Article • Binasal Occlusion for the Treatment of Visual Motion Sensitivity after Traumatic Brain Injury: A Case Series

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ABSTRACT

Background: Symptoms of visual motion sensitivity (VMS) secondary to traumatic brain injury (TBI) have previously been reported to improve with binasal occlusion (BNO). In previous literature, BNO has improved ambulation and gait in those with VMS. The aim of this case series was to demonstrate how BNO improves symptoms of VMS during ambulation in a military population with history of TBI.

Methods: Three patients with a history of TBI and suspected VMS underwent a neuro-optometric examination, including BNO applied to spectacles during ambulation in a clinical setting. Gait was subjectively evaluated by the examiner, and symptoms of VMS were assessed by the patient.

Results: Subjective improvement in gait and symptoms of VMS were appreciated in three TBI patients with VMS. Two subjects also demonstrated improved versional function with BNO. All three subjects were subsequently prescribed BNO and neuro-optometric vision rehabilitation. One subject was lost to follow-up, and two subjects reported improved visual function with BNO several months after its initial application.

Conclusion: BNO is an effective tool for treating VMS symptoms secondary to TBI. The effect of neuro-optometric vision rehabilitation in conjunction with BNO for VMS is yet to be determined.

Keywords: binasal occlusion, neuro-optometric, post-trauma vision syndrome, traumatic brain injury, vision rehabilitation, visual motion sensitivity

Introduction

The United States Centers for Disease Control and Prevention consider traumatic brain injury (TBI) to be a serious public health concern.[1] Around 2.8 million TBI-related hospitalizations, emergency department visits, and deaths occurred in the US in 2013.[2] TBI was diagnosed in 383,947 US service members between 2000 and 2018,[3] and it has been the most prevalent injury among warfighters during the military operations Iraqi Freedom, Enduring Freedom, and New Dawn.[4]

Brain injury can be caused by a focal contact injury such as a contusion, a laceration, or an intracranial hemorrhage. It can also be due to an acceleration-deceleration injury, which results in diffuse axonal injury or brain edema.[5] TBI is defined as a bump, blow, or jolt to the head or a penetrating head injury that disrupts the normal function of the brain.[1] Visual sequalae of TBI include dysfunctions of vergence eye movements,[6-9] dysfunctions of versional eye movements,[9-11] accommodative dysfunction,[8,9,12] abnormal spatial sense,[7] visual

Figure 1. *Gibsonian optic flow. Arrows indicate vectors of relative motion from the perspective of the observer. When the observer walks down the hall, perceived motion is greatest closest to the observer. This hallway was used for gait evaluation of patients in this case series.*

field defects,[10,13,14] and visual motion sensitivity (VMS).[15,16] These individuals are at greater relative risk for optic atrophy, cataracts, vitreous degeneration, dry eye, and corneal abrasion.[17]

Post-trauma vision syndrome (PTVS) has been documented in the literature as an ambient processing disorder,[18] whereby brain injury victims may experience abnormal spatial sense or oculomotor, attentional, and/or cognitive problems.[7] This condition has been theorized to lead to the clinical findings of strabismus, convergence insufficiency, accommodative insufficiency, and oculomotor dysfunction.[18] PTVS is usually treated using a top-down approach, which means that vision rehabilitation incorporates "strategic and voluntary control of visual processes and decision-making."[19]

VMS, also known as visual motion hypersensitivity, visual vertigo, and space and motion sickness, is associated with PTVS and occurs when ambient and peripheral visual motion result in imbalance, dizziness, and/or nausea.[20] Gibsonian optic flow (Figure 1) is when objects closer to the observer appear to move at a faster velocity than objects farther away than the observer,[21] such as when walking down a busy supermarket aisle,[16] executing vergence eye movements,[22] or riding in the back seat of a car.[16] Conditions that generate Gibsonian optic flow can generate symptoms of dizziness, nausea, and imbalance.[16] VMS can be treated with spectacle filters, visual motion desensitization therapy, and binasal occlusion (BNO).[23]

Laboratory studies in subjects with a history of TBI and VMS have quantified the objective effect of BNO using the measurement of the visually-evoked potential (VEP). VEP measures the electrical signal at the scalp over the occipital lobe in response to a light stimulus.[24] Padula, Argyris, and Ray found that subjects with TBI alone and no distinct report of VMS demonstrated increased VEP amplitude with BNO and base-in prism relative to non-TBI patients.[18] Both Ciuffreda, Yadav, and Ludlam[25] and Yadav and Ciuffreda[15] found that while wearing BNO alone, VEP amplitude increased in TBI patients with VMS and decreased in the visually normal. Yadav and Ciuffreda investigated the combination of base-in prism and BNO versus BNO alone. The results revealed that BNO alone is more consistently associated with increased VEP amplitude in TBI patients with VMS.[15]

