ABSTRACT

Background: Wallenberg Syndrome (WS), or lateral medullary syndrome, is a rare condition that occurs after an infarction or stroke in the lateral medulla oblongata in the brainstem. Due to the many structures, nuclei, tracts, and fibres that compose the lateral medulla, WS has a characteristic constellation of symptoms. This may consist of eye movement abnormalities, visual midline shift syndrome (VMSS), Horner’s syndrome, speech and language deficits, vertigo, nausea, and vomiting, along with varying degrees of head/body/limb sensory-motor dysfunctions that can affect posture and balance. This case report analyzes and discusses the clinical findings and the acute optometric treatment and neuro-visual processing rehabilitation (NVPR) that were provided to a patient in Canada with Wallenberg syndrome, as well as how NVPR facilitated functional recovery.

Case Report: A 67-year-old Caucasian male presented with acute diplopia, a left droopy lid, and listing to his left in his wheelchair. Six days previously, he was admitted to Joseph Brant Hospital after having a two-day history of unsteady gait, nausea/vomiting, and spinning sensation. He was diagnosed with Wallenberg lateral medullary syndrome. A neuro-visual processing evaluation revealed Horner’s signs, visual midline shift syndrome, post-trauma vision syndrome, visual-vestibular dysfunction, deficits in visual figure-ground, and cerebellar dysfunction. Acute optometric care was provided, with artificial tears, sector occlusion, and spectacles with yoked prism prescribed, followed by 20 sessions of in-office NVPR. Following this course of treatment, the patient was able to ambulate without walking aids.

Conclusion: Patients with WS have physical limitations and vision problems that vary in severity. This syndrome will affect balance and ambulation. Acute management with NVPR can improve functional vision and help physical rehabilitation.

Keywords: neuro-visual processing rehabilitation, post-traumatic vision syndrome, visual midline shift syndrome, Wallenberg syndrome

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Introduction

A 67-year-old Caucasian male was referred by his in-patient rehabilitation occupational therapist (OT) from the local Joseph Brant hospital for evaluation of acute double vision to the right of midline and a droopy left upper lid following a medically diagnosed stroke. He arrived with his rehabilitation OT in a wheelchair for assessment. Six days before, he was admitted to the emergency department with a two-day history of unsteady gait, nausea/vomiting, and spinning sensation.

An MRI revealed an acute/subacute infarct of the posterior left lateral medulla. He was diagnosed with Wallenberg syndrome (WS) and was transferred to in-patient rehabilitation four days later. A full patch was initially worn over one eye to alleviate his diplopia, but was discontinued at rehabilitation.

His OT reported that he had mild dysphagia (difficulty swallowing), mild dysarthria (slurring of speech), mild tongue weakness, ataxia, improving parasthesia and weakness (right worse than left side), mild left facial numbness, and left lateropulsion when he would stand during mobility rehabilitation.

Post-cerebral vascular accident (CVA), he noticed general blurriness at near with his spectacles. He had a history of multifocal contact lens use and he had an additional pair of distance-only spectacles that were prescribed by his primary care optometrist. Other symptoms reported included nausea, dizziness, vertigo, light sensitivity, difficulty tracking words on the page, left eye discomfort, and decreased balance.

He was taking medications for ischemic heart disease, hypertension, and dyslipidemia. He also had a history of four coronary artery bypass grafting
procedures with stenting, as well as rib surgery for a benign tumour. He was an ex-smoker, did not take recreational drugs, and reported drinking socially. There was a family history of transient ischemic attack on his father's side, and his uncle died of a heart attack around his mid-50s. Prior to his CVA, he was a bachelor, worked in an office at a land development company, and lived alone.

**Background**

Strokes of the medulla oblongata are rare, with WS making up only 2% of ischemic infarctions and Dejerine medial medullary syndrome 1%. Collateral involvement of WS with the medial medulla and bilateral involvement of the medulla are even rarer. This is due to the unique vascular supply to the medulla.

In lateral medullary syndrome, there is an occlusion and/or ischemia of the posterior inferior cerebellar artery; while in medial medullary syndrome, the problem lies in the anterior spinal artery. Both arteries are branches of the larger vertebral arteries, and large vertebral artery related diseases such as atherosclerosis in older patients and dissection in the younger population account for the majority of WS. Conditions such as heart disease, hypertension, a family history of stroke, and smoking are high risk factors.

In medullary syndromes, the pattern of signs and symptoms is unique, unlike in other brainstem disorders, because of the many decussating pathways to and from the brain and spine, and the vestibular nuclei, as well as varying degrees of the medullar areas (lateral, medial, upper, lower) that are affected.

