

Pediatric acquired brain injury

Marie I. Bodack, O.D.

Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio.

KEYWORDS

Acquired brain injury;
Traumatic brain injury;
Cerebral vascular
accident;
Pediatric;
Eye;
Vision;
Strabismus;
Visual field defect

Abstract

BACKGROUND: Although pediatric patients are sometimes included in studies about visual problems in patients with acquired brain injury (ABI), few studies deal solely with children. Unlike studies dealing with adult patients, in which mechanisms of brain injury are divided into cerebral vascular accident (CVA) and traumatic brain injury (TBI), studies on pediatric patients deal almost exclusively with traumatic brain injury, specifically caused by accidents.

CASE REPORT: Here we report on the vision problems of 4 pediatric patients, ages 3 to 18 years, who were examined in the ophthalmology/optometry clinic at a children's hospital. All patients had an internally caused brain injury and after the initial insult manifested problems in at least one of the following areas: acuity, binocularity, motility (tracking or saccades), accommodation, visual fields, and visual perceptual skills.

CONCLUSION: Pediatric patients can suffer from a variety of oculo-visual problems after the onset of head injury. These patients may or may not be symptomatic and can benefit from optometric intervention.

Optometry 2010;81:516-527

There are various methods of classifying types of acquired brain injury (ABI).^{1,2} The first method classifies injuries as to whether the insult is internal or external.¹ External insults to the brain, also known as *traumatic brain injury* (TBI), can result from motor vehicle accidents, falls, sports injuries, assaults, and blunt or penetrating trauma. Internal insults to the brain can result from cerebral vascular accidents (CVA), brain surgery, or arterio-venous malformations (AVMs). The second method of classification uses the mechanism of injury to the brain, either a mechanical force or the interruption of the oxygen supply.² Mechanical force injuries can result from blows to the head, motor vehicle accidents, assaults, and penetrating trauma. However, they can also result from internal forces such as a spontaneous hemorrhage. Interruptions of the

oxygen supply can result from mechanical force injuries, as well as from ischemic or embolic stroke, strangulation, smoke inhalation, and near drowning. In some cases there can be a "vicious cycle" of mechanical force injuries leading to interruption of the oxygen supply and vice versa.²

As with adults, pediatric patients can suffer a brain injury from any of the above mechanisms. Studies on patients with ABI have found that refractive, binocular, accommodative, or ocular motility problems develop in many patients.³⁻⁸ Other studies have found that ABI patients demonstrate visual field defects and visual processing difficulties.^{4,5,7,9,10} Although pediatric patients were included in some studies on ABI, few studies dealt solely with patients younger than age 19.³

According to the Centers for Disease Control and Prevention, approximately 475,000 children younger than age of 14 suffer from a TBI each year.¹¹ Approximately 37,000 to 50,658 of these children are hospitalized.^{11,12} An overall incidence of approximately 70 to 80 patients

Corresponding author: Marie I. Bodack, O.D., Cincinnati Children's Hospital, MLC 4008, 3333 Burnet Avenue, Cincinnati, OH 45229-3039.
E-mail: marie.bodack@cchmc.org

per 100,000 has been reported for pediatric TBI hospitalizations.¹¹⁻¹³ Some of the variability in rates can be attributed to varying definitions of TBI as well as the age range included as pediatric. Studies agree that boys are more likely to suffer from a TBI than girls.¹¹⁻¹⁵ Motor vehicle accidents (including motor vehicle–pedestrian accidents) and falls are the 2 most common causes of pediatric brain injury.^{3,11,13-15} With teenagers, sports injuries, especially from football, become increasingly common.^{14,16}

In younger children, abuse is a frequent cause of brain injury. The incidence of inflicted TBI during the first year of life compared with that in the second year of life has been reported as 29.7 per 100,000 compared with 3.8 per 100,000.¹⁵ Similarly, Reece and Sege¹⁷ found that children with brain injuries secondary to abuse were younger than children with brain injuries from accidents (0.7 years of age compared with 2.5 years). If the etiology of brain trauma in an infant is suspect, the presence of subdural hematomas and/or retinal hemorrhages may be helpful. Reece and Sege¹⁷ found subdural hematomas in 54% of abuse cases and in only 10% of accidental brain injury cases. Retinal hemorrhages were also more common in abuse cases than in accidental cases (33% vs. 2%).

In addition to classification by etiology, cases of ABI can be classified by severity. The Glasgow Coma Scale was developed in the 1970s to assess impaired consciousness.¹⁸ The scale grades a patient's verbal response, motor response, and eye-opening ability. A modified Glasgow Coma Scale for pediatrics exists for preverbal children.¹⁴ The total score for all 3 areas ranges from 3 to 15. Severe brain injury is a score of 3 to 8, moderate is 9 to 12 and mild is a score of 14 to 15. There is some disagreement as to whether a score of 13 is mild or moderate.¹⁴

Although the Glasgow Coma Scale assists in grading impairment of consciousness, the American Congress of Rehabilitative Medicine has developed more specific definitions of what constitutes mild, moderate, and severe traumatic brain injury for everyone. For example, a mild TBI can include a loss of consciousness, a loss of memory for events immediately preceding or after the accident, an alteration of mental state during the accident, and focal neurologic defects.¹⁹ Studies have found that the majority of cases (81% to 90%) of pediatric TBI are mild, 8% are moderate, 6% severe, and 5% fatal.^{4,20}

Vision problems after ABI

Patients who experience a brain injury may complain of numerous visual symptoms. Sabates et al.⁴ found the most common presenting complaints of TBI patients, ages 5 to 74 (average age, 31), presenting to a neuro-ophthalmological clinic, were blurred vision (46%), followed by diplopia (30%) and headaches (13%). In spite of these complaints, they found 88% of patients' visual acuities were able to be corrected to 20/30 or better. Similarly, Poggi et al.³ reported visual

acuity decreases are the most common complaints in pediatric traumatic brain injury cases; however, this study included children with pathological causes for decreased acuity, including optic atrophy.

Suchoff et al.⁵ also reported that blurred vision is a common presenting complaint of ABI patients. The authors noted that a change in refractive error may be the cause, but that damage to the oculomotor nerve (CN III) or medications, which can affect accommodation, can also result in blurred vision, especially at near. Patients may also report blurred vision in the presence of good acuity because of contrast sensitivity problems.

Reports on adults have shown that 90% of patients with a TBI and 87% of patients with a CVA manifest an oculomotor dysfunction after the injury.⁶ Oculomotor dysfunction is a general term for a myriad of conditions including disorders of accommodation, version, vergence, strabismus, and cranial nerve palsies. Interestingly, Ciuffreda et al.⁶ found that the most common anomaly varied depending on the type of brain injury. For patients with TBI, vergence disorders were the most common, affecting 56.3% of patients, whereas for patients with CVAs, version problems were most common, affecting 56.7%. Many studies have found that exo deviations, such as convergence insufficiency or intermittent exotropia, are the most common vergence problems affecting ABI patients.^{3,7,8} Poggi et al.³ found that the duration of coma correlated with the difficulties with convergence. Older children and children with cortical atrophy, subacute, subcortical, or diencephalic lesions were more likely to have convergence difficulties. He also found that visual acuity loss and ocular motility problems were more common than vergence problems in younger children.