There is a relative scarcity of case studies that document the subjective effect of BNO on PTVS and/or VMS. Proctor documented that a forty-six-year-old Caucasian male TBI victim reported dizziness when objects moved around him and experienced difficulty walking in public places if others were around him.[26] With BNO, this patient reportedly seemed more confident moving past people who walked by him and had improved mobility. Gallop reported that when a patient with a suspected TBI wore BNO, the patient immediately noticed the elimination of pain around their eye and the dizziness that typically followed fast eye movements.[27] Gallop also reported a separate TBI case where BNO resulted in immediate, subjectively reported, 80% improvement in balance problems and disorientation associated with busy environments.[27] Lee presented a case report in which the patient reported less blurred vision, dizziness, and vertigo and an increased VEP amplitude with BNO.[28]

Aside from the aforementioned case studies, very little has been documented about the use of BNO for VMS in the clinical setting. Nor has there been much documented on its effect on gait, which is an activity where patients are often subjected to peripheral motion in their surroundings, such as when walking in crowds or in a supermarket. Yadav and Ciuffreda found that improvement in gait occurred in their laboratory study,[15] and one case study reported improvement in ambulation and walking through hallways.[26] Currently, there are no publications that discuss the use of BNO in a military population. The aim of this study, therefore, is to evaluate how BNO improves symptoms of VMS during ambulation in a military population.

Methodology

As a part of this case series, brain injury patients at the Intrepid Spirit Brain Injury Center at Womack Army Medical Center in Fort Bragg, NC were evaluated in two visits. The first visit included the completion of an intake questionnaire (Appendix A), refraction, and a comprehensive eye exam with a dilated fundus evaluation. Refractive error was treated with spectacles. Photosensitivity was treated with patient-preferred filters based on reduction of symptoms. Ocular pathologies, such as ocular surface disease, were treated with therapeutics and/or additional ocular health testing.

At the follow-up visit, after the patient adapted to the most-appropriate distance and near spectacle correction for at least two weeks, a neuro-optometric sensorimotor exam and a visual field were performed. Additionally, tint demonstrations, spectacle prescription trials, ancillary testing, and ocular health evaluations occurred as needed at the follow-up visit.

If the patient reported any balance concerns, dizziness, nausea, or uneasiness in crowded or busy environments, either in the case history or in the intake questionnaire, the examiner assessed the effect of BNO while the patient ambulated in the hallway of the eye clinic. First, the patient was asked to walk down the 20-meter clinic hallway, turn around at the end of the hall, and walk back to the examiner. While the patient walked down the hall, the examiner evaluated the gait of the patient and asked questions about balance, dizziness, and peripheral vision. The examiner also looked for indicators of imbalance, such as veering, swaying, grasping for the walls, or excessive depression of the head. During and after the walk, the examiner asked questions about how the patient felt and whether the patient felt dizzy or imbalanced during their walk. Second, the examiner applied BNO, with the border of the translucent tape at or around the nasal canthi on the spectacle lens, and then asked the patient to repeat the walk. The occlusion was adjusted so that the patient did not notice the tape while looking in primary gaze, the position was subjectively comfortable, and the occlusion was not distracting. If a subjective improvement in gait, visual clarity, or sensation of imbalance occurred, the walk was again repeated without the BNO. BNO was then included as part of the VMS treatment.

Results

Case Study #1

A 39-year-old Caucasian male soldier, HF, presented to the eye clinic with a chief complaint of reduced vision at distance and near since his most recent head injury three months prior. Whether his visual blur occurred