In medial medullary infarct, the pattern is associated with the clinical triad of contralateral cross-pyramid hemiparesis, contralateral medial lemniscus sensory loss, and ipsilateral CNXII palsy.

In WS, the most prevalent signs and symptoms, according to the largest review of 130 WS cases, are ataxia (gait 92%, limb 55%), sensory problems (96%), dysphagia (55%), and dysarthria (22%). The most common signs and symptoms related to the eyes include Horner's syndrome (88%), vertigo (57%), nystagmus (57%), nausea (52%), headache (52%), and diplopia (32%). Two studies by the same researchers found about 94 to 100% of patients reporting deviation of the subjective visual vertical as well as ocular tilt reaction (cyclorotation in one eye) and skew deviation occurring in 41 to 50% of cases. Other vision abnormalities such as oculomotor dysfunctions, saccadic dysmetria, ocular lateropulsion (eye movements biased to one side), and lateropulsion (body leaning to one side without paresis) have also been described, with implications that abnormal slow/fast eye movements and gaze-evoked nystagmus are caused.

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<th>Table 1. Demographic Data of Students with Refractive Error</th>
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<td><strong>Change in Refraction</strong></td>
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<td><strong>Accommodative Dysfunctions</strong></td>
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<td><strong>Oculomotor Dysfunctions</strong></td>
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<td><strong>Binocular Vision Disorders</strong></td>
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<td><strong>Visual Perceptual Disorders</strong></td>
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<td><strong>Dysfunction in Visual-Vestibular-Proproprioeception Integration</strong></td>
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<tr>
<td><strong>Visual field deficits</strong></td>
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<td><strong>Other</strong></td>
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<td><strong>Symptoms</strong></td>
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by impairments and/or disruptions in the cerebellum and related pathways.\textsuperscript{13}

Undoubtedly, these signs and symptoms can significantly impact a person's daily functioning and quality of life.\textsuperscript{14,15} In the field of neuro-optometric rehabilitation, post-traumatic vision syndrome (PTVS)\textsuperscript{16,17} is a diagnosis often made after neurological events, such as traumatic brain injuries or strokes, that describes the possible manifestations that occur when the visual processing pathways are not functioning properly (Table 1).\textsuperscript{18} It is important to note that PTVS is a clinical term that may not have widespread recognition in all medical or scientific circles, but the concept has been developed or explored in more recent research or clinical practice. The theory behind PTVS centres around the hypothesis that the ambient visual process, which is responsible for pre-conscious peripheral vision, localization/orientation in space, motor planning, and coordinating balance, is compromised after brain insults. This results in the focal visual process, responsible for conscious central vision necessary for object identification and comprehension, going into overdrive or becoming more dominant. It becomes unbalanced with the ambient system on which it depends. The signs and symptoms relate to this disorganization in functional vision, spatial vision, balance, and even memory and visual perception/cognition.\textsuperscript{18} Moreover, visual midline shift syndrome (VMSS) can emerge, whereby the ambient system, failing to match its spatial vision with the incoming vestibular and sensorimotor information, will try to change the visual perception by shifting the egocentric visual midline to create postural balance. The mismatch can cause posture modifications, such as weight shifting or leaning to one side, of which the individual may or may not be aware. One study showed that VMSS was present in over 50% of post-stroke subjects.\textsuperscript{19}

Neuro-visual processing rehabilitation (NVPR) is a dynamic and individualized therapeutic approach that capitalizes on neuroplasticity to optimize visual recovery and integration in individuals with visual deficits caused by brain injuries or neurological disorders.\textsuperscript{20} It frequently is provided in conjunction with other rehabilitation and healthcare professionals. Neuroplasticity refers to the brain’s ability to reorganize its structure, function, and connections in response to learning, experience, and injury.\textsuperscript{21,22} It is a fundamental property of the brain that allows it to change and adapt throughout life, and it is crucial for learning, memory, and recovery from brain injuries.\textsuperscript{23,24} It is a central concept in understanding how rehabilitation can occur, including NVPR.\textsuperscript{25,26}

The process of neural plasticity in NVPR involves targeted stimulation, repetition, and adaptation of visual pathways to improve visual function and processing.\textsuperscript{26,27} The neural plasticity process works in NVPR as follows:

- **Assessment and Evaluation:** The process begins with a comprehensive assessment of the individual's visual system and neurological function. This may include evaluating visual acuity, eye movement control, focusing ability, peripheral vision, and other aspects of visual processing. Additionally, the individual's specific neurological condition or injury is considered to understand the underlying challenges.

- **Customized Treatment Plan:** Based on the assessment, a personalized treatment plan is developed. This plan outlines the specific visual deficits and challenges to be addressed and identifies the most suitable interventions to promote neuroplastic changes, including projected outcomes.

