Visual Consequences of Optic Tract Damage

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Abstract
Background: Optic tract lesions can have devastating visual consequences. When insult is isolated to the optic tract, central acuity is typically spared and color vision is usually normal; the visual manifestation of damage is a visual field deficit.

Optic tract damage as a cause for homonymous hemianopia is relatively rare compared to other visual pathway lesions producing such a field defect, and presents with characteristic features. These features include a relative afferent pupillary defect in the eye contralateral to the lesion and pallor of that optic disc in a “band” or “bow-tie” configuration. Case Reports: Two patients with optic tract damage of different etiologies were examined. The first patient presented with concerns about blackouts and peripheral field deficits. Neuroimaging demonstrated a suprasellar meningioma compressing the left optic nerve and part of the optic tract. Prior to meningioma resection, threshold visual field testing revealed the presence of a right incongruous incomplete homonymous hemianopia.

The second patient sustained severe brain injury during a motor vehicle accident which left him in a coma for over one month. Upon awakening, he reported left-sided vision loss. Magnetic resonance imaging revealed damage involving the right optic tract, corresponding with a left homonymous hemianopia found on threshold perimetry. Conclusions: The optometrist should bear in mind that homonymous hemianopic visual field deficits can be caused by lesions in the optic tract, although damage to this portion of the visual pathway occurs infrequently. The unique clinical presentation of optic tract lesions enables the optometrist to make a preliminary diagnosis as to the location of the insult. This is especially important since lesions of the optic tract may be difficult to detect with neuroimaging.

Optometrists should also be aware of the impact that homonymous field loss can have upon activities of daily living and of the various management options that are available.

Introduction
Of all retrochiasmal lesions, optic tract damage remains relatively rare. Zhang and colleagues studied 904 retrochiasmal lesions. The location of the lesion was definitively determined in all cases using neuroimaging — either computed tomography (CT) or magnetic resonance imaging (MRI).1 They found the most frequent location for retrochiasmal insult to be the occipital lobe, accounting for 45% of lesions.1 In contrast, only 10.2% of lesions were at the optic tract. Multiple other studies show similar data, although not all used a definitive method, such as neuroimaging, for determining the area of insult. In a study of 140 patients with retrochiasmal damage, Fujino and colleagues found that only sixteen of these patients (approximately 11%) had lesions in the optic tract.2 Conversely, 71 of the cases in this study (51%) had insult to the occipital cortex.2 In this study, the location and properties of the insult were confirmed via surgery or CT in about half of the cases, while the others were deduced based on mode of onset, symptoms and visual field appearance.2 Smith’s well-known 1962 report of 100 cases of homonymous hemianopia mentions that only 3% of those cases were due to optic tract lesions, although the location of lesion was deduced based on the clinical features of those cases.3

Given the special way in which retinal ganglion cell axons are arranged, brain injuries affecting the visual pathway often produce characteristic defects helpful in localizing the lesion even before neuroimaging is performed.

As the retinal ganglion cell axons exit the globe as the optic nerve, they travel to the optic chiasm where fibers from the nasal retina, approximately 53% to 55% of all axons cross to the join the temporal fibers of the contralateral eye.4,5 Thus, lesions that affect the chiasm where the nasal fibers are crossing will often affect the temporal fields of both eyes, resulting in a bitemporal hemianopia.6,7 In contrast, a lesion affecting the visual pathway posterior to the chiasm results in a homonymous visual field defect, with the same half — either right or left — of the visual field being affected in both eyes.6,7 The optic tract is the segment of visual pathway that goes around the cerebral peduncle and lies...
between the chiasm and lateral geniculate body.\textsuperscript{4,7} It represents
the first location in which, upon insult, a homonymous field
defect will occur.\textsuperscript{5}

