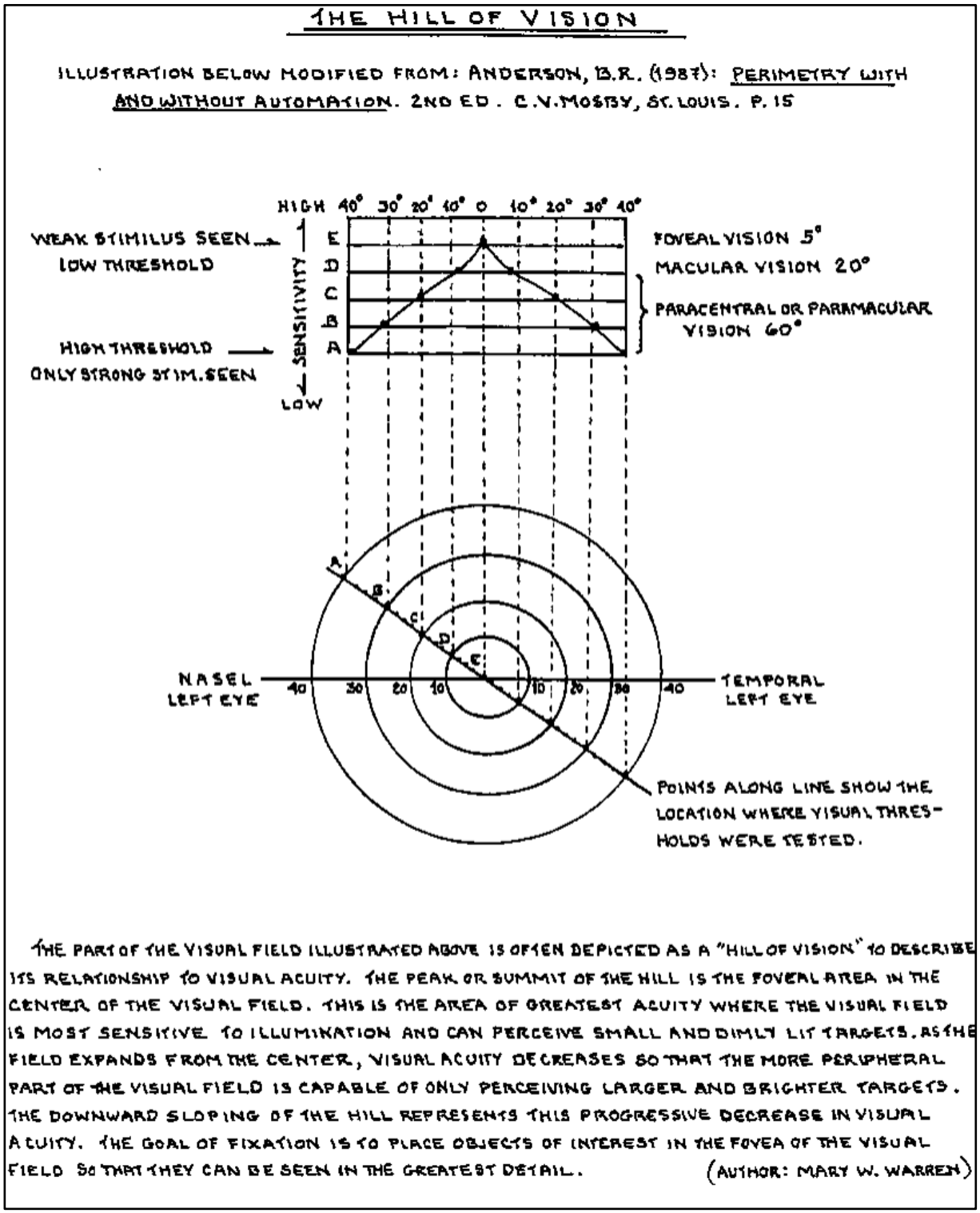

Figure 2.3: The “hill of vision” depicting the relationship between visual acuity and visual field. Illustration courtesy of Josephine C. Moore OT, PhD.

2.4.2 Deficits in the Visual Field from Acquired Brain Injury

Visual field deficits (VFD) occur from injury to the retinal photoreceptor cells or to the optic pathway that relays retinal information to the cortex.[86] Figure 2.4 illustrates this visual pathway as it transitions from the optic nerve to the optic tract to the geniculocalcarine tract (GCT). This pathway transects the entire brain from the eyes to the back of the head and its length makes it vulnerable to brain injury from stroke, TBI, tumor and neurodegenerative diseases including MS and Alzheimer dementia.[54, 86, 147] The location and type of the visual field deficit depends on where damage occurs along the pathway. Although any type of visual field deficit is possible following brain injury, homonymous hemianopia (HH) is the most identified deficit, occurring in approximately two thirds of persons with visual field deficit from stroke[195] and a quarter of persons with TBI.[34] Hemianopia (hemi = half; anopsia = blindness) means that there has been a loss of vision in one half of the visual field in the eye. Damage to the visual pathway posterior to (e.g., behind) the optic chiasm always causes a visual field deficit in both eyes. The term homonymous is used to indicate that the deficit is the same in both eyes.

Because of the crossing of optic nerve fibers at the optic chiasm, the geniculocalcarine tract (GCT) in each hemisphere carries information from one half of the visual field in each eye. The GCT in the right hemisphere carries a representation of the left half of the visual field in each eye and the GCT in the left hemisphere carries a representation of the right half of the visual field from each eye. A lesion occurring posterior to the chiasm within the right hemisphere causes a left homonymous hemianopia; the same lesion in the left hemisphere causes a right homonymous hemianopia.[86]