- **Stimulation of Neural Pathways:** NVPR involves a series of visual activities that are carefully designed to stimulate specific neural pathways and connections such as, but not limited to, eye movement training to help improve reading, tracking, and overall eye coordination or gaze stability training to improve the ability to maintain a steady gaze which can be important for balance and spatial awareness. The activities are strategically chosen to challenge and engage the visual system at various levels of complexity.

- **Repetition and Progression:** Repetition is a key component of neuroplasticity. By consistently exposing the brain to specific visual stimuli and tasks, the targeted neural pathways are repeatedly activated, leading to gradual rewiring and strengthening of connections.\textsuperscript{22} As the individual progresses and adapts, the exercises become more challenging, encouraging further neural adaptation.

- **Feedback and Error Correction:** Immediate feedback and error correction are important during NVPR. This helps the brain to identify and correct errors in visual processing by promoting more accurate and efficient neural connections.

- **Cross-Modal Integration:** NVPR may involve integrating visual information with other sensory modalities, such as proprioception (sense of
body position) and vestibular (balance and spatial orientation) input. This cross-modal integration helps the brain create a more global understanding of the environment and enhances overall sensory processing.

- Prism, Lenses, and Visual Aids: Prism, lenses, and other visual aids such as bi-nasal (BNO) sector occlusion, low power base-in prisms, tinted lenses and stress-relieving low-power near-point correction, may be used strategically to manipulate visual input and challenge the brain to adapt. These tools can help address specific visual deficits and promote neuroplastic changes. For VMSS specifically, the use of yoked prisms have been shown to shift weight bearing or the centre of gravity in order to influence and modify spatial perception and body posture while sitting, standing, and in locomotion. The aim is to re-establish the visuo-spatial perception to what it was before the brain insult.19,28

- Syntonic Phototherapy: a non-invasive therapeutic method that utilizes specific wavelengths of light to treat various visual and neurological conditions. It is based on the principle that light can stimulate the visual system and enhance visual function.29 The therapy involves the use of coloured filters or lights that are applied to the eyes or surrounding areas. These lights can be pulsed, flickered, or held steady depending on the specific treatment protocol. It is speculated to affect the balance between the sympathetic and parasympathetic nervous systems, which can have an impact on a range of visual and systemic functions.30

- Neurocognitive Engagement: Many NVPR activities involve cognitive processes such as attention, memory, and problem-solving. Engaging these cognitive functions alongside visual tasks enhances the brain's overall processing capabilities.

- Functional Application: As visual skills improve through NVPR, the individual is guided to apply these skills to real-world situations. This helps consolidate the learned neural pathways and ensures that the improvements translate into daily activities and tasks.

- Ongoing Assessment and Adjustment: The treatment plan is continuously monitored and adjusted based on the individual's progress. This dynamic approach ensures that the activities remain appropriately challenging and aligned with the individual’s evolving needs.

Of course, a multidisciplinary approach involving other rehabilitation and medical disciplines, such as physiotherapists, occupational therapists, cardiologists, and naturopaths, is necessary to manage co-morbid health conditions and to facilitate recovery.

**Clinical Assessment**

On initial examination, the patient was leaning to his left side in his wheelchair. The left upper lid was visibly ptotic, with lagophthalmos in the resting blinking state and according to the OT, no anhidrosis. Orbicularis function was normal, as he was able consciously to squeeze both his lids shut. Pupils (left smaller than right) were reactive to light/accommodation without an afferent pupillary defect in either eye. Confrontation testing, including extinction field testing, was normal.

With his correction (OD: -1.25-0.50x040, OS: -1.00-0.50x120), his entering static visual acuities were OD 20/30-2, OS 20/40-2, and OU 20/40 for distance and OD 0.62M, OS 1.00M, and OU 0.62M OU at near. Unaided visual acuities at near were OD 0.50M, OS 0.75M-, and OU 0.50M+. Dynamic visual acuities were OD 20/50, and 20/50-1 with him reporting discomfort bordering on nausea and requesting to discontinue.

Ductions were within functional limits in each eye. During binocular eye movement testing, he lost fixation and was hypometric when saccading to his right and hypermetric going to his left. There was ocular lateropulsion on vertical saccades, where his eyes executed an oblique misdirection to the left on the upper portion of the vertical saccade. A right-beating nystagmus and internuclear ophthalmoparesis or a 3/4 left adduction deficit on right lateral gaze, were also noted. Sector occlusion (translucent tape) over the nasal portion of the left lens alleviated his diplopia on right gaze. DEM testing produced below-average vertical (V) and horizontal (H) times and a below-average H/V ratio score of 2.959. There were no obvious deficits noted on visual scanning except for a vertical scanning behaviour on the Davis Visual Scan test.