If the defects found in each eye are the same in size, shape, and
depth, the field is said to be congruous. Likewise, if there are
differences in size, shape, and depth between the visual field
defects of the two eyes, the field can be called incongruous.\textsuperscript{4}
A more anterior lesion classically produces an incongruous
defect, while a congruous homonymous hemianopia will
typically be localized to a lesion further posterior in the visual
pathway, at the visual cortex.\textsuperscript{3,4,6,7}

Two patients with optic tract lesions are presented. The first
case involves an extensive suprasellar meningioma; the second
case was a patient who sustained optic tract damage from a
severe head injury. The clinical features of optic tract lesions,
causes of optic tract insult, and management of patients with
hemianopic visual field deficits secondary to optic tract lesions
are discussed.

**Case Reports**

**Case 1**

A 55-year-old male presented to Neurosurgery Clinic with
a chief concern of lower back pain for the past two years.
Additionally, he reported that over the past year, he had
progressively worsening left lower extremity numbness and
right leg weakness with "foot drop," as well as visual deficits of
the left eye. The episodes of left eye vision loss were described
as "blackouts" occurring three to four times per day, sometimes
lasting all day, with headaches during these episodes. He
described the headaches as "throbbing" and located in the
left frontotemporal area. The leg weakness had deteriorated
to the point where he needed to walk with a cane. His wife
noted that his memory had been declining. His medical history
was otherwise remarkable for hypertension, benign prostatic
hypertrophy, degenerative disc disease, hyperlipidemia, and
obstructive sleep apnea. He was taking cyclobenzaprine
as a muscle relaxant, naproxen, simvastatin, and terazosin.
After further testing in clinic, the neurologist suspected a

multifactorial cause for the patient’s symptomology. Along
with orthosurgery, urology, and psychiatry consults, magnetic
resonance imaging (MRI) of the brain was ordered. The MRI
revealed a meningioma in the suprasellar cistern extending to
the hypothalamus and likely compressing the left optic nerve
along its cisternal segment, and the end of left optic tract could
not be visualized separately from the tumor (Fig. 1). Due to the
size of the tumor, an emergency neurosurgery consult was
requested, as well as an ophthalmology consult for perimetry.

Prior to the ophthalmology exam, the patient was seen by
neurosurgery, which evaluated the patient and recommended
resection of the meningioma. The risks and benefits were
discussed and the patient elected to proceed with the surgery.

At his ophthalmology examination, the patient reported left
eye and periorbital area pain which he rated a 3 out of 10. He
also reported headaches and that his left eye was bloodshot.
Entering visual acuity without correction was found to be
6/7.5 (20/25) OD and 6/12- (20/40-) OS. Pinhole yielded no
improvement in visual acuity in the right eye, while the left eye
improved to 6/9 (20/30). Extraocular muscle testing revealed
full versions. Confrontation fields were full, and the patient
reported equal saturation between eyes in the red-cap test.
Pupils were found to be normal and the patient did not have
an afferent pupillary defect (APD). A resolving subconjunctival
hemorrhage was discovered on the left eye with slit lamp
biomicroscopy and trace nuclear sclerosis of the lens was
noted in each eye; the anterior segment was otherwise normal.
Fundoscopic examination revealed a normal vitreous, macula
and periphery. Sectoral disc edema was noted in both eyes, but
there was no optic nerve head pallor. A Humphrey central 30-2
threshold visual field test showed a hemifield defect respecting
the vertical midline in both eyes (Fig. 2). The patient was started
on artificial tears, and had already been scheduled for resection
of the meningioma.

Following the suprasellar meningioma resection, he was noted
to have paresis of the right upper and lower extremities and
was not following commands. Computed tomography (CT)
performed the day following the surgery showed increased hypotenuation of the anteromedial left temporal lobe and posterior limb of the left internal capsule, suggesting a subacute infarct. A cerebral angiogram revealed focal stenosis of the proximal left M1 segment of the middle cerebral artery (MCA) in the region of the tumor and possible infarct at the left internal capsule. He was followed as an inpatient as his neurological status slowly improved.

