Neuro-Optometric Treatment of Complications from a Cerebellar Astrocytoma
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ABSTRACT

Background
Insult to the brain, whether from trauma or other etiologies, can have a devastating effect on an individual. Symptoms can be many and varied, depending on the location and extent of damage. This presentation can be a challenge to the optometrist charged with treating the sequelae of this event as multiple functional components of the visual system can be affected.

The views expressed herein are those of the author and do not reflect the official policy or position of the U.S. Army Medical Department, Department of the Army, Department of Defense, or the U.S. Government.

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Keywords: cerebellum, diplopia, Fresnel prism, neuro-optometric rehabilitation, pilocytic astrocytoma

Case Report
This paper describes the diagnosis and subsequent ophthalmic management of an acquired brain injury in a 22 year old male on active duty in the US Army. After developing acute neurological symptoms, the patient was diagnosed with a pilocytic astrocytoma of the cerebellum. Emergent neurosurgery to treat the neoplasm resulted in iatrogenic cranial nerve palsies and a hemispheric syndrome. Over the next 18 months, he was managed by a series of providers, including a strabismus surgeon, until presenting to our clinic. Lenses, prism, and in-office and out-of-office neuro-optometric rehabilitation therapy were utilized to improve his functioning and make progress towards his goals.

Conclusions
Pilocytic astrocytomas are the most common primary brain tumors, and the vast majority are benign with excellent surgical prognosis. Although the most common site is the cerebellum, the visual pathway is also frequently affected. If the eye or visual system is affected, optometrists have the ability to drastically improve quality of life with neuro-optometric rehabilitation.

INTRODUCTION
Occam’s razor suggests that the simplest explanation is often the most likely and encourages clinicians to look for a single unifying cause to signs and symptoms. At times, this approach can result in substandard patient care because additional etiologies are not considered. Conversely, Hickam’s Dictum states that “patients can have as many diseases as they [darn] well please,” to remind us that often a constellation of symptoms and signs can come from multiple etiologies. In cases of brain damage, Occam tends to be correct as there is typically a single event (trauma, tumor, etc.) that causes a potentially wide range of seemingly unrelated signs and symptoms. Optometrists are often confronted

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The neurosurgeon performed a twist-drill craniostomy into the right lateral ventricle, implanted a catheter, and drained 60 cc of CSF to relieve the elevated pressure. He then drained the cystic space inside the cerebellar hemisphere through a suboccipital incision. Abnormal tissue was resected and the wound was closed. A pathology report of the abnormal tissue was consistent with pilocytic astrocytoma. A postoperative MRI was performed and two images from this scan are shown (Figures 1 and 2).

Postoperatively, RW began to manifest a variety of new signs and symptoms that were likely iatrogenic in nature. He complained of constant horizontal diplopia worse in right gaze. This was attributed to a right VI nerve palsy from an insult to the VI nerve’s pathway during neurosurgery. He manifested a complete right facial paralysis, including a lagophthalmos-induced exposure keratopathy causing pain and blurry vision at all distances in the right eye. This was attributed to a right VII nerve palsy also associated with his neurosurgical procedure. He additionally had balance deficits secondary to cerebellar ataxia, right-sided intention tremor, right hemiparesis, dysphagia, and dysarthria. These symptoms are consistent with cerebellar hemispheric syndrome, the unilateral triad of hypotonia, disequilibrium, and dyssynergia that results from an insult to one of the cerebellar hemispheres.

He was sent to an inpatient Veteran’s Administration (VA) Polytrauma Center where he engaged with multiple rehabilitation specialties in an interdisciplinary setting. At an optometric evaluation about one month after his neurosurgery, his complaint of constant horizontal diplopia remained. The optometrist identified a 20 prism diopter (pd) constant right esotropia with 5 pd right hypotropia at distance in primary gaze. This right hypoesotropia was also present at near but the magnitudes were not documented. Fresnel prism of 25 base out (BO) and 5 base

with multiple ophthalmic complications that require comprehensive evaluation and creative problem solving. This case report discusses the clinical course and treatment of cerebellar pilocytic astrocytoma in a young adult. In this case neurosurgery to excise the tumor resulted in a host of iatrogenic symptoms that required interdisciplinary rehabilitation, including neuro-optometry.

