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Occurrence of oculomotor dysfunctions in acquired brain injury: A retrospective analysis

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Strabismus;
Accommodation;
Eye movements;
Cranial nerve palsy

Abstract

BACKGROUND: The purpose of this retrospective study was to determine the frequency of occurrence of oculomotor dysfunctions in a sample of ambulatory outpatients who have acquired brain injury (ABI), either traumatic brain injury (TBI) or cerebrovascular accident (CVA), with associated vision symptoms.

METHODS: Medical records of 220 individuals with either TBI (n = 160) or CVA (n = 60) were reviewed retrospectively. This was determined by a computer-based query spanning the years 2000 through 2003, for the frequency of occurrence of oculomotor dysfunctions including accommodation, version, vergence, strabismus, and cranial nerve (CN) palsy.

RESULTS: The majority of individuals with either TBI (90%) or CVA (86.7%) manifested an oculomotor dysfunction. Accommodative and vergence deficits were most common in the TBI subgroup, whereas strabismus and CN palsy were most common in the CVA subgroup. The frequency of occurrence of versional deficits was similar in each diagnostic subgroup.

CONCLUSION: These new findings should alert the clinician to the higher frequency of occurrence of oculomotor dysfunctions in these populations and the associated therapeutic, rehabilitative, and quality-of-life implications.

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Acquired brain injury (ABI) typically includes both traumatic brain injury (TBI) and cerebrovascular accident (CVA), more commonly referred to as stroke.[1] Statistics regarding the frequency of occurrence and impact in the United States are striking. Approximately 8 million people per year suffer a TBI, with 1.5 million of those injuries categorized as "major."[2] About 60% of those affected do not return to the workforce, with an estimated national economic loss of $4 billion.[3] The findings are similar for CVA. Stroke is the leading cause of chronic disability,[4] affecting 500,000 individuals per year.[5] Only 50% of those affected return to the workforce with little, if any, residual disability.[6] Hence, both TBI and CVA, and, more broadly, ABI in general, are major economic, social, medical, and public health concerns.[7]

Because of the global nature of a brain injury, many brain areas and their associated functions are adversely affected.[3] One such area is vision, a primary sensory modality; half of the cranial nerves relate to vision. Injury to vision-related areas of the brain can result in a range of dysfunctions, including the oculomotor, color vision, and visual field systems.[8-10] An important area of vision-based concern is the oculomotor system, which broadly includes

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Table 1 Age range, mean, and standard deviation of the subgroups

Subgroup	Age range (yr)	Mean (yr)	Standard deviation (yr)
TBI (n = 160)	8 to 91	44.9	15.8
CVA (n = 60)	24 to 90	61.2	14.7
ABI (n = 220)	8 to 91	49.3	17.1

the versional, vergence, and accommodative systems.[11] Resultant symptoms are diverse and may include diplopia, blur, difficulty following targets, oculomotor-based reading problems, and asthenopia.[12] While producing vision discomfort and possible loss of visual efficiency (e.g., reading speed and reading duration),[13,14] oculomotor problems may negatively affect the overall rehabilitative process (e.g., cognitive therapy)[15,16] thus impacting adversely on an individual's quality of life.

The frequency of occurrence of oculomotor dysfunction in these studies is dependent on the tests used, the method of categorizing the deficits, and the subgroupings used. Despite methodologic differences, the frequency of occurrence of oculomotor dysfunctions in these populations has been found to be universally high in earlier studies. In the TBI population, convergence insufficiency was found to be about 40%[9,17]; some type of oculomotor dysfunction was 60% to 85%[18,19]; cranial nerve (CN) palsy was 33%,[20] and accommodative dysfunction was about 20%.[21] In the ABI population, accommodative dysfunction ranged from 10% to 70%.?[9,22] For example, a recent study in this area was conducted by Suchoff et al.[9] Their adult (ages 19 to 70 years) ABI population (n = 62) was derived from 2 extended-care facilities, and subjects were unselected with respect to suspected vision problems and related symptoms. All vision examinations were part of their routine annual physical examinations and were performed at least 6 months after injury. They found considerably increased frequency of occurrence of dysfunctions in all oculomotor areas tested compared with a non-ABI cohort: strabismus and convergence insufficiency (approximately 45%), abnormal oculomotor tracking (approximately 40%), and impaired accommodation (approximately10%).