Two weeks following the surgery, the patient was referred by neurosurgery to ophthalmology for evaluation of conjunctival chemosis of the left eye which started following the meningioma resection. The primary care team had prescribed ophthalmic lubricating ointment. The patient’s visual acuity was 6/30+ (20/100+) in the right eye and hand-motion in the left eye, although it was difficult to test given the patient’s aphasia. A left extraocular muscle restriction was present, but resolved by a follow-up visit five days later. In addition, a 1+ left APD was noted. Anterior segment exam using an indirect ophthalmoscope and twenty diopter power condensing lens revealed normal lids and lashes, a clear cornea, and a quiet anterior chamber. The conjunctiva was noted to have marked chemosis inferiorly OS. A left hemifield defect was found on Humphrey visual field testing. Dilated fundus examination revealed a normal disc, vitreous, macula, and periphery, although it was thought to be too soon after the surgery to determine what optic nerve changes may have resulted from the tumor. The conjunctival chemosis was presumed to be due to surgical manipulation in the left orbital apex. A conservative approach was taken, with the patient instructed to continue with the lubricating ointment four times daily (QID). In addition, an MRI with contrast using thin orbital cuts was ordered to rule out optic nerve compression.

The patient was seen by the neuro-ophthalmology service one week later to follow up on the chemosis and review the neuroimaging. At this visit, a 2+ APD was noted. Versions were full OD, but grossly limited in all fields OS and the patient was observed to have persistent severe left conjunctival chemosis. The neuroimaging showed extensive progression of pre-existing chiasmal compression postoperatively and a left internal capsule infarct. It was recommended that the patient continue with liberal application of lubricating ointment QID. In addition, it was suggested that cellophane (Saran) wrap be taped over the involved eye to maximize moisture retention. The patient’s visual prognosis was deemed to be poor.

Two weeks later, the chemosis still had not resolved and yellow-white mucoid discharge was also noted in the left eye. The left eyelid was taped closed to expedite resolution of the chemosis and all left eye medications were discontinued. The next day, the tape was removed and erythromycin ointment was started at a q2h frequency.

After more than one month of aggressive lubrication with erythromycin ointment and a combination of lid taping and shielding the eye, the chemosis resolved. Given the patient’s right hemifield defect, a referral was sent to the Low Vision Rehabilitation service for assistance with mobility and visual tasks.

Upon presentation to Low Vision Clinic approximately thirteen months post-operatively, the patient reported bumping into things and concern about falling due to inability to see objects on right side of his visual field. He also had difficulty reading and writing, specifically with signing documents.

His best-corrected visual acuity had improved to 6/6 (20/20) OD and 6/7.5+ (20/25+) OS. A complete right homonymous hemianopia post-surgically was demonstrated by both Humphrey threshold static perimetry and Goldmann kinetic perimetry with a III4e isopter, consistent with the patient’s history and the most recent post-operative MRI (Figs. 3, 4, 5). Dilated fundus examination revealed sectoral temporal disc pallor in the right eye and diffuse 2+ pallor in the left eye (Fig. 6). The macula, periphery, and background were normal.

To aid in field awareness, Fresnel prisms were applied to his spectacles in varying powers and positions. The patient had some success in-office with 30 prism diopeters base out over
the right half of his right spectacle lens, and he wore the prism home for an extended trial. Unfortunately, he was unable to adapt.

In addition to attempting field enhancement, recommendations were given to aid in reading and writing, such as highlighting the borders of paragraphs so he would know when he reached the end of a line of text and use of typoscopes for writing. He was also referred to Blind Rehabilitation Outpatient Services for a white cane and orientation/mobility training.

The patient's visual acuity and visual field loss remained stable over time, although after nearly two years post-operatively, he transferred his eye care to a clinic located closer to his home. Neuroimaging has remained stable as well, and he was recommended to be followed by neurology with a new MRI obtained every 6 months to check for regrowth of the tumor.