Table 1. Summary of Exam Findings

Patient	HF	LB	ML
Gender	Male	Female	Male
Age	39	39	47
Best-corrected spectacle prescription (D)	OD: +0.25 SPH OS: +0.75-1.00x167 Add: +0.75	OD: +0.75-1.25x175 OS: +0.75-1.25x175 Add: None	OD: -0.50-0.75x105 0.5Δ BO OS: -0.50-0.75x003 0.5Δ BO Add: +1.50
NPC with spectacle correction break/recovery (cm)	8.5/12*	12/19*	6/8
Distance/near Von Graefe phoria with best-corrected spectacle prescription (Δ)	Not assessed	Orthophoria / 4 base in	6.5 base out (distance)
Near base in	6 base in	3 base out	18/24/18
Blur/break/recovery (Δ)	9/18/16	12/18/12	18/28/18
Near base-out blur/break/recovery (Δ)	x/11/6*	x/12/5	Not assessed
NRA/PRA (D)	+1.75* / Plano*	+1.25* / -1.75*	Not assessed
Near vergence facility	10*; base out more difficult to fuse	4*; base out more difficult to fuse	Not assessed
12 base out/3 base in (cpm)	Not assessed	3; ±1.50 D* plus more difficult	Not assessed
Binocular accommodative facility (cpm; power)	Not assessed	OD: 5; ±1.50 D* plus more difficult OS: 3; ±1.50 D* plus more difficult	Not assessed
Monocular accommodative facility (cpm; power)	OD: 3.75*~	OD: 5.25 OS: 5.75	Not assessed
Amplitude of accommodation (D)	OS: 3.00*~	3*	Not assessed
Saccades (1-5 scale) NSUCO seated OU	Not assessed	3+*	3*
Pursuits (1-5 scale) NSUCO seated OU	Not assessed	Not assessed	3*
Maddox rod testing of horizontal alignment in 9 fields of gaze	Range of 3-6$^{\Delta}$ BO (comitant)	(Comitant)	10$^{\Delta}$ BO all gazes
King-Devick^	64.93* seconds	112.41* seconds	69.62* seconds

* Reduced from normal value
~ Reduced relative to age-predicted accommodation values from Hofstetter.[29]
^ Not tested in neuro-optometric exam; recorded at separate visit by occupational therapist trained in neuro-optometric vision rehabilitation; cpm = cycles per minute; NPC = near point of convergence; NRA = negative relative accommodation; PRA = positive relative accommodation. NSUCO= Northeastern State University College of Optometry version testing

under static or dynamic conditions was not discussed. The patient had a history of multiple head injuries that included blast exposures during training and combat operations, a motor vehicle accident (MVA), and a head injury during paratrooper operations. In our intake survey (Appendix A), his most significant symptoms included constant distance blur, constant photosensitivity, headaches after reading, and dry eyes. His ocular history was unremarkable. His systemic history was significant for hypertension, migraine headaches, and hypogonadism. His medications included rizatriptan, naproxen, metoprolol tartrate, and testosterone. The subject saw a neurologist two years earlier; that visit indicated overall normal neurological findings. No vestibular deficits or specific testing was reported on the neurology record.

A summary of HF's neuro-optometric exam findings is included in Table 1. Uncorrected entrance testing indicated normal extraocular muscles (EOMs), normal pupil function, and full confrontational visual fields (CVF). His entrance static visual acuity (VA) at distance was 20/20 (6/6) OD (right eye) and OS (left eye) with his habitual spectacles. At near, his static VA with his habitual correction was 20/20 (0.4/0.4) OD and OS. The ocular health of the anterior and posterior segments was unremarkable, except for reduced tear break-up times in both eyes (OU). Single-vision distance and single-vision near spectacles were ordered with anti-reflective coating in order to maximize his vision.

Prior to his follow-up exam, HF was evaluated by physical therapy. The patient was diagnosed with a possible right posterior semi-circular canal cupulolithiasis. Frenzel goggle vestibular testing indicated negative gaze-evoked nystagmus, right-beat nystagmus with reported dizziness from headshake testing, upbeat nystagmus with right Dix-Hallpike testing, and upbeat nystagmus with left torsion from left Dix-Hallpike. Frenzel goggle vestibular testing also indicated negative return from left and right Dix-Hallpike and negative horizontal roll. Magnetic resonance imaging (MRI) of the brain conducted without contrast performed after his initial exam indicated normal brain appearance but noted a few punctate nonspecific and clinically insignificant white matter foci.

Six weeks later, at his follow-up visit, the patient had adapted to his new glasses. His chief complaint was clipping doorways and veering while walking. He reported persistent photosensitivity, as well as two out of ten pain on a zero-to-ten pain scale, which was qualified as a dull pressure behind the OS. Sensorimotor exam findings can be found in the Table 1.

Due to his chief complaint of imbalances while walking, his gait was evaluated. During his evaluation, he brushed against a doorway, depressed his head while walking, and reported that the walls were "wobbling in and out." BNO was applied to the nasal areas of the patient's spectacle lenses, and the gait evaluation was repeated; his head and chin elevated, he walked through doorways with greater ease, and he reported that the hallway was more stable. The BNO was removed, and the patient immediately reported that his "vision was off." Tints were trialed to aid in photosensitivity, and the patient noted improvement indoors with 85% amber tint versus blue and rose tints. In order to address his ocular surface disease, one drop of preservative-free artificial tear solution was instilled into the left eye, and the patient reported an improvement in his symptoms.