Although these studies provided valuable information, most of them had several limitations: (1) some had small sample sizes, (2) some lacked diagnostic subgroupings, (3) not all patients tested had vision-based symptoms, (4) there was broad categorization of the oculomotor dysfunctions, and (5) CVA was not assessed separately as a subgroup. In the current retrospective study, all of these limitations were addressed.

Methods

A computer-based query was obtained for ABI patients examined between October 1, 2000, and October 7, 2003, using either the 99203 (new patient evaluation) or 99213 (established patient evaluation) procedure codes. All patients were ambulatory outpatients with vision-based symptoms. Optometrists from the Raymond J. Greenwald Rehabilitation Center (RJGRC) at the State University of New York (SUNY) State College of Optometry performed the vision examinations. The majority of patients were referred from rehabilitation professionals at the following institutions: Rusk Institute of Rehabilitative Medicine at NYU Medical Center, Bellevue Hospital at NYU Medical Center, Department of Rehabilitative Medicine at Mount Sinai Medical Center, Lenox Hill Hospital, New York Hospital, and the International Center for the Disabled. Other referrals were made by rehabilitation professionals in private practice in the greater New York City area. Referrals were also received from other services within the SUNY College of Optometry's University Optometric Center including primary care, low vision, contact lenses, and ocular disease and special testing. Referred patients were not limited to those with either TBI or CVA; individuals with other neurologic conditions that affect the visual system, such as vestibular dysfunctions, cranial postsurgical complications, and brain tumors, comprised a sizeable patient base. Table 1 lists the age characteristics of the patients in each group at the initial evaluation at the RJGRC. Table 2 describes the range of years after injury and the mean for each category at initial presentation at the RJGRC.

The computer query yielded 486 records, of which 300 were selected randomly. Each of 3 members of the RJGRC's clinical staff then randomly chose 100 of the records. Of these, only those patients with either TBI (n = 160) or CVA (n = 60) were reviewed. Several patients whose records were selected for the retrospective review and analysis had received dilated fundus examinations 4 months or less before their evaluations at RJGRC, and, as such, a dilated fundus examination was not repeated.

The RJGRC's diagnostic evaluation included assessment of the following areas: distance and near visual acuity, distance and near refraction, distance and near binocular and oculomotor status, color vision, visual fields, and ocular health. In some instances, not all areas could be evaluated because of limitations in the patient's cognitive status, language ability, or physical state.

The 5 major categories of oculomotor dysfunction investigated were accommodation, version, vergence, strabismus, and CN palsy. Conditions included under each category were determined by consensus. Criteria for inclusion into 1

Table 2 Range and mean of the number of years (postinjury) upon initial presentation for the subgroups

Subgroup	Range (yr)	Mean (yr)
TBI (n = 160)	0.1 to 42.0	4.5
CVA (n = 60)	0.1 to 18.0	2.7
ABI (n = 220)	0.1 to 42.0	4.0

Table 3 Summary of the percentage of individuals in each subgroup (where for TBI n = 160 and for CVA n = 60) within a given category of ocular motor dysfunction and the most common anomaly present

Ocular motor dysfunction	TBI (%)	Most common anomaly (TBI)	CVA (%)	Most common anomaly (CVA)
Accommodation	41.1	Accommodative insufficiency	12.5	Accommodative infacility
Versional	51.3	Deficits of saccades	56.7	Deficits of saccades
Vergence	56.3	Convergence insufficiency	36.7	Convergence insufficiency
Strabismus	25.6	Strabismus at near	36.7	Strabismus at far
CN palsy	6.9	CN III	10	CN III