**Case 2**

A 58-year-old Caucasian male presented to the optometry clinic with concerns about decreased near vision. During the case history, he also reported constant loss of the "nasal" visual field since waking from a 37-day coma following a motor vehicle accident. The accident had occurred more than one year prior, and he reported that he had "split his skull open above his eyes."

His medical history was significant for longstanding depression and alcohol abuse, with three previous suicide attempts. In one of the attempts, he drove his vehicle off a cliff, which resulted in traumatic brain injury and the coma he described in the case history. His medications included acetaminophen, amitriptyline, aripiprazole, citalopram hydrobromide, guaifenesin, and mirtazapine.

The patient's unaided entering visual acuity was 6/12 (20/40) in the right eye and 6/15 (20/50) in the left eye. Versions were full. Confrontations were normal. Pupillary reactions were normal and no APD was present. With best spectacle refraction, the patient was 6/6 (20/20) in both the right and left eyes.

Slit lamp biomicroscopy revealed 2 papillomas on the right lower eyelid and mild blepharitis in both eyes. The patient also had diffuse superficial bilateral punctate keratitis. Intraocular pressure by Goldmann applanation tonometry was 10 mm Hg in the right eye and 12 mm Hg in the left. A Humphreys Central 30-2 threshold visual field test revealed an incongruous, partial left homonymous hemianopia (Fig. 7).

On dilated fundus examination, the right and left optic nerves were found to have mild pallor equal between the two eyes, indicating optic atrophy (Fig. 8). In addition, the patient had
small scattered “punched-out” chorioretinal lesions at the equator in both eyes; these were presumed to be ocular histoplasmosis. However, no peripapillary atrophy or choroidal neovascular membrane was present.

The patient was prescribed glasses for distance and near. The results of an MRI of the head that had been performed two weeks earlier were reviewed. This study was reported to demonstrate mild microvascular ischemic changes, but no evidence of post-traumatic encephalomalacia. Noted, however, was that gradient reverse echo (GRE) MRI images demonstrated hypointense foci within the left frontal subcortical white matter, right cerebral peduncle, and left mesial temporal lobe. These findings were reported to be consistent with hemosiderin staining from old diffuse axonal shear injury and possibly an old contusion. A CT of the head with and without contrast was ordered. The CT showed no evidence of head trauma; nothing was found that would correspond to the visual field loss.

The MRI was subsequently reviewed more closely with a staff neuroradiologist. She identified a small area of hyperintensity in the right occipital lobe; however, this was deemed insufficient to cause the visual field defect. No other lesions were found in the optic radiations or lateral geniculate nucleus that would account for the field defect; therefore, the neuroradiologist suspected that the lesion was located at the optic tract. The GRE MRI was then reviewed by the neuroradiologist, who explained that this particular type of MRI highlights hemoglobin, and therefore blood. On this scan, an area of old hemorrhage in the midbrain, adjacent to the right optic tract, was discovered (Fig. 9).

The neuroradiologist concluded that either this hemorrhage had affected the tract, or that axonal shear forces at the time of the head trauma resulted in damage to the tract. Six months later the patient returned to the clinic for a follow-up visit. An attempt was made to plot the patient’s visual field defect, but with a Goldmann III4e isopter, the field showed only generalized constriction, not homonymous hemianopia. Photographs of the optic disc were also taken at this visit to document optic nerve pallor. The patient was referred for low vision rehabilitation to explore options for field enhancement, but failed to return as recommended and was subsequently lost to follow-up. Of note, a PET scan ordered by the patient’s primary care provider approximately one year after the CT for long-term memory problems was found to be normal.

**Discussion**

**Clinical Signs of Optic Tract Insult**

There are various clinical signs with which a patient can present when there has been optic tract damage. These include homonymous hemianopia, retinal nerve fiber layer (RNFL) changes and optic atrophy, and relative afferent pupillary defects (RAPD).