HF was diagnosed with mild convergence insufficiency, fusional vergence dysfunction, PTVS, dry eye syndrome, photosensitivity, and VMS. He was prescribed BNO and 85% amber-tinted lenses for PTVS, photosensitivity, and VMS. Dry eye syndrome was treated with artificial tears. For his convergence insufficiency, fusional vergence dysfunction, PTVS, and VMS, he was prescribed home and office neuro-optometric vision rehabilitation. His neuro-optometric vision rehabilitation included integration of head movement, incorporation of the vestibular system, and inclusion of other neurological systems for a top-down approach, as described by Chang, Cohen, and Kapoor.[19] Occupational therapy performed his vision rehabilitation at the direction of the optometrist. The order of activities performed in vision rehabilitation was determined by the occupational therapist and was supervised by the optometrist. The occupational therapist measured his King-Devick score to be reduced at 64.93 seconds. During testing, he wore single-vision distance glasses without BNO. HF returned to the clinic eight months after his initial sensorimotor exam and after eight in-office neuro-optometric vision rehabilitation sessions. At that visit, BNO still provided symptomatic relief. Eleven months and 16 neuro-optometric vision rehabilitation sessions after his initial exam, he reported that he had been applying his own BNO with translucent tape. He also reported he had self-tapered the BNO to a thinner width, as he found that the original width became annoying over time. He reported that he tried to function without the BNO but that he felt dizzy and nauseous without it. After 22 sessions of neuro-optometric vision rehabilitation and 18 months since his initial neuro-optometric exam, he still reported symptomatic relief with the BNO. His vergence facility and near vergence ranges had not improved relative to baseline. His version testing was near-normal without BNO, but he reported improved comfort when retested with BNO. At this 18-month follow-up visit, the patient was prescribed continued neuro-optometric vision rehabilitation with an emphasis on vestibular and versional exercises.

Case Study #2

LB was a 39-year-old Hispanic female soldier who lost her balance, fell, and hit her head during military training 6 years prior to the exam. Her chief complaint was foreign body sensation of the right eye, which occurred once a week upon wakening, accompanied by redness, which worsened as the day progressed. Her most significant complaints in the clinic's intake survey included constant dizziness in crowds, constant distance and near blur, headaches after reading, and difficulty with night driving. The context of whether her visual blur occurred under static or dynamic conditions was not discussed. Her ocular history was significant for a history of Graves' ophthalmopathy with bilateral orbital decompression surgery two years prior to the exam. She denied any diplopia with spectacle correction. She also had a history of dry eye syndrome, which was treated with lubricating ointment and artificial tears. Her systemic history was significant for migraine headaches, depression, anxiety, gastroesophageal reflux disease, irritable bowel syndrome, seasonal allergies, vitamin D deficiency, insomnia, hypertension, Graves' disease, and overactive bladder. Her medications included duloxetine, topiramate, esomeprazole, dicyclomine, clonazepam, ranitidine, fluticasone nasal spray, cetirizine, rizatriptan, vitamin D, zolpidem, polyethylene glycol, spironolactone, and solifenacin.

The patient's neuro-optometric exam findings are summarized in Table 1. The patient's entering static distance VAs were 20/20 (6/6) OD and OS with habitual correction. Uncorrected VA was not assessed. At near, her static VA was 20/20 (0.4/0.4) OD and OS with habitual correction. The patient's pupils, EOMs, and CVF were normal. Pertinent anterior segment findings included a mildly inflamed pinguecula OD, reduced tear break-up times OU, and trace punctate epithelial defects OD. There was no indication of exophthalmos or lagophthalmos in either eye. Her posterior segment ocular health was unremarkable. She was diagnosed with dry eye syndrome OU and pingueculitis OD. She was prescribed preservative-free artificial tears four times daily in both eyes, was educated to continue nighttime lubricating ointment in both eyes, and was told to start fluorometholone ophthalmic suspension three times daily OD.

At LB's follow-up evaluation, her chief complaint was blurred vision at distance only with some persistent balance complaints; her anterior segment complaints OD had resolved, and her near blur had improved with the new spectacles. A summary of her exam findings can be found in Table 1. Her fixation was tested and was reduced with head movement and excessive blinking. Her pursuit function was reduced and included excessive head movement; additionally, she blinked excessively during testing, suggesting difficulty processing the test and/or refixations. She requested to end pursuit testing early due to discomfort. Her saccadic function was slow and also included excessive head movement. Her distance blur complaint resolved when -0.25 sphere was placed over her habitual glasses at distance, suggesting that she was intolerant to her full hyperopic correction.