Note: The "n" represents the number of persons tested for accommodation, which only included those under the age of 40 years (i.e., prepresbyopic), TBI = 51 and CVA = 8.

or more of the oculomotor dysfunction categories were per conventional clinical standards.[23-25]

The reviewers then recorded the frequency of occurrence (percentage) of the targeted conditions that were diagnosed at the patient's initial evaluation. These conditions were tabulated separately for the TBI and CVA subgroups.

Results

The percentage of individuals in the 2 subgroups manifesting the 5 basic categories of oculomotor dysfunctions are presented in Table 3. The majority of individuals with either TBI or CVA exhibited some type of oculomotor dysfunction. This ranged from 6.9% to 56.3% in the TBI subgroup and from 10.0 to 56.7% in the CVA subgroup. Deficits in accommodation (41.1%) and vergence (56.3%) were more prominent in the TBI subgroup, whereas those of strabismus (36.7%) and CN palsy (10%) were more prominent in the CVA subgroup. The frequency of versional deficits was similar in each subgroup (approximately 55%). When assessed across the 5 categories, 90% of the TBI subgroup manifested some type of oculomotor dysfunction, whereas 86.7% of the CVA subgroup manifested the same.

The number of individuals under the age of 40 years with 1 or more types of accommodative dysfunctions is presented in Table 4. More than 40% of those with TBI exhibited an accommodative dysfunction, with nearly all showing accommodative insufficiency (AI). In contrast, only 1 in 8 (12.5%) patients with CVA exhibited an accommodative deficit, specifically a slowed dynamic facility.

The number of individuals with 1 or more vergence dysfunctions is presented in Table 5. Convergence insufficiency (CI) was the main dysfunction found in both subgroups, occurring in 42.5% and 35% of the TBI and CVA patients, respectively. Other diagnostic categories with high frequency of occurrence were binocular instability (BI; i.e., restricted vergence ranges) in TBI (10%) and basic esophoria in CVA (18.3%). Furthermore, the presence of each abnormal vergence type was reasonably well segregated by diagnostic subgroup.

The number of individuals manifesting 1 or more versional oculomotor dysfunctions in each subgroup is presented in Table 6. The overall percentage within each subgroup was similar (approximately 55%), except for nystagmus, which was nearly 30 times more frequent in CVA than in TBI.

The number of individuals with strabismus in each subgroup is presented in Table 7. Strabismus was found in 25.6% of the patients with TBI and in 36.7% of the patients with CVA. Two of the strabismic categories did not reflect the predicted ratio of the dysfunction based purely on subgroup sample size (i.e., 160TBI:60CV = 2.66). There was a higher relative frequency of a hyper component (ratio = 4.75) and a nearly equal relative frequency of an esophoria component (ratio = 0.83).

The number of individuals manifesting CN palsy in each subgroup is presented in Table 8. The most common deficits were CN III and IV for TBI and CN III for CVA. The

Table 4 Individuals in each subgroup with accommodative dysfunction

Subgroup	No. with accommodative insufficiency	No. with accommodative infacility	No. with accommodative excess	No. with ill-sustained accommodation	Total no. with accommodative dysfunction
TBI (n = 51)	19	2	2	0	21
CVA (n = 8)	0	1	0	0	1

Note: Some persons presented with more than 1 accommodative dysfunction. The "n" represents the number of persons tested for accommodation, which only included those under the age of 40 years (i.e., prepresbyopic).

≥21/51 = 41.1% of persons with TBI presenting with accommodative dysfunction.

≥1/8 = 12.5% of persons with CVA presenting with accommodative dysfunction.