Clinical findings are summarized in Table I.

According to Trobe, an optic tract lesion is expected to produce an incongruous homonymous hemianopia, as axons from the corresponding retinal regions of the two eyes are not yet arranged retinotopically at this point in the visual pathway. However, in 2006, Zhang and colleagues studied homonymous hemianopia in 850 stroke patients and noted that every type of homonymous field defect, other than unilateral loss of temporal crescent and homonymous sectoranopia, could originate from lesions located throughout all areas of the retrochiasmal visual pathway. Additionally, a retrospective study reported in 2007 by Kedar and colleagues evaluated 530 patients with homonymous hemianopia to determine a relationship between lesion location and congruency of defect. They found that lesions involving the occipital cortex do characteristically produce congruent defects; however, 50% of lesions in other locations also produced congruous defects, especially if the etiology of the lesion was cerebrovascular accident.

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<th>Table 1. Clinical signs of optic tract insult</th>
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<td><strong>Entity</strong></td>
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<td>Visual field</td>
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<td>Visual acuity</td>
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<td>Color vision</td>
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<td>Retinal nerve fiber layer</td>
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<td>Optic nerve</td>
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<td>Other signs</td>
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Figure 9. Case 2: GRE-MRI (axial view) shows blood adjacent to optic tract.
Savino and colleagues reported in a review of 21 with optic tract lesions that 17 had decreased central visual acuity either unilaterally or bilaterally. One additional patient had decreased acuity but had a history of retrobulbar optic neuritis, and only three had normal acuity. However, Levin held that loss of vision is due to the effects of the lesion on the optic nerve; unilateral lesions of the visual sensory pathway posterior to the chiasm generally do not result in loss of visual acuity in and of themselves. There are multiple studies of homonymous hemianopia due to insult to optic tract or other postchiasmal lesions in which the patients retained normal visual acuity. Smith found that in 100 cases of homonymous hemianopia, visual acuity was within normal limits in every instance, although only 3% of those patients were determined to have tract damage. Newman and Miller studied tract lesions and found normal acuity in nine of ten patients. Bell and Thompson’s case series of four patients with optic tract syndrome (OTS) all had normal visual acuity; as did all three patients with optic tract lesions in a study by O’Connor and colleagues. In addition, a series of two cases of OTS studied by Takahashi and colleagues both showed normal acuity.

Similarly, color vision typically remains normal following retrochiasmal insult. Case studies by Bell and Thompson and Takahashi and colleagues found color vision to be normal in all cases. O’Connor and colleagues reported color vision for only one of three cases in their study, it was found to be normal. Additionally, Margo and colleagues looked at two patients with congenital optic tract syndrome. Color vision was reported to be normal for one patient, but it was not reported for the second. Newman and Miller did report one patient among a series of ten cases who had abnormal color vision. However, this patient’s lesion was thought to involve not only the optic tract, but the optic nerve as well, which may have accounted for the color vision defect. The other nine cases reported all had normal color vision.

Retrograde degeneration of the axons following optic tract insult may lead to optic atrophy. The eye contralateral to the lesion will often show a pattern of bow-tie, wedge, or horizontal band of pallor indicating atrophy due to loss of fibers from the nasal retina. Levin also stated that patients with optic tract damage may present with generalized pallor of the ipsilateral disc, representing severe loss of the arcuate bundles.

This finding of optic atrophy following optic tract insult has been demonstrated clinically in multiple studies. In Newman and Miller’s case series of ten patients, six of the patients showed “band” or “bow-tie” optic atrophy in the eye contralateral to the lesion. Takahashi also documented loss of RNFL and optic atrophy in patients with tract lesions via clinical observation which included the aid of red-free filter. Tatsumi and colleagues were able to confirm this loss quantitatively by measuring RNFL thickness in a patient with optic tract syndrome using optical coherence tomography. It is thought that retrograde axon degeneration following a pre-geniculate lesion takes approximately six weeks to develop.