He had flat fusion with the Worth 4-dot at distance and at near. Wirt circle stereopsis was 100". NPC repeated several times was receded (>10cm) with observed OS deviating.
An updated best sphere prescription of OD: -1.75, OS: -1.75 Add +2.50 OU yielded acuities of OD 20/20-1, OS 20/30-, and OU 20/20 at distance and OD 0.50M, OS 0.63M, and OU 0.50M at near.

Eye position testing with cover test and phorometry were similar for distance at 2^ exophoria and for near at 9^ exophoria, with slower fusion ability noted in the left eye with the alternating cover test. He had a mild 1^ right hyperphoria that was worse on left gaze. Fusional vergence ranges for distance were positive fusional vergence (PFV) x/20/OD suppression, negative fusional vergence (NFV) x/6/OS suppression. Fusional vergence ranges for near were PFV x/14/OD suppression, NFV x/18/0.

Slit lamp biomicroscopy revealed exposure keratitis OS.

He demonstrated slightly below-average laterality and directionality with the Jordan Left Right Reversal Test. Groffman visual tracing was below average. It was observed the patient had a tight facial expression, and he complained of dizziness and headache.

Collier’s functional field testing wearing Bagolini striated glasses showed normal retinal correspondence, and no suppression with the projections or streaks on his right side to be generally higher than those on his left side. With the modified Brock string test, he demonstrated exo projection with intermittent OS and near vision suppression. Tapping his left shoulder for kinaesthetic feedback did not help.

Cerebellar function testing showed that he had weakness with error touching his left finger to his nose and weakness making alternating rapid movements on his lap with his left hand. There were no benefits with Streff Wedge probing.

Using the visual midline shift testing protocol developed by Padular,20 he demonstrated a perceived visual midline shift to his left. When 5^ base right (BR) yoked prisms were trial framed after extensive trial and error, an upright posture in his wheelchair was achieved. Probing with additional yoked prisms in different magnitudes and directions was not beneficial. While wearing the 5^ BR yoked prisms and having an OT as backup support, he was able to stand and walked cautiously in the examination room without leaning to his left. Other ambulation assessments (modified Romberg, Dynamic Gait Index) were not performed due to exhaustion; the patient asked to stop. In general, the patient was alert, oriented, pleasant, and cooperative, and he demonstrated good understanding of what was asked of him.

**Diagnoses/Impressions**

The patient was diagnosed with the following: WS with associated Horner’s signs, exposure keratitis, PTVS (increased myopia, oculomotor dysfunction, binocular vision dysfunction: convergence insufficiency, suppression), VMSS, visual-vestibular integration dysfunction, mild laterality/directionality problems, deficits in visual figure-ground, and cerebellar dysfunction.

This patient had incomplete Horner’s syndrome because he had ipsilateral ptosis, miosis and, no anhidrosis or enophthalmos.31 Although the apraclonidine test was not done, It is important to note that while this test can be informative in diagnosing Horner’s syndrome and differentiating between preganglionic and postganglionic causes, it may not be accurate in acute stages, such as in this case, because the denervation hypersensitivity might not have fully developed yet. Therefore, the apraclonidine test should be interpreted in conjunction with other clinical information and tests to arrive at an accurate diagnosis of Horner’s syndrome.32

When performing extinction field testing to rule out unilateral spatial inattention of his left side, ipsilateral lateropulsion was observed. Vestibular system involvement was based on his dizziness, vertigo symptoms, and dynamic visual acuity testing results. The oculomotor dysfunction was supported by the results found on the DEM and the vertical scanning pattern observed on the Davis scan test. Although there are no oculomotor nuclei in the medulla, their projections from the ascending and descending tracts to/from the brain and spine are very complex. The sector occlusion applied to the nasal portion of the patient’s left lens alleviated the diplopia when he gazed into his right, because the tape obscured the image from his left eye, and he was monocular on right lateral gaze.

There is strong overlap in the ocular and visual symptoms caused by the common pathophysiology between TBI and stroke, the increase in his myopia was most likely caused by pseudomyopia related to the PTVS. According to London et al., many patients exhibit a myopic shift in refractive error following TBI, which they term to as “post-traumatic pseudomyopia.”33 One of the major visual problems observed in 161 patients with head injuries, according to Kowal, was a 19% prevalence rate of pseudomyopia...
The oblique cylindrical powers were removed, albeit small, from his spectacles to eliminate any inherent spatial distortions that they might have caused and strived to give him symmetrical perception from each eye. His best corrected visual acuity in his left eye was reduced, likely secondary to the exposure keratitis from the lagophthalmos.