Table 5 Individuals in each subgroup with vergence oculomotor dysfunction

Subgroup	No. with CI	No. with CE	No. with DE	No. with DI	No. with BI	No. with basic exo	No. with basic eso	Total no. with vergence dysfunction
TBI (n = 160)	68	4	0	2	16	2	3	90
CVA (n = 60)	21	0	0	0	1	0	11	22

CI = convergence insufficiency; CE = convergence excess; DE = divergence excess; DI = divergence insufficiency; BI = binocular instability; basic exo = basic exophoria; basic eso = basic esophoria.

Note: Some persons presented with more than 1 vergence dysfunction. The "n" represents the number of persons tested for vergence oculomotor dysfunctions, which includes the entire sample for each subgroup.

≥90/160 = 56.3% of persons with TBI presenting with vergence oculomotor dysfunction.

≥22/60 = 36.7% of persons with CVA presenting with vergence oculomotor dysfunction.

frequency of occurrence of CN palsy was 6.9% in TBI and 10.0% in CVA.

Discussion

The current retrospective analysis conducted in a large sample of ambulatory outpatients with either TBI or CVA and related vision symptoms supports previous reports of the markedly increased frequency of occurrence of oculomotor dysfunctions in these populations versus the non-ABI population.[7,9,28] Furthermore, it extends these studies to include CVA, because CVA had not been investigated previously as its own subgroup.

The frequency of occurrence of oculomotor dysfunctions in the TBI and CVA populations is much larger than that found in their non-ABI cohort.[26-28] For example, in a nonpresbyopic clinic population with near work symptoms,[26] convergence insufficiency was found in about 4% of the cases, whereas in the current study, it was found in 43% of the TBI subgroup and in 35% of the CVA subgroup, with similarly symptomatic individuals. The same was true for accommodative insufficiency, which was found in 9% in the non-ABI population[26] and in 40% of the current TBI population. Lastly, the overall frequency of occurrence of oculomotor dysfunctions in the non-ABI symptomatic sample was 20%[26] verses 90% in the current study, representing a 4.5-fold increase in frequency in the brain-injured sample. Thus, in the TBI and CVA populations, if some type of an oculomotor dysfunction is not found after careful and comprehensive testing, it is unexpected and represents an exception to the rule.

Underlying neurophysiology of the oculomotor system and its dysfunctions resulting in diffuse versus local brain insult

Within the TBI and CVA populations, the frequency of occurrence of specific oculomotor conditions appeared to be dependent on the nature of the neurologic insult: diffuse versus localized. Because of the global coup-contrecoup nature of the injury in TBI, brain consequences tend to be diffuse. In contrast, because of the more regional vascular nature of the injury in CVA, brain consequences tend to be more localized.

Accommodation. The innervation for accommodation is comprised of premotor and cortical neural components.[2] The premotor neural component for accommodation is the autonomic nervous system, with the parasympathetic system initiating the accommodative response and the sympathetic system assisting in maintaining the response. The cortical innervation for accommodation begins with fibers from the primary visual cortex (V1) going to the parieto-temporal area and the cerebellum. The fibers continue on to the Edinger-Westphal nucleus in the pretectum, where input

Table 6 Individuals in each subgroup with versional oculomotor dysfunction

Subgroup	No. with deficits of saccades	No. with deficits of pursuit	No. with saccadic intrusions	No. with nystagmus	Total no. with versional dysfunction
TBI (n = 160)	62	52	19	1	82
CVA (n = 60)	23	13	6	10	34

Note: Some persons presented with more than 1 versional oculomotor dysfunction. The "n" represents the number of persons tested for versional oculomotor dysfunctions, which includes the entire sample for each subgroup.

≥82/160 = 51.3% of persons with TBI presenting with versional oculomotor dysfunction.

≥34/60 = 56.7% of persons with CVA presenting with versional oculomotor dysfunction.