The presence or absence of a relative afferent pupillary defect (RAPD) in optic tract lesions has been exhaustively researched. Since the optic tract is composed of ganglion cell axons from the contralateral nasal retina and the ipsilateral temporal retina, and the nasal visual field is significantly smaller than the temporal visual field, Bell and Thompson hypothesized that an isolated tract lesion should produce an RAPD in the eye with the temporal field loss. They evaluated four patients with isolated optic tract lesions and found all to have such an RAPD. Savino and colleagues however, found that a relative APD was present only in patients with substantial unilateral vision loss. None of the patients studied with normal bilateral visual acuity had a RAPD, regardless of the incongruity in their field loss. The authors postulated that the RAPD thought to be associated with optic tract lesions was actually a result of simultaneous optic nerve and chiasm involvement. However, many other studies have shown the presence of RAPD in the context of normal visual acuity.

There have been a few proposals as to the origin of the RAPD in tract lesions. In earlier observations, the RAPD was thought to stem from the fact that there are more nasal decussating fibers than there are temporal fibers, or that the temporal visual field is larger in extent than the nasal visual field. Some case reports of optic tract homonymous hemianopia have indicated that relative afferent pupillary defects seemed to correspond with the size of the visual field defect. In 2006, Kardon, Kawasaki and Miller evaluated the pupil responses of five patients with unilateral optic tract lesions. Using binocular infrared pupillography, they compared pupil response as a function of stimulus light intensity. They discovered that the severity of relative APD (in log units) did not correlate with the estimated percentage of decussating pupil fibers. Therefore, they felt that the relative APD in optic tract lesions does not stem from the difference between numbers of fibers contributing to the temporal and nasal visual fields. They stated that pupil light sensitivity does not necessarily correlate to number of neurons, and that the RAPD is due to the difference in sensitivity of the functioning hemifields rather than the amount of fibers present. An interesting note from Newman and Miller’s case series of ten patients: one patient who initially presented with a RAPD showed resolution of this defect as the visual field improved over time. Conversely, one of the patients who initially did not present with an RAPD developed one as the visual field defect progressed.

Other pupillary phenomena have been postulated to be associated with optic tract damage. Behr’s pupil is said to occur in patients with anisocoria following optic tract lesion. Lowenstein also observed anisocoria clinically following optic tract insult; however, he stated that it was likely caused by an additional lesion in the third cranial nerve nucleus, efferent third nerve pathway on the side of the larger pupil, or sympathetic pathway which supplies the smaller pupil.
Pupillary hemiakinesia with hemianopia, or Wernicke's sign, was described by Wernicke in 1883. It is the diminished or absent pupillary reaction in a hemianopic patient when light is projected onto the non-seeing retina, with a normal reaction to light presented to the seeing visual field.\textsuperscript{1,17,19,20} However, this observation is of little clinical value given intraocular light scatter.\textsuperscript{17} In addition, Wernicke's pupil likely is not pathognomonic for an optic tract lesion, as it has been noted to be present in suprana- and infra-geniculate lesions.\textsuperscript{17,20}

Whether via infarction, trauma, or compression, events which cause insult to the optic tract may also damage nearby structures. The patient therefore may present with a variety of accompanying neurological deficits.\textsuperscript{4,6} Signs and symptoms that have been reported in optic tract case series include headache, hemiparesis and hemisensory loss, various disorders of mentation, diabetes insipidus, esotropia from sixth nerve palsy, amenorrhea, paresthesias, absent abdominal reflexes, seizures, and dysphasia.\textsuperscript{4,6,6,10}