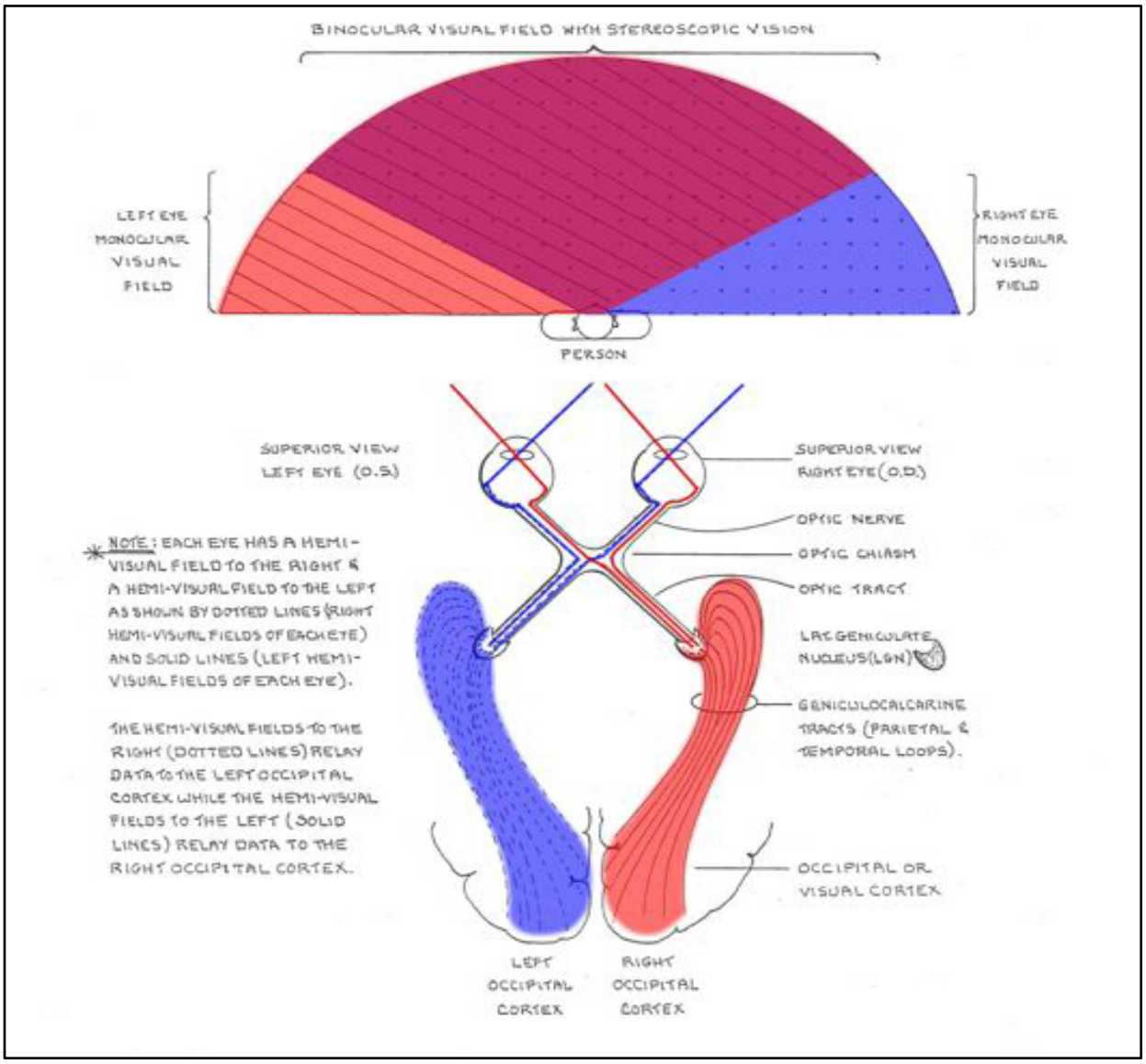

Figure 2.4: The visual field pathway. Illustration Courtesy of Josephine C. Moore OT, PhD. B**lue** shaded areas show pathways conveying visual field information captured in the retina of the **left eye**. R**ed** shaded areas show pathways conveying visual field information captured in the retina of the **right eye**. Note: nasal fibers of the optic nerve of each eye **cross over** at the **optic chiasm** to join the temporal fibers of the optic nerve of the other eye to create the **optic tract**. The optic tract now carries information from the left or right side of the visual field in each eye. The optic tract continues onto the **lateral geniculate nucleus** (LGN) in the thalamus. From the LGN the pathway continues onto the occipital cortex via geniculocalcarine tracts in the right and left hemispheres. The top of the diagram shows how the visual fields of each eye overlap to provide a binocular visual field in the center of a person's vision. The far ends of the field retain their individual color to show how the far peripheral field can only be seen by one eye as the nose occludes the other eye's view.

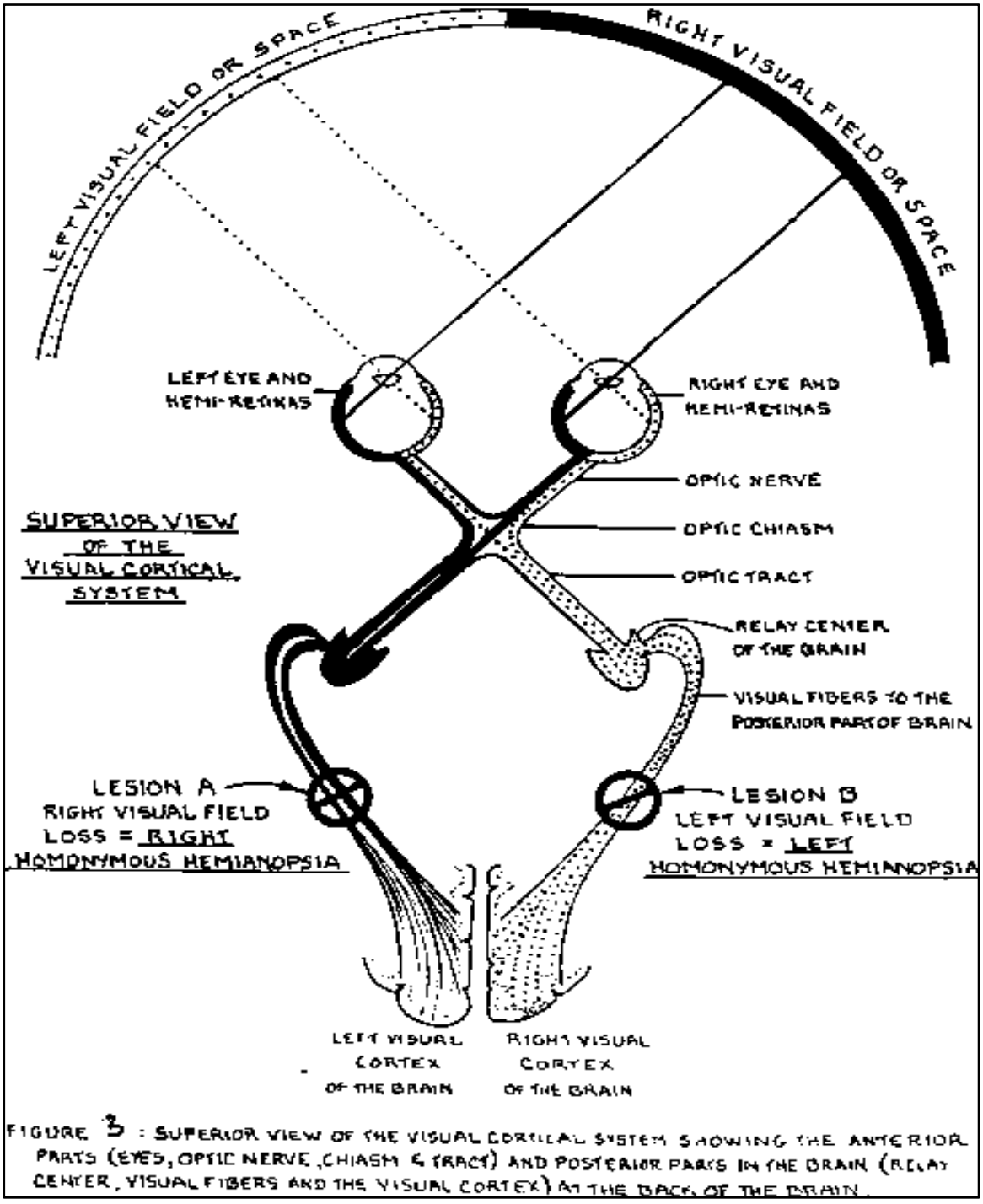

Figure 2.5: The visual field pathway showing the lesions causing a right hemianopia (lesion A) and left hemianopia (lesion B). Illustration courtesy of Josephine C. Moore OT, PhD.