Cranial nerve palsies have been reported to affect 7% to 43% of patients with ABI.^{3,6,20} The percentages vary depending on the type of brain injury, patient age, and the clinic setting in which the patients were examined. One study looking specifically at cranial nerve palsies in patients with closed head trauma found that patients who had a cranial nerve palsy generally had lower Glasgow Coma Scores than patients without cranial nerve palsies and that patients with a third cranial nerve palsy had the lowest Glasgow Coma Scores.²¹ Up to 75% of patients show a partial or complete resolution of the palsy without treatment.^{20,22}

In a study on pediatric patients with cranial nerve palsies, the most commonly affected cranial nerve was sixth (55%), followed by third (22%), fourth (12%), and finally multiple nerves (11%).²² In all groups, the most common etiology for the cranial nerve palsy was trauma (42%). Other etiologies were neoplasm (17%), postoperative (4%), and meningitis (3%).

Many ABI patients receive rehabilitative treatments such as speech, occupational, or physical therapy. Additionally, some patients are referred for psychological or educational testing. In many respects, the primary care optometrist can assist in the treatment of these patients by

diagnosing vision problems that can affect the patient's overall functioning.

Case 1

In November 2004, a 16-year-old white girl suffered an anoxic brain injury secondary to an unintentional drug overdose. Magnetic resonance imaging (MRI) of the brain found "extensive insult to the central and peripheral white matter of the cerebral hemispheres." During her hospitalization, she was examined by an ophthalmologist. At that examination (the latter part of 2004), her visual acuity was 20/400 in each eye, correctable to 20/20 in each eye with a refraction of $-3.75-1.00 \times 180$ in the right eye (O.D.), $-4.50-0.75 \times 180$ in the left eye (O.S.). (She did have eyeglasses, which were lost during her hospitalization admission. Her prior prescription was not known.) She did not have any strabismus. A dilated examination did not find any anterior or posterior segment pathology.

The patient was hospitalized frequently during the next year and was eventually referred to an outpatient rehabilitation center for occupational, physical, and speech therapy. She was also seeing a psychologist for mental health treatment. The patient was wheelchair-bound secondary to motor damage to her lower extremities. She was receiving physical therapy for mobility but was not able to walk without assistance. She was receiving occupational therapy for fine motor issues and for activities of daily living. Her occupational therapist (OT) diagnosed visual perceptual problems, specifically problems with visual memory, and was providing treatment in this area. Twenty-two months after the initial brain injury (September 2006), her OT referred her for an optometric examination because when copying pictures and doing desk work, the patient "would ignore the left side or omit the left half."

First optometric eye exam

At the time of the optometric examination, the patient reported blurry distance vision, difficulty with concentration, and reading problems, specifically a loss of place and horizontal diplopia. Her current medications included baclofen, dantrolene, risperidone (Risperdal®; Janssen, Titusville, New Jersey), and sertraline (Zoloft®; Pfizer, New York, New York). Of these medications, dantrolene (diplopia, visual disturbance), risperidone (abnormal vision) and sertraline (disorder of vision) have given noted ocular side effects.²³

An optometric examination found a visual acuity of 20/40 in each eye at distance and 20/20 in each eye at near with her current prescription. Cover test showed orthophoria at distance and 10Δ exophoria at near with a receded near point of convergence ($8''/12''$ with diplopia). Manifest refraction found 20/20 acuity in each eye at distance with the following, $-5.00-0.50 \times 180$ O.D., $-5.50-0.50 \times 180$

O.S. Ocular motility testing found jerky pursuits, inaccurate saccades, and poor fixation. Confrontation visual field testing found extinction to the left side; the patient was able to identify a single target presented to her left, but she could not identify a target on her left when presented simultaneously with one on her right. Automated visual field testing found some scattered central defects, although the visual field was flagged as unreliable (see Figure 1). (To determine if the defects were reproducible, a repeat visual field was ordered. On 2 separate attempts, the patient was not able to complete the visual field test because of fatigue and frustration with the task.) Pupil testing did not find any defects. Phorometric testing did not find any anomalies at distance but did find a convergence insufficiency at near, with a von Graefe phoria of 8Δ exo and a poor base out (positive fusional vergence) recovery of $X/26/-2$. Accommodative and stereopsis testing results were within normal limits (see Table 1).

Based on the history of behavior on drawing tasks and desk work and the performance during confrontation visual field testing, the patient had unilateral spatial inattention (USI), myopia, oculomotor dysfunction, and convergence insufficiency diagnosed. An updated eyeglasses prescription was given to the patient for full-time use. The patient would work with optometry and occupational therapy services on oculomotor and visual scanning activities to help alleviate the ocular motility and USI problems. Therapy for the USI would incorporate yoked prisms to attempt to reorganize the patient's visual space to help her become more aware of the left field. The convergence insufficiency would be reevaluated later.

Therapy

The USI was present when the patient was reading and drawing, in other words, when doing tasks that were within arm's reach or in peripersonal space.^{24,25} Therapy would start with the patient wearing full-field yoked prisms during these tasks. The goal was to use the prisms to encourage the patient to become aware of her left field. (Suchoff and Cuifreda²⁴ note that generally 20 prism diopters yoked prism is the minimum amount needed to bring a response in these patients.) In this case, treatment started with 20 prism diopters base left yoked prism. During work with the prisms, the patient was asked to look at a page of letters and find specific letters only on the left side of the page, first without, and then with the prisms. Similar tasks using pictures instead of letters and doing work with computer searches were also attempted. The patient became very frustrated during these tasks and would frequently remove the prism glasses. To determine if the patient was bothered by the distortion of the prism, 10 prism diopters yoked prism was also used in therapy. The patient continued working with the prisms in weekly therapy for approximately 6 weeks. Regardless of the power used, she did not show an objective or subjective improvement in her