**Causes of Optic Tract Insult**

There have been a multitude of causes cited for optic tract damage. Among the more common causes are neoplasm, trauma, and vascular or ischemic events.\textsuperscript{1,19,21} In Zhang and colleagues’ study of 904 patients with homonymous hemianopia, 40% of the patients determined to have optic tract lesions sustained the damage due to infarction and nearly 32.3% were caused by tumors.\textsuperscript{1} Trauma was the least frequent cause, accounting for just 16.7% of the optic tract lesions in this study.\textsuperscript{1} In another case series, Savino and colleagues found neoplasia to be the most common etiology of tract lesions.\textsuperscript{9} Kedar and colleagues found homonymous hemianopia was caused most frequently by infarction (63%) in adults. Conversely, the most frequent cause in pediatric patients in their study was traumatic brain injury (34%), followed by neoplasia (27%).\textsuperscript{21} Of note, this study included homonymous hemianopia secondary to all retrochiasmal lesions, not just tract damage.\textsuperscript{12} Neoplastic lesions affecting the tract are typically nonmetastatic tumors. However, infrequently they can be secondary to metastasis from another site, as in a case reported by Groom and colleagues.\textsuperscript{22} The patient presented with difficulty with reading and was discovered to have a right homonymous hemianopia on confrontation fields and automated perimetry. The patient had been previously diagnosed with metastatic breast cancer, and an MRI of the brain confirmed an optic tract and lateral geniculate nucleus tumor consistent with metastasis.\textsuperscript{22}

Optic tract syndrome can also be congenital. The pathogenesis for congenital OTS can be linked to developmental failure of the optic tract, which is typically fully formed by the thirteenth week of gestation, or secondary atrophy due to focal injury to the tract during the perinatal period.\textsuperscript{15,22} Aberrant vasculature development leading to insufficient blood supply to the tract could also result in ischemic insult to the optic tract, although this is less likely to cause congenital OTS due to the dual blood supply to the tract.\textsuperscript{15} The tract is supplied mainly by the anterior choroidal arteries that originate from the internal carotid arteries, with additional supply from the posterior communicating artery.\textsuperscript{4,15,23}

Additionally, there have been observations of optic tract damage from more infrequent causes such as multiple sclerosis, aneurysm and arteriovenous malformations, inflammation, and even iatrogenic damage from placement of intracranial catheters.\textsuperscript{24-29}

**Prognosis and Management of Patients with Homonymous Visual Field Loss from Optic Tract Injury**

Hemianopic visual field loss can be very debilitating for patients, as it may interfere with activities of daily living (ADLs). The prognosis for recovery of the field is unclear. Pambakian reported that up to 50% of patients have spontaneous improvement in the visual field to varying degrees, although less than 10% of patients recover the full field.\textsuperscript{30} He stated that the degree of recovery is thought to vary based on the underlying pathology: field deficits of vascular origin have poor prognosis for recovery, and recovery is thought to be maximal within the first 48 hours following the event.\textsuperscript{30} However, in traumatic hemianopia, large areas of the field frequently recover some light perception.\textsuperscript{30}

In contrast, Zhang and colleagues studied 254 cases of homonymous hemianopia of various etiologies to characterize the evolution of the field deficits over time.\textsuperscript{31} They stated that "no patient, lesion, or visual field characteristic was found to correlate with the final outcome."\textsuperscript{31} They reported that spontaneous improvement, defined as a difference on consecutive fields of 10 degrees in the horizontal field or 15 degrees in the vertical field, was seen in almost 40% of the cases.\textsuperscript{31} They also noted that the probability of improvement decreased with time, and that spontaneous improvement 6 months or longer after the injury is unlikely.\textsuperscript{31}

It is important to note that patients with hemianopic field defects may also develop extensive and rapid scanning eye movements to cope with the field loss, and as this skill develops the field may appear to improve when it actually has not.\textsuperscript{32} There are many different devices and techniques to help people cope with the loss of a visual hemifield, including optical aids, oculomotor training, and computer training. Unfortunately, most research shows these therapies to be of little value functionally, even if a clinical improvement in the visual field can be demonstrated.