Table 7 Individuals in each subgroup with strabismus

Subgroup	No. with an intermittent strabismus	No. with a constant strabismus	No. with a unilateral strabismus	No. with an alternating strabismus	No. with a distance strabismus	No. with a near strabismus	No. with an exophoria component	No. with an esophoria component	No. with a hyper component	No. with a hypo component	Total no. with strabismus
TBI (n = 160)	20	20	20	20	23	31	25	5	19	6	41
CVA (n = 60)	10	10	10	11	14	11	13	6	4	2	22

Note: Some persons presented with more than 1 type of strabismus. The "n" represents the number of persons tested for strabismus, which includes the entire sample for each subgroup.
≥41/160 = 25.6% of persons with TBI presenting with strabismus.
≥22/60 = 36.7% of persons with CVA presenting with strabismus.

is received and processed from the autonomic nervous system to form the motor command.

From the pretectum, the motor fibers innervating accommodation travel with the oculomotor nerve (i.e., CN III) on to the ciliary ganglion. Finally, these motor fibers continue to travel with the short ciliary nerve to the ciliary muscle to produce a change in accommodation.

Thus, the accommodative pathway is susceptible to diffuse axonal injury, with its numerous stages of neural motor innervation being prone to impact on neurologic insult. However, it is also possible to have a localized lesion (for example in the pretectum), which would paralyze accommodation.[29] Finally, there is normal physiologic reduction of accommodative amplitude and dynamic accommodative facility as one ages, which may confound the contribution of the neurologic injury on accommodation, especially for persons between 35 and 45 years of age (i.e., incipient presbyopia).[29] Moreover, this process may be exacerbated in TBI patients, especially those with hyperopia.[10,30]

Vergence oculomotility. The neuromotor control for vergence oculomotility is less clearly elucidated.[11,31] The premotor neural components in the brainstem are located in the mesencephalic reticular formation 1 to 2 mm dorsal and dorsolateral to the nucleus of the oculomotor nerve. Three types of vergence cells have been isolated: tonic, burst, and burst–tonic. Tonic cells are correlated with changes in vergence angle, whereas burst cells are correlated with changes in vergence velocity. The burst–tonic cells respond to combined vergence angle and vergence velocity. The additional premotor neural components include the medial longitudinal fasciculus, cerebellum, and frontal eye fields. Finally, to elicit convergence at the peripheral level, decreased stimulation to the bilateral abducens nerves and increased stimulation to the inferior division of bilateral oculomotor nerves are evident, with the converse being necessary to elicit divergence.

Thus, with its numerous premotor and motor contributions for vergence, there are multiple axonal pathways susceptible to the diffuse axonal injury pathophysiology of traumatic brain injury. However, localized brainstem lesions to cranial nerves III, IV, or VI, as well as either localized lesions or diffuse axonal shearing along the motor pathways of cranial nerves III, IV, and VI in the cavernous sinus, will result in a restriction of ocular motility to one or both eyes, thus producing a CN palsy/paresis and strabismus.

Versional oculomotility. Versional oculomotility includes components such as fixations, saccades, and pursuit, among others.[11,31] With respect to fixation, it is the most poorly understood of the versional oculomotor pathways. The premotor neural components have been specified as being the frontal eye fields, supplemental eye fields, parietal area, right prefrontal cortex for attentional aspects, and right posterior parietal cortex for attentional aspects.[32] With re-

Table 8 Individuals in each subgroup with CN palsy

Subgroup	CN III	CN IV	CN VI	INO	WEBINO	Total no. with CN palsy
TBI (n = 160)	6	5	2	0	0	11
CVA (n = 60)	6	1	0	1	0	6

Note: Some persons presented with more than 1 CN palsy. The "n" represents the number of persons tested for CN palsy, which includes the entire sample for each subgroup.

≥11/160 = 6.9% of persons with TBI presenting with CN palsy.