Her gait in the clinic hallways appeared relatively normal, with the exception that she veered left and right during her walk. After applying translucent BNO at approximately her nasal canthi on her glasses, she walked straight and reported that she felt better. Her performance with saccades and pursuits improved with BNO. Saccadic performance improved from 3 to 4 with trace head movement. Improvement with patient attentiveness and pacing during saccade testing was also noted. Pursuit function improved from 3+ to 4+ with trace head movement and reduced discomfort reported.

LB was diagnosed with mild convergence insufficiency, fusional vergence dysfunction, saccade and pursuit dysfunction, accommodative infacility, fixation dysfunction, PTVS, and VMS. She was prescribed BNO to aid her VMS and neuro-optometric vision rehabilitation with a top-down approach for her accommodative, version, and vergence dysfunctions. Occupational therapy recorded her King-Devick score to be reduced at 112.41 seconds (s) while wearing distance glasses. LB attended only 3 sessions of vision rehabilitation since she was discharged from military service.

Case Study #3

ML, a 47-year-old African American male soldier, presented to the eye clinic complaining of constant blurred vision at distance for one year that improved with squinting. ML had a history of head trauma secondary to sports and ejection from a vehicle during a mortar attack during combat with loss of consciousness; the dates for these incidents ranged from 37 years prior to the exam up to less than one year before. He also reported dizziness and balance difficulties and was scheduled to be evaluated by a vestibular physical therapist in one week. ML reported regular migraine headaches that were associated with photosensitivity. The significant complaints reported in his intake survey included frequent photosensitivity, constant dizziness and nausea in crowds, and constantly losing his place while reading. He had an unremarkable ocular history and a systemic history significant for depression, hypertension, and anxiety. His medications included buspirone, sertraline, bupropion, and clonidine. The patient had an MRI of the brain without contrast several days before the exam that indicated a left cerebellar supratentorial region nodular, but no parynchemal mass, midline shift, or suggestion of restricted diffusion was noted. Isointensity to subtle hyperintensity within the transverse sinus was noted by radiology. A follow-up computed tomography image without contrast indicated that there was no obvious acute parynchemal hemorrhage and that a left cerebellar supratentorial region calcification corresponded to the previously noted nodular imaged on MRI. A follow-up magnetic resonance venography indicated that there was no evidence of stenosis in the major structures of the dural sinuses.

The patient's initial neuro-optometric exam findings are summarized in Table 1. His pupils, EOMs, and CVF were normal. His NPC with distance correction, which broke at 11 cm and recovered at 22 cm, was within normal limits based on Ostadimoghaddam et al.[30] His indoor photosensitivity improved with 85% blue lenses. Aside from pingueculae, his anterior and posterior health were unremarkable.

The patient's now-completed physical therapy evaluation indicated a possible right unilateral vestibular hypofunction. The physical therapist also reported that the patient had a normal Romberg test result but drifted left with his eyes closed. Additionally, the physical therapist reported that with tandem stance testing, the patient experienced two losses of balance out of three attempts and three out of three losses of balance with his eyes closed. With functional gait assessment, physical therapy reported tthat he patient earned a mildly reduced score of 25 out of a maximum 30 points. Frenzel goggle testing was negative for gaze-evoked nystagmus, negative headshake test, negative right and left Dix-Hallpike test, negative return from left and right Dix-Hallpike, and negative horizontal roll. The record did not indicate whether the patient wore spectacles.