The geniculocalcarine tracts are divided into two loops: the temporal loop and the parietal loop (Figure 2.6). The loops are named for the cortical areas traversed by the pathways. Input from the superior visual fields is carried through the temporal loop fibers. A lesion along this part of the tract causes a quadrantanopia in the superior visual field. Information about the inferior visual field is carried through the parietal loop fibers; damage along this pathway results in a quadrantanopia in the inferior visual field. If the lesion is large enough to damage the pathways of both loops, a hemianopia would occur with involvement of both the inferior and superior visual fields in one half of each eye.[70] Hemianopia with macular sparing can occur when the lesion occurs in the occipital lobe.[108] Clients with this type of field deficit retain 5-25 degrees of central vision but lose the peripheral visual field. Stroke-because it generally causes damage in only one hemisphere, most often causes the classic left or right hemianopia or quadrantanopia with or without macular sparing.[108] Bilateral damage to the occipital lobe-which can occur with trauma, hypoxia or inflammation-may cause deficits throughout both visual fields, significantly reducing the client's field and visual acuity and often leaving them with little usable vision. Spontaneous partial recovery of the visual field has been shown to occur in approximately half of persons with hemianopia, but a complete recovery of the visual field is uncommon.[274, 277] Pouget et al.[177] in a large study estimated that only approximately 10 percent of persons with

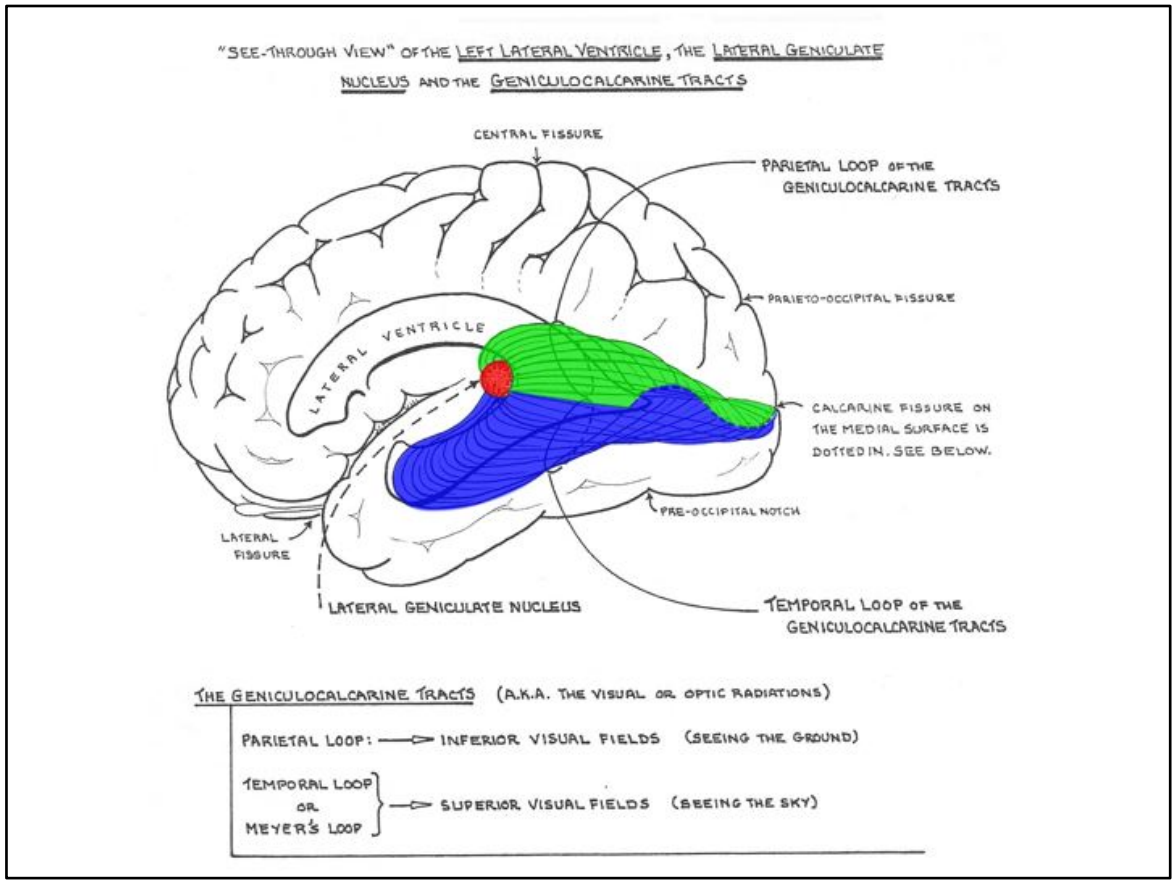

Figure 2.6: The temporal and parietal loops of the geniculocalcarine tract in the left hemisphere. Illustration courtesy of Josephine C. Moore OT, PhD.

homonymous hemianopia recover a full visual field. Most recovery occurs within the first 4 weeks after onset and the likelihood of improvement decreases significantly beyond 8 weeks.[277] Due to the low rate of complete recovery, hemianopia is generally considered a permanent visual impairment.[274]

2.4.3 Occupational Limitations Caused by Visual Field Deficit

Visual field deficit can limit participation in a significant number of daily living activities. The type of limitation depends on the extent of the field deficit and whether it affects the central or peripheral visual field. The brain relies on two forms of visual recognition to complete daily occupations. Recognition of "*where*" an object is in the environment alerts the brain to the presence of objects and is a precursor to the recognition of "*what*" the person is seeing.[69, 152] To be aware that there are objects in the environment does not require as precise visual information as that needed to identify the object. The brain only must be able to detect form, motion, and gradations in shading to detect the general location of an object. The rod photoreceptor cells comprising the peripheral retinal field are adept at capturing this information and the brain primarily relies on the peripheral visual field to complete "where" processing. A visual field deficit in the periphery diminishes the ability to detect motion and form causing the client to have trouble orienting to surroundings. In contrast, the ability to recognize "what" is being viewed requires extremely precise visual information. The cone photoreceptors in the central field that detect color, small details and contrast supply this information. A central visual field deficit can cause the client to have difficulty visually identifying objects.