≥6/60 = 10.0% of persons with CVA presenting with CN palsy.

spect to saccades, the premotor neural area differs for vertical versus horizontal saccades; for vertical saccades, it is the rostral mesencephalon (RM), whereas for horizontal saccades, it is the paramedian pontine reticular formation (PPRF). And, with respect to horizontal pursuit, the premotor neural components include pursuit neurons in V1, the medial vestibular nuclei (MVN), and the prepositus hypoglossi. The associated integrated premotor neural areas for vertical saccades, horizontal saccades, and horizontal pursuit are similar and include the frontal eye fields, parietal area, basal ganglia, superior colliculus, and cerebellum.

For horizontal saccades and horizontal pursuit, there is a common motor neural pathway for the motor fibers traveling with the inferior division of the oculomotor nerve. In addition, recent evidence suggests a shared saccade/pursuit premotor neural pathway.[33] The joint premotor pathway includes common inhibitory omnipause neurons and common saccade/pursuit neurons in the PPRF responsible for modulating velocity of the motor response.[11,33] Thus, with its multiple localized premotor neurologic substrates, as well as multiple neural motor cortical axonal pathways for these 3 components of versional oculomotility, control of versions appears to be prone to both localized and diffuse axonal injury.

Comparison of oculomotor dysfunctions for the 2 subgroups of ABI. Individuals with TBI presented with an increased frequency of accommodative and vergence deficits relative to that found in individuals with CVA. This may be because of the involvement of multiple premotor and motor neurologic sites for both accommodation and vergence, which makes sensorimotor vision aspects particularly susceptible to diffuse axonal injury. However, local lesions are possible, with their more discrete and restricted motor involvement.

Individuals with CVA presented with an increased frequency of strabismus and CN palsies. The 3 cranial nerves responsible for innervating the extraocular muscles are particularly vulnerable to localized lesions and disturbances at the level of the brainstem cranial nerve nuclei as well as in the cavernous sinus just before innervating the extraocular muscles. This may account for the increased frequency of occurrence of strabismus and CN palsies in those with CVA versus TBI.

Deficits of versional oculomotility were present with similar frequencies in TBI and CVA. The similarity may be attributed to the fact that versional oculomotility can be impaired by either localized lesions or diffuse axonal injury. Localized infarcts to any of the premotor neurologic substrates, such as the frontal eye fields, the parietal lobe, and the cerebellum, may be evident in those with CVA. However, diffuse axonal injury could occur in those with TBI just as frequently, thus resulting in shearing of the axons for cranial nerves III, IV, and VI caused by a coup-contrecoup injury,[3] with consequent less accurate and poorly sustained versional oculomotor responses.

Knowledge of expected oculomotor sequelae. Optimal management of individuals with either TBI or CVA requires that the clinician be aware of the expected oculomotor dysfunctions found in these patients as well as the potential adverse effects on basic eye tracking, reading, visual scanning, and higher-order visual information processing (e.g., perceptual interpretation). Thus, the case history and diagnostic evaluation should be tailored with this notion in mind. For example, the expected occurrence of nystagmus in the CVA population is 17% based on the current study. This is nearly 30 times greater than expected in a matched TBI cohort (0.625%). Hence, when examining a CVA patient, if nystagmus is not obvious by gross visual observation, then more sensitive techniques should be used, such as high magnification biomicroscopy and careful visuoscopy. Then, if found, the negative impact of acquired nystagmus on reading ability[34,35] and other visual tasks must be addressed with respect to vocational, avocational, and rehabilitative aspects.

Impact on global rehabilitation. Most types of brain injury rehabilitation involve the visual system, as it is a primary sensory modality. For example, cognitive rehabilitation and speech/language rehabilitation involve saccadic visual search and visual scanning activities in conjunction with intervening periods of accurate fixation. Vestibular rehabilitation also involves dynamic interaction with the vergence and accommodative systems as targets are displaced in depth.[11] The notion that the presence of an oculomotor dysfunction may adversely affect the progress of brain injury rehabilitation is well accepted.[15,16] This is important because approximately 90% of individuals in each of the TBI and CVA subgroups in the current study had at least 1 type of oculomotor dysfunction. Thus, nearly

all patients in each subgroup presented with some type of oculomotor dysfunction. For example, convergence insufficiency, evident in the majority of those with TBI, may produce intermittent diplopia during near work. Saccadic deficits, found in the majority of those with CVA, may result in inefficient and error-prone saccadic tracking.