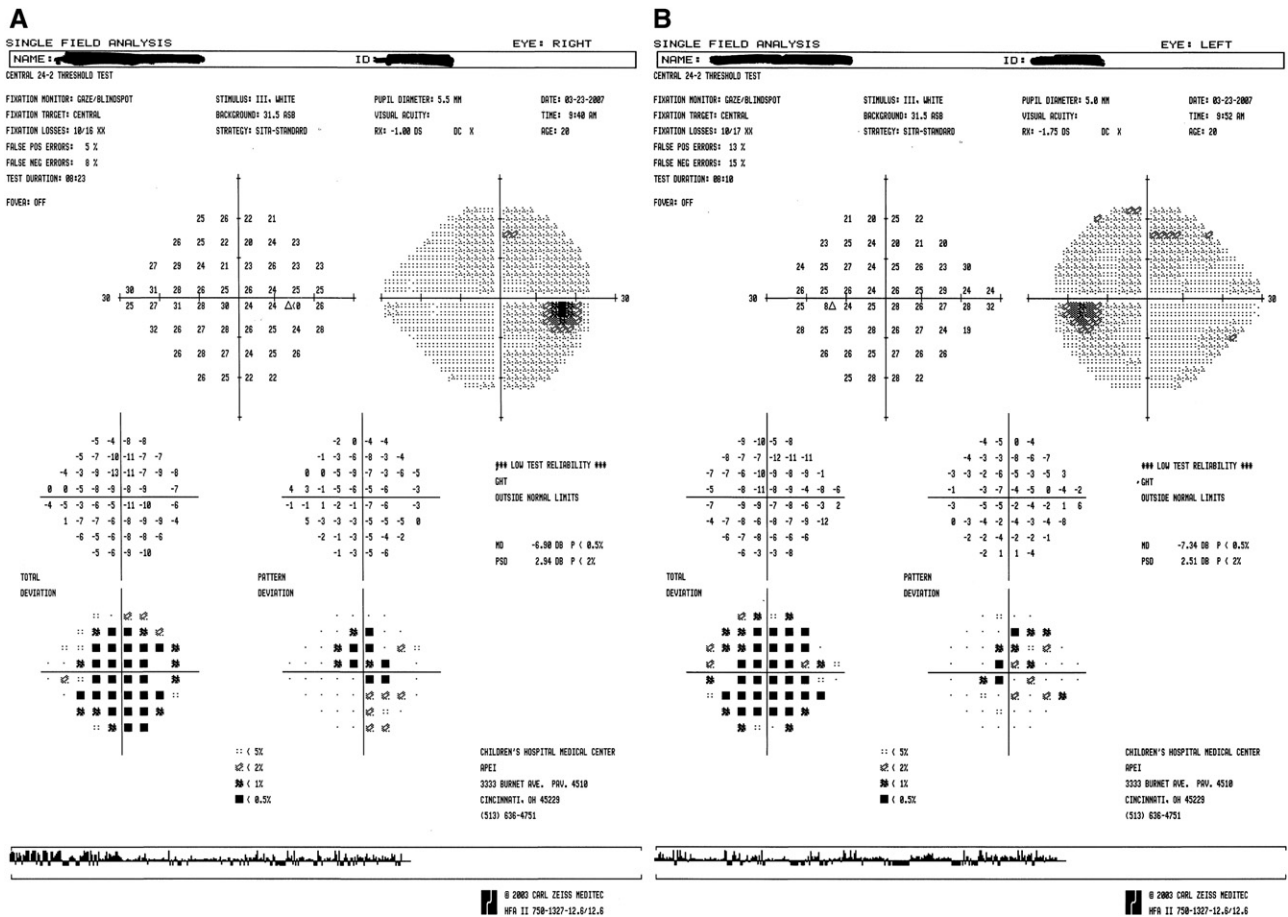


Figure 1 Automated visual field, patient 1.

ability to complete the tasks with the prisms. She no longer wished to continue therapy with the prism glasses but was willing to continue therapy for her oculomotor problems and for strategies to help with reading.

Follow-up

When the patient returned for a 3-month follow-up examination, she reported improved vision with her new glasses. She also reported that her initial complaints of losing her place and diplopia when reading were no longer present. Drawing tasks and visual field testing indicated that the USI was still present. A re-evaluation of the visual findings indicated a visual acuity of 20/20 in each eye with her new glasses. Fixation, saccades, and pursuit movements all improved. Binocular testing still reflected a convergence

insufficiency; however, she showed suppression on near photometric testing O.S. This suppression at near would explain the lack of symptoms to her convergence insufficiency. Vision therapy was discussed with the patient to treat the convergence insufficiency. However, therapy was deferred by the patient and her caregivers because the patient was still undergoing multiple therapies at another outpatient facility where vision therapy was not offered, and transportation was an issue. The patient will be re-evaluated when these therapies become less frequent.

Case 2

In November 2006, a 16-year-old white girl suffered from a ruptured arteriovenous malformation with intraventricular and subarachnoid hemorrhage affecting the right occipital-

Table 1 Additional binocular findings from patients

| | Distance phoria | Distance base out/in | Near phoria | Near base out | Amps | Randot stereo | Wirt stereo |
|--------|-----------------|----------------------|-------------|---------------|---------------|---------------|-------------|
| Case 1 | Ortho | | 8 exo | X/26/-2 | 11.50D OD/OS | 250" | 50" |
| Case 2 | 6 exo | Diplopia | 12 exo | X/18/0 | 13D, 12.75D | 250" | 30" |
| Case 3 | Ortho | | 3 exo | X/20/14 | 15.00 D OD/OS | 250" | 40" |

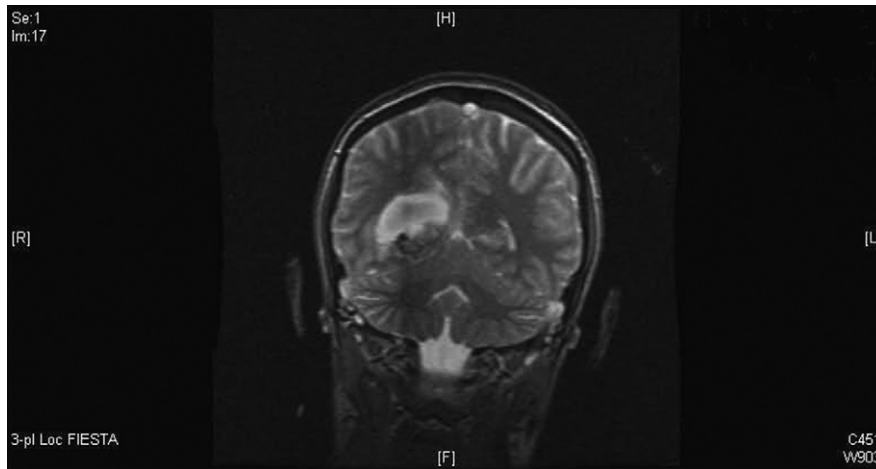


Figure 2 MRI shows intraventricular and subarachnoid hemorrhage affecting the right occipital-temporal lobes, patient 2.

temporal lobes (see Figure 2). The AVM was treated surgically by a resection, and the patient was hospitalized for 6 weeks. The patient was seen postsurgically by a neuro-ophthalmologist who diagnosed a left homonymous hemianopsia (see Figure 3). She was referred to an outpatient rehabilitation center for speech and occupational therapy, which she began at the time of her discharge from the hospital.

Her OT diagnosed visual motor integration and visual perceptual problems and was providing therapy in these areas. However, the OT was also concerned about peripheral vision loss, especially when the patient was looking in the distance, and referred the patient for an optometric examination. There were no concerns about the patient's reading ability or vision at near. The patient was examined by an optometrist 2 months after the surgery (January 2007). The