CASE REPORT

A 22 year-old white male, RW, was referred from his primary concussion care manager to the neuro-optometry clinic for evaluation of ophthalmic complications after removal of a cerebellar tumor. His case was complex with multiple rehabilitation providers addressing a constellation of sequelae that occurred after removal of the tumor.

One year prior to his first exam with the author, RW presented to his primary care clinic complaining of an intermittent, severe headache with dizziness, nausea, and vomiting that had been worsening over the previous three months. Computed tomography (CT) of the head revealed a low-density mass in the right cerebellar hemisphere extending into the vermis at the midline of the cerebellum. The mass blocked the cerebrospinal fluid (CSF) at the level of the foramen magnum causing enlargement of the lateral and third ventricles and displacement of the fourth ventricle. This obstructive hydrocephalus was an emergent condition and the patient was sent directly to a neurosurgeon.

Magnetic resonance imaging (MRI) of the brain performed prior to neurosurgery was remarkable for a 6.5 cm cystic mass centered at approximately the right middle cerebellar peduncle. There was mass effect on the posterior fossa structures and brainstem, displacing the cerebellar tonsils approximately 1.3 cm below the foramen magnum. The optic nerves and sheaths demonstrated tortuosity, likely secondary to the hydrocephalus.

The neurosurgeon performed a twist-drill craniostomy into the right lateral ventricle, implanted a catheter, and drained 60 cc of CSF to relieve the elevated pressure. He then drained the cystic space inside the cerebellar hemisphere through a suboccipital incision. Abnormal tissue was resected and the wound was closed. A pathology report of the abnormal tissue was consistent with pilocytic astrocytoma. A postoperative MRI was performed and two images from this scan are shown (Figures 1 and 2).

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Table 1. Cover test, right and left eye horizontal muscle restrictions, and prism rescribed for each exam.

<table>
<thead>
<tr>
<th>Location</th>
<th>Cover Test D</th>
<th>Cover Test N</th>
<th>R Abduction Restriction</th>
<th>R Adduction Restriction</th>
<th>L Adduction Restriction</th>
<th>L Abduction Restriction</th>
<th>Prism in Glasses</th>
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<td>Texas Polytrauma</td>
<td>CRET’, R Hypo</td>
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<td>0%</td>
<td>100%</td>
<td>0%</td>
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<td>Not documented</td>
<td>55 BO</td>
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<tr>
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<td>40 BO</td>
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<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>40 BO</td>
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<td>0%</td>
<td>0%</td>
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<td>40 BO</td>
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<td>Neuro-Optometry s/p second procedure</td>
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<td>25%</td>
<td>50%</td>
<td>50%</td>
<td>20 BO, 4 BU OD</td>
</tr>
</tbody>
</table>

Figure 1: Sagittal T1 image of RW’s right side, status post neurosurgery. The red arrow is pointing to the section of cerebrum missing after the creation of the ventriculoperitoneal shunt entering the right lateral ventricle. The blue arrow shows a small section of missing cerebellum after the tumor was resected.

Figure 2: Another T1 sagittal view, this one closer to midline but still on the right side. The red arrow again points to the channel from the shunt, and the blue arrow points at the much larger midline area of missing cerebellum.
performed but magnitudes were not measured. With the Fresnel in place he demonstrated a right hyperesotropia at distance and near, suggesting overcorrection of his vertical deviation and undercorrection of the eso deviation. His prism was reassessed and changed to 40 BO OD and 3 BU OD, which resulted in “slightly overlapped” images. A 100% abduction deficit was still present OD, but the OS now demonstrated “difficult adduction” with no specific magnitude documented.

Two weeks later RW reported that the images were further apart again. Cover test with the Fresnel prism removed revealed approximately 45 pd esotropia at distance and near. His vertical component was not documented. The vertical prism was removed from the right lens and replaced with a 15 BO for a total of 55 BO and no vertical. This resulted in overlapped images with no vertical component. Eye movement testing showed a stable 100% abduction deficit OD with no documentation concerning the OS. The patient was discharged from the polytrauma center with no additional eye exam recommendations.