Impact on quality of life. The first step in patient management for any vision dysfunction is to identify and understand the basic abnormal vision condition along with its associated symptoms. Only then can appropriate therapy be implemented for its remediation (e.g., vision therapy) or a proper referral made for additional guidance and assistance (e.g., neuropsychological evaluation). With success in this management phase, an increased quality of life can result.

If an appropriate vision diagnosis is not made, the patient's visually based symptoms will persist and perhaps even become exacerbated. Thus, the patient will likely continue to have difficulty reading, writing, scrutinizing written instructions, and ambulating through complex environments.[7,10]

Future directions. The frequency of occurrence for the various categories and types of oculomotor dysfunctions in the TBI and CVA subgroups has been determined. The next logical step is to investigate therapeutic efficacy using a large sample size, with emphasis on both clinical and laboratory-based neurophysiologic aspects (e.g., functional magnetic resonance imaging), as well as a study of the impact on vocational, avocational, and other aspects of overall quality of life. Correlation of damaged structure and related degree of dysfunction, using neuroimaging techniques, would be informative in terms of predicted adverse effects and subsequent treatment outcomes.

References

1. Suchoff IB, Kapoor N, Ciuffreda KJ. An overview of acquired brain injury and optometric implications. In: Suchoff IB, Ciuffreda KJ, Kapoor N, eds. *Visual and vestibular consequences of acquired brain injury*. Santa Ana, CA: Optometric Extension Program Foundation, 2001:1-9.
2. D'Angelo CM. An overview of enhancement techniques for peripheral field loss. *J Am Optom Assoc* 1994;64:60-70.
3. Zost MG. Diagnosis and management of visual dysfunction in cerebral injury. In: Maino DM, ed. *Diagnosis and management of special populations*. St. Louis: Mosby, 1995:75-134.
4. Kent T, Hart M, Shries T. *Introduction to human disease.* New York: Appleton-Crofts, 1979.
5. Alexander L. Pre-stroke signs and symptoms. *Rev Optom* 1978;8: 45-53.
6. Kelley RE. Cerebrovascular disease. In: Weiner WJ, Goetz CG, eds. *Neurology for the non-neurologist*. Philadelphia: Lippincott, 1989: 52-66.
7. Suchoff IB, Ciuffreda KJ, Kapoor N. *Visual and vestibular consequences of acquired brain injury.* Santa Ana, CA: Optometric Extension Program Foundation, 2001.
8. Hellerstein LF, Freed S, Maples WC. Vision profile of patients with mild brain injury. *J Am Optom Assoc* 1995;66:634-9.
9. Suchoff IB, Kapoor N, Waxman R, et al. The occurrence of ocular and visual dysfunctions in an acquired brain-injured patient sample. *J Am Optom Assoc* 1999;70:301-9.
10. Kapoor N, Ciuffreda KJ. Vision disturbances following traumatic brain injury. *Curr Treat Options Neurol* 2002;4:271-80.
11. Ciuffreda KJ, Tannen B. *Eye movement basics for the clinician.* St. Louis: Mosby Yearbook, 1995.
12. Suchoff IB, Gianutsos R, Ciuffreda KJ, et al. Vision impairment related to acquired brain injury. In: Silverstone B, Lang MA, Rosenthal B, et al., eds. *The Lighthouse handbook on vision impairment and rehabilitation*. New York: Oxford University Press, 2000: 549-73.