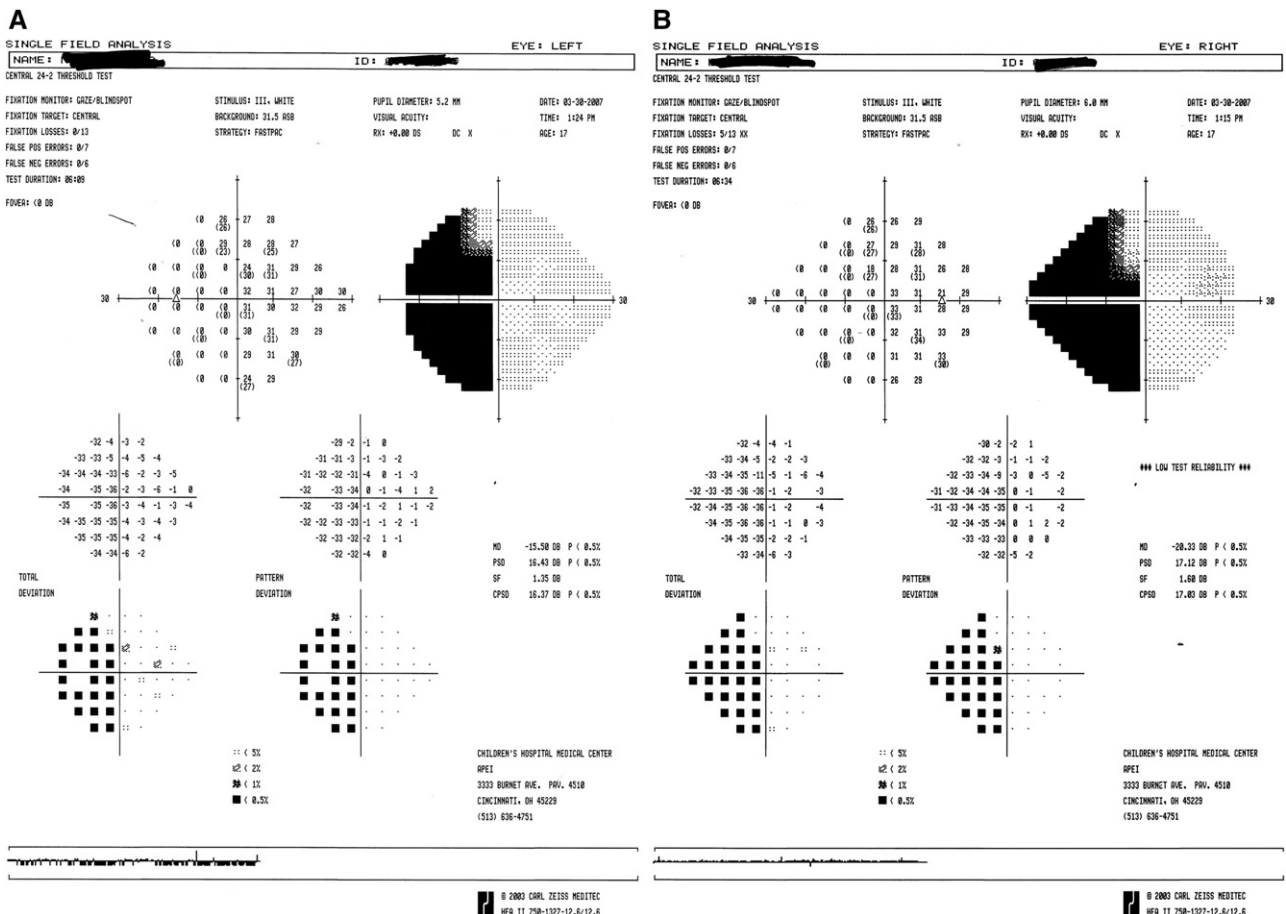


Figure 3 Automated visual field, patient 2.

patient's complaints during the examination were blurry distance vision and problems remembering things she learned in school. Her current medications included dilantin, which does not have reported visual side effects.²³

Initial optometric examination

Optometric testing found uncorrected distance visual acuities of 20/30— in each eye, correctable to 20/20 with -0.50 sphere in both eyes (OU). Distance and near cover test found an intermittent right exotropia of 10Δ . Near point of convergence was to the nose. Photometric testing found 6Δ exo at distance and 12Δ exo at near. Base out ranges showed diplopia at distance and X/18/0 at near. Pursuits, saccades, stereopsis, and accommodative amplitudes were normal (see Table 1).

The patient had myopia, intermittent right exotropia, a left homonymous hemianopsia, and probable visual memory problems diagnosed. Continuation of visual perceptual therapy was recommended, and glasses were prescribed for distance use.

Therapy

To address the visual field loss, a visual scanning program was recommended. The program would be done in conjunction with optometry and the patient's OT. Binocular sectoral prisms for visual field enhancement were recommended to allow the patient to more easily access visual information in the hemianopic field during visual scanning activities. Sectoral Fresnel press-on prisms (base left) were applied to the patient's glasses approximately 2 mm from the border of the seeing field (the fitting technique described by Weiss and Brown²⁶). Investigators generally recommend 12 to 20 prism diopters yoked prism

for distance work when a hemianopsia is present.²⁶⁻²⁹ In this case, the prism power was determined by holding loose prisms, base left, in front of each eye while the patient fixated at a distance target. The patient was asked to report when she was able to see some objects placed to the left of the target that she could not see without the prism. Initially, 10 prism diopters was used, with the prism power being increased incrementally until, with 15 prism diopters, the patient noted an ability to see some objects on the left. Training, both in office and home, was attempted with the prisms; however, the speed at which the patient was able to locate targets, and the patient's perception of the ease of locating the target, did not change with the prisms.

Follow-up visit

The patient returned for a follow-up visit 4 months later. She reported improved visual acuity with the glasses. She was compensating well for her visual field loss without the prisms. According to her occupational therapist, the visual motor integration and some visual perceptual scores improved, but additional treatment was required. The patient would continue with therapy in these areas. Although the intermittent exotropia was still present, the patient was not symptomatic, and clinical findings were essentially unchanged from her prior examination. Vision therapy was discussed with the patient and was recommended. The patient was referred to a vision therapist closer to her home for continued therapy.

Case 3

In December 2006, an 8-year-old black boy was admitted to a hospital with seizures secondary to herpes simplex virus

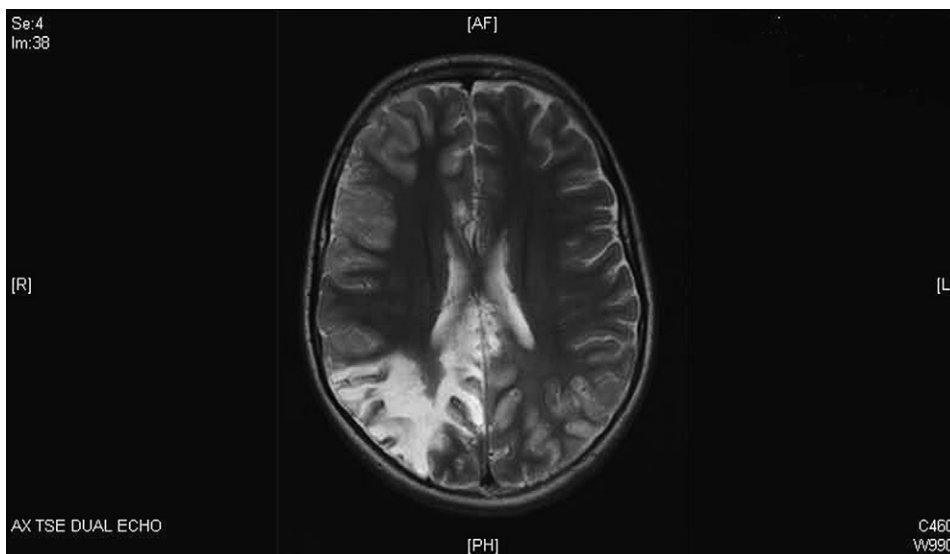


Figure 4 MRI shows necrotizing encephalitis affecting the right parietal and temporal lobes, patient 3.

(HSV) encephalitis. An MRI scan found necrotizing encephalitis affecting the right parietal and temporal lobes (see Figure 4); he was hospitalized for 45 days. As an inpatient, he was evaluated by an ophthalmologist who found an uncorrected visual acuity of 20/20 in each eye, no significant refractive error, strabismus, or pathology, but showed a left hemianopsia on confrontation testing. After his discharge from the hospital, the patient was referred to an outpatient rehabilitation clinic for occupational therapy. The OT diagnosed visual motor integration problems and was providing therapy in this area. Additionally, the OT had concerns because the patient omitted details when copying pictures, and the OT referred the patient for an optometric examination.

Five months after the seizures started (April 2007), the patient was examined by the optometry service. The patient had no complaints. His mother was concerned about handwriting and reading ability. The patient's current medications included divalproex (Depakote®; Abbott Laboratories, Abbott Park, Illinois) and methylphenidate (Concerta®, McNeil Pediatrics, Fort Washington, Pennsylvania). Of the medications, divalproex (amblyopia, blurred vision, 16% diplopia) and methylphenidate (visual disturbance) have visual side effects.²³

Initial optometric examination

Entering uncorrected visual acuities at distance and near were 20/20 in each eye. Cover testing found orthophoria at distance and near. Retinoscopy found low, age-appropriate hyperopia. Pursuit testing findings were normal. Saccadic testing found frequent loss of fixation with slow alternation between targets. Near tests did not find any binocular or accommodative anomalies (see Table 1). Automated visual field testing found an incomplete left hemianopsia, greater superiorly than inferiorly (see Figure 5). Drawing tests were completed. When copying pictures, the patient did not consistently omit objects on the left or the right side but omitted details throughout (see Figure 6). Additionally, when asked to write his name, the patient displayed poor spacing between letters and an inability to stay on the line. During the examination, he frequently lost interest in the task and would become easily distracted. His mother reported that although he had a diagnosis of attention deficit disorder and was on Concerta before the infection, he has had increased problems with "staying on task" since the hospitalization.