His binocular vision dysfunction was supported by the clinical findings: reduced stereo-acuity, remote NPC, convergence insufficiency, alternating suppression during fusional vergence testing, and exo deviation on Brock string test with suppression. Collier's functional field testing demonstrated that he had skewed projections, with suppression and spatial localization errors.

Yoked-prism probing was done with flippers in various magnitudes: 0.50\(^\wedge\), 1.0\(^\wedge\), 2.0\(^\wedge\), 5.0\(^\wedge\), and 10\(^\wedge\). 5\(^\wedge\) BR yoked prisms, what was prescribed, probably could have been refined. With the next increase in prism power of 10\(^\wedge\) BR yoked that was available, he did not lean to his right, but he also did not like the distorted visual perception or non-uniformed magnification as described by Ogle from the higher-powered yoked prisms.

In addition to the diagnosis of WS, it was suspected that the medial part of this patient's left medulla was also likely affected due to 1) the contralateral weakness; 2) improving contralateral paresthesia (heightened proprioception), commonly seen in acute strokes before the heightened proprioception turns to decreased proprioception; and 3) tongue weakness, all of which comprise the characteristic clinical triad presentation of medial medullary infarct. There was also suspected concomitant, partial left medial longitudinal fasciculus involvement because his eyes moved independently on ductions, but he could not synchronize horizontal conjugate eye movement to the right. About 1/3 of internuclear ophthalmoplegia (INO) cases are caused by infarctions in the older population, while another 1/3 are from demyelinating disease like MS in younger people.

**Case Conference & Treatment/Management Plan**

The patient and his OT were counselled about the diagnoses and no known cure for the Horner's signs. He was instructed to continue to lubricate his left eye to prevent infection. He acknowledged that, as a bachelor, he frequently ordered takeaway, which typically has much higher levels of saturated and trans fats than home-cooked meals. He was advised due to his history of bypass surgery and stenting to consult a dietician/nutritionist to help develop better eating habits and to plan healthier meals. It was strongly recommended that he return for a dilated fundus examination to complete his ocular health assessment as soon as he was able.

In the interim, preservative-free HYLO eye drops were prescribed q2h OS and Vigamox, 1gtt, TID OU for 7 days to prevent infection in his left eye and as prophylaxis in his right eye. The use of artificial tears should help improve his best corrected visual acuity in his left eye, in the presumed absence of structural anomalies within the eye. He was advised to practice squeezing his lids shut several times per day and to consider taping his left upper lid during sleep.

Prescriptions for three pairs of spectacles were issued. First were distance spectacles with nasal sector occlusion on the left lens, 10% blue tint, and 5\(^\wedge\) BR yoked prisms to be used for mobility rehabilitation. Second, low plus +0.75 OU was given for reading, with sector occlusion on the left lens. The third prescription was for single-vision polarized distance sunglasses without yoked prisms for his photosensitivity. Progressive lenses and multifocal contact lenses were not recommended until his ambulation improved due to the inherent spatial distortions that they cause.

- The final power and direction of the yoked prisms were based on the left visual midline shift. They were prescribed to align his posture and facilitate orientation/mobility rehabilitation with his OT. The patient and the OT were reminded that as his posture and balance improved with vision guiding his mobility rehabilitation, his brain would continue to reorganize with other sensorimotor systems. It was important that the patient be monitored on a weekly basis to update the prism magnitude and/or direction as needed based upon changes observed.
- Uni-nasal sector occlusion was applied to the left lens for diplopia management on mid-right to right lateral gaze. The width of the translucent tape was expected to be tapered as his oculomotor skills improved.
- The blue tinting was prescribed based on clinical experience and from positive anecdotal reports from the majority of patients. Acute neurological cases tend to appreciate the blue end of the spectrum, while chronic cases prefer the FL41 tints. Shorter wavelengths focus in front of the retina and can act as low-plus lenses to help increase VOR gain. In this case, the patient was
suspected to be focally bounded and would not accept anything less than 20/20; therefore, lowering his Rx was not his preference, and adding the blue tint was the compromise.

- Low-plus readers were prescribed to increase, or to normalize, the VOR gain and to reduce motion sensitivity;\textsuperscript{37} this was based on the dynamic VA testing at distance and the assumption that the dynamic VA testing at near, which was not performed, would also be positive. The amount prescribed was verified by the overall brighter and more brilliant retinoscopy reflex colour indicative optimum engagement and cognitive processing with the left eye being slightly dimmer and irregular from the exposure keratitis. It should also be noted that the power needed could also be presbyopia-related.

- The patient decided not to have yoked prisms in the polarized sunglasses due to the associated replacement expenses and the potential reduction in the magnitude of yoked prisms with ambulation rehabilitation.