Simple optical aids such as monocular mirrors and prisms may be used to expand the patient’s field. In the monocular mirror technique, a mirror is mounted onto the frame of the patient’s glasses at an angle, on the same side as the field defect.\textsuperscript{30} The patient looks into the mirror to view the hemianopic field.\textsuperscript{30,33,34}

Reverese telescopes are another strategy that has been employed in the past. They expand the visual field by minifying objects so that more of them will be contained in the functional...
visual field. However, the inconvenience of the telescope, along with decreased visual acuity and distortion when looking through the telescope, has prevented its widespread use.

Many variations on prisms have been used for field enhancement. With an older technique, low to moderate powered Fresnel prisms were placed on the patient’s spectacles with the base in the same direction as the field loss. One or both lenses could be fitted, and the prism shifts the hemianopic field slightly to bring part of it into view. However, improvement with this strategy is anecdotal only. A controlled trial of Fresnel prisms demonstrated no functional improvement, and the patients often got confused by the double images.

In 2000, Peli introduced a different strategy for prism correction of homonymous hemianopia. A high-powered elliptical prism segment of 30-40 prism diopters was placed across the upper side of the spectacle lens on the side of the field loss. Of the 12 patients in the study, 11 were found to have the expected field expansion of 20 degrees when measured with perimetry. Subjectively, the majority of the patients were pleased with the prism. Three of the treated patients were followed for more than one year and all reported that the prism was beneficial.

More recently, Bowers and colleagues tried a modified version of Peli’s design. They fit 40 prism diopter Fresnel prism segments above and below the visual axis with 11 mm interprism separation. They fit the upper prism first, and the patient wore it for two weeks to enhance adaptation before adding the lower prism. The approach in this study showed much promise. The success rate of the prisms was 74% at 6 weeks of use and after a year, 47% of the participants were still choosing to wear the prism glasses as a mobility aid. In addition, while this study was in progress, a permanent version of the press-on Fresnel prism was developed by Chadwick Optical, providing a better option for optical clarity and long-term prism wear.

Saccadic and oculomotor training strategies have also been employed for hemianopic patients. Saccadic training is based on the assumption that in patients with homonymous hemianopia, scanning eye movements are less regular, accurate and systematic than patients with a normal visual field. Therefore, the goal of this therapy is to increase the patient’s ability to direct gaze movements toward the blind hemifield and efficiently explore it. Patients first practice making large, quick saccades into the blind field, then they are taught to scan for targets in a systematic way. A study by Pambakian and colleagues found that saccadic training improved performance in activities of daily living and reported significant subjective improvements. However, this study did not include an untreated control group for comparison.

Vision Restitution Training (VRT) is another training technique for hemianopes. This computer-based training involves repetitive stimulation of the transition zone between the blind and normal visual field. The data for VRT is mixed. In a study on the efficacy of VRT, Reinhard and colleagues found that two-thirds of the patients reported subjective improvement following VRT; and reading performance also significantly improved in one of two reading tests given. However, Schreiber and colleagues examined 16 patients with stable homonymous visual field defects. Using static automated perimetry, they tested the field before and after visual restitution training and found that VRT had little effect on the field defect.

Conclusion

Although relatively rare, optometrists should be aware of the potential for optic tract damage in patients presenting with homonymous field loss. Characteristics of a homonymous hemianopia can often be helpful in localizing a brain lesion. As discussed, there is a high likelihood that a patient with an incongruous homonymous hemianopia, especially in conjunction with a relative afferent pupillary defect, has visual pathway damage located at the optic tract.

Neuroimaging should always be obtained to confirm the diagnosis, as every type of homonymous hemianopia, other than unilateral loss of temporal crescent and homonymous sectoranopia, has been correlated with lesions located throughout all areas of the retrochiasmal visual pathway.

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