Using Principles of Neuroplasticity and Visual-Vestibular Function in the Treatment of 6th Cranial Nerve Palsy or Paresis

Jill K. Schultz, OD, FAAO, FCOVD, FNORA
Clinical Director and Residency Coordinator of Bright Eyes Vision Clinic affiliated with Pacific University College of Optometry
Clinic locations in Otsego and Minnetonka, MN

ABSTRACT
Background
Sixth cranial nerve (abducens) palsy is a common manifestation of acquired brain injury. Abducens nerve damage results in esotropia and horizontal diplopia. The current standard of care appears to be no treatment initially other than monocular occlusion or limited use of Fresnel prism, followed by Botox or strabismus surgery at 6-12 months post injury.

Case Report
The case presented demonstrates the use of neuro-optometric rehabilitation, which includes Fresnel prism, prescribed home techniques and office visits to monitor responses. These treatments are effective for patients experiencing diplopia as a result of sixth nerve palsy.

Conclusion
A treatment protocol that mirrors other sensory motor impairments and incorporates concepts of neuroplasticity and visual-vestibular function is described. Early treatment will better position the patient for fuller recovery and improved quality of life.

BACKGROUND
The abducens nerve is very susceptible to damage and the most common adult cranial palsy (followed by oculomotor and trochlear nerves) due to the long and winding course of the peripheral nerve. This nerve feeds the lateral rectus extra ocular muscle, which is responsible for abduction of the eye. Pathology involving the midbrain, cavernous sinus and anywhere along the path can result in nerve damage. Non-traumatic cases of abducens nerve palsy especially warrant neuroimaging of the brain. In cases of normal neuroimaging, evaluation of cerebrospinal fluid may be recommended. Common causes of damage can include congenital, inflammatory, vascular, traumatic, infectious and space-occupying pathology. An analysis of 4373 cases of acquired nerve palsies determined that 1918 were involving the sixth cranial nerve. Of these 1918 cases, 503 had an undetermined cause, 417 were classified as ‘other’ while the remaining causes were neoplasm (413), head trauma (287), vascular (240) and aneurysm (58).

Acquired brain injury can create a host of ocular and neurological manifestations. Clinical presentation of abducens paresis or palsy includes esotropia and associated horizontal diplopia. The magnitude of deviation increases further into the affected field of action and results in a non-comitant pattern along with abduction deficit due to paralysis of the lateral rectus muscle. Anomalous compensatory head posture is often present in the direction of the underacting muscle. There is some confusion in the literature regarding...
nomenclature as paresis is a partial palsy and palsy is often used for any level of loss, but some say palsy is a complete loss of function. For ease of communication, we will use the terms palsy and paresis interchangeably.

Extraocular muscles provide proprioceptive input and play a role in spatial awareness, localization, motor planning and depth perception. Extraocular muscles contain palisade tendon organs that serve as their primary proprioceptors. This information is fed to the spinal trigeminal nucleus via the ophthalmic branch of the trigeminal nerve and the Gasserian ganglion, or through the ocular motor nerves. Proprioceptive information is sent to structures involved in ocular motor control (ie., superior colliculus, vestibular nuclei, nucleus prepositus hypoglossi, cerebellum and frontal eye fields). This information is also important to structures involved in visual processing, such as the lateral geniculate body, pulvinar and visual cortex.5,6 A feedback system involving the dorsal stream of vision is important to reach and grasp, spatial localization and parietal lobe function.7,8,9 This process of vision is often described as the “where is it” or “how to” system as perception impacts action.8,10

Diplopia and impaired function of the abducens nerve are likely to cause deficiencies in visual perception, which can impair function especially when combined with other neurological deficits that often accompany acquired brain injury.11,12,13 Untreated double vision and poor spatial skills are likely to lengthen rehabilitation treatment, reduce outcomes and place patients at risk for subsequent injuries. This article will not address differential diagnosis or other visual deficits, such as oculomotor efficiency, accommodative, egocentric midline shift and visual information processing impairments that commonly accompany acquired brain injury12,14-19 which may also occur concurrently with sixth nerve palsy. Instead, the focus will be on basic motor recovery to provide a stable platform for oculomotor function, fusion and the ability to derive meaning and direct action. This is critical for the rehabilitation process when these areas have been compromised.