To maximize recovery and performance, a NVPR program was strongly recommended. The ultimate goal was to improve and to synchronize the foundational visual process, oculomotor/accommodative/binocular vision functions, and visual perceptual skills with the integration of other sensorimotor functions. NVPR includes training in awareness, interpreting what is seen, solving sensory-motor challenges, and providing repeated feedback for the highest level of cognition (attention, concentration, thinking, comprehension), as well as for the most favourable motor executions for spatial orientation, movement in space, and balance in life.

It should be noted that although heath care services are excellent in Canada, NVPR is not an insured service. An individual participating in a government-funded in-patient rehabilitation program does not usually participate in in-office NVPR, until he is discharged from rehabilitation or is willing to pay out-of-pocket or has private insurance for the NVPR and travel expenses. Instead, the basics of NVPR are often prescribed and reviewed via telephone conferencing for the rehabilitation OT to administer. Even though there is no revenue generated in this type of patient management arrangement, the referrals from working with the rehabilitation OT and from the patients who do decide to continue to participate after they are discharged are well worth it. The OT is fully aware that sector occlusions, spectacles, tints, etc. are prescribed only by, and cannot be provided without, the neurovisual optometrist. This relationship has worked out well. In-office follow-ups, on the other hand, are based on the financial resources that the patient has and are often not on the recommended re-scheduling. Given that this patient had no family support, extraocular range of motion activities (oculomotor smooth pursuit and visual-vestibular activities including gaze stabilization, head/eyes moving in the same direction) were prescribed, with a follow-up in one week either in person or by teleconferencing with the OT.

The sessions were performed with the patient’s yoked prisms first to stabilize posture, then with his multifocal contact lenses as he improved, always barefooted with occasional Voox socks to increase proprioceptive feedback. The environment went from quiet and uncluttered to noisy, busier scenes with the therapist creating distractions through singing, humming, or tidying up nearby. The patient’s posture started off supine, then progressed from sitting to standing to walking with different magnitudes of lenses and/or yoked prisms added to increase the challenge. Gaze stabilization was initially done with, then without, tactile support. Peripheral awareness activities were done with Binovi Touch, Sanet Vision Integrator, flashlight pointing, MacDonald cards, etc.\textsuperscript{38} Binocular vision development (monocular to bi-ocular to “monocular fixation in a binocular field” to binocular, from “seeing/feeling” gross to fine motor discrimination in eyes/body) started with oculomotor, using Hart chart saccades, coin circles, Marsden ball tracking, mazes, and line tracings, then accommodation (feeling soft/hard, mental minus, lens sorting, monocular accommodative rock). This was followed by bi-ocular, anti-suppression, monocular fixation in a binocular field activities (red colouring, Squinchel, dowel sticks with vertical dissociating prisms, direct/indirect cheiroscopic tracings, and physiological diplopia) and finished off with binocular function (binocular accommodative rock, look & catch ball, ball bunting, slap-tap, distance/near tranaglyphs & vectograms, aperture rule, chioptic/orthoptic fusion with opaque/clear life saver cards, Brock string, BIMBOP, rotator Ts).\textsuperscript{37,38}

**Prognosis**

Any rehabilitation using vision simultaneously to guide motor movement has the potential for positive outcomes due to neural plasticity, even if there is brain damage from trauma, disease, age, drugs,
mental illness, or stress. The tenets of NVPR are based on the brain's ability to change, reorganize, develop, and enhance neuronal pathways throughout life. Hence, NVPR can be engaged at any age. The patient's motivation, commitment, and positive mindset or attitude are also keys to meaningful and successful NVPR. However, if there is a concurrent structural demyelination condition, then the prognosis will be guarded, and remediation/rehabilitation will depend on neural plasticity.

**Follow-ups**

The patient returned three weeks later for a follow-up evaluation (Table 2). Leading up to this appointment, the weekly telephone conversations with the OT were positive. The yoked prism spectacles allowed the patient to stand upright and to perform trunk/leg strengthening exercises. He was also able to walk with his 4-legged cane when he was wearing his MFCLs. He used the wheelchair only when he was tired. He saw an ophthalmologist at the hospital and was told to continue with the artificial tears prescribed. His ocular health was normal for his age. His speech was no longer slurred, but it was broken because he had to take break periods to swallow. He described that he was not as light sensitive, and his left eye was not as dry with the drops. He was seeing better with his new spectacles but still reported diplopia if he looked to his extreme right. Dizziness without vertigo was still present when he moved his head, especially when he read.