At ML's follow-up sensorimotor exam, he reported that his distance blur was resolved with his new spectacles, and photosensitivity was improved with the 85% blue lenses. The context of whether blur worsened with head movement was not discussed. His pursuit function was tested, with reduced accuracy; the testing resulted in nausea, for which the patient needed to take breaks. His saccades were relatively slow and induced nausea. His NPC improved with his updated spectacles to a break value of 6 cm and a recovery of 8 cm. The Worth four-dot test indicated that he suppressed his right eye at distance, but no suppression or diplopia was found at intermediate or near distances. Due to his previous complaint of dizziness, his gait was evaluated, and he veered left and stumbled when he turned in the hallway. He reported that the walls wobbled, and he felt un-centered in the hall. After applying BNO to his glasses at the nasal canthi, ML was able to walk straight. He reported that the walls no longer moved, the hallway "opened up," and he felt more centered. The examiner noted that he still stumbled when making turns, but less than without the BNO. The patient appreciated the improvements in his comfort and gait. His saccade and pursuit functions were retested, and his performance improved. His saccade function overall improved from a 3 to a 4 out of a maximum of 5+, with reduced dizziness and increased speed, although some nausea from earlier testing lingered. His pursuit function increased from 3 to 4, with persistent reduced accuracy but improved ability. The patient stated that he was more comfortable during testing with BNO on his glasses. This patient was diagnosed with pursuit and saccade dysfunction, VMS, and PTVS. For these conditions, he was prescribed BNO and neuro-optometric vision rehabilitation, with an emphasis on "top-down" therapy and with a focus on divergence and version dysfunction. Occupational therapy measured his King-Devick time without spectacle correction; it was reduced at 69.62 seconds. ML attended three vision rehabilitation sessions but stopped attending. He returned to the clinic three months after his initial evaluation, and the BNO still provided subjective symptomatic relief.

Discussion

All three of these cases suggest that BNO can subjectively improve VMS symptoms and can objectively improve visual function in a military TBI population diagnosed with VMS. This is the first case series that uses gait as an evaluation criterion for establishing the efficacy of using BNO in the treatment of VMS. Case studies by Gallop[27] and Lee[28] did not comment on any in-office evaluation of gait or supervised trial of ambulation in an environment that stimulates Gibsonian optic flow, such as a clinic hallway. Proctor did supervise gait and noted that the patient seemed "more confident moving through doorways and past those walking by him" but did not evaluate balance or specifically mention whether symptoms of dizziness or vertigo improved.[26] Additionally, Yadav and Ciuffreda noted in their study that mild TBI patients with symptoms of VMS reported greater comfort and confidence walking down a hallway while wearing BNO.[15]

The mechanism of BNO and its clinical effect is not well understood. Rummell indicates that patients with ambient visual dysfunction "find it difficult to visually sort things out in a busy visual environment" and that BNO "allows the patient to have reduced ambient input."[31] Gallop indicated that BNO may serve as an "anchor for the interface between the external environment and internal visual process" and that it "places a marker in the environment" that remains the same size when the patient processes oculocentric and egocentric localization information.[27] Gallop also postulated that the occlusion removes "intense" binocular information and "relieves stress," which would imply that the processing load of an injured brain would be reduced.[32] Lastly, Gallop indicated that by occluding the nasal field, the temporal fields are subject to preferential stimulation. Gallop also indicated that the occlusion of the binocular nasal fields removes stress associated with integrating binocular images, enabling the peripheral vision to function appropriately.[32]

When it comes to the laboratory studies using VEP, the effect of BNO on magnocellular deficits has been evaluated with greater depth. Padula, for instance, believed that the relative increase in VEP amplitude associated with base-in prism and BNO with brain injury patients implies that the primarily focal binocular cells of TBI patients are compromised.[18] Padula postulated that BNO improved VEP function in TBI subjects by providing structure to the ambient visual process, thus increasing binocular cortical function.[18] In the second recorded laboratory study performed by Ciuffreda et al. it is theorized that without BNO, patients with VMS attempt to suppress peripheral motion and that this suppression spreads into contiguous areas tested by the VEP.[25] Ciuffreda et al. theorized that wearing BNO reduces the need to suppress peripheral motion, which prevents inaccurate suppression from spreading into the area tested by the VEP, resulting in an increase in cortical excitation and subsequently an increase in the measured VEP amplitude.[25] Ciuffreda et al. also argued that it is possible that mild TBI victims do not filter irrelevant information as well as those without mild TBI and that BNO improves the filtering of irrelevant information.[25] Yadav and Ciuffreda noted that visual attention has an effect on VEP amplitude in those with mTBI[33] and postulated that the BNO shifts visual attention to central vision tested by the VEP.[15] It is also theorized that there may be a mismatch between object- and self-motion in cortical areas V6 and V6a that may elicit the symptoms of VMS.[16]

VMS is described as a symptom of PTVS,[7] and PTVS has been linked to other deficits found in a neuro-optometric exam. Such deficits include convergence insufficiency, strabismus, accommodative insufficiency, and version dysfunction.[18] The comorbidity of oculomotor dysfunction with VMS and how BNO affects oculomotor function, however, have not been thoroughly documented. In two cases where BNO was used in TBI patients, Gallop did qualify some exam findings but did not quantify oculomotor exam findings, list diagnoses, or report the effect of BNO on oculomotor function.[27] Proctor's case included detailed and quantified exam records; however, Proctor did not discuss the effect of BNO on these deficits.[26] King-Devick testing was reduced in all subjects in this case series; all subjects scored more than 20 seconds worse than normal. Anderson and Biely found that in a sample of 243 subjects with an average age of 40.46 years of age, the average KD time was 42.2 seconds.[34] This case series did not test King-Devick with BNO. Versional eye movement function, as measured by Northeastern State University College of Optometry version testing[35] performed seated, improved with BNO in two subjects in this case series. It is not known whether vergence, accommodation, or other functions may improve with BNO. Future investigations on whether BNO affects visual performance are merited.