Common Treatment of Abducens Palsy

Common treatments of 6th nerve palsy include monocular occlusion or no treatment for 6-12 months until extraocular muscle surgery or Botox is considered. It appears that a “wait and see” approach along with medical treatment is common practice.1-4,20-26 Untreated double vision leads to poor spatial skills, poor quality of life and the possibility in this case for secondary contracture and atrophy. Visual deficiencies are likely to lengthen rehabilitation treatment time and costs, reduce long-term outcomes and place patients at risk for subsequent injuries. Not surprising, the literature also suggests that visual conditions are associated with adverse psychological sequelae,27 which can potentially further complicate prognosis and recommended treatments.

Other sensory motor systems are generally not treated with a “wait and see” approach. In fact, inpatient rehabilitation is standard16,28-32 for function loss and physical, occupational and speech/language therapies are provided as soon as possible after injury. Many studies show these interventions improve outcomes and are cost effective.32-36 In contrast, visual manifestations of acquired brain injury are typically managed differently. In addition, treatment of diplopia secondary to acquired brain injury is being mentioned in the literature.37-39 This shows that fortunately, this trend is changing. It is the author’s opinion that sixth nerve palsy can and should be treated as other sensory systems post-injury to improve functional outcomes and quality of life.

Proposed Treatment Protocol

A proposed treatment protocol is suggested based on principles of:

- motor rehabilitation used in other disciplines, particularly for hemiplegia,
- vision therapy/rehabilitation and understanding of developmental vision,
- visual-vestibular concepts to engage reflexive eye movements,
- voluntary eye movements, incorporating fast and slow eye movements, and
Proposed Treatment Options and Considerations for CN 6

1. Minimal to no compensatory monocular occlusion: Full field patching does serve the purpose of eliminating diplopia, but there are reductions to binocular input, a shift in visual midline/egocenter, reduced visual field and potential contracture of the affected musculature. All of these side effects can negatively impact rehabilitation outcomes and increase potential for injury or falls. However, short-term monocular occlusion can have a place in protecting the patient from hazardous situations due to diplopia. Minimal and judicious occlusion is recommended only when needed. Spot, binasal or sector occlusion allow the patient to reduce diplopia, but still offer peripheral cues and have the maximum visual input for comfort. (see photos 1A and 1B: binasal occlusion). If full field compensatory occlusion is needed for safety, alternation of the patch is recommended to promote equality. In contrast, alternate occlusion is recommended during most therapeutic techniques to increase nerve input until symmetry is achieved. Fortunately, monocular occlusion is rarely required due to sector or binasal occlusion.

2. Fresnel prism. Fresnel prism is a relatively inexpensive and quick way to move the image to where the eye is pointing, providing fusion and thus alleviating diplopia. Reducing diplopia allows for more accurate binocular vision, increased visual field compared to occlusion, improved spatial information and reduces the possibility that the patient will have sensory adaptations. This will allow patients to have increased safety and more consistent vision. Many patients require other therapies such as occupational and physical therapy, which would be more effective with efficient visual skills as they improve sensorimotor skills. However, use of prism does not address that the eye is in an adducted position and there is potential for contracture of the muscle. For this reason prism is not recommended as a stand-alone management tool; binasal occlusion and active therapies should be offered as further described. Fresnel prism can be prescribed in a compensatory manner with the goal of reducing the power as skills are normalized. Fresnel prism may also be used therapeutically to enhance tonic fusional vergence during later stages of treatment. (see photo 2: Fresnel prism).

3. Voluntary eye movement therapy. a. Pursuit eye movement control. Monocular and binocular pursuit activities are used to improve function of the impaired
visual system. Starting monocularly, the patient is encouraged to look or “stretch” the eye as far as possible. Binasal occlusion conveniently allows for monocular therapeutic conditions during lateral eye movements. The goal would be to move the eye as far as possible in each direction, but emphasizing range of motion in the affected field (abduction of the involved eye). This is a technique that most patients can complete independently and using objects in the room (e.g. door frame, lamp) can provide feedback to the patient about how far they are stretching. Other techniques such as chalkboard circles or Groffman Visual Tracing 40 can be prescribed to provide supplemental improvement of coordinated pursuit eye movements. Once the performance in each eye is relatively equal, binocular therapy can begin. Improvements can be measured by the patient in the amount of field they can recover as they “stretch” into new space. Improvements can also be measured in the office using a Vision Disk (available from Bernell www.bernell.com) (see photo 3a and 3b: Vision Disk).