The patient had oculomotor dysfunction, a left hemianopsia, visual motor integration problems, and visual

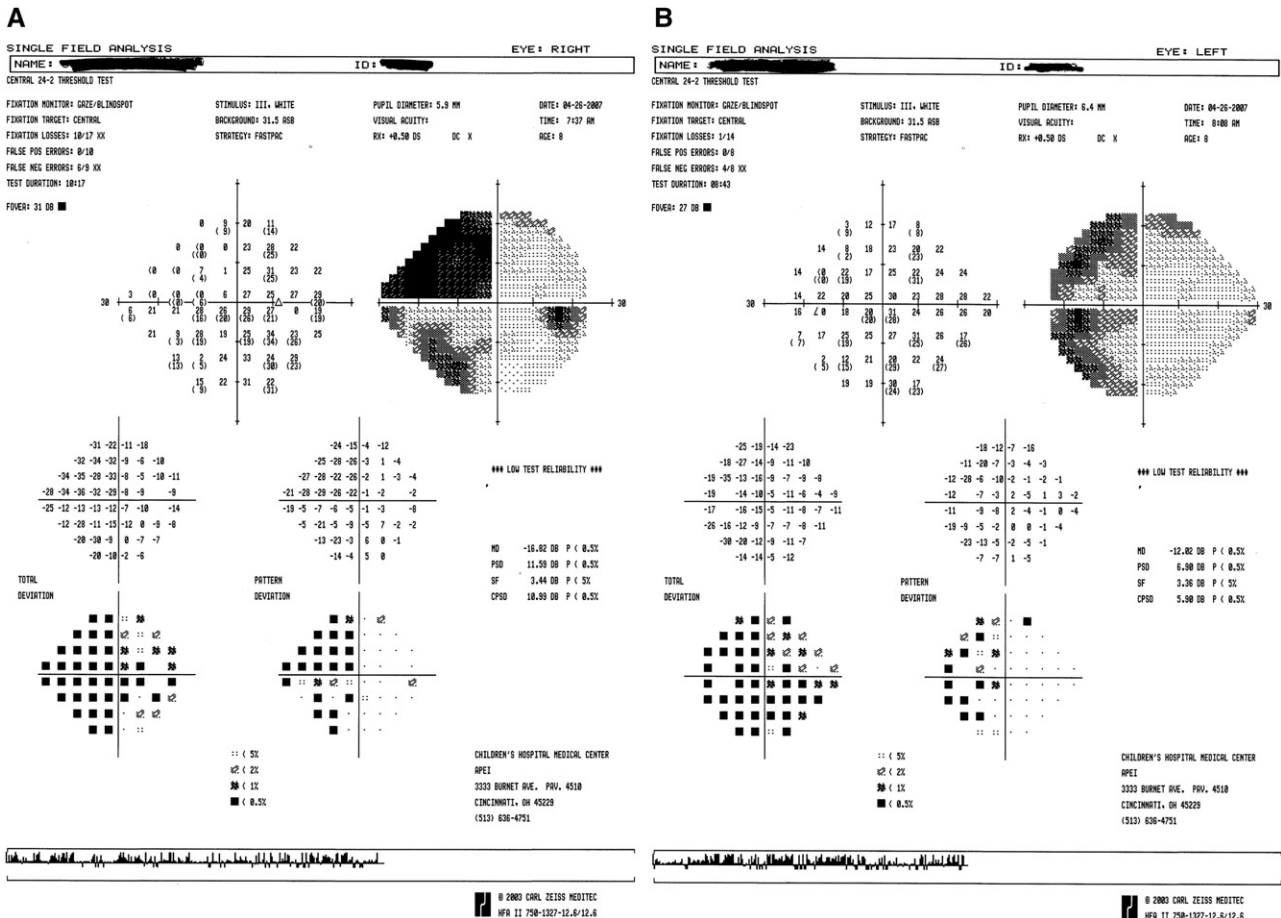


Figure 5 Automated visual field, patient 3.

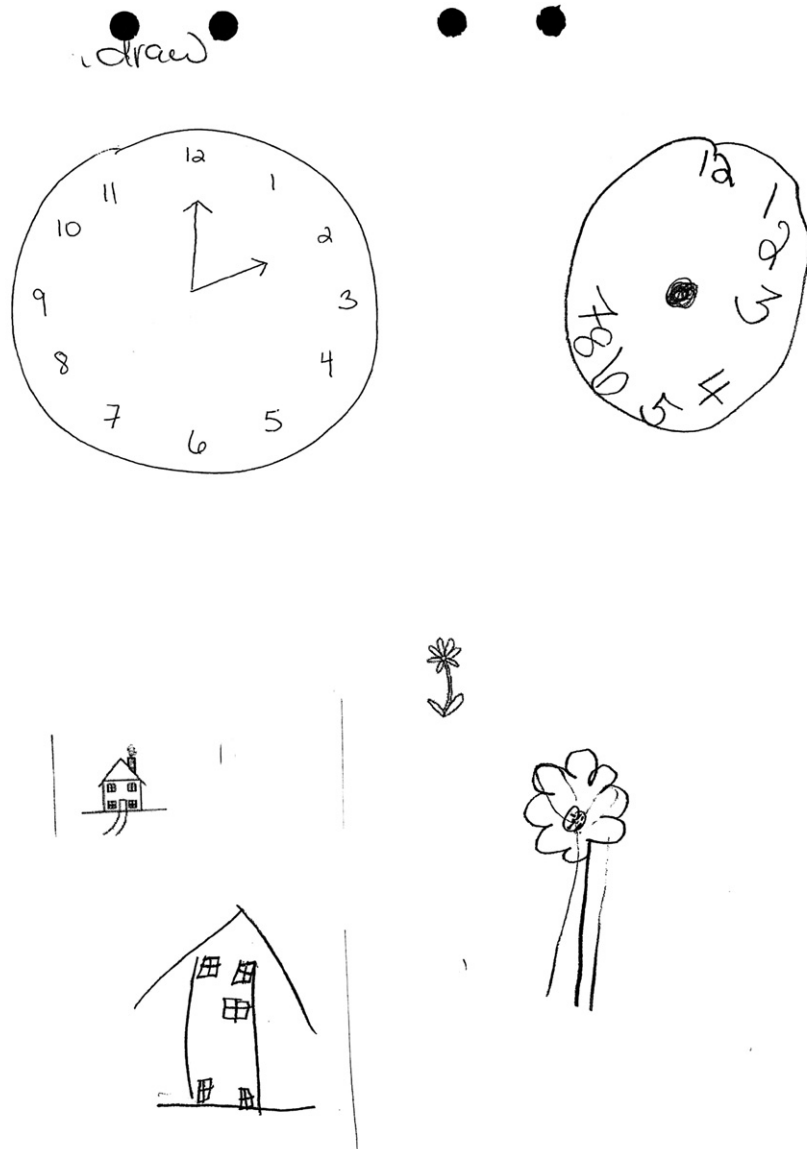


Figure 6 Picture copying task, patient 3.

attention problems diagnosed. Oculomotor and scanning therapies were prescribed for the oculomotor dysfunction and hemianopsia, respectively. The patient was instructed to continue visual motor integration therapy with his OT and was referred for neuropsychological testing because of his attention issues. A follow-up examination 6 months later found improvement in attention and reading skills and stable binocular findings. The patient is currently undergoing neuropsychological testing, and his dosage of methylphenidate has been adjusted since his last eye examination.