Cases by Lee,[28] Proctor,[26] and Gallop[27] did not specifically mention any history of blast exposure. Only one of the fifteen subjects in the laboratory study by Yadav and Ciuffreda had a reported head injury due to a blast.[15] The improved symptoms associated with two subjects in this cases series, who both suffered blast injuries as well as other non-blast injuries, suggest that BNO may be efficacious after blast exposure as well.

Two of the patients presented were 39 years old, relatively young compared to other case reports evaluating BNO. While Proctor's[26] patient was 46 years old, Lee's[28] patient was 62, and Gallop's[27] patient was 67. Compared to laboratory studies, the patients in these three military case studies were generally older: the mean age in Yadav and Ciuffreda's[15] study was 35,[2] and the mean age in Ciuffreda et al.'s study was 28.9 years.[16] The success of BNO for VMS in two 39-year-old patients suggests that BNO is likely clinically efficacious in a wide age range, but more research to determine clinical efficacy is likely indicated.

The placement of the BNO does have some reported variation. When treating esotropia, BNO is usually applied at the nasal pupillary border.[32,36] When treating VMS and PTVS, it is typically applied in thinner sections, usually medial to the nasal limbus. Proctor indicated that it should be applied nasal to the limbus,[26] and Gallop indicated that it should be applied at the inner canthi or more medial.[27] However, Rummel indicated that it should be placed at a 30 degree angle above the pupil margin and should be reduced over time.[31] Laboratory studies seem to occlude more of the nasal visual field; Padula et al. generally indicate that it should be applied to the nasal field.[18] Additionally, Ciuffreda et al. applied it at the superior-nasal pupillary margin at a 15 degree angle from the vertical.[25] Much like the other case studies, all patients in this case series had the BNO applied in a region that was more nasal than the pupillary border, further reinforcing that the clinical effect can be attained with a relatively conservative degree of occlusion.

The type of material used for occlusion has varied in the literature. Previous authors have suggested translucent material.[15,18,26,31,32] All three of the cases in this series reported improved symptoms using translucent occlusion; opaque occlusion was not trialed. However, opaque occlusion has also been reported to have some efficacy.[15,25] Ciuffreda et al. argued that opaque occlusion is preferable to translucent occlusion and that the minimum level of opacity that elicits the desired symptomatic response should be determined in the future.[25] Whether the type or degree of occlusion incurs a different effect is yet to be established.

While previous studies have evaluated the efficacy of using BNO in brain injury populations, the diagnosis was likely VMS but was not always labeled as such. For instance, Proctor indicated that PTVS is a common condition after brain injury. She also identified PTVS as a dysfunction of the magnocellular pathway.[26] Magnocellular dysfunction can cause vertigo or dizziness with movement through crowds, but Proctor was never more specific than labeling the patients' disposition as PTVS. Gallop simply identified that the patients in his case report had "acquired brain injury" without discussing a specific diagnosis beyond a suspected "head injury" for one patient and "traumatic brain injury" for another.[27] Lee, on the other hand, diagnosed her patient with the synonymous term "visual vertigo."[28] Visual vertigo has been specifically defined as "dizziness provoked by visual environments with large-size (full-field) and repetitive or moving visual patterns" by Bronstein,[37] which was not indistinct from the synonymous description of visual motion hypersensitivity by Winkler and Ciuffreda.[20] At this time, there is not a universally accepted diagnosis that describes and defines the sensation of disequilibrium with visual motion after brain injury. This shortcoming likely explains why the label of the conditions being treated has varied. Healthcare practitioners and patients alike would benefit if the international classification of diseases included "visual motion sensitivity" as an identified diagnosis.