b. Saccades using eye stretches and voluntary eye movement therapy. Margolis has promoted a training method called the Margolis Eye Throwing Technique. The patient is trained to generate a monocular saccade from the most extreme lateral position of the affected field over to the unaffected position. Next, the patient learns how to “throw” his or her eyes as far into the extreme abducted position as possible. The technique is performed with the eyes closed and then later with the eyes open to develop proprioceptive and kinesthetic awareness.16 The patient can complete this activity sitting in a familiar room (e.g. living room) and can have a goal to move the eyes to a specific object that is slightly out of the normal range of vision. It should be noted how far the patient can move eyes out and effort should be made to exceed goal each time. This provides feedback to the patient and a goal to exceed prior range of motion. The ballistic nature of this activity and emphasis on motor planning mirrors research demonstrating increased motor neuron excitation.41

c. Providing patient feedback with proprioceptive localization included to regain function. Techniques to encourage movement and nerve function can be used to enhance recovery. Syncing motor and visual information with feedback of accuracy is helpful in the rehabilitation process. Many studies demonstrate the role that visual pathways play in reach and grasp type activities7-10,28,42 along with the visual relationship of
the “where is it” system. Sharma et al describes the importance of motor planning that precedes movement and how imagery can increase neuron impulse. Work by Callow et al also supports the therapeutic use of imagery for sports related injuries. For decades, developmental optometrists have been using motor activities and spatial matching techniques to strengthen impaired ocular function. Visualization techniques can be very helpful in coaching patients to reach increased levels of motility.

An example of motor feedback during saccade and pursuit therapy we use in our office is called Eye Stretches with Finger Touches, which uses proprioceptive localization to support accurate visual localization. The activity is performed monocularly with a therapist or helper. The patient will fixate on a target (ie. sticker, toy, small object, pen cap) while the therapist moves the target in a variable fashion and into the affected field. The patient is instructed to accurately touch the target on command. The therapist will vary the location of the object and provide experiences so that roughly 80% of the targets are accurately localized. Roughly 20% of the targets should be just beyond the patient’s level of accuracy in an effort to push the patient towards improvement of matching visual and motor input. This technique can also be used to emphasize pursuit and saccade eye movements. There are many other opportunities for feedback within the context of an in-office vision therapy/optometric rehabilitation program, such as use of lenses, prism, after-image transfer, filters, etc.

4. Vestibular driven eye movements. The visual and vestibular systems are anatomically, functionally and neurologically paired. The semicircular canals are arranged in planes oriented to a single pair of extraocular muscles, which results in a synchronous movement of the eyes in the same plane as head motion. For example, stimulation of the left horizontal canal will result in both eyes turning right by activating the right lateral rectus and left medial rectus while inhibiting opposing extraocular muscles. This visual-vestibular interweaving is the anatomical and physiological basis for the Laws of Hering and Sherrington. Visual-vestibular input is provided to the brain and paramount to postural stability. This elegant design lends itself to symptoms when one system is damaged, but also allows for pathways of rehabilitation due to sensory redundancy.

a. Visual Ocular Reflex (VOR): The angular VOR serves to maintain stable vision during head motion, which can be leveraged to improve function. Head movement to the right will cause compensatory conjugate eye movements to the left due to contraction of the left lateral rectus and right medial rectus. Clinically, this “doll’s eye technique” utilizes the reflexive visual ocular reflex (VOR) to increase range of motor and function. The patient is seated in a swiveling office chair (in the case of a child, the patient may need to sit on parent’s lap) with direct attention to a distant target, such as a television or a mirror. Gently rotate patients chair slowly to right and then left. Repeat the rotation back and forth. Head will move, but eyes should stay straight and stay fixated on target. In some cases it may be necessary to gently hold patient’s head with palms of helper’s hands to stabilize head. Perform for 15 min, twice a day or as tolerated. This technique should be performed monocularly with each eye to enhance effect. (see photo 4A and 4B: VOR).

b. Optokinetic nystagmus (OKN): The OKN is a type of reflexive eye movement induced by motion of a large visual scene that mimics the sensation of physical rotation. This nystagmus eye
movement can be stimulated clinically using an optokinetic drum or cloth to generate motor movement in the direction of underaction. For example, a striped target is rotated in a clockwise direction to stimulate smooth pursuit and optokinetic systems, which will produce a counterclockwise fast nystagmus repetitive movement. This technique should also be performed monocularly with each eye to enhance effect.