We need both areas of the visual field to participate fully in activities and environments. Persons with central field deficits will have difficulty seeing small visual details and color which can impair reading, writing, and fine motor coordination. Occupations dependent on these skills include meal preparation, medication management, financial management, grooming and shopping. They may also have difficulty quickly identifying the details of landmarks and obstacles and experience difficulty participating in community activities such as driving, shopping, and social events. Persons with involvement of the peripheral visual field will have difficulty quickly detecting objects that are moving or blend into the background and objects in environments with low lighting. The person may not develop the big "picture" needed to accurately map the environment. This can affect the ability to recognize surroundings (orientation) and detect obstacles or landmarks fast enough to safely navigate environments (mobility). Without an intact peripheral field, the person is forced to use central vision as an anchor to guide ambulation, which leads to behaviors like shoe gazing (e.g., looking at the floor/feet when walking) or fixating a distant central target and walking towards it without looking to either side. Either behavior increases the risk of collisions or becoming lost while moving.

2.4.3.1 The Influence of Perceptual Completion on Visual Search

Although often considered mild when compared to the dramatic loss of the use of the limbs, homonymous hemianopia can significantly impact the client's ability to complete daily living activities and safely navigate environments. Hemianopia can alter the person's visual search pattern creating a slow disorganized search pattern characterized by multiple fixations that results in difficulty locating objects on the blind side.[274] These visual search changes can be at least partially explained by the influence of a visual perceptual process known as *perceptual completion*. The prefrontal lobes are tasked with directing visual search of the environment to quickly locate and catalog the items/features needed to complete occupations. The prefrontal lobes direct visual search by constructing an impression (picture) of the client's surroundings, and then directing search towards specific targets based on anticipation of where they would be found in the environment. One way the prefrontal lobes could construct this picture of the environment, would be to painstakingly locate and foveate each item in the person's surroundings, but this would require way too much time to enable successful adaptation to dynamic environments. So instead, the prefrontal lobes direct the eyes to sample locations in the environment and fill in (e.g., perceptually complete) the rest of the visual scene based on memory and expectation.[67, 86, 87, 100, 150, 181] The prefrontal lobes are amazingly skilled at using this method to build an accurate picture of the location of the items/features in familiar environments. For example, let's say I am lecturing to you in a hospital auditorium. You haven't been in this auditorium, but you have spent plenty of time sitting in lecture halls attending CEU events. Now I ask you if there is a fire extinguisher in the room. You probably didn't search for a fire extinguisher when you entered the auditorium, but your experience with hospitals prompts your prefrontal circuitry to direct your search towards the walls. Why? because your experience suggests that is where fire extinguishers are typically located in a room like this, and chances are you would be correct.

The speed that perceptual completion provides enables us to safely drive and participate in dynamic environments, but it can create challenges for the client trying to adapt to vision loss in the early stages of recovery. Research has shown that the prefrontal circuitry can perceptually complete the visual field even when the client has lost 50% of their vision due to hemianopia.[51, 210, 215] It is common for a person with hemianopia to be initially unaware of vision loss in an area of their field because they see a perceptually completed visual field without gaps or missing information.[45, 51, 102, 210] Perceptual completion creates two big challenges for the client. First, the prefrontal circuitry cannot include an unanticipated object in the perceptually completed scene unless the object was seen during the sampling process. As a result, the client may collide with a recently moved chair or trip over a toy in left on the floor. Secondly, perceptual completion makes it difficult for the person to determine the actual border between the seeing and non-seeing areas of the visual field or where a target might be located within the blind field. Without this information, the person is unable to confidently execute a single accurate saccade to locate the target in the blind field. Instead, the person often adopts a strategy of making repeated short "stair step" saccades towards the target until it is located.[144, 224, 274] To help you understand what a client might experience when searching the blind field, picture yourself in a tunnel completely devoid of light. You are instructed to run

as fast as you can towards a wall at the end of the tunnel. You are told not to worry, as you will locate the wall when you run into it. In this situation would you run quickly towards the unseen wall or run slowly stopping frequently to put your hands out to feel for it?

2.4.3.2 The Effect of Visual Field Deficit on Performance Skills

During the initial stages of recovery, before conscious awareness of the field deficit, the client will experience the odd perception of seeing a complete visual scene where objects on the blind side are always appearing, disappearing, and reappearing, without warning.[52, 84, 138, 210, 274] This often causes the client to move slowly and tentatively when navigating environments and to rely on others to lead them through an unfamiliar environment. Even when the person becomes aware of a hemianopia, visual search into the blind field remains slow and delayed [51, 169, 224, 274] Slowness searching towards the blind side adds to client's challenges in navigating the environment and locating objects for daily activities.[51, 74, 102, 122, 151, 170, 256] Newer research suggests that persons with hemianopia may also search their intact (seeing) field more slowly although compared to their blind side, the person's slowness searching is milder and interferes less in daily activities.[44] These search changes are more pronounced in persons with left hemianopia and compound the person's difficulty in locating information from the environment. Additional research is needed to verify this initial finding, but it reinforces the importance of carefully assessing the client's ability to quickly search *both sides* of the visual field during high-risk activities such as driving.

The changes in visual search that accompany a complete homonymous hemianopia limit three important performance skills: mobility and orientation, reading, and eye hand coordination.[141] These are critical components of many daily occupations and improving them is a primary intervention focus.

- *Mobility and Orientation*: Persons with hemianopia experience frequent actual or near collisions with objects and a tendency to get lost especially in unfamiliar and crowded environments.[59, 102, 274] They often demonstrate behaviors during navigation that indicate they are experiencing stress and uncertainty.[274] Key behaviors to observe for include: using a stiff, short, and uncertain gait; staring fixedly at a distant target and moving towards it without looking at their surroundings or staring at the support surface immediately in front of them (shoe gazing). Shoe gazing is especially prominent during transitions such as walking through a doorway or down steps or a ramp. To remain oriented the client may extend their hand and use their fingers to trail along a wall using tactile input to maintain their position in space. Following is another often subtle behavior used to maintain orientation. The client lags slightly behind another person during ambulation to follow and use them as a guide. Difficulty monitoring the environment may cause the client to experience anxiety in crowded and dynamic environments. Sometimes the stress can be severe enough to provoke a panic reaction with sweating, heart palpitations, nausea, and hyperventilation. One client with hemianopia aptly described this sensation as "crowditis", reporting that he became physically ill if he had to go into a crowded environment like a grocery store or sports

venue. The anxiety can become debilitating, leading to withdrawal from community activities and social isolation.[102, 256]