Entering VAs with his new multifocal contact lenses from his primary care optometrist were 20/20 OU at distance and 1.25M- OU at near. Adding +1.50 OU over his contacts improved his near VA to 0.62M+. Dynamic VAs at distance and at near were 20/25 OU. His EOM testing results were more accurate until he moved his head. He demonstrated 90% adduction deficit right gaze. R beating nystagmus on right gaze was present. BVD: CI Not assessed. N: 9 exo, suppression with vertical phoria right gaze. NPC was still remote (>10cm) with OS deviation. His visual midline was slightly shifted to his left. He needed 2^ BR yoked prisms for immediate balance standing and walking.
without rocking side-to-side. Probing with syntonic filters did not yield a positive change.

Since he was comfortable wearing his multifocal contact lenses for general vision, the patient was instructed to update his distance spectacles with decreased yoked prisms and to use them only for mobility training with his OT at out-patient rehabilitation. A new prescription for distance spectacles with $2^\circ$ yoked prisms was issued due to the immediate steady, upright posture observed without his 4-legged cane. It was also recommended that he update the add in his multifocal contact lenses with his primary optometrist for better near vision. He was told that there was no reason for him to look to his extreme right except for his ocular calisthenics warm-ups every morning. The second phase of VOR adaptive activities was prescribed three days/week for three weeks. He enrolled in an NVPR program and came every two weeks to see the vision therapist.

After 10 sessions of in-office NVPR, he was reassessed. He reported that his posture, gait, ambulation, and balance were improving at out-patient rehabilitation. He used a regular cane now as security when he was wearing his multifocal contact lenses. He could read up to 30 minutes without fatigue. Sector occlusion on his left lens was removed because the range of horizontal motion to his right was within normal limits, and he did not report diplopia even though there was a persistent right-beating nystagmus. His visual midline was still shifted slightly to his left, and the Romberg test was positive with eyes closed and feet shoulder width apart standing on a hard surface.

After 20 sessions of NVPR, he arrived without any assistive devices. His best spherical prescription reduced to -1.25 OU with 20/20 visual acuity in each eye at distance. There was still a one-line difference on DVA testing at distance only. EOM testing was within normal ranges, with a small right-beating nystagmus on right gaze. His stereo acuity improved to 30 seconds of arc. NPC was within normal limits with slight recession over time from fatigue. There was no vertical phoria on right gaze. His score on the Jordan Left Right Reversal Test and Groffman Visual Tracing normalized. His functional field testing showed normal projections with central fixation without suppression. The score on the Dynamic Gait Index, a tool used to assess gait, balance, and risk for falls with changing walking demands, was 20/24, with the lowest scores in tasks involving speed and pivot turning with weight shifted slightly to his left. (Below 21 is indicative of increased risk for falls; the lower the number, the higher the risk) Romberg testing was still positive with eyes closed, feet shoulder width apart. Cerebellar testing showed the same weakness touching his left finger to his nose and mild weakness making alternating rapid movements on his lap with his left hand.

Upon completion of his NVRP, he was very happy with the nearly resolved dizziness and improved balance. He required his regular cane only when he was very tired. He was more accepting of his permanent left lid ptosis and was glad that it was not interfering with his reading and tracking ability on the computer. He was not as light sensitive and could wear his polarized sunglasses over his multifocal contact lenses outdoors. There was still noted weakness on his right side with loss of temperature sensation. He also had persistent left facial numbness, which he said he could live with. He was seeing his cardiologist regularly and had a new nutritionist. The final spectacle prescription issued was -1.25, add +1.75 OU with 1.00$^\circ$ BR yoked prism, with instructions that they be worn on a tapering basis when attending his maintenance sessions with his physiotherapist every two weeks. Despite his residual physical limitations, the patient was managing well.

At his three-month post-therapy assessment, he was asymptomatic, and he only wanted his regular eye examination performed due to limited financial resources. He said that the multiple spectacles that he was purchasing were bankrupting him. He rarely noted dizziness now and could quickly regain his stance when there was a loss of balance. He had stopped using his cane since the last visit and had discontinued the wear of his yoked prism spectacles for about three weeks. His physiotherapist told him that his core muscles were stronger with persistent weakness on his right side. His clinical findings were within normal ranges, except for the ptosis and miosis left eye and early nuclear sclerotic changes.

The timeline from the assessment to the three month post therapy progress evaluation is depicted in Figure 1.

**Future Plan**

The patient was instructed to see his primary care optometrist for routine eye health and vision care. He was also asked to continue to see his cardiologist for
cardiovascular health and blood pressure regulation, his physiotherapist for cerebellar function and muscle/balance/swallowing training, and his nutritionist to maintain healthy meal plans.

Discussion

Strokes happen in an instant, with aggressive neurological damage and rapid changes at the beginning. The condition then evolves to become a progressive disorder as the brain stabilizes, with temporary or long-lasting functional disabilities and/or neurological sequelae.