c. Post-rotary nystagmus (PRN): VOR allows clear vision by matching motion with the vestibular signal during head movement. The semicircular canals sense input about head velocity, which enables the VOR to generate an eye movement that matches the movement of the target of regard. This synchrony allows for clear sight during head movement. Once head movement ceases, the VOR response continues due to hair cell recovery (velocity storage) and PRN is present. Therefore, stimulation of the semicircular canals will result in a nystagmus eye movement in the direction of underaction.6,28 This reflexive eye movement can be used to drive motor recovery of abducens paresis. Rotation of the patient using a platform swing (available from Southpaw Enterprises www.southpawenterprises.com) or a rotating board (available from Astronaut Boards www.astronautboards.com) can provide a strong stimulus for motor excitation into the paretic field of action. Starting monocularly, with occlusion of the non-paretic eye, rotate the patient into the paretic field up to 10 rotations at a speed of about 0.5-1 cycles per second. Suddenly stop the rotation and you will observe the PRN driving a strong and slow response in the direction of the paretic action. This is a strong subcortical drive that can be
combined with cortical localization activities with motor feedback ("Eye Stretches with Finger Touches") as previously discussed. This treatment should be administered carefully to prevent a disequilibrium response or a fall.

5. Traditional optometric vision therapy techniques. Once fusion can be achieved and range of motion improved, vergence and oculomotor techniques can be introduced to normalize visual function. Examples include: monocular prism jumps, after-image transfer, Brock string, fusion targets and vectograms to further enhance function and comfort of the visual process. These techniques should be directed from where fusion is easily established and move into the paretic field of action. The goal is to extend the binocular range further outward. In-office visual rehabilitation is the ideal modality to provide appropriate feedback to the patient for maximum recovery.

CASE REPORT

On 4/21/2010, JK, a 41-year-old man presented for a visual examination due to recent onset of double vision. He was self-referred and lived very close to the office. He stated that the right eye doesn’t see as well and “doesn’t move”. On the morning of 3/8/2010, he woke as normal, drove to work and en route he developed horizontal binocular diplopia, which started at about 8:30 AM. At about 11:30 AM he noted slurred speech. He denied any other symptoms, such as numbness, tingling, headache, lack of coordination, dizziness, cognitive issues, chest pain or loss of consciousness. He had never experienced a similar event in the past. He had not been using a patch and was finding it difficult to function due to diplopia.

JK’s past ocular history has been unremarkable other than glasses and contact lenses for refractive error. JK works as a printer. He has a medical allergy to ibuprofen. Past medical history was significant for restless leg syndrome, a current sinus infection, smoking one pack of cigarettes per day for the last 20 years and occasional drinking. He reported taking prednisolone, cephalexin bid, ropinirole qhs and triamcinolone acetonide bid. Family history was significant for coronary artery disease.

Per the patient, he had a neurological evaluation on 3/9/2010, which found no determinable cause of his double vision and he was being referred to a...
larger institution for more testing. It was noted that JK’s condition has responded to steroid therapy. His imaging was normal. Records were not available at the time of the evaluation.

Presenting aided distance visual acuity was 20/30- in the right eye and 20/30- in the left eye. Near corrected visual acuity was 20/20. Unilateral cover testing at far revealed constant right esotropia of 14 prism diopters and at 40 centimeters constant right esotropia of 14 prism diopters. Alternate cover test was performed in nine positions of gaze and an increase in magnitude to the right at 20 prism diopters and a reduced magnitude of five prism diopters to the left, indicated noncomitant deviation. Extraocular muscle examination demonstrated an underaction of the right lateral rectus muscle with limited abduction past the midline. Pursuit eye movements were uncoordinated due to gaze evoked nystagmus. JK was unable to appreciate random dot or lateral disparity stereopsis. Uncrossed diplopia at far and near was found with Worth 4 dot testing, which increased in right gaze. Near point of convergence was to the nose. Manifest refraction was -2.00 -1.00 X 001, 20/20 for the right eye and -1.75 -1.75 X 170, 20/20 for the left eye. Trial framing of prism yielded best fusion, binocularity and comfort with 14 base out prism diopters. Color vision testing was significant for deficiency in red-green color discrimination. Confrontation visual fields, external and internal ocular health tests were unremarkable. A gross gait assessment indicated no obvious anomalies.