- *Reading*: When the border of the hemianopia comes into fovea, all or part of a targeted object may fall into the blind field. This can create significant challenges in reading.[25, 51, 102, 104, 151, 170, 177, 196, 212, 256, 274] Normally sighted readers view words through a "window" or perceptual span that allows them to see approximately 18 characters (letters/numbers) with each fixation of the eye.[196] The reader moves the eye from the center of one word to the center of the next using a series of alternating fixations and saccades. Each fixation lasts approximately 250 miliseconds (ms) and generally only one fixation is required to decipher the word.[182] The brain uses 50 ms to decode the word and 200 ms to plan the saccade to the next word in the sentence.[182] The saccade of a person reading English will move fixation 8-9 characters to the right which is the typical length of English words. The remaining partially decoded letters on the right side of the perceptual span are used to plan the next saccade.[182] The left side of the span is used to accurately identify words and locate the next line of text to navigate through text.

  Hemianopia shortens the perceptual span on the side of the deficit causing the client to see only part of a longer word during fixation and or even skip a small word.[135, 212, 274] For example, a client with a left hemianopia may read the sentence, "She should not shake the juice" as "He should not make the juice," transforming "she" into "he" and "shake" into "make." A right hemianopia can especially hinder reading. The shortened width of the span on the right side causes the person to miss letters on the right. For example, "She should not shake the juice" might be read as "She should not share the juice." More importantly a right hemianopia disrupts the person's ability to plan and execute an accurate saccade to the next word. The eye may land off-center so that only a few letters are seen or entirely miss the next word as in "She should share juice."[135, 212] Each time a word is seen incorrectly, the client must stop and re-read the word to identify it. Re-reading is called a regression and regressions significantly slow reading speed and reduce reading accuracy.[25, 274] The client also experiences difficulty accurately reading numbers and this can be particularly problematic. A nonsensical sentence will alert the client to errors made in reading words, but numbers often lack a precise context causing mistakes to go unnoticed. For example, a credit card bill for $288.00 may be misread as $266.00 and the error missed until a notice of insufficient payment is received. Clients who make numerical errors quickly lose confidence in their ability to pay bills and manage their checkbook, accurately read a recipe, or set of instructions, or complete medication management and often turn over these important daily occupations to someone else.[102, 151, 256]

- *Eye/Hand Coordination*: When the hemianopia has occurred on the same side as the dominant hand, the client may have trouble visually guiding the hand in fine motor activities. Writing legibility is often reduced.[151, 256] The client often cannot visually locate and maintain fixation on the tip of the writing instrument as the hand moves into the blind visual field. This may cause the client's handwriting to drift up down on the line or

the client may improperly position handwriting on a form. The client also has difficulty completing activities like mending, sewing, pouring liquids, measuring, dialing numbers on a smartphone, texting and other fine motor activities that require visual monitoring of the hand.[256] Research shows that persons with HH have trouble completing daily living activities that are dependent on these performance skills.[102, 151, 256, 274] Reported ADL limitations include medication management, financial management, communication using computers, smart phones and tablets; meal preparation, home management, viewing TV and videos, and yardwork.[30, 51, 53, 102, 151, 170, 256] Generally, the more dynamic the ADL environment and the wider the field of view required to complete the task, the greater the limitation. Therefore, only minor limitations are experienced in a few selfcare activities (mostly grooming) compared to significant limitations in shopping, driving, and participating in community events. The client's ability to resume driving is always questioned although research has shown that some persons with hemianopia can safely resume driving with specific training.[30]

2.5 Visual Attention

2.5.1 What is Visual Attention?

Visual attention is the ability to closely observe objects to discern information about their features and their relationship to self and other objects in the environment. It requires focusing the brain, ignoring irrelevant sensory input and random thoughts, and sustaining this focus over a period of several seconds to minutes.[181] The ability to attend closely is significantly influenced by the environment and context and the person's motivation to acquire information and achieve a goal.[130] Visual attention is broadly divided into *selective* visual attention and *global* visual attention. Selective attention focuses on visual details such as differences between letters, numbers, and faces. It is used to recognize and identify objects.[69, 90] Global attention focuses on getting the big picture-the location of objects in the environment and their proximity to the person. Its job is to ensure that a person is oriented and moves safely through space; without it, collisions and disorientation when moving would be the norm.[69, 81] To be able to fully engage and learn from the environment, a person must simultaneously employ these two modes of visual attention at all times. The contribution of each is equally important to visual processing.

Coordinating visual attention requires an extensive, well-connected neuro network distributed through all areas of the cortex, brainstem, and cerebellum. Important hubs within this network include the visual cortical relay centers (circuitry in the occipital lobe and posterior areas of the temporal and parietal lobes), the frontal eye fields and prefrontal cortex, the hippocampus, amygdala, cingulate gyrus, and the brainstem reticular activating system. These hubs form network connections that feed forward information (this is what I am seeing) and feedback information (this is what I should see next) to direct our attention (see Figure 2.6).[101] The visual cortical relay centers beginning with the lateral geniculate nucleus and primary visual cortex refine the raw input from the retina to enhance critical features needed to complete the desired task. The refined information is then simultaneously sent to the posterior temporal and

posterior parietal areas for further processing. Visual input processed through the posterior temporal circuitry links language to visual images to label and classify objects. This area utilizes the precise information supplied by cone cells and obtained via selective attention to identify visual details like color, shape, size, and juxtaposition to identify objects so they can be recognized, and their purpose identified. The posterior parietal circuitry links spatial visual information with movement to prepare the body to orient and move through space. Global attention to, and awareness of, the space surrounding the body and the body's relationship to that space is the function of this circuitry. To accomplish this, the posterior parietal area creates internal spatial and temporal maps of the body and maps of surrounding environments, space, and time.[81, 96] The maps are dynamic and continuously updated as the person moves through space in order to direct attention to the critical features needed to navigate space and manipulate objects.