13. Kapoor N, Ciuffreda KJ, Han Y. Oculomotor rehabilitation in acquired brain injury: a case series. *Arch Phys Med Rehabil* 2004;85:1667-78.
14. Ciuffreda KJ, Han Y, Kapoor N. Oculomotor rehabilitation for reading in acquired brain injury. *Neurorehabil* 2006;19:1125-38.
15. Reding MJ, Potes E. Rehabilitation outcomes following initial unilateral hemispheric stroke: life table analysis approach. *Stroke* 1988;19: 1354-8.
16. Grosswasser Z, Cohen M, Blankstein E. Polytrauma associated with traumatic brain injury: incidence, nature, and impact on rehabilitation outcome. *Brain Inj* 1990;4:161-6.
17. Cohen M, Grosswasser Z, Barchadski R, et al. Convergence insufficiency in brain injured patients. *Brain Inj* 1989;3:187-91.
18. Lepore FE. Disorders of ocular motility following head trauma. *Arch Neurol* 1995;52:924-6.
19. Schlageter K, Gray K, Shaw R, et al. Incidence and treatment of visual dysfunction in traumatic brain injury. *Brain Inj* 1993;7:439-48.
20. Sabates NR, Gonce MA, Farris BK. Neuro-ophthalmological findings in closed head trauma, *J Clin Neurol Ophthalmol* 1991;11:273-7.
21. Al-Qurainy IA. Convergence insufficiency and failure of accommodation following medical trauma. *Br J Oral Maxillofac Surg* 1995;63: 564-8.
22. Gianutsos R, Ramsey G, Perlin R. Rehabilitative optometric services for survivors of acquired brain injury. *Arch Phys Med Rehabil* 1988; 69:573-8.
23. Griffin JR, Grisham JD. *Anomalies of binocular vision: diagnosis and management*, 3rd ed., Boston: Butterworth-Heinemann, 1995.
24. Scheiman M, Wick B. *Clinical management of binocular vision*, 3rd ed. Philadelphia: Lippincott, 2002.
25. Caloroso E, Rouse M. *Clinical management of strabismus.* Boston: Butterworth-Heinemann, 1993.
26. Hokoda SC. General binocular dysfunction in an urban optometry clinic. *J Am Optom Assoc* 1985;56:560-2.
27. Ciuffreda KJ. The scientific basis for and efficacy of optometric vision therapy in non-strabismic accommodative and vergence disorders. *Optometry* 2002;73:735-62.
28. Ganley JP, Roberts J. Eye conditions and related needs for medical care among persons 1-74 years of age: United States, 1971-72. *Vital and Health Statistics* 1983;Series 11(228):1-69.
29. Ciuffreda KJ. Accommodation, the pupil, and presbyopia. In: Benjamin WJ, ed. *Borish's clinical refraction.* Philadelphia: WB Saunders, 1998:77-120.
30. Kapoor N, Ciuffreda KJ. Vision problems. In: Silver JM, Yudofsky SC, McAllister TW, eds. *Neuropsychiatry of acquired brain injury.* Arlington, VA: American Psychiatric Publishing Inc., 2005:405-17.
31. Leigh RJ, Zee DS. *The neurology of eye movements.* 2nd ed., Philadelphia: F.A. Davis, 1999.
32. Petit L, Dubois S, Tzourio N, et al. PET study of the human foveal fixation system. *Hum Brain Mapp* 1999;8(1):28-43.
33. Keller EL, Missal M. Shared brainstem pathways for saccades and smooth pursuit eye movements. *Ann N Y Acad Sci* 2003;1004:29-39.
34. Ciuffreda KJ. Reading eye movements in patients with oculomotor disturbances. In: Ygge J, Lennerstrand G, eds. *Eye movements and reading*. New York: Pergamon Press, 1994:163-88.
35. Taylor EA. *The fundamental reading skill.* Springfield, IL: Charles C. Thomas, 1966.