One month later RW presented to the local military optometry clinic for prism re-evaluation. He again complained of constant horizontal diplopia. Cover test with no prism in place was documented as greater than 60 right esotropia at distance and near. His vertical component was not documented. The vertical prism was removed from the right lens and replaced with a 15 BO for a total of 55 BO and no vertical. This resulted in overlapped images with no vertical component. Eye movement testing showed a stable 100% abduction deficit OD with no documentation concerning the OS. The patient was discharged from the polytrauma center with no additional eye exam recommendations.

One month later RW presented to the local military optometry clinic for prism re-evaluation. He again complained of constant horizontal diplopia. Cover test with no prism in place was documented as greater than 60 right esotropia at distance and near with no vertical deviation. A 100% right abduction deficit was still present though no left adduction deficit was noted. A total of 80 pd BO was split between the two eyes (40 pd OD and 40 pd OS), which produced an approximate sensory alignment response. Fresnel prisms were dispensed with 40 pd BO OD and 40 pd BO OS. The patient was instructed to follow up with an eye care provider after his impending move to Fort Bragg, North Carolina.

He presented to the neuro-optometry clinic at Fort Bragg six weeks later where a resident other than the author examined him. His symptoms of constant binocular diplopia were unchanged. The Fresnel was not present up (BU) over the right eye (OD) placed the images from the two eyes overlapping but not fused. The prescribing doctor did not offer a hypothesis for the reduced fusion; it may be a combination of reduced VA in the OD along with reduced control of the extraocular muscles. Cover test findings and Fresnel prism prescribed can be seen in Table 1, which organizes pertinent exam data by visit.

A right gaze palsy was present, consisting of a complete abduction deficit OD and complete adduction deficit OS. Pursuits were jerky and saccades were reduced in velocity. In primary gaze, a binocular downbeat nystagmus was present, which worsened with occlusion of the OD more than the OS. He also had gaze-evoked nystagmus with a torsional component greater in magnitude OD than OS.

Entering VA with habitual correction was OD: -5.50-0.50x015 20/60+2 and OS: -5.50-0.50x175 20/60+2 with no improvement on pinhole. Best corrected vision on refraction was 20/40 OD with -6.75-1.25x172 and 20/30+2 OS with -7.25-1.00x170. Historically, his refraction upon entry to the Army was OD: -5.00-1.75x005 20/20- and OS: -5.25-1.25x175 20/20. Damp autorefraction revealed a prescription of -4.00-1.00x006 OS and no data OD which was attributed to the disrupted ocular surface. This suggested that the patient was significantly overminused due to excessive accommodation. Ocular health examination revealed 3+ conjunctival injection and 3+ punctate keratitis with incomplete blink OD and 1+ injection with trace keratitis OS. The doctor prescribed Fresnel prism (25 BO and 5 BU on the right lens) on the habitual glasses, along with ocular lubrication and moisture chamber goggles. RW was also referred to occupational therapy to work on eye movement procedures. The Polytrauma Center did not have a comprehensive vision rehabilitation therapy program and as a result, the therapy he received was limited in scope.

At follow up two weeks later RW reported worsening horizontal diplopia. Cover test was
on the left lens. The record did not discuss the missing Fresnel prism on the left lens, but one interpretation is that the visual blur in the better seeing OS induced by the Fresnel prism was an intolerable functional limitation and the patient removed it. It is also possible that reducing the total prism increased separation of the diplopic images rendering the blurry OD image easier to ignore. Cover test through the 40 pd BO Fresnel on the right lens revealed 7 pd right esotropia at distance and near in primary gaze with no vertical. His right abduction deficit was stable with full ductions OS. These exam findings resulted in the prescription of 46 pd BO; 6 pd BO ground-in OD and subsequent application of 40 pd BO Fresnel OD. The patient had been selected for an interdisciplinary rehabilitation program at a different VA polytrauma center and would be gone for a couple of months to complete that program. He was instructed to follow up with neuro-optometry when he returned.

At the polytrauma center, he attended a few sessions of limited vision rehabilitation consisting mainly of Brock string procedures, eye movement procedures, and binasal occlusion. After 9 weeks his diplopia remained unchanged and binasal occlusion did not decrease the amount of strabismus. The optometrist at the center performed an examination 8 weeks after RW arrived and recommended glasses with applied Fresnel prism along with a strabismus surgery consult due to the limited success with other treatments. Cover testing revealed 53 pd right esotropia at distance and 45 right esotropia at near without prism in place. The right abduction deficit remained 100% with full movement of the left eye.