Together, these posterior areas of the cortex provide a "library" of visual images integrated with movement and language to the prefrontal lobes to use in directing actions and achieving goals.[90] The prefrontal areas operate as the CEO of the brain. They establish the plan, identify the information needed to implement the plan, gather this information from the environment, posterior library, and body, and coordinate neural structures to execute the plan. Like any good CEO, the prefrontal cortex determines the best course of action based on input received from all of the sensory systems combined with memory and past experience. The frontal eye fields within the prefrontal cortex direct the search for specific objects in the environment to accomplish the goal. This visual search is guided by the anticipation of where the desired object typically would be found in the environment and based on visual memory.[70] Recall in the example in section 2.4.3 of searching for a fire extinguisher in a lecture hall. The visual memories from your previous experience with fire extinguishers directs your attention and search towards the walls of the room instead of the ceiling or floors. The prefrontal cortex also regulates visual attention on tasks through working visual memory.[90, 223, 275] Working memory is the ability to hold more than one piece of information "on deck" in memory and ready for immediate recall to assist in completing a task.[190] *Working visual memory* is the specific ability to hold on deck a picture of an object and its location while completing a task.[190] An example of working visual memory would be holding in mind a picture of a specific brand of canned tomatoes along with its location in the aisle while shopping for the ingredients to make chili. Its "working" memory in the sense that once you accomplish the task, you discard the memory of the tomatoes and replace it with the next item on your list. We use working memory to sequence and stay on task while completing activities.

Older areas of the cortex-the amygdala and hippocampus-also assist in directing visual attention. The amygdala reacts to all incoming visual input and assigns to it a level of emotional relevance ranging from no relevance to significant relevance (e.g., pay *ATTENTION to THIS*!). Connecting a strong emotion to a visual image strengthens the motivation to attend to it.[181] For example, a piece of chocolate sitting on a plate next to someone who LOVES chocolate tends to rivet their attention to the plate. In contrast, an object (kale?) without emotional relevance will go unnoticed. One of the jobs of the hippocampus is to create a context for incoming visual images by linking them to past experience. The hippocampus keeps potentially important visual

input based on the context buffering in temporary memory to be reinstated later if needed. For example, you walk into a room and notice that a normally closed window is open (context) and thirty minutes later you realize that you haven't seen the cat-your helpful hippocampus jumps in and directs your attention back to the window to investigate.

The brainstem also contributes to cortical attentional processing through activation of the body's 4-step arousal sequence that goes back and forth between asleep, awake, alert and attending. The arousal sequence energizes the cortex and helps to maintain vigilance when attending.[117] It responds to tonic and phasic influence. *Tonic arousal* is strongly influenced by the sleep-wake cycle and circadian rhythms; some persons are early birds who are most alert in the morning and others are night owls who have difficulty waking up and attending until after the sun goes down. *Phasic arousal* is triggered by a specific event. The attention level of early birds or night owls would immediately increase upon hearing a fire alarm go off in their building.

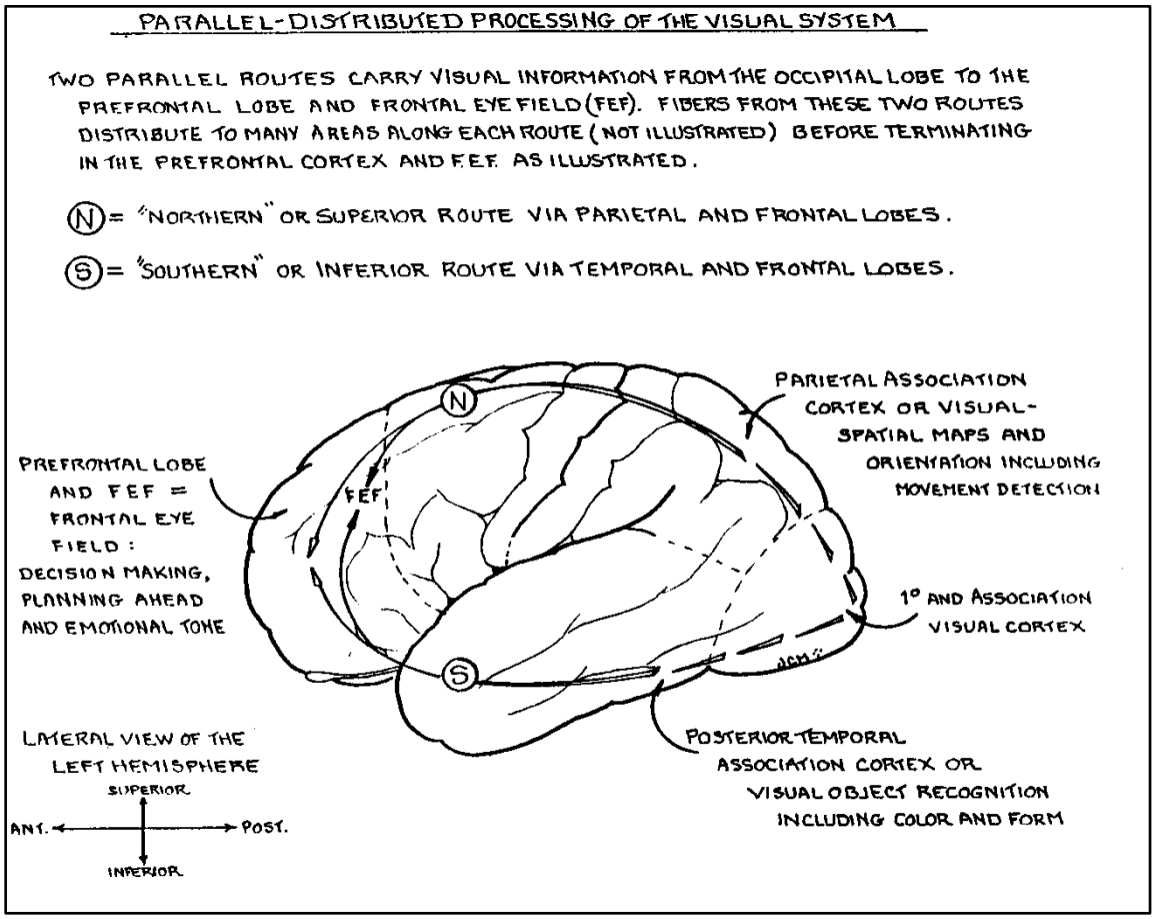

Figure 2.6: Major cortical centers Involved in modulating visual attention. Illustration courtesy of Josephine C. Moore OT, PhD.

2.5.2 Deficits in Visual Attention from Acquired Brain Injury

2.5.2.1 Neuro Networks that Control Attention

Long white matter pathways connect the key hubs to create an extensive neural network that controls attention.[5, 65] The extensiveness of this network means that any form of brain injury is likely to alter visual attention to some degree due to pathway or structural damage. Pathway damage (as occurs from diffuse axonal injuries sustained in TBI) can be particularly disruptive because it disconnects hubs from each other, including those that received no structural damage from the brain injury.[65, 92] Injury to the brainstem may reduce visual attention by diminishing the client's general level of alertness. The client can't be aroused sufficiently to attend and/or has difficulty sustaining sufficient attention to complete an activity. Injury along the visual cortical relay pathway that delivers visual input to the cortex, may degrade the quality and quantity of visual input available for processing. The client may not attend to certain objects or features in the environment because they weren't seen clearly enough to engage attention and unlock prediction and sequencing.