There is no standard for the long term-efficacy of BNO for the treatment of VMS. Two of the three patients in this case series found that the BNO was beneficial months after it was initially applied. Proctor reported that her patient found benefit six months after its initial application. Gallop reported that the BNO was efficacious after one week for one patient but did not follow forward with the second.[27] Lee reported that her patient was able to conduct activities of daily living with BNO, implying that the efficacy extended beyond just the office evaluation. Ciuffreda et al. and Yadav and Ciuffreda did not follow the subjects in their laboratory studies over time.[15,25] The typical recovery timeline for mTBI is three months,[38] although symptoms can persist for longer periods of time due to post-injury psychological adjustment, premorbid vulnerability, and changes in brain function post-injury.[39] Future research that investigates the long-term effect of BNO is indicated.

The effect of combining vision rehabilitation and BNO is not well understood. Visual and vestibular therapy has been shown to improve VMS symptoms without BNO.[40] The combination of prescribed vision rehabilitation and BNO, much like in this case series, has also been previously reported, but with limited understanding of the long-term outcome. Gallop treated two patients with BNO and vision rehabilitation but did not report on the outcome after therapy was initiated or completed.[27] Additionally, Proctor's patient only wore BNO for two weeks in conjunction with therapy.[26] All three patients in this case series performed vision rehabilitation with BNO, but whether the therapy independently improved any VMS symptoms is not known. It is yet to be determined what effect vision rehabilitation has on the BNO's effect on VMS symptoms. A future study investigating whether vision rehabilitation has a confounding effect is indicated.

Ciuffreda reported that the diagnosis of "visual vertigo syndrome" could be made with a rotating optokinetic drum in the periphery of a patient's peripheral visual field.[22] Examiners did not perform testing with the optokinetic drum on subjects in this case series. Therefore, it is possible that the positive response to binasal occlusion in the subjects in this case was due to other pathology. Two of the three subjects in this case series were diagnosed with possible vestibular dysfunction by a physical therapist; it is possible that some of the dizziness and vertigo symptoms reported during optometric examination were due to a vestibular dysfunction and not to VMS or perhaps to a combination of VMS and vestibular dysfunction. It is also possible that the symptomatic relief reported from BNO may have improved symptoms associated with a separate vestibular disorder and not VMS. Further investigation on whether BNO might treat vertigo not associated with VMS is indicated. Collins et al. report that there are multiple clinical trajectories for concussions, which can overlap.[41] These trajectories include anxiety/mood, vestibular, oculomotor, cognitive, post-traumatic migraine, and cervical. It is possible that the subjects in this study suffered any one of these types of concussions or a combination of multiple types, thus possibly eliciting their complaints and clinical findings from a different source. It is not known whether specific subtypes elicit positive responses to BNO or optokinetic drum testing.

Conclusion

BNO is a technique that clinicians should consider trying on their brain-injured patients complaining of symptoms of VMS. It has been effective on patients of multiple mechanisms of head trauma. The improvement in symptoms is typically immediate, and the amount of occlusion necessary is generally minimal, usually at the nasal canthi or less. Translucent occlusion was successful in reducing VMS for patients in this case series, but more research is indicated to determine whether translucent or opaque BNO is more efficacious. Gait evaluation is a diagnostic measure that clinicians should consider when using BNO on patients complaining of symptoms of VMS. Further information is needed to investigate the long-term efficacy of BNO, whether BNO improves symptoms of vertigo associated with vestibular dysfunction, and whether concurrent neuro-optometric vision rehabilitation has any effect on recovery time.

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Appendix A

WAMC NEURO-OPTOMETRY INTAKE QUESTIONNAIRE

	Never	Not Very Often	Sometimes	Fairly Often	Always
Do you bump into objects and walls more often now than before your injury?					
Do you (or has anyone told you) sway or bump into people when you walk since your injury?					
Does walking seem harder now or require more concentration than before your injury?					
Are you more sensitive to light (-- indoors -- outdoors -- both) since your injury?					
Do you find yourself dimming lights or lowering brightness on electronics since your injury?					
Do crowds bother, overwhelm, or cause dizziness since your injury?					
Do loud noises bother, overwhelm, or cause dizziness more since your injury?					
Do you get nauseous or uneasy in busy or crowded environments since your injury?					
Have you noticed a change in your vision since your injury?					
Do you cover or close one eye at times since your injury?					
Is your vision blurry at -- distance, -- near, or -- both since your injury?					
Have you had any double vision since your injury?					
Have you noticed any changes in your peripheral vision since your injury?					
Have you noticed a change in your ability to read since your injury?					
Do you lose your place while reading more now than before your injury?					
Do you get headaches during/after reading more now than before your injury?					
Do you have more difficulty with night time driving now than before your injury?					
Do you have burning, itching, redness, or tearing of eyes more now than before your injury?					
Other visual symptoms not listed:					