After his discharge from the VA, the patient had strabismus surgery on the right eye. It consisted of transposition of the superior and inferior recti to address the vertical deviation and recession of the medial rectus to address the abduction deficit. At the surgeon’s three month follow up, he documented that RW manifested a 40 pd right esotropia in all nine positions of gaze at distance without prism correction even though he also documented that RW’s right abduction remained 100% restricted with unrestricted ductions OS. These two findings are contradictory, as the restricted abduction should produce a large noncomitant deviation. With a 50 pd BO prism in place the patient reported single vision from far left gaze to 25 degrees across midline into right gaze. The surgeon recommended an additional procedure in one month to reduce the deviation further.

Before his second surgery occurred, he returned to the neuro-optometry clinic and was seen by the author for the first time. He reported a slight constant horizontal binocular diplopia that he was gradually learning to ignore. He used artificial tears during the day and moisture chamber goggles with ointment at night. His symptoms of dry eye were tolerable. Through his habitual glasses with 40 BO prism, a 14 pd constant right esotropia was identified at distance and 6 pd constant right esotropia at near. The right abduction deficit was stable. On Worth 4 Dot testing while wearing the Fresnel prism with normal room illumination the patient suppressed the OD at distance, had uncrossed diplopia at intermediate range and had overlapping images at 14 cm. Minimal nystagmus was noted in primary gaze that increased to an obvious left-beating nystagmus in left gaze. In right gaze no nystagmus was noted OS while the OD remained at midline. Pursuits were not smooth with several saccadic intrusions. As he was already scheduled for more surgery, further testing was not performed since his alignment would change significantly. The patient was instructed to return to clinic three weeks after this second surgery.

At the post-surgical evaluation, RW presented reporting constant diplopia in all fields of gaze and at all distances. He also reported a cosmetic improvement in the misaligned eyes. He had undergone a
rerecession of the right medial rectus and a new recession on the left medial rectus. He had no Fresnel prism on his glasses. Cover test revealed a 20 pd constant right esotropia with 4 pd right hypotropia at distance and near. Ductions had changed significantly after the second procedure. The OD was now 75% restricted for abduction and 25% restricted for adduction. The OS was 50% restricted for both. On Worth 4 Dot, he reported uncrossed diplopia at all distances. Worth 4 Dot was repeated with 20 BO and 4 BD OS prism and he reported completely normal fusion at all distances. His new manifest refraction was OD: -5.25-1.50x003 20/25 and OS: -5.75-1.00x003 20/20-2. This was more consistent with his pre-injury refractive status and vision. The treatment plan was to order glasses with 2 pd BU OD and 2 pd BD OS ground into the lenses. Fresnel prism would be applied to compensate for the horizontal deviation. We intended to reduce the magnitude of horizontal prism over time as neuro-optometric rehabilitation therapy improved RW’s binocular fusion ability.

The patient spent the next four months attending once weekly in-office therapy sessions for an hour, with concurrent out-of-office therapy three days a week for 15 minutes. His out-of-office therapy consisted of three procedures. First, monocular post-it saccades in all directions OD and OS. Second, spoon pursuits with horizontal and vertical target movement, first with eye movement only and then with head movement only (to engage the vestibulo-ocular reflex). Lastly, Brock string to improve his range of binocular fusion. When his fusional abilities improved, he added jump ductions with left and right head turns to engage vergence outside of primary gaze.

In office, each session began with using the Brock string to delineate his range of binocular fusion. At his first measurement, he was able to fuse successfully without suppression only between 50 and 75 cm from his nose. After 4 months of treatment, his best-recorded values were 5.5 cm at near and 126 cm at distance, meaning that he had single vision within and just beyond arm’s reach. He was also able to maintain single vision with a head turn of 10 degrees to the right and 20 degrees to the left for 40 cm beads.

His saccadic training was performed using the Sanet Visual Integrator Saccades I program. Initially this was done monocularly, but after the patient’s Brock string range improved and he was single within arm’s reach it was changed to a binocular procedure. The targets were weighted 75% to the right in order to encourage more abduction in the OD. He alternated between letter and number targets that persisted, switching hands with each target press to work laterality, cognitive ability, and motor coordination on his weaker right side. After a few months, his balance was improving with physical therapy and a balance board was incorporated for further vestibular integration.