Structural damage, as occurs in stroke and moderate-severe TBI-can disrupt the function of key hubs that contribute to attention. Damage to the posterior temporal cortex may disrupt object identification. The client may have difficulty pulling details together to form meaningful patterns or link language to visual images to classify patterns, resulting in an agnosia or alexia.[93] Damage to the posterior parietal cortex may disrupt the ability to map the body and surrounding space. The client may not attend to important environmental features and landmarks and experience disorientation-unable to discern where they have been or where they are going. Injury to prefrontal areas may alter the client's ability to procure or use visual input to accomplish a task and achieve a goal. Visual search may become random because the client is unable to anticipate where to find an object and efficiently direct attention to that location. The client may have difficulty examining visual input to decide what is important to complete the task at hand. The client may indiscriminately attend to any object within their field of view exhibiting impulsive field-dependent behavior. Working memory may be diminished impairing the ability to stay focused on a task. Without the plan generated by working memory, the client maybe easily distracted by external and internal input. Damage to the older cortical structures-the amygdala and hippocampus-will affect memory. The person may lack the motivation to attend to and subsequently lay down new memories of objects. The object/event must have significant emotional relevance to register in memory or trigger memories to guide actions.

Visual attention is expressed though search and scanning. Therefore, a change in visual attention will be observed as a change in how the client searches for visual information. Normal search strategies are intentional, purposeful, and driven by the need to obtain specific information.[133, 157] The type of search pattern the person uses depends on the demands of the task. For example, a person reading English would use a left-to-right and top-to-bottom linear strategy. Whichever pattern the person uses, it will be efficient, symmetrical, and comprehensive, aimed at acquiring the greatest amount of information in the least amount of

time. Although eye movements are used to carry out visual search it's important to remember that visual search is driven by attention.

2.5.2.2 Visual Spatial Neglect

Visual inattention commonly occurs following brain injury and may range from mild to severe.[90, 188] *Visual spatial neglect* is the most studied attention deficit in persons with acquired brain injury from stroke or head trauma. Visual neglect is predominantly caused by injury to the occipital, parietal, temporal and prefrontal areas of the right hemisphere.[188, 244] Left hemisphere cortical lesions can cause right neglect.[227] Right neglect causes less severe and obvious changes in behavior and is most apparent when the client is required to multi-task.[227]

Kerkhoff and Schenk [124] defined neglect as the "impaired or lost ability to react to or process sensory stimuli [visual, auditory, tactile, olfactory] presented in the hemispace contralateral to a lesion of the human right or left cerebral hemispheres." (p. 1072). There are several key words in this definition that delineate the features of neglect. First, the ability to attend can be "impaired" or "lost" implying that there are degrees of severity of the condition ranging from mild to severe. Second, persons can have difficulty "reacting" to stimuli (e.g., noticing and responding to it) and difficulty "processing" stimuli, (e.g., using the sensory information to complete a task). Third, persons can experience inattention to different forms of sensory input- not just visual. Finally, persons experience inattention to sensory input on the side of the body "contralateral" (e.g., opposite) to the location of the brain injury. This means that persons with right hemisphere lesions experience difficulty attending to visual input from the left side of the body/space and vice versa.

There is consensus among researchers that neglect represents a heterogeneous disorder characterized by a complex and diverse set of behaviors.[188, 125, 244] These behaviors can be generally grouped into three broad categories: 1) a lateralized spatial bias in attending to and searching space, 2) a lateralized inability to map and build internal mental representations of space on the left side of the body, and 3) a non-lateralized attention deficit that impairs arousal and the ability to focus, shift, and sustain attention.[2, 16, 188, 244 ,245]

- Spatial bias restricts the person's ability to explore the space contralateral to the side of the brain injury. It is the most prominent and consistent behavior associated with neglect from right hemisphere lesions and shows up as difficulty or inability to explore space on the left side of the body.[163, 188, 244, 245] The person may make no attempt to search the left side or make fewer visual fixations and slower eye movements towards the left contributing to slow and incomplete search of left space.[129, 163, 254] The person demonstrates a bias towards the right side that creates an asymmetrical search pattern where the person initiates and confines search to the right side of a visual array.[163] This rightward bias can deprive the client of needed information on the left and impair their ability to complete ADLS that require attention to both sides of the body such as dressing, bathing, eating, grooming, meal preparation, home management, shopping.[10, 129, 130] The strong association of spatial bias with right hemisphere lesions is thought to occur because of a difference in the way the hemispheres direct visual attention.[65, 244,

[245, 276] As illustrated in Figure 2.7 the left hemisphere directs attention toward the right half of the visual space surrounding the body, while the right hemisphere directs visual attention towards both the right and left halves of space. If a lesion occurs in the left hemisphere, visual attention and search toward the right side are diminished, but the right hemisphere still provides some attentional capability. A similar lesion in the right hemisphere may completely eliminate attention toward the left because the left hemisphere does not direct attention toward the left side.

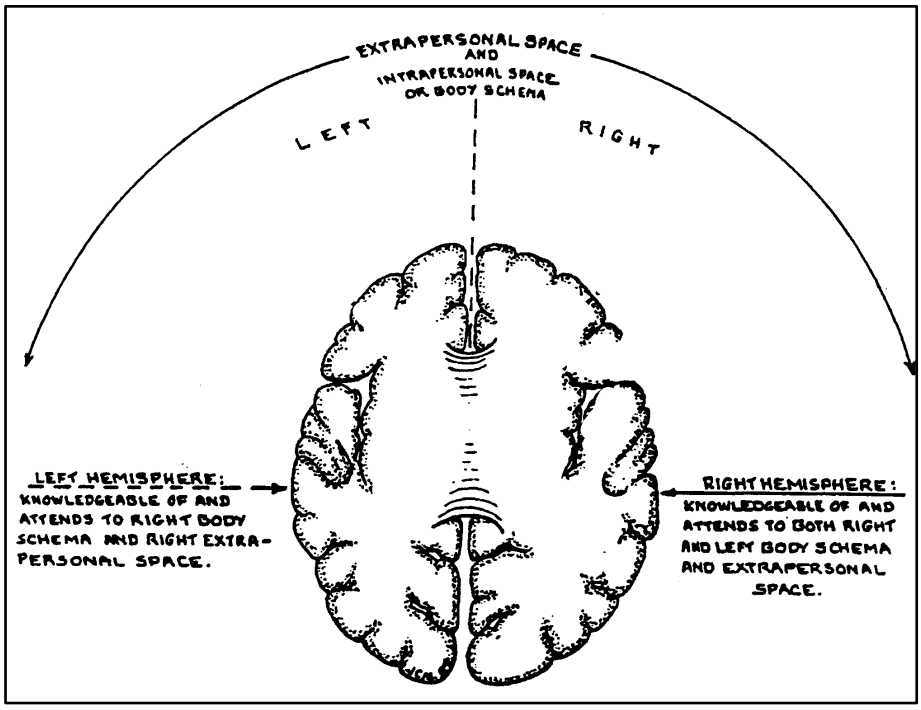

Figure 2.7: Differences in the direction of visual attention between the two hemispheres. Illustration courtesy of Josephine C. Moore OT, PhD.