Using neural plasticity as its foundation, this case report illustrates the importance of including NVPR early after a CVA to help alleviate acute visual symptoms that can impact physical rehabilitation. A 13-year follow-up of a case by Ciuffreda and Tannen support the importance of early and focused rehabilitation and intervention for patients with this syndrome. The results of this first-person account offer important insights into the long-term effects of WS on oculomotor function and neural plasticity. While the initial decade of intensive rehabilitation following the diagnosis showcased the remarkable adaptability of the brain to rewire itself in response to the neural damage, it appears that the capacity for further improvement may be limited. Additionally, the observed age-related changes in oculomotor responsivity serve as a reminder that our bodies and brains are in a constant state of change, influenced by both intrinsic and extrinsic factors. The findings of this longitudinal study emphasize the need for future research to better understand the interaction between age and neural recovery.

In general, if the two eyes are not pointing in the same direction, there will be localization errors.

**Figure 1. Timeline summary**
as to where the patient thinks he is in space. This affects the vestibular and somatosensory cues and reference frames. The two systems then compensate with additional errors, and the structure or body becomes unbalanced with body lateropulsion for example. When the visual process is rehabilitated and enhanced through NVPR, it can visually guide and support gait, posture, and mobility challenges and can sometimes even hasten recovery. Further, outside of the physical limitations that medullary strokes bring about, cognition is not affected, which allows the patient to engage fully in NVPR.

Observing and understanding the postural modifications that a person makes after a CVA was the key in recognizing that this patient had VMSS. Yoked prisms were used to modify spatial perception, improve postural tone, and facilitate weight shift towards a balanced, upright posture.

A multidisciplinary approach was ideal in this case. Together with the OT administering the basics of NVPR as directed and facilitating mobility/movement while wearing the yoked prism spectacles prescribed, this patient regained control of much of his voluntary movement, including strength and flexibility in his limbs and trunk. He was able to progress from sitting in a wheelchair to standing with the aid of a cane, to standing without that assistance. His participation in the in-office NVPR helped synchronize his visual, vestibular, and somatosensory maps toward easy and effortless movements in all stances and in all environments over time.

If he had had more stamina at his initial assessment, the following would have been done or considered:

1) Eye movement recordings to objectively track and analyze his eye movements in order to gain a better understanding of the relationship between the visual symptoms and the underlying oculomotor processes. This information, together with the Double Maddox Rod and supine/upright testings for skewed deviation, can provide insights into the anatomy and neurological mechanisms at play and contribute to a more comprehensive understanding of the disorder’s progression and recovery.\textsuperscript{41-43} It is interesting to note that clinical observations of visually guided eye movements with MRI data can provide valuable insights into the classification of WS - one with brainstem lesions and another with both brainstem and cerebellar lesions\textsuperscript{44} or gaze lateropulsion or ocular tilt reaction, is indeed a neurological sign associated with lateral medullary disease.\textsuperscript{45} These distinctions could be important for understanding the underlying pathology and guiding treatment strategies for patients.

2) Van Orden Star cheirometric drawing to give further insight as to his visual projection and to look for any imbalances between his cognitive and visual grasping, as well as for evaluation of a visual midline shift

3) Bihemispheric Dissonance Test (BDT) from Dr. Merrill Bowan to check “visual aliasing.”\textsuperscript{46}

4) VEP to see the waveform representing his visual processing

5) Motor Free Visual Perceptual Test to obtain a baseline of his visual cognitive processing skills and to see whether there were any perceptual areas that required attention. The activities selected for his NVPR were appropriate, and even though MVPT was not assessed, the NVPR incorporated selected activities that challenged his visuo-cognitive skills.

Conclusion

The visual process can go awry just as suddenly as the brain insult or CVA that caused it. Even as rare a condition as Wallenberg lateral medullary syndrome can impact the function of the eyes, body, and mind in varying degrees. It is comforting to know that the visual process is remarkably adaptable and recoverable due to neural plasticity. It is on a continuum and does not require a beginning or an end and can be stimulated and/or initiated at any time. This case report shows that NVPR, especially when used early after the neurological event, was effective in treating a patient with VMSS and PTVS when the visual process was not working well. The development of the internal asymmetry that manifested as a postural misalignment was visible, and the appropriate and timely use of NVPR helped augment other mobility and movement rehabilitation/training and allowed the person to go from sitting in his wheelchair pre-NVPR to walking without any aids post-NVPR. Therefore, the treatment/management approach for all brain injury cases should be multidisciplinary and include NVPR as the first line of rehabilitation so that the most dominant sense, vision, can guide and coordinate all of the other sensory-motor maps in the body for the most congruent and optimum human performance.
References

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