At week 4, the Quoit vectogram was attempted. However, RW suppressed his OD constantly and he was not able to successfully perform the procedure. RW started anti-suppression training with GTVT cards to encourage simultaneous perception. On his second session using the GTVT card, he was able to sit at a distance of about 4 feet away and see both sets of letters simultaneously. After this occurred, vectograms were reintroduced and RW was able to maintain a single image without suppression between E (BI) and 7 (BO). Additionally, he reported noting changes in the spatial localization of the target, though no change in the image’s size.

The patient also performed accommodative therapy using monocular accommodative flippers. With each eye, he was able to successfully clear a +/-0.75 flipper by the end of his four months of rehabilitation therapy. RW found this procedure difficult as it depends upon clear vision in order to know when to flip the lens. With the right eye, RW was always slightly blurry (20/25) which led to frequent uncertainty if he had accurately
accommodated. The Fresnel prism was removed for monocular procedures and did not play a role in the blur.

Lastly, his horizontal prism was reassessed at week seven. We were able to reduce the Fresnel prism from 20 BO to 18 BO with no loss of comfort. The vertical prism remained stable. These glasses were ordered with the prism ground in for clearer vision OD, which likely contributed to his ability to report fine fusion in the subsequent weeks of treatment.

Based on the progress observed, a re-evaluation of RW will be completed with another reduction of the horizontal compensating prism if possible. Vision rehabilitation will continue to focus on building his fusional vergence ranges with vectograms in primary, left, and right gazes. Pursuits, saccades and accommodation will also be addressed as secondary areas of focus. RW’s goals at his first exam with the author included driving and going back to college to study engineering. The addition of stable single vision at distance would aid RW during both of these activities. Though he has demonstrated remarkable improvement, maintaining single vision in primary gaze at all distances continues to be our overarching goal. Three to four more months of vision rehabilitation were recommended to maximize his field of single vision and normalize his accommodation and eye movements.

DISCUSSION
The brain and central nervous system are the most common sites for pediatric (age 0-19) neoplasm development with involvement in these locations about 25% of the time. For adults (age 20 and up) it is one of the least common areas of involvement. Pilocytic astrocytoma (PA) is the most common histological type of neoplasm accounting for 15-20% of primary brain tumors in those under the age of 20. The annual incidence rate is between 8.4 to 8.7 per million per year for ages 0-19 with gender preference either nonexistent or slightly leaning towards males. PA can occur at any age, but the incidence decreases tenfold from the 0-14 age group (9.3 per million) to the age 40+ group (0.9 per million). The cerebellar hemispheres are the most common location for PA’s at 42% but they also occur at the optic chiasm and hypothalamus, other sections of the optic pathways, cerebral hemispheres, spinal cord, and brainstem.

Pilocytic astrocytoma is a World Health Organization (WHO) grade I tumor on a scale from I to IV. The WHO grading is an estimate of malignant potential with grade I being essentially the same as benign. Surgery is the treatment of choice as the vast majority of these tumors can be surgically resected successfully with 5 and 10 year survival rates greater than 95%. However, location of the tumor affects survival rate with cerebellar tumors showing the best rate and tumors of the hypothalamus and optic chiasm showing the worst. This is most likely related to the ability of the surgeon to completely resect the tumor, as total resection significantly improves prognosis. Based on anatomy, it is much easier to gain access to the cerebellum on the posterior aspect of the skull than the hypothalamic region in the center of the brain. Recurrence rates are 10-20% if a complete resection occurs but increases to 50-80% with partial resection.

Less than 20% of all cerebellar astrocytomas are considered “diffuse,” which means they are either a separate class of tumor (diffuse astrocytoma (DA), WHO grade II) or are PAs with a diffuse growth pattern (pilocytic astrocytoma, diffuse variant). Diffuse astrocytomas are slow-growing but have a higher likelihood of malignancy, resulting in grade II classification. PA, diffuse variant, has similar outcomes to standard PA. PA diffuse variant is morphologically similar to DA when examined, which means genetic testing of a biopsied sample is required to properly diagnose.