JK was diagnosed with a right abducens nerve palsy resulting in diplopia secondary to right esotropia, associated dysarthria and normal ocular health. The patient had an excellent response to prism and a compensating Fresnel prism of 12 diopters was applied to the right lens of his habitual spectacles. Initially, 14 prism diopters was desired, but was out of stock. Fortunately, he was fusing beautifully, and very happy with the 12-prism diopter Fresnel. (Note: often a Fresnel prism used in these conditions provides some blurring of the target which allows the patient to more effectively use peripheral fusion to obtain single vision. As the Fresnel prism is reduced the blur reduces, emphasizing more central fusion.) Vision exercises were prescribed to rehabilitate the paretic neuromuscular condition. JK was instructed to perform VOR/Doll’s eye exercises 2-3 times daily for 10-15 minutes, eye stretches with Eye Stretches with Finger Touches with a helper and a modified Margolis Eye Throwing technique several times per day. JK was asked to return to the office in one month for assessment of his response and hopeful reduction of the prism. Records were requested from his neurologist. JK returned as recommended, but had a setback in his health. He still did not know his medical diagnosis, but doctors had reduced his prednisolone dose, which appeared to worsen his double vision. Examination revealed that his esotropia was indeed worse and increased to 26 prism diopters. An additional Fresnel prism was applied to the left lens and the same at-home exercises were recommended. (Note: The Vision Disk can be used to more precisely quantify range of movement and equality of the left and right abduction ability. A single nasal strip of occlusion could have been applied to promote abduction range of movement and reduce diplopia.] JK returned for four more visits approximately every 3-12 weeks over the next year to assess his visual response to medical treatment, Fresnel prism and prescribed home visual-vestibular exercises. He improved greatly and finally was able to achieve full range of motion and was diplopia free with no prism on 5/12/2011. A small amount of base in prism was prescribed for one hour a day to improve negative fusional vergence ranges. JK was given the option of in-office vision therapy/neurooptometric rehabilitation, but declined further treatment. The patient progressed beautifully with the treatment given and denied symptoms, such as eye strain, diplopia, inaccurate spatial judgments, loss of place when reading and headache.

CONCLUSIONS

Vision is a neurological sensory motor process that provides stabilizing input and feedback for other sensory motor systems. A poorly functioning or inconsistent visual system can impair other abilities related to motor, balance, vestibular, attention and
communication. Treatment of visual impairments, such as esotropia secondary to abducens nerve palsy, can and should be implemented as soon as possible to enhance visual skills, but also other skills that depend on a fully functional visual system. The case report illustrates the need for improved referral protocols. Despite the presenting visual symptom of diplopia and examination by at least 21 physicians, there was no record of referral or management for his visual rehabilitation. Optometrists that specialize in neuro-rehabilitation have an opportunity and responsibility to educate physicians involved in rehabilitation that vision treatment can help support the entire rehabilitative process and thus get the patient back to work and play sooner. The suggested treatment protocol is more consistent with other rehabilitation disciplines, is cost-effective and increases the potential to avoid surgery or more invasive medical treatment. Providing the above opportunities for improvement will position the patient for maximum recovery in both ocular range of motion, but also a more ideal platform to successfully engage in other therapies.

REFERENCES


AUTHOR BIOGRAPHY:
Jill K. Schultz, OD, FAAO, FCOVD, FNORA Otsego and Minnetonka, Minnesota, USA

Dr. Jill Schultz is a Minnesota native and practices in Otsego and Minnetonka, MN. After her undergraduate studies at the University of Minnesota-Duluth, she obtained a Doctorate of Optometry degree with honours from Pacific University College of Optometry. During her training, she developed a greater interest in children’s vision and strabismus, which led her to pursue a residency in Pediatric Optometry and Vision Therapy from the Southern California College of Optometry. Her interests and experience include pediatric eye care, binocular vision, pediatric and adult strabismus, neuro-rehabilitative optometry, myopia management, dry eye, nutrition and evaluation of those with special needs.

Dr. Schultz has served on the MOA board, the MOA Children’s Vision Committee, as a Clinical Director of the Special Olympics Opening Eyes Program and NORA board. She is also a Fellow of the American Academy of Optometry, Fellow of College of Optometrists for Vision Development and Fellow of the Neuro-Optometric Rehabilitation Association. She is also an Adjunct Clinical Professor and Residency Director affiliated with Pacific University College of Optometry. Dr. Schultz enjoys spending time with her three busy children and husband, camping, crafting and volunteering her skills to the community/church.