Left neglect can be confused with a left hemianopia because clients with either condition show spatial bias and limited search of the left space. Although they can occur together, hemianopia and neglect are distinct conditions that disrupt search performance differently.[276] Hemianopia is a *primary sensory loss* that limits the amount of visual input into the cortex. It interferes with the client's ability to locate objects in the blind field but does not alter the brain's ability to direct attention. In fact, client with a left hemianopia *depends* on visual attention to compensate for vision loss by directing eye movements towards the left to gather visual information from the blind side. Unfortunately, perceptual completion (see section 2.4.3) can disrupt the client's ability to move the eyes far enough into the blind visual field to successfully locate objects making it appear that the client neglects the left side.[276] In contrast, a client with neglect loses the *attentional mechanisms* that drive the search for visual information on the left. The client (even if they have an intact visual field) makes little attempt to search the left side of the visual space.[163] Visual search is most significantly impaired when the two

conditions occur together.[244] In this case the client does not receive visual input from the left side because of the hemianopia and does not compensate for the loss of visual input by directing attention toward the left side. Clients with both conditions show greater inattention toward the left visual space when completing ADLS[10] and experience more difficulty moving the eyes or the head towards the left side.[1, 124, 188, 244]

- Impaired mental representation of space: Internally generated cognitive maps create a mental representation of the space surrounding the body that person uses to orient to space.[18, 81, 89, 109] The maps are continuously updated as the body moves. Persons with neglect can experience a disruption in the mapping of space on the left side so severe that objects on the left simply disappear from their concept of space. According to Becchio and Bertone,[18] it is as though the mental map of space on the left side did not exist in the past, doesn't exist in the present, and will never exist in the future. As a result, the client doesn't attend to landmarks on the left side and does not build a map of environments on the left side. This diminishes the ability to orient to and maintain orientation in space and clients are often observed to literally be "lost in space" as they try to navigate environments.[26] Difficulty conceptualizing left space may explain the consistency of the client's inattention towards the left despite repeated cuing from the OT.[18] It may also explain revisiting-another commonly observed neglect behavior-where the person repeatedly reexamines objects located on the right side of a visual array while ignoring objects on the left.[188, 225] For example, when asked to locate the cup of pudding sitting on the left side of the dinner tray, the client repeatedly searches the right side of the tray even when cued to search towards the left.

- *Non-lateralized attention deficit*: Clients-especially those with persistent (also called chronic) neglect-often have difficulty generating adequate levels of alertness and sustained attention.[185, 223, 235, 237, 244, 245] Their inattention is **non**-lateralized in that it does not differ between the body sides and instead affects the brain's overall ability to receive and process visual information. This generalized diminishment of attention impairs the ability to begin and complete tasks.[2, 185] Clients with low arousal may have difficulty generating sufficient attention to engage in a task unless the energy requirements are amped up.[180] For example, the client must be seated on the edge of the mat instead of the wheelchair, so that the threat of falling over increases their alertness. Clients with non-lateralized inattention may have difficulty disengaging focus on one object to shift attention to another object, slowing their search performance even when attending to a task.[245] This slowness increases the length and effort required to complete a task, causing fatigue and poor performance.[244] Inability to sustain attention is particularly detrimental to successful completion of daily occupations as even basic ADLs require sequencing steps over time. When the client loses focus and drifts off during an activity, their lapse of attention disrupts working visual memory requiring the OT to provide continuous redirection to the task.

The incidence of visual spatial neglect averages between 50-70% in the early stages of recovery from a right hemisphere brain injury.[83, 120, 244] Fortunately, most neglect resolves during the first

year of recovery and is significantly diminished by three months post injury in most persons.[83, 120, 161, 244] Disruption of the pathways that connect the frontal, temporal, parietal and occipital lobes together may account for the initially high incidence of neglect immediately after injury and the good potential for recovery.[65] Persons whose neglect persists beyond three months may have significant and persistent deficits that reflect structural rather than pathway damage within the brain.[120]

2.5.3 Occupational Limitations Caused by Visual Inattention

Because visual attention is modulated through an extensive neural network, some capacity for visual attention generally is retained even in cases of severe brain trauma.[150] On the other hand, changes in visual attention almost always occur with any brain injury, even a mild injury. Inattention creates asymmetry and gaps in the visual information gathered through scanning.[27, 179, 191, 242, 244, 193] The brain requires complete visual information delivered in an organized fashion to make appropriate decisions and direct actions. Without it, the person's ability to efficiently and successfully complete occupations decreases. Whether a client's inattention impairs performance depends on the task. Different tasks require different types and levels of attention. For example, reading a highly technical textbook can require enormous amounts of selective visual attention, but you can enjoy the comics page while listening to the TV and eating breakfast. Driving requires continuous global attention and vigilance to monitor the speed and position of other vehicles and objects and only sporadic selective attention to landmarks, street signs and traffic lights.

It's important to remember that how and whether inattention manifests following brain injury depends on the demands of the task and environment. This is an important consideration in evaluation and intervention. All ADLS require attention to both sides of the body and environment and the ability to sequence multiple steps over time. Tuning out and drifting off disrupts ADL performance as does attending to only one side of the body/space. Performance limitations observed during ADLs include difficulty locating items, sustaining attention to task completion, multi-tasking, and rapidly and accurately assessing situations to complete activities in dynamic environments. The more dynamic the environment and the more ambiguous the task and outcome, the greater the client's attentional limitations will impair task performance. Performance limitations will be most noticeable in daily activities that require significant working visual memory and sustained attention. Examples include participating in driving, shopping, sports, work, and other I-ADLS. These limitations may persist even in a client who appears to have completely recovered from neglect. For example, a client with good recovery, may show no inattention to the left and drive safely along familiar country roads going to and from the small town he grew up in, but show marked inattention to the left and dangerous driving maneuvers when attempting to drive to an appointment in a larger, less familiar city.

It's important that the client recovering from neglect reclaim as many occupations as possible. The most informative evaluation includes 1-2 more challenging I-ADLS to provide a complete picture of the client's attentional capabilities and limitations. This will help the OT set the most appropriate goals and timeframes for the client. When providing intervention, the OT must

carefully examine the requirements of the targeted ADL and determine if the client will be able to successfully complete it in all situations or in some situations. In the case of the driver, he does not need to completely give up driving if he is willing to limit driving to environments that match his attentional capabilities.