Histologically, PAs have well-defined borders, low to moderate cellularity, elongated
nuclei, cells with bipolar processes, Rosenthal fibers, and mucoid material with microcysts.\textsuperscript{2,3,11} The term “pilocytic” comes from the fact that the cells’ bipolar processes resemble hairs.\textsuperscript{2,3} On CT, PA is a well-defined lesion that enhances well with iodine contrast and is iso- or hypodense. Similarly, they are iso- or hypointense on T1 weighted MRI, hyperintense on T2, and enhance well with gadolinium.\textsuperscript{3}

Symptoms of PA in the cerebellum can include ataxia and symptoms from increased intracranial pressure such as cranial nerve palsies, headache, nausea, and vomiting.\textsuperscript{3} This was the case for RW, who developed hydrocephalus secondary to his PA and then rapidly developed these symptoms. Tumors in the cerebellar hemispheres, where PAs normally form, can produce a hemispheric syndrome which includes the cerebellar dysfunction triad of hypotonia (loss of muscle tone), disequilibrium, and dyssynergia (loss of muscle coordination). Dyssynergia includes nystagmus and hypo- or hypermetric saccades.\textsuperscript{12} If the PA affects the vestibulocerebellum, located in the ventral portion of the cerebellum abutting the pons, a number of eye movement abnormalities can occur. These include multiple types of nystagmus when looking to the same side as the lesion, a downbeat nystagmus that often worsens when looking laterally and downward, reduced smooth pursuits, vestibulo-ocular reflex reduction, and drifting away from fixation after making a saccade.\textsuperscript{13}

The systemic disorder most commonly associated with PA is Neurofibromatosis Type 1 (NF1). NF1 is the most common genetic disease affecting the nervous system and causes tumors to grow in any part of the nervous system. Ocular signs include Lisch nodules on the iris, as well as astrocytic neoplasms anywhere along the visual pathway.\textsuperscript{14} About 10\% of all PAs are secondary to NF1, and about 15\% of NF1 patients have PA.\textsuperscript{2} When associated with NF1, PA can show spontaneous regression without surgery or with partial resection.\textsuperscript{9}

**SUMMARY**

Pilocytic astrocytoma, though the most common brain tumor, is frequently benign and carries an excellent prognosis. As the visual pathway and cerebellum are two of the more commonly affected locations, there is potential for this neoplasm to cause visual dysfunction. Careful attention to neurological symptoms, eye movements, optic nerves, and visual fields can assist your diagnosis of a serious etiology that requires imaging and possibly an emergent referral. Following surgical intervention, optometrists are poised to help these patients recover from the neurological sequelae of excised tumors by utilizing all of the options at our disposal: lenses, prisms, filters, and vision rehabilitation.

In this case, RW suffered from multiple complications affecting his eyes and visual system. This required management across the spectrum of optometric scope of practice. Glasses and prism laid the groundwork by providing maximally clear images to each eye and bringing the images as close together as possible. Treating the secondary exposure keratopathy improved the comfort and clarity of the right eye, improving the stimulus to fusion. Finally, training accommodation, vergence, eye movements, and balance improved RW’s functioning from constant diplopia to clear and single vision at near.

Patients like RW are not simple cases. Severe brain injury, whether traumatic or acquired, can result in massive deficits across multiple areas of functioning. The treatment goal for these types of patients is rarely to return them to normal functioning; this is not possible for the vast majority. Instead, therapeutic interventions must aim to improve their visual system as much as possible and celebrate the small victories. This is similar to the low-vision specialist who does not cure the retinal disease but instead uses strategies and tools to allow the patient to live with their disease better. If RW had not made his way to the neuro-optometric service, he would be constantly diplopic for
the rest of his life. With neuro-optometric and interdisciplinary intervention, he is able to go to school, meet his goals, and have confidence with navigating the spatial world. His trajectory is forever changed.

This case highlights the importance of neuro-optometric rehabilitation as an integral part of the interdisciplinary team treating brain injury. No other specialty is poised to truly manage visual sequelae after severe injury in the same way. Optometrists seeking to treat these patients should not be discouraged by the complexity of the patients; instead, manage what you are able to the best of your ability. Addressing even relatively minor visual symptoms can have a profound impact on patients’ quality of life and functioning.

REFERENCES


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