Case 4

A 3-year-old white boy had right-sided cerebral vascular accident (CVA) diagnosed in utero. He subsequently had cerebral palsy and seizures diagnosed. At the age of 2, in February 2006, he was examined by a pediatric

ophthalmologist and had a left hemianopsia diagnosed. In November 2006, the patient underwent a functional hemispherectomy to help control his seizures. For this procedure, the right parietal, temporal, and frontal lobes were removed (see Figure 7). In February 2007 he was examined by the same ophthalmologist and referred for an optometric evaluation of the visual field defect.

Initial optometric examination

At the optometric examination, the patient’s parents had no concerns other than the child’s overall left-sided weakness. He had been seizure free since the surgery. His parents reported that he would occasionally bump into objects on his left. Since birth, they had been aware of possible sequelae of the CVA and had been working on improving left awareness (i.e., placing objects on the patient’s left

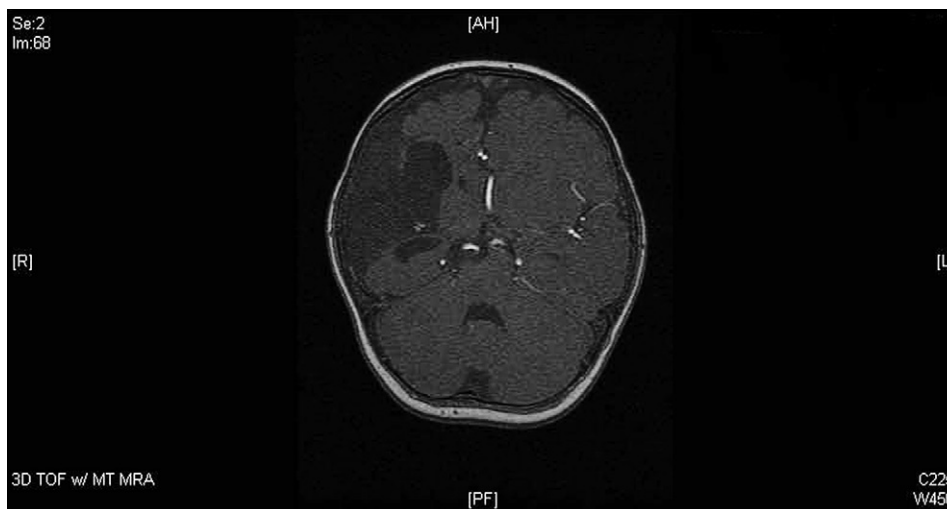


Figure 7 Computed tomography scan of patient status post right hemispherectomy of parietal, temporal, and frontal lobes, patient 4.

and encouraging him to find them, having him wear a band on his left hand). His current medication included dilantin.

Uncorrected entering visual acuities were 20/25 in each eye with the HOTV letter matching chart. Cover testing showed orthophoria at distance or near. Near point of convergence was to the nose. Motilities were full and comitant. Refraction showed age-appropriate hyperopia and astigmatism. Visual field testing with finger puppets showed a left hemianopsia greater superiorly than inferiorly. Stereopsis testing revealed 200" Randot with the Lang test. The patient displayed no postural shifts or body tilts when sitting or walking.

The patient had a left hemianopsia and overall left-sided weakness diagnosed. The patient's parents were instructed to continue working on increasing the patient's left-sided peripheral awareness. At that time, it was felt that much of the patient's left-sided weakness was associated more with his cerebral palsy than with the visual field defect, although the visual field defect could be contributory. The patient was referred for physical therapy, and the physical therapist was educated about the visual field defect.

Discussion

The 4 patients in this report all suffered from internal causes of brain injury and interruptions of the oxygen supply: hypoxia, CVA (in 2 patients), and encephalitis. Additionally, 2 patients underwent brain surgery to treat the initial disorder.

CVAs in childhood can be divided based on age into neonatal/perinatal stroke, occurring between 28 weeks of gestation and 28 days postnatally, and childhood stroke, occurring between 29 days and 18 years of age.³⁰ The rate of neonatal stroke (including ischemic and nonischemic) is approximately 1 in 4,000 live births per year. Traditionally, the annual incidence of childhood stroke has been reported to be 2 to 3 per 100,000 children.^{30,31} However, data from

the National Hospital Discharge Survey from 1980 to 1998 reveal higher rates: the overall rate of stroke in children 0 to 18 years is approximately 13.5 in 100,000, with ischemic stroke occurring almost 2.7 times more than hemorrhagic.³⁰ One study from a California-wide hospital database found a fairly equal distribution between the rate of pediatric ischemic and hemorrhagic strokes.³¹ The same study found that males and blacks had a higher risk of stroke than females and other races.

In these case reports, the 2 oldest patients displayed exo-type deviations. One of the patients had a convergence insufficiency and the other an intermittent exotropia. Both patients did complain of decreased visual acuity, which improved to 20/20 with an updated eyeglasses prescription. The 7-year-old child did not display any visual acuity, convergence, or accommodative problems but displayed problems with ocular motility, whereas the 3-year-old child did not display any refractive, binocular, or motility problems. However, all patients had either a visual field defect or unilateral spatial inattention.

In these case reports, 2 of the patients were on medications that have ocular side effects. Both dantrolene and divalproex have diplopia as a side effect, whereas risperidone, sertraline, and methylphenidate have nonspecific side effects listed.²³ The patient taking sertraline was symptomatic for blurry vision and diplopia and was found to have an increase in myopia and a convergence insufficiency. The patient taking the divalproex and methylphenidate was asymptomatic; however, the examination found a disorder of saccadic eye movements.

In a study of brain-injured patients on medication, Han et al.³² found that in patients with a history of CVA (age 24 to 90; average age, 61), 37.5% on medications and 25% not on medications displayed a vergence problem. For patients with a history of TBI (age 8 to 91; average age, 45), 55% of each patient group (those who were on medications and who were not taking medications) displayed a vergence problem. The exact percentages varied by class of medication. The researchers looked at other diagnoses, including

accommodative dysfunction, version disorders, and dry eyes. Overall, the researchers concluded that in most cases, the frequency of symptoms and diagnoses were related more to the brain injury than the medications. They recommended that when examining brain-injured patients, the history and examination should include evaluations of these areas and symptoms.

Visual field defects have been reported in 20% to 41% of acquired brain-injured patients from age 6 months to 70 years.^{3,7,9,10,20} Scattered defects, hemianopsias, and tubular visual fields have been reported.^{10,20} In a study of homonymous hemianopsias in pediatric patients, 34% resulted from traumatic brain injury, 27% from tumor, 23% from infarction, and 7% from cerebral hemorrhage.¹⁰ The authors felt that visual field defects are “underrepresented” in pediatric patients with brain injuries, primarily because children may not complain of vision problems and because many children have more serious neurologic problems that are addressed first. In the same study, 39% of patients showed an improvement of their visual field defects within 3 to 6 months after the injury.

In this case report series, 2 of the 4 patients, specifically the patient with the AVM and the patient with the encephalitis, exhibited hemianopsias. The youngest patient, with a CVA in utero, displayed a suspected hemianopic visual field defect to targets but was too young for formal automated visual field testing. It is expected that this patient will also display a visual field defect on formal testing. The oldest patient, with the history of anoxia from a drug overdose, was thought to have a left hemianopic visual field defect but had USI. In this case, although the visual field displayed scattered central defects, it was unreliable, and the existence of the defects could not be confirmed with repeat testing.

USI, also known as *visual neglect*, is a condition in which a patient is not aware of events occurring in the contralesional side of space.^{24,33} For example, patients may not be aware of impairments to contralesional limbs, they may not acknowledge items presented to the contralesional side, and they might ignore grooming one side of their face. One technique for testing for the presence of USI is confrontation visual fields. Patients with this condition will respond to a target presented in one hemifield but will be less sensitive to a target in the affected hemifield when two hemifields are stimulated simultaneously.³⁴ Because neglect implies willfulness, and because this condition is not willful, the preferred term is *unilateral spatial inattention*.²⁴ USI is more common in patient with a CVA, especially if the middle cerebral artery of the right inferior parietal lobe is affected. It is present in up to 33% of patients 1 year after having a stroke.³⁵

Treatment of adult patients with hemianopic visual field loss and unilateral spatial inattention can include yoked or sectoral prisms or scanning training.^{26-29,34} For hemianopic visual field defects, the recommended prism power varies from 6 to 20 diopters depending on the design of prism fit (e.g., full field, sectoral), whether the prism is fit

binocularly or monocularly, whether the prism is for distance or near tasks, and patient response.^{26-29,34} For a more detailed discussion on the fitting of prisms for visual field defect, the reader is referred to additional sources.^{26-29,34}

In this case series, for the patients with visual field defects, sectoral prisms were only attempted with the 16-year-old patient. Prisms were not attempted with the 8-year-old patient because of his attention or on the youngest child, as he was asymptomatic. The 16-year-old patient did not notice an improvement in her scanning ability with the prism glasses and did well with scanning activities alone. It is possible that the patient was not a good candidate for prism therapy based on her age and binocular status. Cohen²⁸ notes that patients who would benefit from yoked prism lenses are those between the ages of 25 and 65 and those without binocular vision problems. (This patient had an intermittent exotropia.) In this case series, all of the patients with visual field defects who were treated did well with visual scanning activities, showing an improvement in their ability to function, despite their visual field defect, and to develop compensatory strategies.

Prisms were also used with the patient who had the USI in an attempt to improve awareness of her affected side. When treating patients who have USI for activities in peripersonal space, Suchoff and Cuiffreda recommend starting with 20 prism diopter rotating yoked prisms with the base facing the defect (e.g., base left for left USI). As the patient progresses in therapy, the power of the prism can be decreased. He recommends that therapy include pen and paper tasks, reading tasks, and computer programs. Other researchers have found that after 3 minutes of exposure to 20-prism diopter yoked prisms while doing pointing tasks, patients displayed an improvement in drawing and mental imagery 24 hours after prism removal.³⁶ Although many studies used base left prism for cases of left USI, other investigators have used base right prism with the same effect.³⁷

In this case report, in spite of treatment, the patient still manifested USI during various near tasks. One reason for this finding could be that the patient was first evaluated by an optometrist almost a year and a half after the initial injury without prior treatment, and therefore the USI was “embedded.” Suchoff and Cuiffreda²⁴ note that patients with severe USI may not respond well to prism because they “are asked to look into a part of space that for them does not exist.” It is also possible that the patient was sensitive to the blur and distortion from the prisms, perhaps compounded by her convergence insufficiency.

Two of the patients in this case report series displayed an increase in myopia. One patient did not have a prior history of glasses and required a low myopic prescription to see clearly, whereas another had a prior history of eyeglasses for myopia. Suchoff et al.⁷ noted that 50% of patients with TBI had a need for glasses, either first-time glasses, replacement glasses, or a prescription change. More recently, Leslie³⁸ found that 13 of 15 patients (87%) with

a history of TBI and without a history of myopia pretrauma, had myopia ranging between 0.75 and 2.25 diopters after injury. Other investigators have also noted an increase in myopia after brain injury.^{39,40}

Children and acquired brain injury

Children who suffer from acquired brain injury frequently require long-term hospital stays. One study reported that pediatric patients who suffer from a TBI stayed in a hospital an average of 4.5 days; however, the range of inpatient days was 0 to 291.¹² After the initial hospitalization, many patients, including those in this study, receive some type of outpatient rehabilitation, generally occupational, physical, and/or speech.

Traditionally, pediatric patients are thought to have a better prognosis for recovery than adults, with many children showing dramatic recovery during the first year after ABI.^{14,41} A study of patients who suffered mild brain injuries as children and adolescents found that there were no long-term adverse effects on cognition or academic function 25 years after the initial injury.⁴² However, many children later displayed a “halting or slowing in later stages of cognitive, social, and motor development beyond a year after brain injury,” known as *neurocognitive stall*.⁴¹ Researchers hypothesize that neurocognitive stall may manifest in adolescence and is more likely to occur in patients with severe brain injury and in patients with frontal lobe and subcortical lesions.

Other long-term sequelae of brain injury in children and adolescents include later learning difficulties and behavioral problems.^{14,43} Recent research has found that children with a history of severe TBI at ages 2 to 7 may continue to manifest difficulties in attention 5 years after the injury.⁴⁴ Vision problems, though not specifically studied, have been reported to be present years after the injury. One study on pediatric patients 10 years after injury found that self-reported vision problems were the second most common problem (after mobility) in patients with a history of “serious” brain injury.⁴⁵ Unfortunately, the investigators did not define what was meant by “vision problems,” so it is not known if the respondents were having difficulty with acuity, diplopia, contrast sensitivity, or visual fields.

Conclusion

Pediatric patients with acquired brain injury appear to manifest signs and symptoms of vision problems similar to those of adults. In the preceding case studies, the patients presented with problems with visual acuity, visual fields, tracking, and convergence, including exotropia. It is important to note that in this case series there appears to be a correlation between the visual signs and symptoms noted during the examination with the patient’s age at the

time of brain injury. For example, the youngest patient did not present with any oculomotor problems or symptoms. The 8-year-old child manifested problems only with saccades. The teenage children were both very symptomatic, and both manifested problems with convergence and tracking.

Three of the 4 children were having difficulty with academics at the time of their evaluation. In many cases, it is possible that the vision problems were contributing to the academic difficulties, such that a child who is diplopic or who has a visual field defect or tracking problem may have reading difficulties.

These same 3 children were also receiving treatment for visual processing problems by occupational therapists. Visual processing problems include problems with skills such as visual memory, visual discrimination, and visual form constancy and can also contribute to problems with reading ability.^{46,47} As these problems were diagnosed and treated by the occupational therapists, they were not specifically addressed in this report. It is important to recognize that the diagnosis and treatment of visual processing problems is within the scope of practice of optometry and should be addressed in patients with a history of ABI as well.

For the above reasons, children with a history of head trauma should receive a complete optometric evaluation with attention to visual acuity, refractive error, binocularity, accommodation, motilities, and ocular health. Additionally, these children should receive visual field testing (automated, or formal, if possible) to determine if a defect exists. Visual perceptual testing should be performed, if indicated. Once the child’s visual status is known, the optometrist can communicate with other members of the rehabilitation team and make suggestions for therapy or accommodations.

These case reports illustrate that more research on pediatric patients with brain injury is required to determine common visual sequelae and any relationship to age or brain injury severity. It would be interesting to examine children who have had CVAs in utero in a separate study compared with children who suffered CVAs after birth to determine differences and similarities in visual field defects, presentations, and symptoms. Furthermore, long-term studies on pediatric patients with brain injury are needed to understand the prognosis for these patients and to determine whether the visual findings change over time with or without therapy.

References

1. Suhoff IB, Kapoor N, Ciuffreda KJ. An overview of acquired brain injury and optometric implications. In: Suhoff IB, Ciuffreda KJ, Kapoor NJ, eds. *Visual and Vestibular Complications of Acquired Brain Injury*. Santa Ana, CA: Optometric Extension Program, Inc; 2001:1-9.
2. Dimancescu MD. Neurosurgery and acquired brain injury. In: Elbaum J, Benson DM, eds. *Acquired brain injury: an integrative neuro-rehabilitation approach*. New York: Springer; 2007:4-17.
3. Poggi G, Calor G, Mancarella G, et al. Visual disorders after traumatic brain injury in developmental age. *Brain Injury* 2000;14:833-45.

4. Sabates NR, Gonce MA, Farris BK. Neuro-ophthalmological findings in closed head trauma. *J Clin Neuroophthal* 1991;114:273-7.
5. 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.
6. Ciuffreda KJ, Kapoor N, Rutner D. Occurrence of oculomotor dysfunction in acquired brain injury: A retrospective analysis. *Optometry* 2007;78:155-61.
7. 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.
8. Hellerstein LF, Freed S, Maples WC. Vision profile of patients with mild brain injury. *J Am Optom Assoc* 1995;66:634-9.
9. Barlow K, Thompson E, Johnson D, et al. The neurological outcome of non-accidental head injury. *Ped Rehab* 2004;7:195-203.
10. Kedar S, Zhang X, Lynn MJ, et al. Pediatric homonymous hemianopia. *JAAPOS* 2006;10:249-52.
11. Langlois JA, Rutland-Brown W, Thomas KE. *Traumatic brain injury in the United States: Emergence department visits, hospitalizations and deaths*. Atlanta (GA): Centers for Disease Control and Prevention, National Center for Injury Prevention and Control; 2006.
12. Schneier AJ, Shields BJ, Hosteler SG, et al. Incidence of pediatric traumatic brain injury and associated hospital resource utilization in the United States. *Pediatrics* 2006;118:483-92.
13. Reid SR, Roesler JS, Gaichas AM, et al. The epidemiology of pediatric traumatic brain injury in Minnesota. *Pediatrics* 2001;155:784-9.
14. Atabaki SM. Pediatric head injury. *Pediatr Rev* 2007;28:215-23.
15. Keenan HT, Runyan DK, Marshall SW, et al. A population-based study of inflicted traumatic brain injury in young children. *JAMA* 2003;290:621-6.
16. Agran PF, Winn D, Anderson C, et al. Rates of pediatric and adolescent injuries by year of age. *Pediatrics* 2001;108. e45.
17. Reece RM, Sege R. Childhood head injuries. Accidental or inflicted? *Arch Pediatr Adolesc Med* 2000;154:11-5.
18. Teasdale G, Jennett B. Assessment of coma and impaired consciousness: a practical scale. *Lancet* 1974;2:81-4.
19. Kay T, Harrington DE, Adams R, et al. Definition of mild traumatic brain injury. *J Head Trauma Rehabil* 1993;8:86-7.
20. Kraus JF, Rock A, Heymari P. Brain injuries among infants, children and adolescents. *Psych Med* 1989;7:11-6.
21. Dhaliwal A, West AL, Trobe JD, et al. Third, fourth, and sixth cranial nerve palsies following closed head injury. *J Neuro-Ophthalmol* 2006;26:4-10.
22. Kodsri SR, Younger BR. Acquired oculomotor, trochlear, and abducent cranial nerve palsies in pediatric patients. *Am J Ophthalmol* 1992;114: 568-74.
23. Micromedex Health Care Series. Available at: <http://www.thomsonhc.com/hcslibrarian>. Last accessed September 4, 2009.
24. Suchoff IB, Ciuffreda KJ. A primer for the optometric management of unilateral spatial inattention. *Optometry* 2004;75:305-19.
25. Stein JF. Representation of egocentric space in the posterior parietal cortex. *Q J Exp Physiol* 1989;74:583-606.
26. Weiss NJ, Brown WL. Uses of prism in low vision. In: In, Cotter SA, eds. *Clinical uses of prism—a spectrum of applications*. St. Louis: Mosby; 1995:279-99.
27. Cohen JM. An overview of enhancement techniques for peripheral field loss. *J Am Optom Assoc* 1992;63:60-70.
28. Cohen AH. Management of patients with hemianopic visual field loss. *J Optom Vis Dev* 2003;34:111-8.
29. Lee AG, Perez AM. Improving awareness of peripheral visual field using sectoral prism. *J Am Optom Assoc* 1999;10:624-8.
30. Lynch JK, Hirtz DG, DeVeber G, et al. Report of the national institute of neurological disorders and stroke workshop on perinatal and childhood stroke. *Pediatrics* 2002;109:116-23.
31. Fullerton HJ, Wu YQ, Zhao S, et al. Risk of stroke in children: Ethnic and gender disparities. *Neurology* 2003;61:189-94.
32. Han MH, Craig SB, Rutner D, et al. Medications prescribed to brain injury patients: A retrospective analysis. *Optometry* 2008;79:252-8.
33. Driver J, Vuilleumier P. Perceptual awareness and its loss in unilateral neglect and extinction. *Cognition* 2001;79:39-88.
34. Gianutsos R, Suchoff IB. Visual fields after brain injury: management issues for the occupational therapist. In: Scheiman M, ed. *Understanding and managing vision deficits—a guide for occupational therapists*. 2nd ed. Thorofare, NJ: Slack; 2002:247-63.
35. Allegri RF. Attention and neglect: Neurological basis, assessment and disorders. (Abstract). *Rev Neurol* 2000;30:491-4.
36. Rode G, Rossetti Y, Boisson D. Prism adaptation improves representational neglect. *Neuropsychologia* 2001;39:1250-4.
37. Padula WV. Neuro-optometric rehabilitation for persons with a TBI or CVA. *J Optom Vis Dev* 1992;23:4-8.
38. Leslie S. Myopia and accommodative insufficiency associated with moderate head trauma. *Opt Vis Dev* 2009;40:25-31.
39. Suter PS. Rehabilitation and management of visual dysfunction following traumatic brain injury. In: Ashley MJ, Krych DK, eds. *Traumatic brain injury rehabilitation*. Boca Raton: CRC Press; 1995:187-219.
40. Padula WV. Chapter VI: Neuro-optometric rehabilitation examination. In: Padula WV, ed. *Neuro-optometric rehabilitation*. 3rd ed. Santa Ana: Optometric Extension Program; 1988:78-87.
41. Chapman SB. Neurocognitive stall: A paradox in long term recovery from pediatric brain injury. *Brain Inj Prof* 2007;3:10-4.
42. Hessen E, Nestvold K, Sundet K. Neuropsychological function in a group of patients 25 years after sustaining minor head injuries as children and adolescents. *Scand J Psychol* 2006;47:245-51.
43. Keenan HT, Bratton SL. Epidemiology and outcomes of pediatric traumatic brain injury. *Dev Neurosci* 2006;28:256-63.
44. Catroppa C, Anderson VA, Morse SA, et al. Children's attentional skills 5 years post-TBI. *J Ped Psychol* 2007;32:354-69.
45. Horneman G, Folkesson P, Sintonen H, et al. Health-related quality of life of adolescents and young adults 10 years after serious traumatic brain injury. *Int J Rehab Res* 2005;28:245-9.
46. Groffman SG. The relationship between visual perceptual problems and learning. In: Scheiman MM, Rouse MW, eds. *Optometric management of learning-related vision problems*. St. Louis: Mosby; 2006:241-80.
47. Garzia RP. *Vision and reading*. St. Louis: Mosby; 1996.